MED +

Royal College of Physicians

Case reports

November 2025

rcpconferences.co.uk/med-plus-2025

Prolonged survival in metastatic triple negative breast cancer: A case series.

Oladapo Adesua | Tanushree Dewan. Glan Clwyd Hospital

Med+ 2025

1. Introduction

- Triple negative breast cancer (TNBC) lacks of oestrogen, HER2 and progesterone receptors. It accounts for 15–20% of breast cancer cases and is generally high grade with elevated proliferation rates and aggressive
- Chemotherapy remains standard therapy (1).
- Metastatic TNBC has a poor prognosis with low survival months despite treatment (2).

2.Method

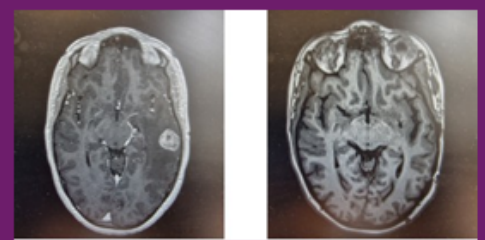
- We look at two women diagnosed 7 years ago with metastatic disease, their management and disease course.

3.

2017: breast lump with biopsy, mammoplasty and diagnosis.

Chemotherapy x6 cycles with adjuvant radiotherapy.

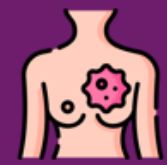
2018: brain metastasis. Steroids and stereotactic radiosurgery



a.2018
b.2025

Treatment complications: Anaphylaxis to Taxotere, neutropenic sepsis, avascular necrosis of femoral head leading to THR.

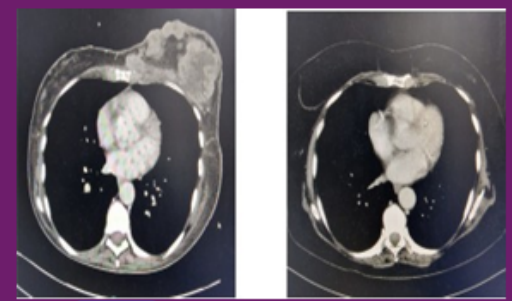
4.



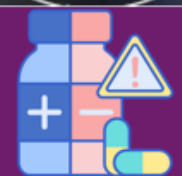
2017: fungating lesion and worsening sciatica. Diagnosis with vertebral metastasis.

On weekly chemotherapy with resolution of symptoms and disease stabilization

Radiotherapy to the spine



a. 2017
b. 2015



Complications: Nausea and initial failed treatment, pulmonary embolism.

5. Discussion

- These two cases notably have surpassed the median survival rate of 8 -13 months. Kesireddy et al.
- Factors identified correlating with improved survival include multiagent therapy and individualized approach.
- Recent advances have led to increased treatment options in TNBC with PARP inhibitors and immune checkpoint inhibitors approved for treatment.
- Overall, this data emphasizes the need for an individualized approach to TNBC management, where therapy is guided by thorough evaluation of patient and tumor characteristics.

6. Conclusion

Though associated with unfavorable prognosis; these cases demonstrate that individualized treatment approaches contribute to prolonged survival. Their outcomes highlight TNBC's heterogeneity and the need to tailor management while advancing novel therapies.

References

1. Zagami P, Carey LA. Triple negative breast cancer: pitfalls and progress. NPJ Breast Cancer. 2022;8:95.
2. Dent R, Trudeau M, Pritchard KI, Hanna WM, Kahn HK, Sawka CA, et al. Triple-negative breast cancer: clinical features and patterns of recurrence. Clin Cancer Res. 2007;13(15):4429–34.
3. Kesireddy M, Smith ER, Johnson K, Patel R, Brown A, Lee J, et al. Overall survival and prognostic factors in metastatic triple-negative breast cancer: a national cancer database analysis. Cancers (Basel). 2024;16(10):1791. doi:10.3390/cancers16101791.
4. Chue BM, La Course BD. Case report of long-term survival with metastatic triple-negative breast carcinoma: treatment possibilities for metastatic disease. Medicine (Baltimore). 2019;98(16):e15302. doi:10.1097/MD.0000000000015302.
5. Dr Julie Jones consultant oncologist

UNMASKING VEXAS SYNDROME THROUGH A CASE OF FEVER OF UNKNOWN ORIGIN

Sourav Hariram [1], Nwe Ni Win [1]
[1] Department of General Medicine, North West Anglia NHS Foundation Trust

Royal College of Physicians

NHS North West Anglia NHS Foundation Trust

INTRODUCTION

- VEXAS syndrome is a recently identified autoinflammatory disorder with distinctive hematological and systemic inflammatory features.
- The syndrome was first described in a study by Beck et al.[1] in 2020 with the acronym VEXAS standing for:

V	E	X	A	S
Vacuoles	E1 Ubiquitin activating enzyme	X-linked	Auto-Inflammatory	Somatic

- Recognition remains limited due to its recent discovery, clinical heterogeneity, and overlap with other conditions.
- This case highlights the VEXAS syndrome as a potential cause of fever of unknown origin (FUO) and emphasizes the diagnostic complexity faced and therapeutic considerations.

PATHOGENESIS OF VEXAS SYNDROME

Somatic UBA1 Mutation (X-chromosome)

p.Met41 variants
↓ UBA1b, ↑ UBA1c → Myeloid Restricted clonal hematopoiesis

↓ E1 Enzyme Function
→ Impaired Ubiquitylation
Protein accumulation → ER stress
→ Unfolded protein response

Systemic Inflammation

Recurrent fevers, fatigue, weight loss, Neutrophilic dermatosis, Relapsing chondritis, Pulmonary infiltrates, arthritis, Elevated CRP/ESR
→ Clinical phenotype of autoinflammation

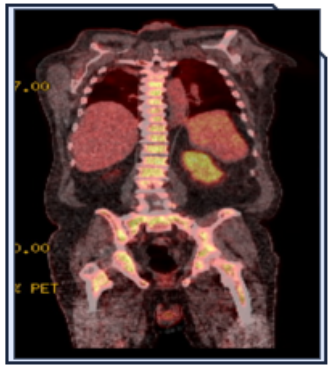
Haematologic Changes

Marrow vacuolization (Promyelocytes, proerythroblasts)
Macrocytosis, cytopenias, Myelodysplastic syndrome (MDS) overlap / clonal dysplasia, Thrombosis (venous and arterial) due to inflammatory and endothelial activation
→ Haematologic and thrombotic manifestations

Systemic Inflammation and Haematological Abnormalities (PET-CT: diffuse marrow hypermetabolism is frequent)

CASE PRESENTATION

Patient Profile	77-year-old Caucasian male
Presenting Complaints (Over 2 months)	Persistent low-grade fevers, profuse night sweats, unintentional weight loss, generalized malaise and fatigue.
Initial investigations	• **Blood cultures and autoimmune screen :** No evidence of infection or autoimmune disease • **CT Chest, abdomen, pelvis :** No signs of malignancy, lymphadenopathy or occult infection • **Quantiferon-TB Gold test :** Negative
Escalated diagnostics due to persistence of symptoms	• **PET-CT scan:** Demonstrated diffuse bone marrow activation • **Bone marrow aspirate and Trephine (BMAT):** Presence of vacuolated megakaryocytes
Definitive Diagnosis	**Next-generation sequencing (NGS):** UBA1 somatic mutation detected
Final diagnosis	VEXAS syndrome



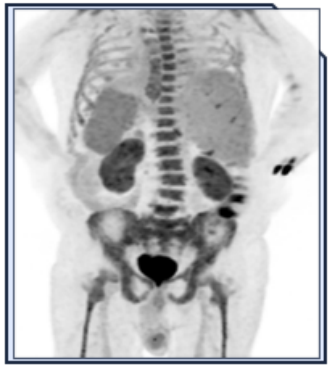


Fig.1 and Fig 2. (above) are PET-CT scans that demonstrate an increased Fluorodeoxyglucose (FDG) uptake in the spleen along with diffuse uptake in the bone marrow as well. This thus confirms hypermetabolic activity in above structures

FOLLOWUP AND DISCUSSION

- The patient was commenced on intravenous corticosteroids which showed a favourable clinical response. (Resolution of fever and systemic symptoms)
- However an attempt to taper the steroids led to the recurrence of symptoms indicating steroid dependency.
- Tocilizumab (IL-6 receptor inhibitor) was initiated following a multidisciplinary team discussion between hematology and rheumatology. This resulted in sustained symptom control.
- The patient now continues under joint follow-up with rheumatology and hematology and is under the respective team surveillance.

Discussion and Key learning points:-

- Diagnosis often relies on high index of clinical suspicion, bone marrow findings and genetic confirmation of UBA1 somatic mutations
- The syndrome carries a high morbidity burden and has a 5 year survival of upto 63% as of latest ongoing studies.[2] Complications that increase morbidity include thromoembolic events and cytopenias.
- Corticosteroids remains first-line and biologics illustrated in above case can offer symptom control, but do not halt disease progression.
- In terms of future directions, clinical trials are essential to study emerging therapies and advance understanding. Allogenic hematopoietic stem cell transplantation (allo-HCT) shows promise as a potentially curative approach, however is currently limited to select cases that have demonstrated remission.[3]

CONCLUSION

VEXAS syndrome should be considered in unexplained febrile illnesses. Being aware of its manifestations, diagnostic workup, symptom directed management and emerging treatments is essential for early diagnosis and optimal care. Multidisciplinary collaboration is essential to optimise outcomes in this complex and emerging disease entity.

References

Scan here to view references

SPORADIC CREUTZFELDT-JAKOB DISEASE: WORKUP AND CHALLENGES TO REACH PREMORTEM DIAGNOSIS WITH THE HIGHEST LEVEL OF CONFIDENCE

AUTHOR: MAHMOUD TEAMA

CO-AUTHORS: KOMOLAFE OLUTOPE, ALEXANDER OGUNWUSI, SHYAMALA MANIBALAN

AFFILIATIONS:

Princess Royal University Hospital, King's College NHS Foundation Trust

INTRODUCTION

Creutzfeldt-Jakob disease (CJD) is a very rare, rapidly progressing, and fatal neurodegenerative disorder. CJD can prove to be very difficult to diagnose in the early stages due to the non-specific nature of its symptoms, which include visual changes, behavioral changes, ataxia, pyramidal/extrapyramidal signs, and rapidly progressive dementia

CASE PRESENTATION

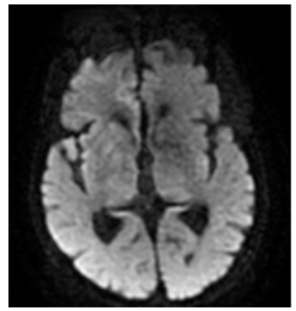
We are presenting a 76-year-old man who presented with a 4-month history of symptoms, which started with dizziness and progressed to progressive confusion. Gradually, the patient started to develop twitchy movements, dysarthria, and a rapid decline in cognitive functions. A series of extensive workup of investigations, and treatment, including a trial of IV steroids for autoimmune encephalitis, to which the patient did not respond. Subsequently, the patient was diagnosed as a case of sporadic CJD by the National CJD Research and Surveillance Unit in Edinburgh, with the highest confidence we have in life following the abnormal hyperintensity signals in his diffusion weight MRI, and positive RT-QUIC in his CSF

DISCUSSION

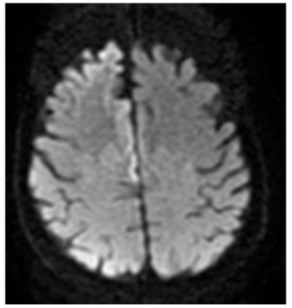
The early presentation of CJD is non-specific as patients typically present with rapidly progressive cognitive impairment, myoclonus, cerebellar ataxia, pyramidal/extra-pyramidal signs, behavioral changes, and visual disturbances. The variety of these symptoms and signs poses a real challenge in reaching a premortem diagnosis of CJD. A high index of clinical suspicion for CJD in patients with rapidly progressive dementia, exclusion of differential diagnoses of cognitive decline, and appropriate use of MRI, EEG, and CSF analysis are crucial in reaching the diagnosis. MRI with DWI and FLAIR should be ordered as soon as a rapidly progressive dementia is suspected, as it classically demonstrates hyperintense signal on DWI and FLAIR in the regions of the cerebral grey matter, striatum, and thalamus. The real-time quaking-induced conversion (RT-QuIC) assays of the CSF have made a considerable impact on the clinical diagnosis of CJD in recent years. Current sensitivity of CSF RT-QuIC undertaken at the UK National CJD Research & Surveillance Unit is 92% and the specificity is 100%. Unfortunately, there is no cure for CJD. Management is focused on symptom management and palliative care. The prognosis of CJD is extremely poor, with death expected in 70% of cases within 1 year.

IMAGING

MRI Head with DWI and FLAIR was requested which demonstrated a high DWI signal in the right cingulate gyrus extending into the frontal lobe, as well as within the right anterior insula and the medial right temporal lobe. There was a mild FLAIR high signal, although less prominent, in the corresponding regions



High DWI signal in the right anterior insula



High DWI signal in the right cingulate gyrus

CONCLUSION

Many diseases may mimic sCJD's early picture, including Alzheimer's disease, fronto-temporal dementia, paraneoplastic encephalitis, and autoimmune encephalitis, which makes the diagnosis of sCJD really challenging. This case illustrates the challenging and heavy workup done to rule out other disorders and confirm the diagnosis of sCJD with specificity approaching 100%

REFERENCE: UTTLEY L, CARROLL C, WONG R, HILTON DA, STEVENSON M. CREUTZFELDT-JAKOB DISEASE: A SYSTEMATIC REVIEW OF GLOBAL INCIDENCE, PREVALENCE, INFECTIVITY, AND INCUBATION. THE LANCET INFECTIOUS DISEASES. 2020 JAN 1;20(1):E2-10.

Pneumococcal purulent pericardial effusion as a presentation of immunocompromise: The importance of asking the right questions at the right time

Authors: Anesa Noor, Kalliopi Ioakim, Ahmed Shahin, Linda Vaccari , Douglas Fink, Niket Patel

Royal Free London NHS Foundation Trust

Background

- Purulent pericarditis is a rare but life-threatening condition, accounting for <1% of pericarditis cases.[1]
- Gram positive cocci (staphylococci and streptococci) are the most common organisms isolated.[1]
- Prompt recognition, antimicrobial therapy, and pericardial intervention are critical to survival.[2]

Case Presentation

- 64-year-old female presented to ED with 1-day history of pleuritic chest pain & SOB
- PMH: HTN & Asthma (both well-controlled)
- Vitals, bloods and CXR all normal → discharged with referral to RACPC
- Represented 4-days later with worsening chest pain
- Vitals: BP 139/86 mmHg , **HR 111 bpm**, **T 37.9˚C, RR 24/min** and SpO2 95%

Initial Workup

- Bloods: Hb 100, **WCC 15**, **CRP 380**, Troponin 20 (14) and D Dimmer 3205ng/mL.
- ECG: STE lead II, aVL and V1-2, global PR depression
- Coronary angiography: unobstructed coronaries
- CTPA: bilateral small pleural & pericardial effusion
- Bedside TTE: **1.1cm pericardial effusion**
- Blood cultures**: Streptococcus *pneumoniae* (+)**

Progress During Admission

- Pt deteriorated despite appropriate antibiotics
- Serial TTEs showed increasing size of effusion
- Inflammatory markers continued to rise
- Persistent anaemia(Hb 70) → **MM screen +ve**
- Pericardial drain inserted Day 13 of admission
- Symptomatic and clinical improvement
- Clinical improvement Drain removed 3 days
- Fluid re-accumulated on repeat TTE
- Transfer to CTS centre for pericardial window

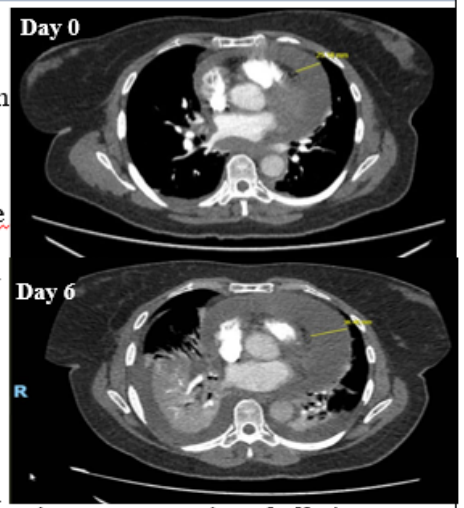
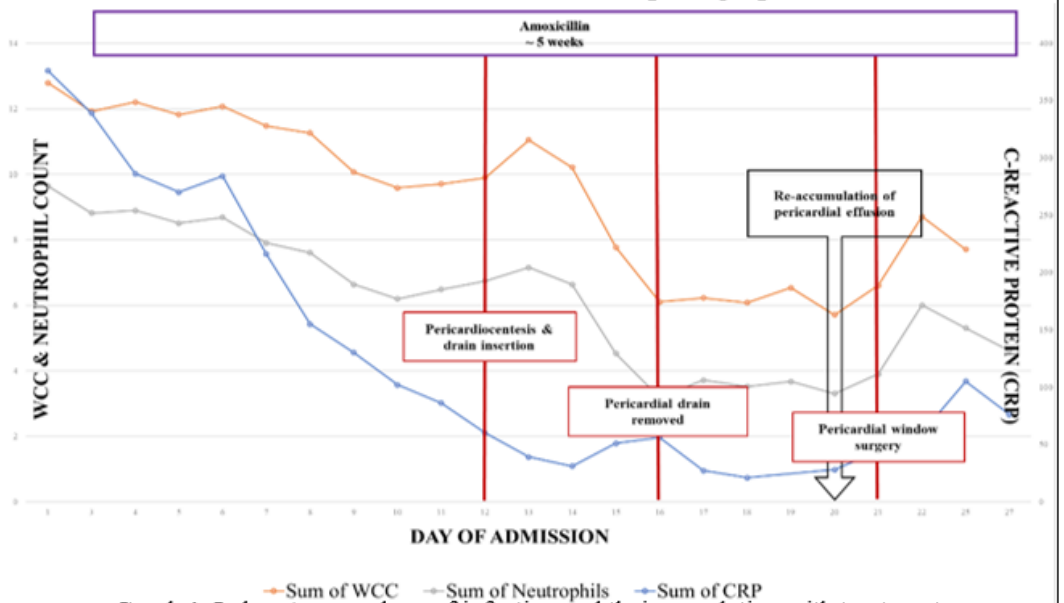


Figure1. progression of effusions on CT



Graph 1. Laboratory markers of infection and their correlation with treatment.

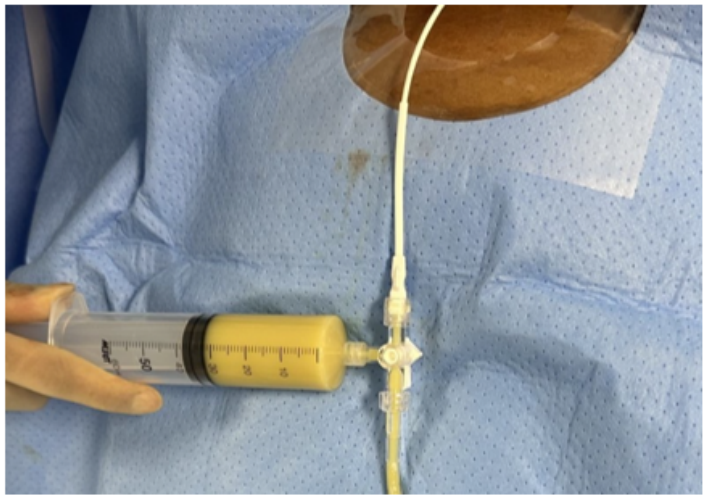


Figure2. Macroscopic appearance of drained pericardial fluid.

Conclusion

- Purulent pericarditis carries significant mortality if unrecognised.
- Early multidisciplinary input, timely pericardial intervention, and attention to underlying risk factors are essential.
- This case emphasises the importance of considering bacterial pericarditis in patients with persistent inflammatory response and pericardial effusion.

References

1. Sagristà-Sauleda J, et al. Purulent pericarditis: review of a 20-year experience in a general hospital. J Am Coll Cardiol. 1993 Nov 15;22(6):1661-5.

2. Parikh SV, et al. Purulent pericarditis: report of 2 cases and review of the literature. Medicine (Baltimore). 2009 Jan 1;88(1):52-65.

* Note this work was presented at ESC Congress 2024

Beneath the surface: Splenomegaly as the first presentation of type II Cryoglobulinaemic Vasculitis (a rare case)

Dr. Nosheen Pervaiz (Internal medicine trainee year 3 registrar)
East Kent university hospitals NHS foundation trust

NHS
East Kent Hospitals University
NHS Foundation Trust

Introduction

Cryoglobulins are immune complexes that precipitate in reduced temperatures & re-dissolve when temperature rises. Splenomegaly is very unusual presentation of Cryoglobulinemia as in our case. In one review, Systemic manifestations included skin lesions, peripheral neuropathy and joint involvement in 71.3%, 42.5%, and 28.7% of cases, respectively. Gut involvement was observed in 11.3% of patients who presented abdominal pain related to mesenteric infarction, intestinal perforation, or intestinal bleeding. Intra-alveolar haemorrhage defining lung involvement and intracerebral haemorrhage were also present in 5% and 1.3% of cases in the setting of the vasculitis flare.J Am Soc Nephrol. 2015 Aug 10;27(4):1213–1224. No cases in literature has been described of Splenomegaly as initial presentation of Cryoglobulinemia. Splenomegaly is well described in association with Cryoglobulinemia -. J Clin Med. 2025 Jan 16;14(2):556.

Case presentation

64 year old lady presented with persistent nausea, abdominal discomfort, and significant weight loss of 32kg over a 3-month period.

Initial labs revealed only microcytic hypochromic anaemia. A contrast-enhanced CT of the thorax, abdomen, and pelvis excluded any overt malignancy but incidentally revealed moderate splenomegaly, with the spleen measuring 187 mm in the craniocaudal dimension. PET-CT revealed interval splenic enlargement (193 mm) and diffusely homogeneous splenic parenchyma without evidence of focal hypermetabolism on FDG uptake. Bone marrow biopsy revealed normal trilineage hematopoietic maturation and no morphologic evidence of dysplasia or infiltrative disease.

Amidst continued diagnostic uncertainty, an elective splenectomy was planned to further evaluate splenomegaly of indeterminate origin and to obtain tissue for definitive

Fig 1. Splenomegaly CT and PET

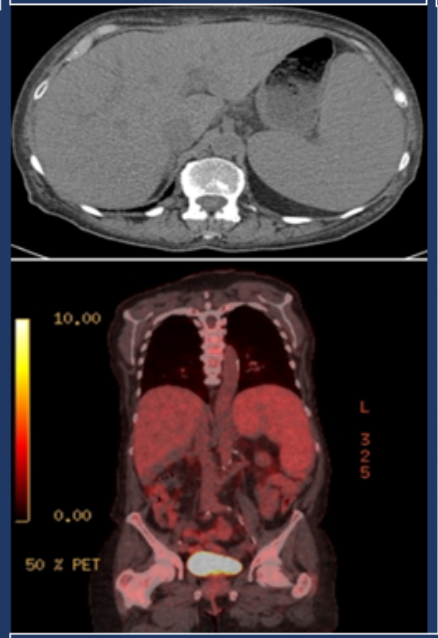


Fig 2. Vasculitis rash on legs

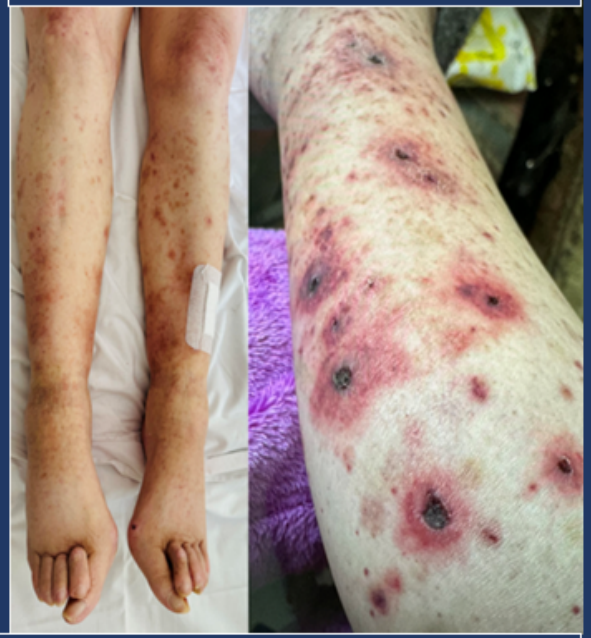


Fig 3. Bilateral patchy ground glass opacities (alveolar haemorrhage)

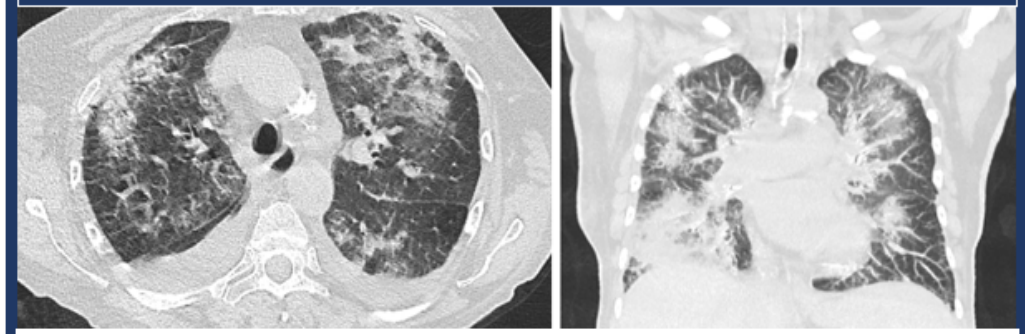


Fig 4. Renal Biopsy report

Supplementary
Immunoperoxidase shows some high background staining, but nevertheless it shows granular mesangial and capillary wall IgM+++, and IgM is taken up by the granular luminal material in the affected glomerular segment. There is also granular mesangial C1q+++. Kappa and lambda are negative.

The appearances are in keeping with focal cryoglobulins in a glomerulus given the clinical scenario, in the setting of some chronic damage, with 4/31 obsolete glomeruli, and a further 6 glomeruli with segmental sclerosing lesions.

Clinical course

While awaiting elective splenectomy, she had an acute presentation with ongoing weight loss, the emergence of a new erythematous, non-blanching ulcerative rash over the lower extremities and peripheral neuropathy. She now disclosed a two-year history of intermittent rash affecting the lower limbs, previously managed in the community as possible psoriasis, awaiting formal assessment by a dermatologist.

Renal function declined markedly (eGFR) to 6 mL/min/1.73 m^2. Anaemia also worsened necessitating transfusion, and she developed haemoptysis with prominent bilateral ground glass opacities on CT thorax, and her spleen was now progressively enlarged to 200mm. She was started on renal replacement therapy followed by Kidney biopsy.

Investigations reported cryoprecipitating IgM kappa, IgG Kappa and IgG Lambda paraprotein with positive rheumatoid factor, low C3 & C4 with some polyclonal IgG also present in the cryoprecipitate, consistent with a TYPE 2 MIXED CRYOGLOBULINAEMIA.

Kidney biopsy confirmed membranoproliferative glomerulonephritis exhibiting prominent immune complex deposition.

She was treated with PLEX, Rituximab and Cyclophosphamide, but unfortunately succumbed to her disease due to multisystem involvement.

With regards to aetiology, the only positive findings included EBV IgG and CMV IgG positivity indicating a possible past infection.

Conclusion

- Cryoglobulinaemic vasculitis can present with heterogenous clinical picture, underscoring the need for high index of suspicion, as early recognition and targeted interventions are key to improving outcomes.
- Clinical research including pathogenesis, investigating novel biomarkers to aid diagnosis and monitoring, and therapeutic advancements is crucial for this rare disease.
- Reporting rare presentations may reveal previously unidentified associations between conditions not well documented in

A Rare Case of Gadolinium induced Polyarthritis: A Case Report

Dr Anooja Anil, Dr Samantha Goh, Dr Nasreen Saleem

NHS Nottingham University Hospitals NHS Trust

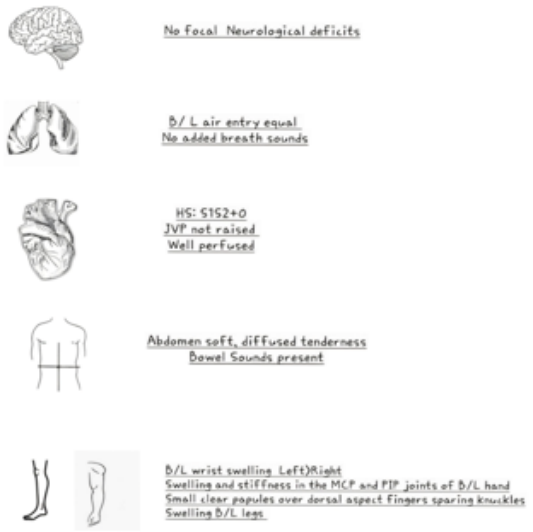
Background

Gadolinium based contrast agents (GBCAs) have been widely used and accepted in MRI imaging and are generally considered safe.

Nephrogenic systemic fibrosis (NSF) has long been a recognised entity in patients with renal impairment(1) however over the recent years, emerging literature, although limited, suggest that gadolinium exposure may be associated with chronic systemic symptoms even in patients with a normal renal function.

Presentation of Symptoms:

30 year old male
Background of Hep C and IVDU.
History of OP contrast enhanced MRI for a longstanding facial swelling.
At the time of MRI developed breathing difficulties which resolved with reassurance.
Few hours later developed –fever and myalgia which was managed with simple analgesia.
The following day developed joint swelling and stiffness in the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints of his hands.
Initially he reported no associated rashes.
Symptoms worsened - stiffness and pain in bilateral hands and legs while mobilization ,abdominal pain and a small rash on his left arm.



Investigations : CK 1659, eGFR >90, CRP 4.9
Nerve Conduction Study and EMG : Normal
MRI contrast : Lipoma confirmed with biopsy later
Management: Expert opinion from Consultants in Neurology, Radiology, Dermatology, Rheumatology taken and treated as Gadolinium-induced Polyarthritis with IV fluids, analgesia and supportive care.

Discussion

This case adds to the growing body of evidence that suggests gadolinium exposure, even in patients with normal renal function may be associated with delayed-onset symptoms affecting the cutaneous and musculoskeletal system. (2)Previous studies have demonstrated gadolinium deposition in multiple organs including the brain, bone and skin.(3) Very few have reported associations with joint involvement. In addition, confirmation of histopathological modifications in such incidences have yet to be established. (4)

Reference:

1.Bernstein EJ, Schmidt-Lauber C, Kay J. Nephrogenic systemic fibrosis: A systemic fibrosing disease resulting from gadolinium exposure. Best Pract Res Clin Rheumatol. 2012 Aug;26(4):489–503.
2. Semelka RC, Ramalho J, Vakharia A, AlObaidy M, Burke LM, Jay M, et al. Gadolinium deposition disease: Initial description of a disease that has been around for a while. Magn Reson Imaging. 2016 Dec;34(10):1383–90.
3. Darrah TH, Prutsman-Pfeiffer JJ, Poreda RJ, Ellen Campbell M, Hauschka P V., Hannigan RE. Incorporation of excess gadolinium into human bone from medical contrast agents. Metallomics. 2009;1(6):479.
4. Fitch G, Williams K, Freeston JE, Dass S, Grainger A, Hodgson R, et al. Ultrasound and magnetic resonance imaging assessment of joint disease in symptomatic patients with cystic fibrosis arthropathy. Journal of Cystic Fibrosis. 2016 Jul;15(4):e35–40.

Delayed-onset Irinotecan-Induced Pneumonitis in a Colorectal Cancer Patient

NHS Wye Valley NHS Trust

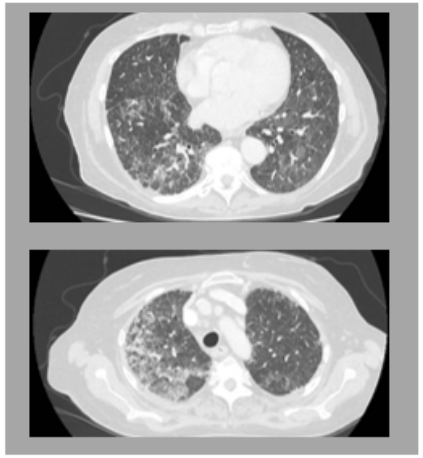
Kenny Ling, Mariam Al-Ani, Justin Choo

BACKGROUND

- Irinotecan is a widely used **chemotherapeutic agent** in the treatment of metastatic colorectal cancer, with established efficacy in combination regimens such as FOLFIRI.(1)
- While its most common toxicities, such as neutropaenia and diarrhoea, are well recognised,(2) irinotecan-induced pneumonitis remains a **rare** but potentially **life-threatening** complication.(3,4)
- Pulmonary toxicity has been reported to occur even **weeks after** administration, and may present subtly, often mimicking infectious causes.
- Prompt recognition and management are essential to **prevent progression** and **optimise outcomes.**

CASE SUMMARY

- **80-year-old female** with metastatic colorectal cancer who developed worsening **dyspnoea** three weeks following her fourth cycle of irinotecan and etoposide.
- CT showed bilateral **ground-glass opacities,** she had **elevated inflammatory markers**, with no microbial growth on blood cultures.
- Empirical **antibiotic therapy** was commenced for presumed atypical pneumonia; however, her **oxygen requirements persisted**. Bronchial washing samples were negative for infectious causes.
- Given the radiological findings, clinical course, and lack of improvement with antibiotics, a diagnosis of **irinotecan-induced pneumonitis** was considered.
- The patient responded well to **high-dose corticosteroids**, with rapid clinical and radiological improvement, and was successfully weaned off oxygen before discharge.



Figures 1&2: HRCT-Chest

REFERENCES

1. Chai Y, Liu JL, Zhang S, Li N, Xu DQ, Liu WJ, et al. The effective combination therapies with irinotecan for colorectal cancer. Front Pharmacol. 2024 Feb 5;15:1356708.
2. Kciuk M, Marciniak B, Kontek R. Irinotecan-Still an Important Player in Cancer Chemotherapy: A Comprehensive Overview. Int J Mol Sci [Internet]. 2020 Jul 12;21(14). Available from: http://dx.doi.org/10.3390/ijms21144919
3. Madarnas Y, Webster P, Shorter AM, Bjarnason GA. Irinotecan-associated pulmonary toxicity. Anticancer Drugs. 2000 Oct;11(9):709–13.
4. Baba K, Matsubara Y, Hirata Y, Ota Y, Takahashi S, Boku N. Case report: Irinotecan-induced interstitial lung disease in an advanced colorectal cancer patient resurfacing decades after allogeneic bone marrow transplantation for aplastic anemia; a case report and narrative review of literature. Front Oncol. 2023 Jun 16;13:1215789.
5. Michielin O, Udry E, Périard D, Matzinger O, Lobrinus JA, Stupp R. Irinotecan-induced interstitial pneumonia. Lancet Oncol. 2004 May;5(5):322–4.
6. Yoshida Y, Sugimoto T, Hosokawa Y, Suma H, Kobayashi H, Ishitoku M, et al. C-reactive protein and ground-glass opacity as predictors for intractable interstitial lung disease in patients with systemic sclerosis under cyclophosphamide treatment regardless of concomitant glucocorticoids. Mod Rheumatol. 2022 Jan 5;32(1):141–8.
7. Shinoda T, 河原秀次郎, カワハラヒデジロウ, Kawahara H, Yoshinaga K, Shioya H, et al. Refractory Irinotecan-Induced Interstitial Pneumonitis after Treatment of Metastatic Lung Cancer form the Rectum : A Case Report (Case Report). Jikeikai medical journal. 2006 Dec 10;53(4):159–62.
8. Chandak TR, Huberfeld SI. Irinotecan(CPT-II)-induced Fatal Pulmonary Toxicity [Internet]. 2010 [cited 2025 Sep 2]. Available from: http://dx.doi.org/10.1164/ajrccm-conference.2010.181.1_MeetingAbstracts.A292

DISCUSSION

- Diagnostic complexity of drug-induced pneumonitis in patients receiving chemotherapy.
- **Non-specific respiratory symptoms**, persistent hypoxia, and radiographic ground-glass changes may initially be attributed to infection, **delaying appropriate treatment.**
- Irinotecan-induced pulmonary toxicity is believed to involve **immune-mediated inflammatory pathways** with a favourable response to corticosteroids (5,6)
- The **delayed onset** of symptoms, occurring weeks after chemotherapy administration, underscores the importance of maintaining a **high index of suspicion** for pulmonary toxicity outside the immediate post-treatment period.
- Severe cases may require mechanical ventilation (7,8), **early recognition,** and **corticosteroid therapy** may prevent clinical deterioration.

CONCLUSION

- Drug-induced pneumonitis should remain a **key differential** diagnosis in patients undergoing chemotherapy who present with **respiratory symptoms.**
- Persistent oxygen requirements, ground-glass opacities on imaging, and raised inflammatory markers unresponsive to antibiotics should prompt consideration of high-dose corticosteroid therapy.
- Need for vigilance as symptoms can appear **weeks after administration.**
- MDT collaboration involving **oncology**, **respiratory** and **radiology** is critical in ensuring quick diagnosis and management of this rare but serious adverse drug reaction.

First Seizure with Olfactory Aura Leading to Diagnosis of Right Medial Temporal Glioblastoma

Olfactory aura = mesial temporal lesion until proven otherwise.

Dr Haliman Khan[1], Dr Karan Jethwa[1]

[1] Department of Medicine & Surgery, Royal Lancaster Infirmary

Background:

Glioblastoma multiforme (GBM) is the most common primary malignant brain tumour in adults with a median survival of 14–15 months despite maximal multimodal therapy. Seizures are the first presenting feature in 30–50% of cases, particularly with temporal lobe involvement. Olfactory aura is a key red-flag sign localising to the mesial temporal region and mandates urgent MRI in adult first seizures.

Case:

A gentleman in his 50s presented with a first focal seizure with behavioural arrest, automatisms and a “burnt rubber” smell. He had no prior seizure history. Examination revealed mild receptive dysphasia and subtle left upper limb weakness. CT suggested a right temporal lesion; MRI confirmed a medial temporal ring-enhancing mass with vasogenic oedema and corpus callosum extension, consistent with GBM.

Management:

He received IV lorazepam, levetiracetam and dexamethasone was started for vasogenic oedema. He underwent right fronto-temporal craniotomy with near-total resection. He is under neuro-oncology review for adjuvant chemoradiotherapy.

Learning Points:

- First seizures in adults require urgent neuroimaging
- Olfactory aura strongly suggests medial temporal pathology
- CT can be normal in early tumour-related seizures — MRI is mandatory
- Early seizure + oedema management is critical
- Multidisciplinary care drives outcomes

MRI:

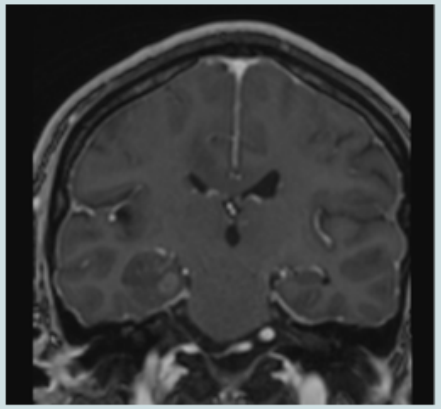


Figure 1: Coronal T1 gadolinium-enhanced MRI demonstrating a right medial temporal ring-enhancing lesion with central necrosis and vasogenic oedema.

Stage	Findings/Actions
Presentation	First focal seizure with olfactory aura, automations, arrest
Examination	Mild receptive dysphasia, subtle left upper limb weakness
Imaging	CT: right temporal lesion MRI: (Figure 1)
Acute Management	IV lorazepam, levetiracetam, dexamethasone, IV antibiotics for aspiration pneumonia
Surgery	Right frontotemporal craniotomy, near total resection

Figure 2: Diagnostic and management timeline

Hanif F et al. Asian Pac J Cancer Prev. 2015;16(9):3617–24 Englot DJ et al. Handb Clin Neurol. 2016;134:263–75 Capampangan DJ et al. Ann Emerg Med. 2010;56(4):374–7 Sullivan J, Fisher R. Epilepsy Behav. 2021;115:107648 Mousinho M et al. Eur Psychiatry. 2024;39(S1):S317 Weller M et al. Lancet Oncol. 2014;15(9):e395–403 Stupp R et al. N Engl J Med. 2005;352(10):987–96 American Association of Neurological Surgeons. Astrocytoma Tumors. 2024 Gaillard F et al. Radiopaedia. 2024 Weller M et al. Nat Rev Dis Primers. 2015;1:15017

Transient Posterolateral Ischemia Due to 5-Fluorouracil-Induced Cardiotoxicity: A Case Report

Muhammad Fahad Saleem[1], Pharveen Jaspal[2], Shoaib Siddiqui[3]

1. Resident Doctor. 2. Resident Doctor and Clinical Education Registrar. 3. Consultant Cardiologist

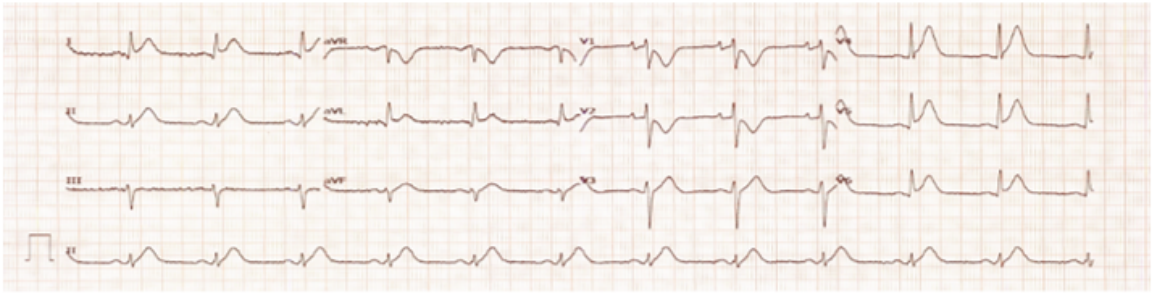
South Warwickshire University NHS Foundation Trust

Introduction

- A 74-year-old man with a history of coronary artery disease developed chest pain after receiving 5 fluorouracil (5-FU) therapy, thought to have occurred due to coronary vasospasm as there was complete resolution of symptoms following cessation of the drug.
- While gastrointestinal side-effects and myelosuppression of 5-FU is common, cardiotoxicity remains a rare but significant complication.

Case Presentation

- A 74-year-old man with a history of coronary artery disease and CABG performed 15 years earlier presented to ED with sudden-onset severe chest pain.
- Alongside a history of hypertension he was diagnosed with anorectal carcinoma, for which he had commenced his first cycle of chemotherapy with 5-FU and mitomycin infusion, 48 hours earlier.
- Having received aspirin and sublingual glyceryl trinitrate (GTN), an electrocardiogram (ECG) performed on arrival (pain-free) demonstrated normal sinus rhythm. Serum troponin level was 52 ng/L.
- Two hours later, he developed chest pain again, and a repeat ECG showed deep ST depression in the anterior precordial leads.
- Following a diagnosis of acute MI, he was given subcutaneous low molecular weight heparin, sublingual GTN, intravenous morphine, and his 5-FU infusion was discontinued.
- This resulted in resolution of symptoms and ECG changes for the next five hours. Another episode of chest pain then ensued, and a repeat ECG showed ST elevation in the lateral leads with reciprocal changes anteriorly, (Figure 1) while the troponin level rose to 184 ng/L. (Table 1)
- Following administration of sublingual GTN, the pain and ECG changes resolved.
- Although the initial working diagnosis was non–ST-elevation myocardial infarction, this was subsequently revised to ST-elevation myocardial infarction secondary to 5-fluorouracil-induced coronary vasospasm, given the transient nature of the ECG changes and chest pain.
- A transthoracic echocardiogram demonstrated regional wall motion abnormalities with an ejection fraction of 39%, without evidence of intracardiac thrombus.
- Coronary angiography further showed patent grafts and no evidence of thrombus.
- After a stable clinical course, the patient was discharged with optimised medications and follow-up; the oncologist advised against future 5-FU, and cardiology specialist review supported coronary vasospasm as a possible cause.



Laboratory Test	Reference Range	30/10/2024 21:15	30/10/2024 23:59	31/10/2024 06:26	01/11/2024 08:15
Troponin (ng/L)	<14	52	57	184	322

Discussion

The exact mechanism underlying 5-FU induced cardiotoxicity remains unclear, the most widely accepted explanation is coronary vasospasm leading to myocardial ischaemia. Hence, given the transient chest pain, ECG changes, and troponin elevation, with no thrombus, patent grafts on angiography, and no prior chest pain before 5FU initiation, the most likely cause is 5FU induced coronary vasospasm affecting territories beyond the grafts and causing transient posterolateral ischaemia.

Conclusion

Given the frequent use of 5-FU, awareness of its cardiotoxicity is essential, and alternative regimens should be considered in patients with preexisting cardiac disease. Early recognition therefore of the offending agent is crucial to prevent adverse outcomes.

References

1. Balaji N, Dixit P, Adams AM, et al. ST-elevation MI due to 5-fluorouracil cardiotoxicity. Cureus. 2024;16(1):e52864. 2.Faheem B, Kania B, Ashkar H, et al. 5-fluorouracil cardiotoxicity with coronary vasospasm. J Community Hosp Intern Med Perspect. 2022;12:71–4.3. Raber I, Warack S, Kanduri J, et al. Fluoropyrimidine-associated cardiotoxicity: a case-control study. Oncologist. 2020;25(3):e606–9.

Severe Adenovirus Pneumonia in a medical registrar : a case report

Dr Kabita Pathak [1], Dr Selina Zakri [1], Dr Kushagra Mathur [1]

Medical Registrar, Acute and General Medicine, William Harvey Hospital [1]

INTRODUCTION

Human adenovirus is known to cause mild to severe forms of respiratory infections. While most of them are self-limiting, specific strains and host factors can lead to severe forms as well [1]. On the week that this case presented, the weekly PCR positivity rate in the UK was 4.05%. [2] The common symptoms of adenovirus infection are runny nose, fever, and cough sometimes with expectoration. It usually presents as a severe infection in immunocompromised individuals, including diabetics, elderly and the ones undergoing chemotherapy. It is also common in individuals with poor lung reserves like chronic smokers, ones with chronic obstructive pulmonary disease and fibrosis.

CASE HISTORY

- A 52-year-old male physician presented with a 7-day history of flu-like symptoms. His past medical history included COPD due to long-term heavy smoking (40–60 cigarettes/day), type 2 diabetes mellitus managed with oral hypoglycaemics , diet-controlled hypertension, hypercholesterolemia, and spinal claudication.
- Initially treated with co-amoxiclav in the community, his symptoms progressed to include fever, productive cough, and dyspnoea. He was admitted for suspected community acquired pneumonia and started on intravenous antibiotics and steroids. Within 24 hours, his condition deteriorated with sudden confusion, hypotension, faecal incontinence, and a fall, necessitating ITU admission
- Laboratory findings were disproportionate to clinical severity: CRP 98 mg/L, normal white cell count, mild lymphopenia (1.2 $\times 10^9$/L), and a normal renal function.
- Chest CT revealed bilateral multifocal consolidations and mediastinal lymphadenopathy (as seen in figures: 1 and 2).
- He was started on Piperacillin-Tazobactam and supplemental oxygen. Respiratory viral PCR was positive for adenovirus with an unusually high viral load (5.2 million copies/mL).
- Based on microbiology advice, he was treated with oral Ribavirin and Clarithromycin.
- The patient showed clinical and biochemical improvement within 72 hours, was weaned off oxygen, and discharged in stable condition.

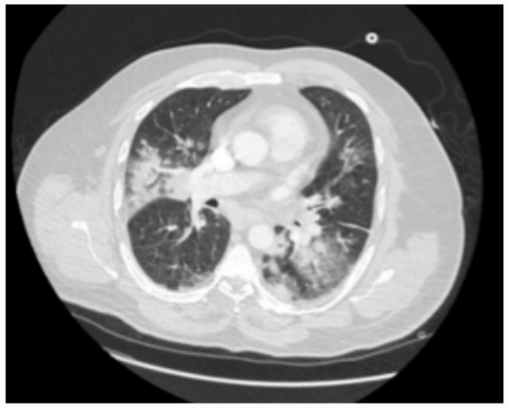


Figure: 1

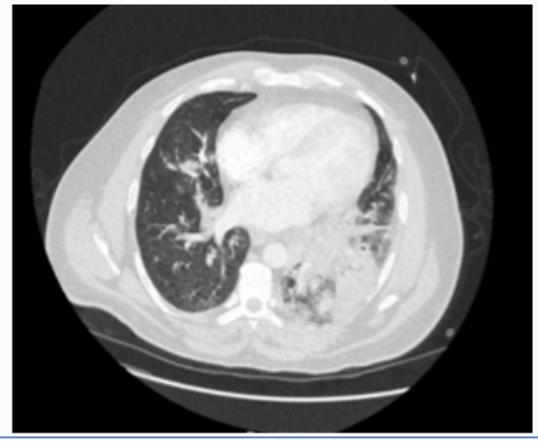


Figure: 2

DISCUSSION

Adenovirus is known to cause mild upper respiratory infections, but it is not unheard to cause lower respiratory tract infections. A case published by Larson et al in 2005 discussed a similar case in a 52-year-old chronic smoker who had severe adenovirus infection, requiring ventilatory support and antibiotics

REFERENCES

1) Hakim FA, Tleyjeh IM. Severe adenovirus pneumonia in immunocompetent adults: a case report and review of the literature. Eur J Clin Microbiol Infect Dis. 2008 Feb;27(2):153-8. doi: 10.1007/s10096-007-0416-z. PMID: 18030505; PMCID: PMC7101838.

3) Zhao B, Yang T, Zheng R. Adenovirus pneumonia should not be overlooked in immunocompetent youths and adults. Epidemiol Infect. 2021 Sep 10;149:e199. doi: 10.1017/S0950268821001485. PMCID: PMC8414599.

4)Larsen IK, Nielsen H. Community-acquired adenovirus pneumonia in a patient with chronic lymphatic leukaemia. Eur J Clin Microbiol Infect Dis. 2005 Mar;24(3):217-9. doi: 10.1007/s10096-005-1288-8. PMID: 15761721; PMCID: PMC7087800.

Seven miscarriages and pain in abdomen: A rare case report

Dr. Kushagra Mathur[1], Dr Snigdha Sharma [1], Dr. Imran Ashraf [2]
SHO, General Medicine, Darent Valley Hospital, Dartford [1]
Consultant,Stroke and Geriatric Medicine, Darent Valley Hospital, Dartford [2]

Dartford and Gravesham NHS Trust

INTRODUCTION

Rheumatoid Arthritis is a chronic, systemic, connective tissue disorder with mainly affects joints and soft tissues but is known to involve organs like liver, spleen or even heart. In many individuals, it can often affect the reproductive system of females leading to a wide range of issues- from mild cervicitis to ovarian tube blockages. There is evidence that the symptoms arise due to a chronic inflammatory condition of the body[1].

Here, we present a case of one such female with atypical presentation

Case

A 32-year-old woman with multiple admissions for abdominal pain, initially treated as acute cholecystitis and recurrent abdominal infections, underwent a CT scan showing perihepatitis and ultrasound showing splenomegaly, despite normal inflammatory markers.

She had a history of 7 miscarriages and 2 successful deliveries. Further evaluation revealed negative miscarriage screens, with mild positivity for anticardiolipin antibodies. However, strong positivity for ANA and rheumatoid factor (30 IU/L) directed us towards the possibility of an autoimmune condition. After multidisciplinary discussions, her liver lesions, splenomegaly, and miscarriages were diagnosed as extra‑articular manifestations of Rheumatoid Arthritis.

CONCLUSIONS

Early diagnosis and targeted treatment for RA have since stabilized the patient's condition, demonstrating the importance of recognizing atypical systemic presentations of RA for proper management and improved patient outcome

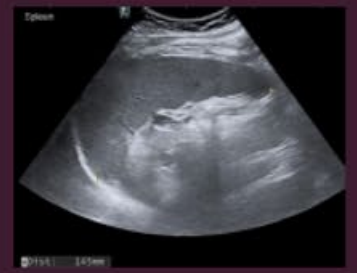


Figure 1 : Ultrasound scan showing splenomegaly (14cm)

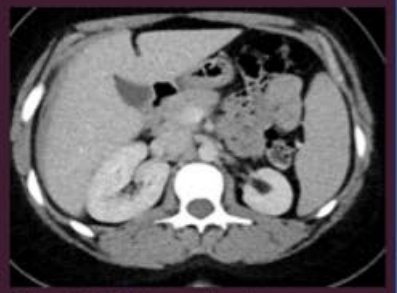


Figure 2 : CT abdomen showing (i)perihepatic inflammation and (ii) significant atrophy of left kidney as compared to right kidney.

DISCUSSION

The patient's presentation of raised rheumatoid factor, strongly positive ANAand CT findings of perihepatic inflammation, splenomegaly, and left renal atrophy were indicative of extra-articular manifestations of rheumatoid arthritis (RA).

Despite initial treatment for recurrent abdominal infections, a multidisciplinary evaluation confirmed the link between her autoimmune condition and these symptoms.

The association of RA with recurrent miscarriages further supports RA's systemic impact beyond joint involvement

REFERENCES

1.Krause M et al. Management of rheumatoid arthritis during pregnancy: challenges and solutions. Open Access Rheumatol. 2016 Mar 23;8:23-36.

2. Guo D et al. Causal association between rheumatoid arthritis and pregnancy loss and intrauterine growth retardation: A bidirectional two-sample Mendelian randomization study. Medicine (Baltimore). 2024 Jan 12;103(2):e36873

Creatinine Kinase Negative PM/Scl-Autoimmune Myositis With Pleural Involvement

Rahul Choudhary, Nain Kamal Aulakh, Ceris Owen
Medway NHS Foundation Trust

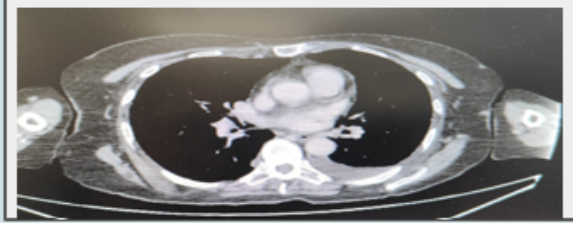
INTRODUCTION

Autoimmune myositis is an uncommon inflammatory muscle disease with potential multisystem involvement. This case highlights diagnostic and therapeutic challenges of creatine kinase–negative myositis with pleural involvement, emphasizing immunosuppression and management in patient declining steroid therapy.

CASE

Female in late 70s came with 4-week history of cough, fatigue, small joint pain, exertional dyspnoea, weight loss (~5 kg), reduced appetite, difficulty rising from chair and unable to lift arms above head.

- Exam: marked proximal muscle weakness with mild tenderness (shoulder abduction & hip flexion 4/5), distal power intact; tone, reflexes,sensation normal.
- Resp: dullness at left base, CVS normal.
- Initial bloods- CRP 165 mg/L, CK 21 IU/L BNP 39
- Extended AI screen- PM/Scl-100 positive, NXP2 positive , other AI antibodies including anti HMG CoA Red – Negative
- Pleural fluid consistent with exudative serositis (raised protein and LDH, negative M-C/S).



HRCT:
Small left pleural effusion

DISCUSSION

- Lung MDT - pleural fluid cytology, microbiology, and CT CAP were discussed; no features of malignancy or infection were identified, supporting a systemic autoimmune aetiology.
- **Management and Outcome** A working diagnosis of PM/Scl-associated autoimmune polymyositis overlap syndrome with systemic features was made. Treatment with IV methylprednisolone followed by oral steroids (prednisolone 60mg daily) was recommended after rheumatology review. Despite initial improvement on IV steroids, the patient declined further oral use due to side-effect concerns. After counselling, she signed a treatment refusal form; mycophenolate is planned to be initiated with ongoing rheumatology follow-up.
- PM/Scl positivity is associated with mixed connective tissue disease and may involve serosal surfaces. The patient's low CK highlights that myositis activity may not correlate with muscle enzyme elevation.
- Steroid hesitancy, while uncommon, can delay critical interventions. This case highlights the importance of clear communication, shared decision-making, and offering alternative steroid-sparing strategies.

LEARNING POINTS

- AI myositis may present with low CK and systemic features requiring MDT review . It may have fluctuating course and can be asymptomatic in initial episodes but still requires testing and regular follow up.
- Single dose of steroids can mask symptoms completely and hamper the clinical diagnosis.
- PM/Scl-100 positivity is associated with overlap syndromes and requires careful systemic assessment. Steroid hesitancy requires early engagement, education, and alternative planning.

REFERENCES

1. Jean-Luc Senécal, Isabelle C, Fritzler MJ, Targoff IN, Goldstein R, Gagné M, et al. An Autoimmune Myositis Overlap Syndrome Associated With Autoantibodies to Nuclear Pore Complexes. Medicine. 2014 Nov 1;93(24):383–94.
2. Satoh M, Tanaka S, Ceribelli A, Calise SJ, Chan EKL. A Comprehensive Overview on Myositis-Specific Antibodies: New and Old Biomarkers in Idiopathic Inflammatory Myopathy. Clinical Reviews in Allergy & Immunology. 2015 Sep 30;52(1):1–19.

Anterior Spinal Artery Infarction in a Middle-Aged Female: A Diagnostic Challenge Mimicking Transverse Myelitis

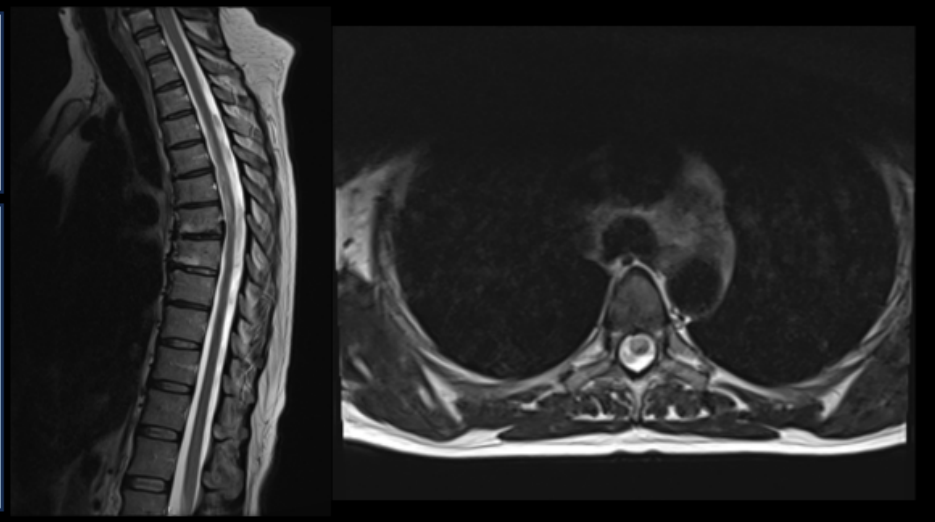
Authors and Co-Authors Details: Bhattacharjee, Santonu1; Todi, Rahul2 University Hospitals Sussex NHS foundation Trust

Background:

Anterior spinal artery (ASA) infarction is a rare but potentially debilitating vascular event resulting from ischaemia to the anterior two-thirds of the spinal cord. Diagnosis is often challenging due to clinical and radiological overlap with conditions such as transverse myelitis and cauda equina syndrome.

Case Presentation:

- A 56-year-old woman presented with **sudden-onset radicular pain** at the T4 dermatome, followed by bilateral lower limb paraesthesia.
- Within hours, she developed **flaccid paralysis** of the right lower limb and weakness in the left, along with **acute urinary retention.**
- Neurological examination showed 0/5 power in the right LL and 2/5 in the left, with preserved deep tendon reflexes. Sensory deficits were noted at the T4–T5 level, with impaired proprioception and vibration sense on the left.



Investigations and Findings:

- Routine blood tests, (CSF) analysis, including inflammatory markers: Normal
- Urgent MRI of the whole spine excluded cauda equina. Imaging showed **extensive high signal** on STIR and T2-weighted sequences from T2 to T8, predominantly involving the **anterior spinal cord**, with **contrast enhancement**. The initial impression was transverse myelitis, and the patient was started on corticosteroids and aspirin, given the possibility of infarction.
- Subsequent neuroradiology review revealed that the hyperintensity was primarily confined to the **anterior third of the cord** on axial images— an atypical pattern for transverse myelitis but characteristic of ASA infarction.
- Neurological reassessment supported this revised diagnosis. Further workup to exclude infectious, inflammatory, and autoimmune causes was negative.
- Lipid profiling revealed **elevated cholesterol and lipoprotein(a)**, a known prothrombotic risk factor
- A repeat MRI ruled out dural arteriovenous fistula

Discussion and Conclusion:

- This case highlights the diagnostic complexity of ASA infarction, especially its mimicry of inflammatory myelopathies on clinical and radiological grounds.
- Recognition of the anterior spinal cord involvement on MRI, in correlation with clinical findings, is crucial to prevent misdiagnosis and inappropriate treatment. The patient was initially treated for both ASA infarction and transverse myelitis. Once transverse myelitis was ruled out and ASA infarction confirmed, corticosteroids were tapered, and stroke-specific management continued. The patient responded well and began physiotherapy-led rehabilitation, with referral to a specialised spinal rehabilitation centre.
- Identifying modifiable vascular risk factors, such as dyslipidaemia and elevated lipoprotein(a), allowed for targeted secondary prevention.
- Early and accurate diagnosis of ASA infarction is essential for appropriate treatment and optimising outcomes, as demonstrated in this case.

References

1. Caton MT, Huff JS. Spinal Cord Ischemia [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2020. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539870/
2. Pawar NH, Loke E, Aw DC. Spinal Cord Infarction Mimicking Acute Transverse Myelitis. Cureus. 2017 Dec 6;
3. Simone CG, Emmady PD. Transverse Myelitis [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559302/

Don't go breaking my bones: A case-control study investigating the risk factors for fractures in patients under 35 years old

K Jethwa[1], V Ready[1] & H Khan[1]

Introduction:

Fractures are a common complication of various rheumatological conditions, and because of this we have a wealth of knowledge relating to the risk factors that increase the likelihood of a fracture in patients with rheumatic diseases. However, we have not previously been able to successful identify the risk factors that increase the risk of fracture in those less than 35 years of age. As a result, the aim of this study is to investigate the risk factors for fractures in patients with rheumatological conditions aged below 35 years.

Methods:

A total of 663 patients with a range of rheumatological disease who had received a DEXA scan when they were 35 or younger were included. The odds ration of fracture was then calculated in patients who developed fractures and those who did not across the risk factors in Table 1.

The statistical significance was then determined by calculating the confidence interval for each factor.

Table 1: Risk Factors for

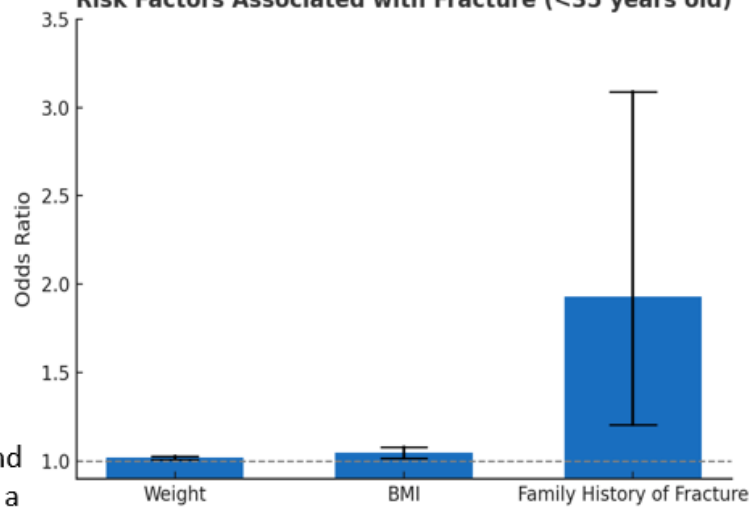
Height	Family History of a Fracture
Weight	Excess Alcohol Intake
BMI	Smoking History
History of RA	Previous Steroid Use
History of PMR	Bone Mineral Density
History of AS	History of IBD
History of PsA	History of Coeliac Disease
History of SLE	Menopausal Status

Results:

Across the 16 risk factors that we assessed, weight with an OR = 1.017555 (CI – 1.007525, 1.027685), BMI with an OR = 1.047332 (CI – 1.017308, 1.078241), and a positive family history of fracture with an OR = 1.92805 (CI – 1.203862, 3.087877) increased the risk of fracture in patients less than 35 years old.

Interestingly, co-morbid coeliac disease was found to be protective with an odds ratio of 0.5874597, notably the CI reflected that this was statistically insignificant (CI = 0.3167937, 1.089381).

Graph 1: Risk Factors Associate with



Discussion:

Across the study population we found that weight, BMI and a positive family history of fractures were associated with a statistically significant increase in risk for fracture, while the remaining risk factors that we assessed did not reflect a statistically significant increase in risk for fracture.

Analysing the positive factors further, those with a higher BMI (BMI>25 kg/m^2) had a greater frequency of fracture, similarly those who had a higher weight were also at greater odds of fracture. Expectedly, if a patient had a positive family history of fracture their odds of fracture were also increased.

Considering this, future avenues of research would be to assess further risk factors in patients under 35 years of age, as well as to reassess the above risk factors to increase validity of these results.

Key Learning points:

1) In this study of patients less than 35-years-of age we found that weight, BMI, and a positive family for a previous fracture of any bone is a statistically significant risk factor for fracture
2) Further avenues for research would be review of the above risk factors, and exploration of further risk factors

Acknowledgement: data kindly provided by M Bukhari, UHMB

✨ An Atypical Presentation of Osimertinib-Induced Pneumonitis with Asymmetrical Radiographic Findings ✨

Ali Hassan[1] (Presenting Author), Ihsan Ullah[2], Nasir Majeed[3] [1]University Hospitals of Morecambe Bay NHS Foundation Trust [2]Manchester University NHS Foundation Trust [3]Blackpool Teaching Hospitals NHS Foundation Trust

Background:

- Osimertinib is a highly effective first-line treatment for advanced EGFR mutation-positive lung cancer.
- Drug-induced pneumonitis is a rare but potentially lethal complication of this treatment.
- We present a case of severe Osimertinib-induced pneumonitis with **an atypical, asymmetrical radiological pattern**.

Case Report:

Presentation:

•A 68-year-old male with EGFR-positive lung adenocarcinoma, post VATS left upper lobectomy, presented with two weeks of dyspnoea, dry cough, and loss of appetite.

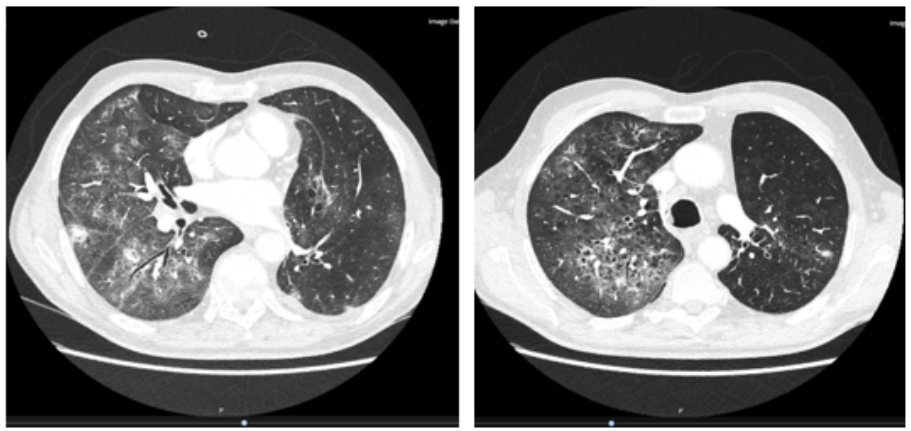
• He began adjuvant Osimertinib three months ago after postoperative histology identified an EGFR Exon 19 mutation.

On Examination:

Dyspnoeic and profoundly hypoxic
15 Litres NRM to maintain saturations.
Afebrile

Work Up:

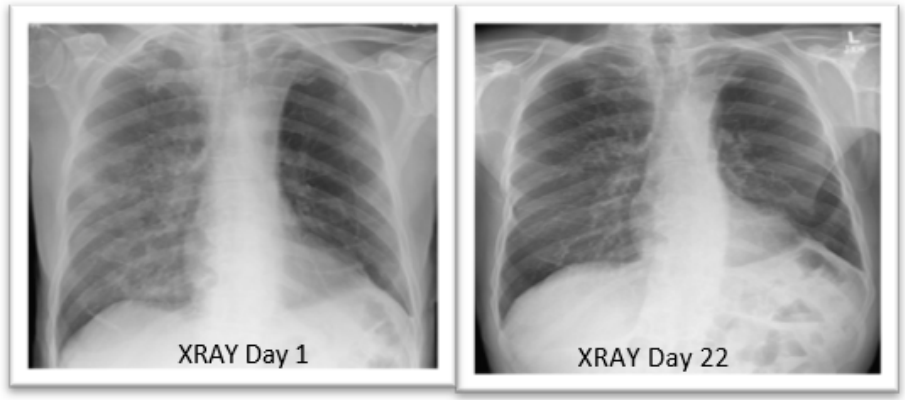
Procalcitonin: <0.03
Mild raised CRP: 20
B- Glucan: Negative
Serum PCP PCR: Negative
Atypical Pneumonia Screen: Negative



A CTPA excluded pulmonary embolism but identified new multifocal, patchy, peri-bronchovascular ground-glass consolidations. These consolidations were asymmetrical, predominantly involving the right lung, and were highly suggestive of drug-induced pneumonitis.

Treatment & Outcome:

The patient was admitted to the Intensive Care Unit and initiated on high-flow nasal oxygen therapy.
High-dose Methylprednisolone (2 mg/kg) was administered alongside prophylactic antibiotics.
Rapid Response: Significant improvement in oxygen requirements was observed within **two days**.
He received 3 days of Methylprednisolone and was started on tapering dose of prednisolone after that.
By Day 4, the patient was transitioned to a 2L nasal cannula and was subsequently discharged on Day 8 with complete symptom resolution.



Discussion and Conclusion:

- Maintain a **high index of suspicion** for DIP in any patient receiving Osimertinib who develops new or worsening respiratory symptoms, particularly when infection is not evident.
- **Early recognition** and the **timely administration of high-dose methylprednisolone** are essential to avoid fatal complications.
- Drug-induced pneumonitis generally exhibits a **symmetrical distribution**.
- This case emphasizes that **asymmetrical or unilateral presentations may also occur**, presenting a significant diagnostic challenge for healthcare providers.

Reference: Sato, Yuki et al. "Drug-Related Pneumonitis Induced by Osimertinib as First-Line Treatment for Epidermal Growth Factor Receptor Mutation-Positive Non-Small Cell Lung Cancer." CHEST, vol. 162, no. 5, pp. 1188-1198.

Progressive Multifocal Leukoencephalopathy As A Stroke Mimic

Dr Thaw Thiri Hlaing, Dr Thet Phyo Maung
Midland Metropolitan University Hospital

NHS Sandwell and West Birmingham NHS Trust

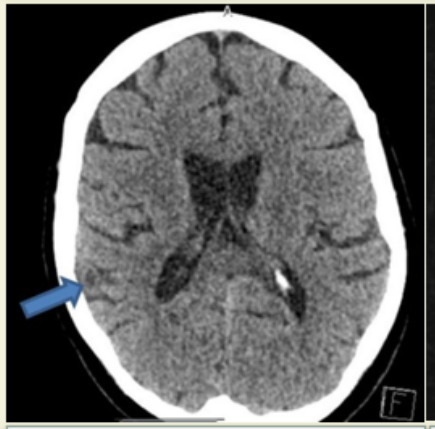
Introduction /

- **Progressive multifocal leukoencephalopathy** (PML) is a rare demyelinating disorder of the central nervous system caused by reactivation of **John Cunningham (JC) virus** in immunocompromised patients.
- It most commonly occurs in advanced HIV infection and carries a high mortality despite treatment.

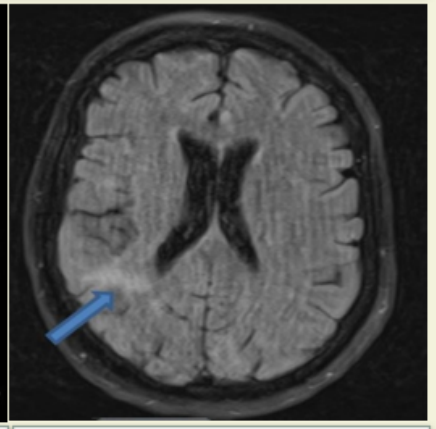
Case Description

- A 56-year-old previously healthy woman presented with a two-week history of progressive left-sided weakness and numbness, initially in the lower leg then involving the entire left side, resulting in wheelchair dependence. Examination showed left visual inattention, hemiparesis (power 2/5), and a positive Babinski sign on the left, with preserved reflexes and no cranial nerve or speech deficits.
- CT head suggested a hypodense area in right posterior temporal region. She was treated as subacute ischaemic stroke initially but the symptoms became progressive.
- Repeat contrast MRI showed diffuse white-matter changes in temporal area concerning for an infiltrative process.
- HIV testing was positive, and CSF PCR confirmed JC virus, establishing a diagnosis of **progressive multifocal leukoencephalopathy (PML)**. Despite antiretroviral therapy, the patient deteriorated and sadly passed away 4 months after the symptom onset.

Investigations



CT Head - patchy hypodense area in right posterior temporal region and parietal gyrus



MRI head - increasing diffusion restriction at white matter around the right parietal gyrus

Discussion

- Discordance between clinical findings and radiology prompted multidisciplinary discussion.
- Discovery of HIV dramatically shifted diagnostic direction from stroke to opportunistic infection.
- PML remains a devastating diagnosis even with early ART initiation.
- Highlights the need for early HIV testing in unexplained neurological presentations.

Lumbar puncture

Test	Result	Units	Reference range
Appearance	Clear color	N/A	N/A
WBC	0	cells/cumm	<5
Red cell	3	cells/cumm	0
Glucose	2.6	mmol/L	2.5-4.5
Protein	0.43	g/L	0.15-0.6
CSF culture	No organism	N/A	N/A
Mycobacterium	Not isolated	N/A	N/A
Enterovirus RNA	Not detected	N/A	N/A
VZ DNA	Not detected	N/A	N/A
BK virus RNA	Not detected	N/A	N/A
Parechovirus RNA	Not detected	N/A	N/A
JC virus DNA	DETECTED	N/A	N/A

References

1. Pathophysiology of progressive multifocal leukoencephalopathy. https://pmc.ncbi.nlm.nih.gov/articles/PMC3072716/

2. Progressive multifocal leukoencephalopathy in a HIV patient: A case report and literature]- https://pubmed.ncbi.nlm.nih.gov/37564607/

3. Berger JR, Aksamit AJ, Clifford DB, Davis L, Koralnik IJ, Sejvar JJ, Bartt R, Major EO, Nath A (2013) PML diagnostic criteria: consensus statement from AAN Neuroinfectious Disease Section. Neurology 80(15):1430– 14. https://www.neurology.org/doi/10.1212/WNL.0b013e31828c2fa14d.

SOCIAL MEDIA SHAPING SALUBRITY

Ali, Warda[1]; Jamson, Brogan[2]; Nalla, Nanda Kishore[1]
[1]NUH -Nottingham University Hospitals NHS Trust ; [2]United Lincolnshire Hospitals NHS Trust

INTRODUCTION

- **Case:** 23-year-old male presented with nausea, vomiting, fatigue, thirst, diarrhoea, and toothache
- **History:** Ingested approximately 1,000,000 units of over-the-counter vitamin D after viewing a misleading social media video promoting its benefits, resulting In severe hypercalcaemia secondary to vitamin D toxicity. [1]

MATERIALS AND METHODS

- **Permission** from the patient via written consent.
- The investigations and management as per the Nottingham University **Trust guidelines**. [2]
- Independent literature search undertaken including two databases: **PubMed and Science Direct**. [3,4]

Further correspondence: Warda Ali
warda.ali3@nhs.net
Acknowledgement: Dr Abilash Sathya

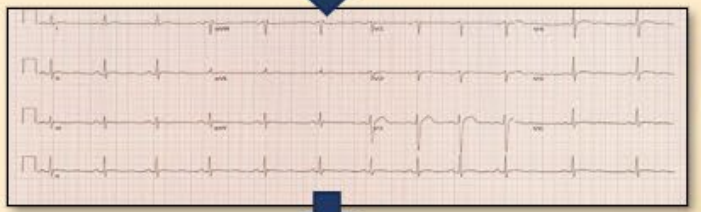
RESULTS AND DISCUSSION

- **Laboratory results on admission:**

-Vitamin D: 2747 nmol/L (toxicity >375 nmol/L)
-Calcium: 4.2 mmol/L (normal 2.2–2.6 mmol/L)
-Parathyroid hormone (PTH): 8.0 pmol/L (normal 1.6–7 pmol/L)

- **Other investigations:** Normal liver function tests, blood film, and cultures. Normal imaging: Chest X-ray, CT thorax, abdomen, and pelvis. ECG: NSR
- **Initial management:** Intravenous 0.9% NaCl fluids, 4–6 L over 24 hours for rehydration and calciuresis. Oral prednisolone 15 mg once daily for 5 days due to slow biochemical response. [2,5]
- **Subsequent course:** Readmission due to decline in renal function after discharge. Treated with IV fluids and weaning regime of prednisolone.[5,6]

FIGURE 1: ECG



- **Highlight**: clinical consequences of excessive vitamin D supplementation in a healthy individual as influenced by non-professional health advice on social media.
- **Social media's growing influence** on health-related matters presents both educational opportunities as well as risks when misinformation occurs. [7, 8]
- **Vitamin D:** vital for calcium and phosphate homeostasis, fat-soluble (accumulates in body tissues, raising toxicity risks)
- **Symptoms of toxicity:** diverse and affects; gastrointestinal, cardiac, renal, and mental health systems. [1]

CONCLUSION

- **This case underscores risks of unsupervised supplementation and highlights the impact of health misinformation on social media.**
- It calls for stricter regulation of digital health claims and improved public education on supplement safety to prevent similar cases

REFERENCES

Scan me!

Therapeutic Dilemma in Lemierre's Syndrome: Anticoagulation Considerations

Dr Wajeeha Latif Internal Medicine trainee year 2; Royal Berkshire Hospital, Reading, Dr Rabail Mustafa ST6 Diabetes and Endocrine trainee; Oxford Centre for Diabetes, Endocrinology and Metabolism, Oxford University Hospital, Dr Maham Khan ST4 Radiology trainee; New Cross Hospital, Wolverhampton

INTRODUCTION

Lemierre's syndrome is a rare septic thrombophlebitis usually arising from oropharyngeal infection, with Fusobacterium necrophorum the predominant pathogen.[1] It is characterised by internal jugular vein thrombosis and septic emboli. Anticoagulation in Lemierre's remains controversial, with limited evidence to guide practice.[2-3] We report an atypical case of Lemierre's syndrome due to Staphylococcus aureus in a middle-aged woman with multiple chronic conditions, where infection management and anticoagulation required careful multidisciplinary decision making.

CASE REPORT

A woman in her early sixties with atrial fibrillation on long-term warfarin, type 2 diabetes mellitus, and hypertension presented with two weeks of worsening confusion, fluctuating consciousness, and new left-sided weakness. On arrival, she was noted to have a supratherapeutic INR of 9, elevated inflammatory markers and normal platelets. Neurological examination showed left hemiparesis and variable GCS.

Urgent CT brain excluded haemorrhage. MRI brain confirmed acute infarcts in the right thalamus, midbrain, and medial temporal lobe, with a right subdural empyema. Extensive odontogenic sinusitis with an oroantral fistula was also evident. Contrast-enhanced sequences revealed multiloculated empyema, early orbital involvement, and a non-occlusive thrombus in the right sigmoid sinus and jugular bulb, consistent with Lemierre's syndrome (Fig1 & Fig2). Blood cultures later grew Staphylococcus aureus.

She was transferred to a tertiary centre for joint management with neurosurgery and ENT/oral-maxillofacial teams and underwent functional endoscopic sinus surgery and tooth extraction. Broad-spectrum intravenous antibiotics were commenced, later tailored to prolonged linezolid therapy.

Repeat imaging post-procedure confirmed cerebral venous sinus thrombosis,possibly due to septic embolization. She was anticoagulated initially with therapeutic dalteparin and later switched back to warfarin. Over the following weeks, she showed neurological recovery and was transferred for inpatient rehabilitation. Interval imaging demonstrated resolution of the empyema and evolving, stable infarcts, with mild dural thickening.

IMAGING

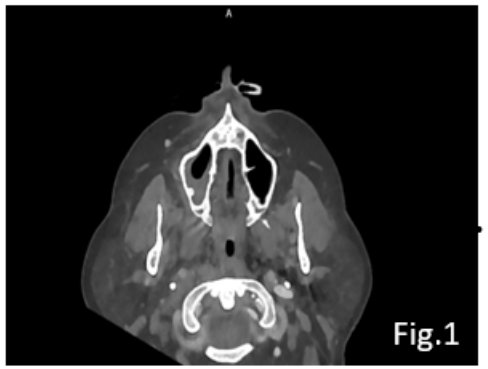
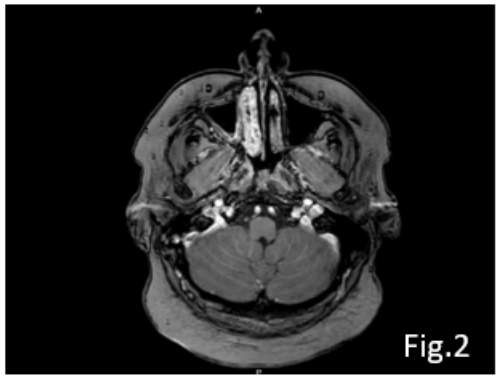


Fig1. Axial CT venogram demonstrating thrombus within the right internal jugular vein at the C1-C2 vertebra level.

Fig2. Axial contrast-enhanced MRI showing thrombus within the right jugular bulb



CONCLUSION

This case highlights the complex therapeutic dilemmas of Lemierre's syndrome in a middle-aged patient with significant comorbidities. While antibiotics remain the cornerstone of treatment, the role of anticoagulation is far less certain. Our patient developed extensive thrombosis despite a supratherapeutic INR, showing that infection-driven endothelial injury, venous stasis, and systemic inflammation can bypass vitamin K–dependent pathways (Virchow's triad).[4] The decision to reverse warfarin, pursue neurosurgical and ENT intervention, and later restart anticoagulation underscores the need for multidisciplinary, patient-centred care.

References 1. Riordan T. Human infection with Fusobacterium necrophorum (Necrobacillosis), with a focus on Lemierre's syndrome. Clinical microbiology reviews 2007;20(4):622–59. 2. Kuppalli K, Livorsi D, Talati NJ, Osborn M. Lemierre's syndrome due to Fusobacterium necrophorum. The Lancet Infectious Diseases 2012;12(10):808–15. 3. Phua CK, Chadachan VM, Acharya R. Lemierre syndrome—should we anticoagulate? A case report and review of the literature. The International journal of angiology : official publication of the International College of Angiology 2013;22(2):137–42. 4. Bagot CN, Arya R. Virchow and his triad: a question of attribution. British journal of haematology 2008;143(2):180–90.

CNS Rickettsial Infection Beyond the Brain :

Case Series Highlighting Ocular and Renal Manifestations

Dr. Alisha Musthafa[1], Dr Rajiv Motiani |Maidstone and Turnbridge Wells NHS Trust, Maidstone, UK | NEO hospital, Noida, India.

Aim:

Highlight atypical ocular and renal manifestations and the value of early therapy.

Introduction:

- **Rickettsial diseases are increasingly recognised across India, with presentations ranging from febrile illness to severe multi-organ dysfunction.**
- **Neurological complications, including meningoencephalitis, remain under-reported and may mimic other infectious or inflammatory conditions, delaying diagnosis and treatment.[1,2]**
- **Early initiation of doxycycline is critical to reducing morbidity and mortality.[3]**

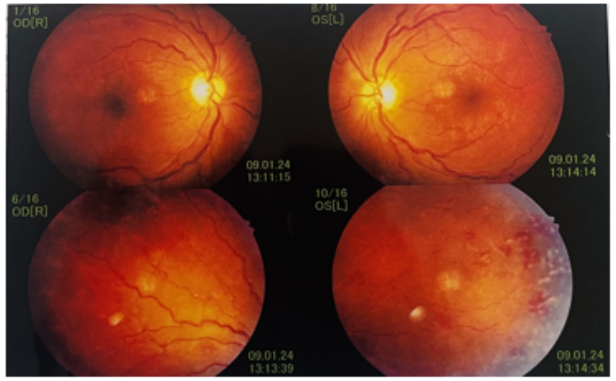
Case 1:

- **A 12-year-old girl presented with fever, photophobia, headache, vomiting, and neck rigidity. Initial testing suggested typhoid fever. MRI brain was unremarkable, but cerebrospinal fluid (CSF) analysis revealed pleocytosis with elevated protein and low glucose. Multiplex PCR detected Rickettsia spp. Ophthalmological review showed bilateral retinal haemorrhages. Broad-spectrum antimicrobials were replaced with intravenous doxycycline, resulting in full recovery.**

Case 2:

- **A 38-year-old man presented with fever, altered sensorium, aphasia, anuria, and metabolic acidosis. Investigations demonstrated acute kidney injury with raised creatinine and blood urea. CSF analysis revealed lymphocytic pleocytosis with raised protein; PCR confirmed Rickettsia spp. He responded rapidly to intravenous doxycycline, with both neurological and renal parameters normalising within five days**

Feature	Case 1: 12 y F	Case 2: 38 y M
Presentation	Fever, photophobia, headache, vomiting, neck rigidity	Fever, altered sensorium, aphasia, anuria, backache
Ocular	Bilateral retinal haemorrhages	Normal fundus
Renal	Normal	AKI (↑ creatinine & urea)
CSF	287 cells/mm^3, ↑protein, ↓glucose, PCR +Rickettsia spp.	55 cells/mm^3, ↑protein, normal glucose, PCR +Rickettsia spp.
MRI Brain	Normal	Normal
Treatment	Doxycycline IV	Doxycycline IV
Outcome	Full recovery	Full recovery



Discussion:

- **These cases demonstrate the diverse neurological and systemic involvement of rickettsial disease.**
- **Vasculitic injury to cerebral endothelium underlies meningoencephalitis, with manifestations ranging from meningism and seizures to visual deficits.[3,5,6] Ocular involvement, such as retinal haemorrhages, is uncommon but documented in association with CNS infection.[7]**
- **Renal dysfunction, including acute kidney injury, has also been described in severe rickettsial disease and may complicate recovery if not promptly treated.**
- **The rarity of combined neurological, ocular, and renal complications highlights the importance of maintaining a high index of suspicion.**
- **PCR of CSF, while not widely available, is a valuable diagnostic adjunct alongside serological assays.[1,2]**
- **These cases also reinforce the necessity of empiric doxycycline therapy in suspected rickettsial infections, as delayed initiation is associated with poor outcomes.[4,6]**

Conclusion:

- **Rickettsial meningoencephalitis, though rare, should be considered in unexplained neuro-ophthalmologic or renal presentations in endemic areas. Our series illustrates the value of early doxycycline therapy and highlights the role of advanced diagnostics in confirming atypical cases.**
- **Increased awareness of these multi-system manifestations can facilitate earlier recognition and improve patient outcomes.**

References:

1. Rathi N, Rathi A. Rickettsial infections: Indian perspective. Indian Pediatr 2010;47:157-64.
2. Rolain JM, Parola P, Cornet JP, Raoult D. Diagnosis of Mediterranean spotted fever by real-time PCR. Clin Infect Dis 2004;38:1428-34.
3. Woods ME, Olano JP. Host defences to *Rickettsia rickettsii* infection contribute to increased microvascular permeability in human cerebral endothelial cells. J Clin Immunol 2008;28:174-85.
4. Tikare NV, Shahapur PR, Bidari LH, Mantur BG. Rickettsial meningoencephalitis in a child: a case report. Trop Doct 2010;40:198-200.
5. Kulkarni R, Khadse S, Kinikar A, Valvi C, Mahajan R. Rickettsial fever with ARDS and meningo-encephalitis: a case report. Paediatr Infect Dis 2013;5:75-7.
6. Shaked Y. Rickettsial infection of the central nervous system: the role of prompt antimicrobial therapy. QJM 1991;79:301-8.
7. Jena SS, Satapathy S, Misra S, Sahu BK. Meningo-encephalitis due to spotted fever group Rickettsia: a rare case report with serial follow-up MRI. Neurol India 2022;70:412-4.

The Heart of the Matter : MELAS Syndrome-induced Cardiomyopathy

University Hospital Southampton NHS Foundation Trust

Dr Rita Omweri, Dr Edna Mensah, Dr Chitsa Seyani, Dr Catherine Mercer, Dr Andrew Flett

Royal College of Physicians

CASE PRESENTATION

- PC: **39yr old** Caucasian male presented to ED with sudden onset central chest pain lasting 5hrs.
- PMH: Childhood Asthma, Type 1 Diabetes complicated with retinopathy.
- Medications: Insulin. NKDA
- FH: Strong family history of cardiovascular death and T1DM was noted.
- Mother passed away from myocardial infarction aged 50, and her identical twin sister from heart failure aged 54. Both were diagnosed with T1DM.
- Patient's sister was diagnosed with Epilepsy and T1DM.
- SH: Accountant. No ETOH. Social smoker & no illicit drug use
- Stable observations. Bibasal crepitations noted on auscultation, otherwise unremarkable examination.

INVESTIGATIONS

- Initial troponin was elevated at **1439ng/l** (local range <12ng/L) and **5369ng/l** at 3hrs.
- ECG: no changes indicating an acute coronary syndrome.
- Lactate was elevated at **4.4mmol/L**, other blood tests were unremarkable.
- CXR: venous congestion, increased cardiothoracic ratio and no pleural effusions.
- TTE: **severely impaired biventricular systolic function**. Left ventricular ejection fraction of **20-25%** (normal range 55-65%).
- Coronary angiogram: unobstructed coronary arteries.
- Cardiac MRI: overall appearances suggestive of a potential inherited cardiomyopathy. Apical transmural scar and microvascular obstruction (MVO) representing previous event.
- **Management**: He was commenced on ACS management and Furosemide 40 mg OD for venous congestion. He was started on heart failure treatment and a CRT-D device was eventually implanted.

CARDIAC MRI AND PEDIGREE

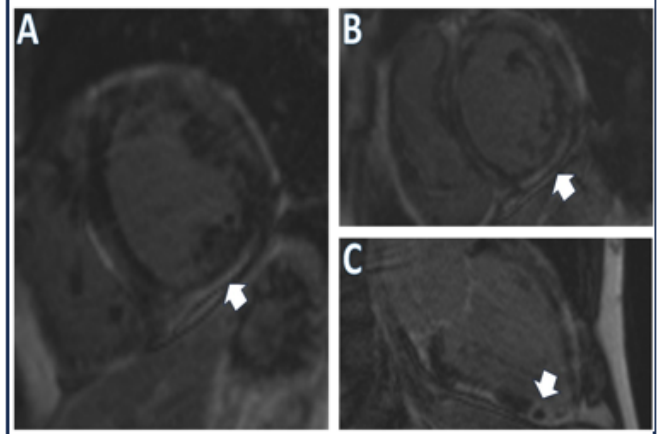


Figure 1: Short & Long Axis Late Gadolinium Enhancement (LGE) Images: Short axis (A&B) and long axis (C) with white arrows indicating areas of LGE. Image C highlights transmural enhancement of the apical inferior wall with a central area of MVO.

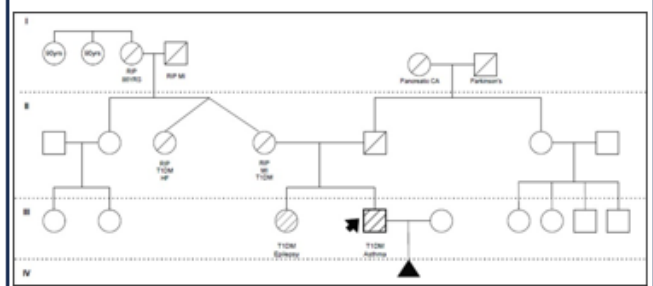


Figure 2: Family tree: Genetic screening which revealed pathogenic mitochondrial DNA variant m.3243A>G in the MT-TL1 gene, confirming the molecular diagnosis of mitochondrial DNA disease.

DISCUSSION

- **Mitochondrial, Encephalomyopathy with Lactic Acidosis and Stroke-like episodes (MELAS) syndrome** is a rare, progressive, neurodegenerative, mitochondrial disorder originally described by Pavlakis in 1984.
- Most common mutation is the A-G substitution at nucleotide 3243 (m.3243A>G).
- MELAS has a highly variable presentation, and it affects tissues with high energy demand.
- Commonly presents with neurological symptoms, a few cases have reported cardiac pathologies such as HCM, DCM and conduction defects.
- Our case highlights a rare case of MELAS with cardiac symptoms, elevated cardiac biomarkers and high serum lactate. Cardiac MRI and family history alluded to an underlying inherited cause.
- **Learning point:** Young patients with heart failure, and a significant family history should be evaluated for an inherited cardiomyopathy. Genetic screening is invaluable in these patients and their first-degree relatives.

Pleomorphic Dermal Sarcoma of the Eyelid:

A Rare Case Report and Review of the Literature

Dr Maria Skaria[1], Dr Mark Awad[1], Mr Matthew Gillam[1]

[1]Royal Bournemouth Hospital, Bournemouth, Dorset, UK

NHS
University Hospitals Dorset
NHS Foundation Trust

INTRODUCTION

Pleomorphic dermal sarcoma (PDS) is a rare, aggressive skin tumour, and periocular cases are uncommon. We report a lower eyelid PDS treated with local excision without complications.

CASE SUMMARY

A 72-year-old man with a background of renal cell carcinoma and Parkinson's disease (PD) presented for follow-up of a left eyelid basal cell carcinoma (BCC). A rapidly growing lesion on the right lower eyelid was noted. Biopsy confirmed **p53 and CD10 positivity** and **negative MNF116, EMA and p63**, consistent with PDS. The case was discussed in local and national MDT sarcoma meetings, and two-stage excision was recommended. Histologically clear margins were achieved. The patient **declined adjuvant radiotherapy** and reconstructive eyelid surgery; however, the site healed by secondary intention with excellent functional and cosmetic outcomes. Follow-up CT imaging showed **no recurrence or metastasis**.

TIMELINE

0 months	New right lower lid lesion noted on follow-up for left eyelid BCC; biopsy advised but patient chose to monitor.
5 months	Lesion enlarged with bleeding noted.
6 months	Incisional biopsy performed.
8 months	Histology suspicious for high-grade dermal sarcoma - referred to local and national MDTs.
9 months	Two-stage excision performed; clear margins achieved.
10 months	MDT confirmed grade 3 PDS. Reconstruction and radiotherapy both declined.
11 months	Wound healing well by secondary intention on follow-up.
13 months	Oncology review - CT-CAP clear. Eyelid healed well.
16 months	CXR clear, surveillance ongoing.

CLINICAL IMAGES

Figure 1: Appearance of right lower eyelid at the point of biopsy.

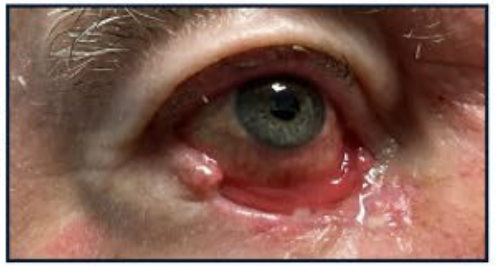


Figure 2: Appearance of right lower lid one month post-operatively.

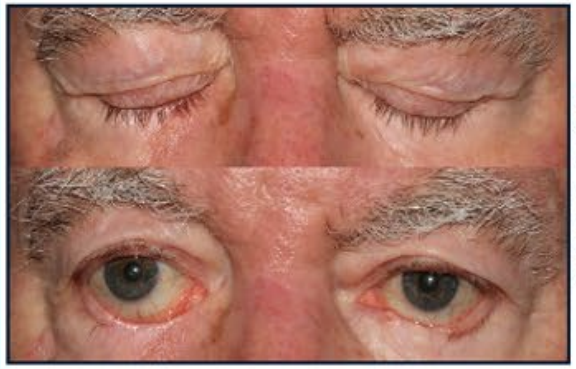


Figure 3: Appearance at 7 months post-op, with good cosmetic and functional outcomes achieved.

DISCUSSION

- Periocular PDS is rare, with **only two cases** in the literature to date.[1,2] Our case highlights a favourable outcome due to **early and incidental detection**.
- Atypical fibroxanthoma (AFX) and PDS exist on a spectrum. AFX is less aggressive, but PDS presents with **high-risk histological features**.[3,4] Early biopsy and immunohistochemistry are therefore essential in its investigation.
- Surgical management of PDS comprises excision with adequate **margin control**.[3] Preserving eyelid **function** and acceptable **cosmetic outcome** is challenging in periocular PDS. Tissue sparing techniques such as Moh's micrographic surgery have been described for eyelid malignancies.[1,5,6] **Radical excision may not be needed** if histologically clear margins obtained.
- Collaborative working between district general hospitals and specialist sarcoma centres facilitated effective and prompt treatment for this patient. Early MDT involvement has been shown to **improve morbidity and mortality** rates in rare cancers.[7]

LEARNING POINTS

1. **Early biopsy and early MDT involvement are crucial if suspecting a malignant eyelid lesion.**
2. **Tissue-sparing techniques for eyelid lesions can maintain both clear margins and excellent functional/cosmetic outcomes.**
3. **A patient-centred approach is key. Good outcomes were still achieved despite the patient declining radiotherapy/reconstructive surgery.**

REFERENCES

1. Wong S, Zhang Y, Li W, et al. Periocular pleomorphic dermal sarcoma: clinical features and management. 2016;17(4):375-82.
2. Lee J, Choi Y, Park J, et al. Diagnostic challenges in rare eyelid tumors. 2018;30(6):1259-65.
3. Kim E, Lee S, Choi H, et al. Management of rare ocular sarcomas: a comprehensive review. 2019;30(9):1325-33.
4. Chang J, Park K, Jang W, et al. Advances in the diagnosis and management of pleomorphic dermal sarcoma in ocular regions. 2020;35(2):231-6.
5. Smith R, Davis J, Jones B, et al. Histological markers for identifying pleomorphic dermal sarcoma. 2019;22(1):67-72.
6. Harris M, Lin X, Richards K, et al. Surgical outcomes in periocular pleomorphic dermal sarcoma: a multicenter review. 2023;10(5):456-65.
7. Yu Z, Wang X, Liu L, et al. Case report on eyelid pleomorphic dermal sarcoma: clinical features and surgical management. 2021;16(3):212-5.

Consent obtained for the use of clinical images.

Severe Pectus Excavatum–Induced Ventricular Arrhythmia & Cardiac Arrest in a Teenager

Authors: Abdalla Reda Mahmoud, Urshila Ramah, Mohamed Ahmed, Periklis Perikleous, Ian Hunt

Introduction:

Pectus excavatum is the most common congenital chest wall deformity, characterized by an inward depression of the sternum [1]. It affects approximately 1 in 400 live births, with a male predominance [2]. While many patients are asymptomatic or present with cosmetic concerns, severe cases may lead to cardiopulmonary compromise, including arrhythmias, right heart compression and exercise intolerance [3]. Rarely, PE may precipitate malignant arrhythmias and cardiac arrest. **We present the case of a teenager who developed ventricular fibrillation (VF) cardiac arrest secondary to severe PE.**

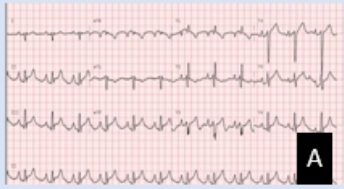
Case presentation & Discussion

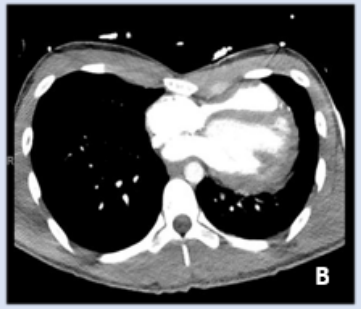
A 17-year-old male collapsed at a construction site with VF arrest. He achieved return of spontaneous circulation after 14 minutes of Advanced Life Support. On admission, he was intubated for low Glasgow Coma Scale. Examination revealed severe PE. The collateral history from his parents revealed that he had been complaining of palpitations for the past 2 weeks and worsening tiredness on physical exertion for the past few months, but he never had any previous episodes of syncope.

Investigations demonstrated elevated troponin (16,000 ng/L), right bundle branch block with right axis deviation on ECG and CTPA findings of pericardial agenesis, right ventricular dilatation, prominent pulmonary artery, and severe PE (Haller index 3.9) (Figure 1). Echocardiography revealed impaired left ventricular systolic function (ejection fraction 45–50%) with extracardiac compression of the right atrium and ventricle (Figure 1C). Cardiac MRI excluded arrhythmogenic right ventricular cardiomyopathy, and Holter monitoring showed ventricular ectopics only.

The patient underwent minimally invasive repair of PE (Nuss procedure) with pericardial patch, relieving cardiac compression. Intraoperatively, a deep asymmetric deformity with left-sided pericardium was confirmed. Due to his VF arrest, a prophylactic single-lead implantable cardioverter defibrillator (ICD) was inserted. Post-operative imaging confirmed satisfactory device and bar position (Figure 2). He recovered well and was discharged home within days.

This case demonstrates the life-threatening potential of severe PE. Mechanical compression of the right heart chambers likely precipitated arrhythmia and cardiac arrest. Surgical correction, combined with ICD implantation, was both therapeutic and preventive. Multidisciplinary evaluation was essential in decision-making.





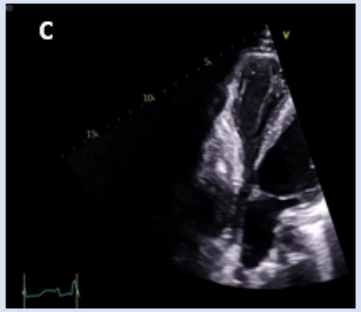
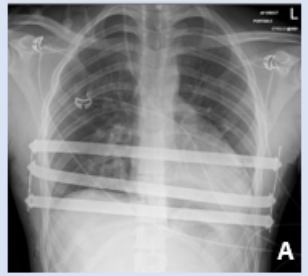
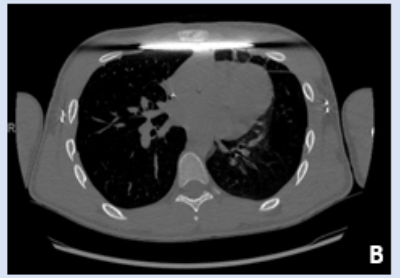


***Figure 1**: Investigations admission. A : ECG with paramedics. B: Cross-sectional image of CT chest on admission C: Apical four chamber view on cardiac echo showing RV compression.*

***Figure 2**: Post operative images. A: Chest Xray done after the Nuss procedure. B: CT scan chest showing Nuss fixation in situ.*





Conclusion

Severe pectus excavatum can extend beyond cosmetic implications to cause malignant arrhythmias and cardiac arrest. Early recognition of symptoms, thorough diagnostic evaluation, and timely multidisciplinary intervention are critical. Definitive surgical correction and arrhythmia protection strategies can be lifesaving in young patients with severe PE.

*References: **[1]** Langan PR, Pectus excavatum, Radiology Case Reports, Vol 6, Issue 1, 2011; **[2]** Diseases of the chest wall. UpToDateONLINE 18.1[Updated 2009 Dec 21]; **[3]** Moossdorff M, Maesen B, et al. (2021). Case report: ventricular fibrillation and cardiac arrest provoked by forward bending in adolescent with severe pectus excavatum. European Heart Journal*

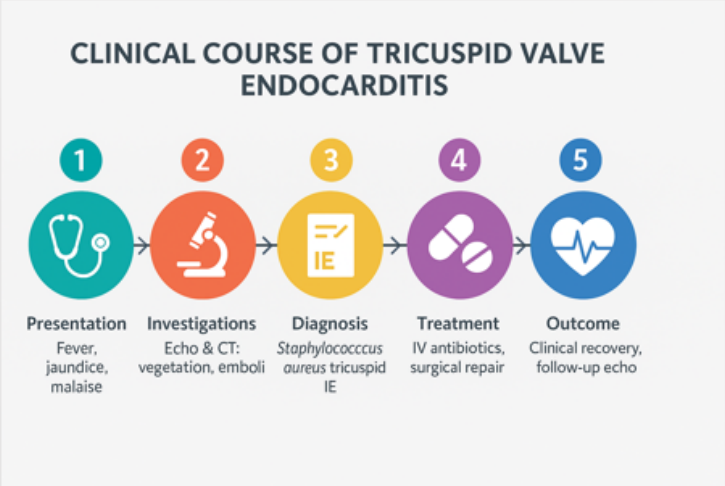
NHS
St George's University Hospitals
NHS Foundation Trust

When Risk Factors Deceive: A Rare Case of Tricuspid Valve Endocarditis

Dr Tanushree Dewan*, Dr Akhil Tomy, Dr Maria John, Dr Cristoss Gregory, Dr Soon Neoh, Dr Chetan Upadhyaya
Glan Clwyd Hospital, Betsi Cadwaladr University Health Board

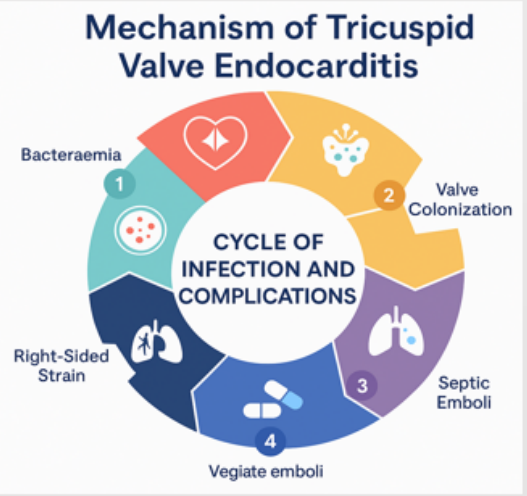
Introduction

- Infective endocarditis (IE) involving the tricuspid valve accounts for 5–10% of IE cases.
- Typically seen in intravenous drug users or those with central venous catheters.
- Right-sided IE in patients without risk factors is rare.
- Highlights importance of early diagnosis and multidisciplinary care.



Discussion

- Non-IVDU tricuspid IE is rare; transient bacteremia and indwelling devices possible risks.
- Staphylococcus aureus is the predominant organism.
- Large vegetations (>2 cm) and septic emboli → surgical indication.
- Early surgery prevents right heart failure, improves survival.
- Atypical presentation causes diagnostic delay.



Clinical Course

History and examination

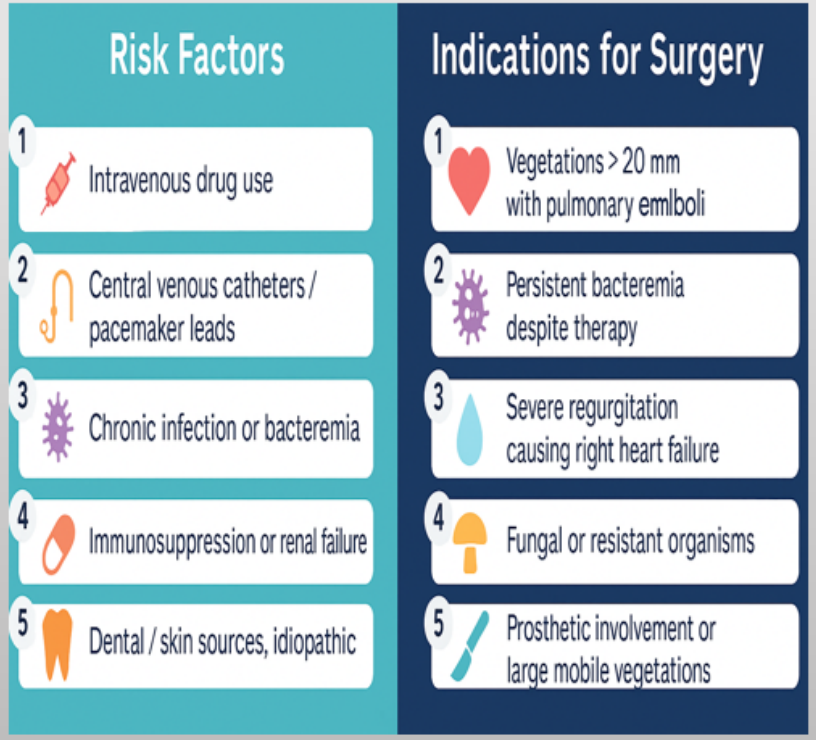
- 47-year-old male with fever, jaundice, and malaise; no IVDU history.
- Systolic murmur, hepatomegaly.

Investigation

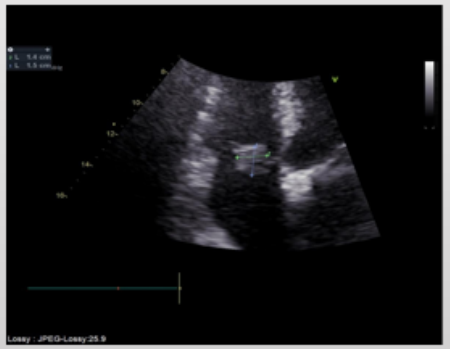
- Labs: leukocytosis, elevated CRP, cholestatic LFTs.
- Blood cultures: Staphylococcus aureus.
- Echo: 2.4 cm tricuspid vegetation, severe regurgitation.
- CT thorax: septic emboli to lungs.

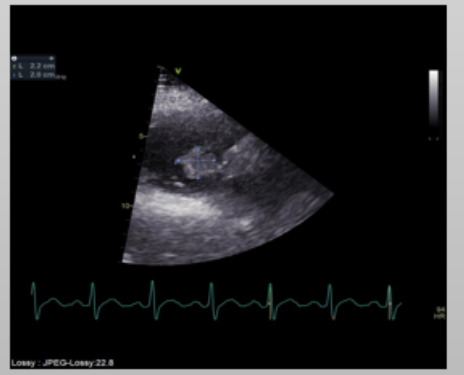
Management

- Managed with IV antibiotics then surgical repair.



Echo Images





Conclusion

- Tricuspid IE can occur in non-IVDU patients.
- Early recognition and multidisciplinary care are key.
- Surgical intervention for large vegetations or persistent sepsis.

References

1. Habib G, et al. Eur Heart J. 2015;36:3075–3128.
2. Nappi F, et al. J Thorac Cardiovasc Surg. 2019;157(1):172–182.
3. Akinosoglou K, et al. J Infect Public Health. 2014;7(6):357–369.
4. Hussain ST, et al. Ann Thorac Surg. 2016;101(1):116–123.
5. Cahill TJ, et al. Lancet. 2016;387(10021):882–893.

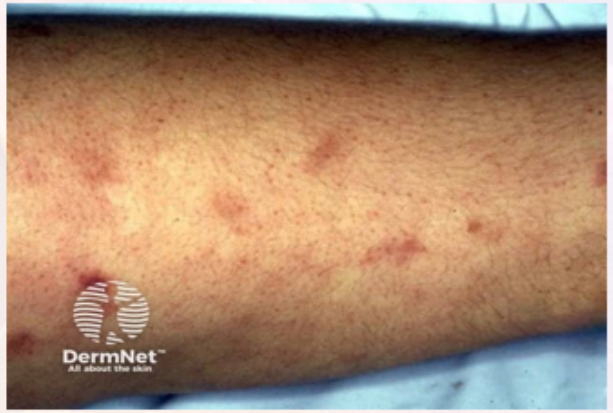
Cutaneous Polyarteritis Nodosa: From Misdiagnosis to Definitive Diagnosis

Dr Tanushree Dewan*, **Dr Maria E John***, Dr Akhil Tomy, Dr Cristoss Gregory, Dr Tahir Aziz
Glan Clwyd Hospital

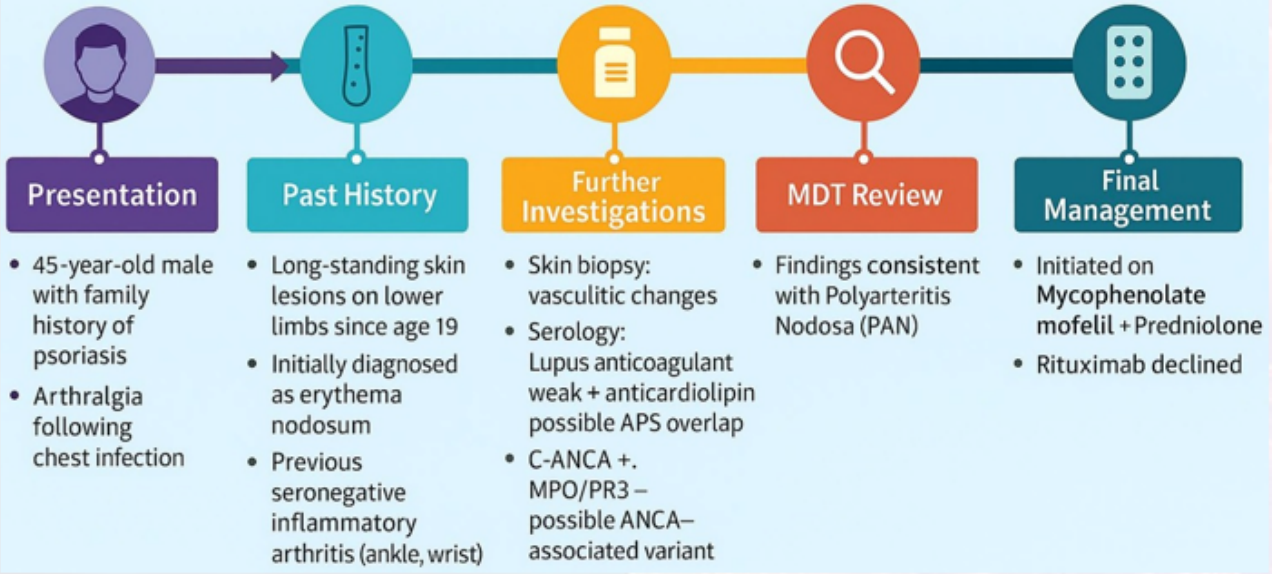
Introduction

Polyarteritis nodosa is a medium vessel neutrophilic vasculitis with systemic or cutaneous forms.
Cutaneous PAN often presents with skin and peripheral nerve involvement[1], occasionally with myalgia and constitutional symptoms.

We report the case of a 45-year-old man with chronic inflammatory arthritis and persistent cutaneous vasculitis lesions.



Case Presentation

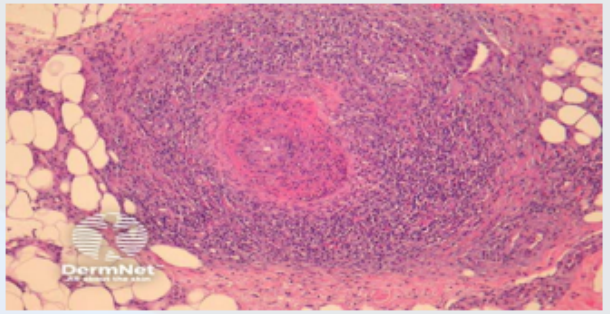


Discussion

Diagnostic challenge:
- PAN can mimic other rheumatologic and dermatologic conditions.
- Cutaneous signs include nodules, ulcers, necrosis, and livedo reticularis[2].
- **Skin biopsy** is key for confirmation[3].

***Clinical features*:**
- Arthralgia may precede other symptoms, causing diagnostic delay[4].
- **Cutaneous vs Systemic PAN:**
- Differentiation is vital; systemic PAN has worse prognosis[5].
- Cutaneous PAN rarely progresses to systemic form[6].

***Treatment*:**
- **Steroids** are first-line.
- **Rituximab** shows mixed results in refractory cases[7].
- **Infliximab** may offer benefit in select cases.
- Biologics hold promise but need further study.



Conclusion

PAN remains a diagnostic challenge due to its variable presentation. This case emphasizes the need for timely MDT input and early diagnosis. It also stresses on the notion of initiating novel treatment modalities and further improving our understanding of their efficacy.

References

1.Matteoda MA et al. Cutaneous polyarteritis nodosa. An Bras Dermatol. 2015;90(Suppl 1):188–90.
2.Saghari S et al. Polyarteritis nodosa with complications: a diagnostic challenge. Cureus. 2023;15(11):e49677.
3.Morgan AJ, Schwartz RA. Cutaneous polyarteritis nodosa: review. Clin Exp Dermatol. 2010;35(5):514–20.
4.Takamatsu R et al. Arthritis as initial manifestation in cutaneous PAN. Case Rep Rheumatol. 2020;2020:8897358.
5.Liang I et al. Spectrum of cutaneous PAN: two contrasting cases. Clin Case Rep. 2022;10(12):e6659
6. Nakamura T et al. Cutaneous PAN: revisiting definition and criteria. J Dermatol. 2008;35(9):543–50.
7. Boukhris I et al. Rituximab efficacy in refractory PAN. Pan Afr Med J. 2023;45:92

Neuropsychiatric adverse effects following Ashwagandha supplementation

A Case of Sleep Abnormalities, Acute Confusion and Retrograde Amnesia temporally associated with ashwagandha ingestion in a Healthy Adult

Dr G. Thushani Anuththara[1], Dr Sarah Amin[1], Dr Benedict Sebastiampillai[1]
[1]Department of Acute Medicine, Peterborough City Hospital

Royal College of Physicians

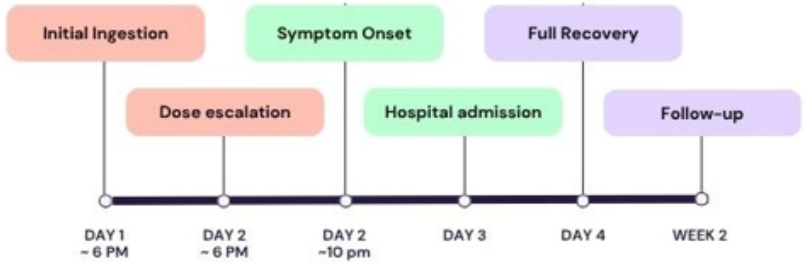
NHS North West Anglia NHS Foundation Trust

Introduction

- Ashwagandha (***Withania somnifera***) is a popular adaptogenic herb
- Extensively used in traditional Ayurvedic medicine
- Ashwagandha supplementation has increased significantly in Western countries

Case Presentation

- Patient: 50-year-old male, previously well
- Clinical features:
  - **Prolonged sleep** (~23 hours)
  - **Intermittent abnormal behaviours during sleep**
  - **Altered mental status on waking**
  - **Transient memory loss**
  - Vitals: haemodynamically stable
  - Neurology: no focal deficits. **4AT- 10**
- Clinical Timeline:



Investigations

- Blood tests:
  - Mild neutrophil leucocytosis
  - Normal inflammatory markers
- Urgent NCCT head: No acute pathology
- MRI Brain: No structural abnormalities
- CSF Analysis: Unremarkable

Management

- Initial Approach:
  - Working diagnoses:
    - **Infective meningoencephalitis**
    - **Drug-induced encephalopathy**
  - Empirical IV antibiotics and antivirals
  - Immediate Ashwagandha discontinuation
  - Supportive care
- Treatment Evolution:
  - Antimicrobials stopped after negative workup
  - Continued monitoring
- Clinical Outcome:
  - Complete resolution by day 4
  - Sustained recovery at 2 week follow up

Discussion

- Evidence suggesting Drug-induced aetiology:
  - Close temporal relationship
  - Recent dosage escalation
  - Rapid resolution post discontinuation
  - **Exclusion of alternative diagnoses**
  - Similar reactions documented in literature
  - **Naranjo Adverse Drug Reaction Probability Scale: 7**
- Probable risk factors:
  - **Individual metabolic variability**
  - **Genetic polymorphisms in drug metabolising enzymes**
  - **Dose escalation patterns**

Key Clinical Messages

- **Comprehensive drug history: consider herbal supplements**
- **Adverse event/drug interaction reporting**
- **Clinician awareness/ Patient education**

References

- Singh N et al., 2011, Afr J Tradit Complement Altern Med, doi:10.4314/ajtcam.v8i5S.9.
- Mishra L et al, 2000; Altern Med Rev, doi:10.21275/SR25520190842
- Fazlani M et al., 2025, Cureus, doi:10.7759/cureus.89104.

Rare Case Of Multiple Cutaneous Lesions From Atypical Carcinoid Tumour Of Unknown Primary

Authors: Fatma Shah, Anesa Noor, Junaid Kayani

Northampton General Hospital NHS Trust

Background

- Carcinoid tumours are rare, slow-growing neuroendocrine neoplasms.
- They most commonly originate in the gastrointestinal (62%) and bronchopulmonary (23%) systems.[1]
- Cutaneous metastases from carcinoid tumours are extremely rare even among patients with advanced disease. [2]

Case Summary

- 37-year-old ♀ with no PMHx
- Presented with progressively worsening abdominal discomfort over a few weeks.
- Associated with **unintentional weight loss** (30Kg over 6 months).
- Examination: **Numerous widespread cutaneous nodules** over her forehead, neck, back, breasts, trunk, and thighs.
- Variable sizes and characteristics (scarring/ ulceration, some were hard and fixed with no clear edges, while others were mobile).
- First lesion reportedly appeared 2 years ago
- Assessed by GP with US → presumed to be sebaceous cysts.
- Despite the extensive metastatic burden, the patient exhibited no typical features of carcinoid syndrome, such as flushing, diarrhoea, or wheezing.

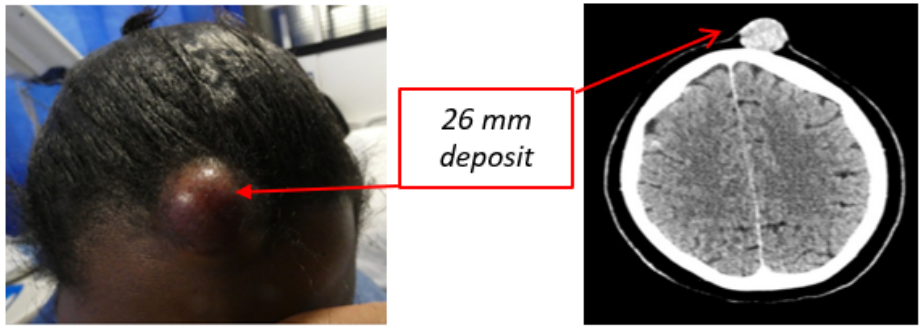


Figure – 1 gross and CT images of 26mm lesion on forehead

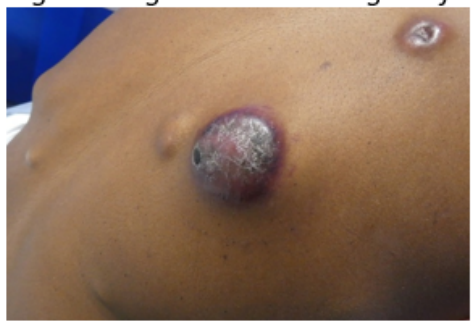


Figure – 3
Gross features of lesions on the back

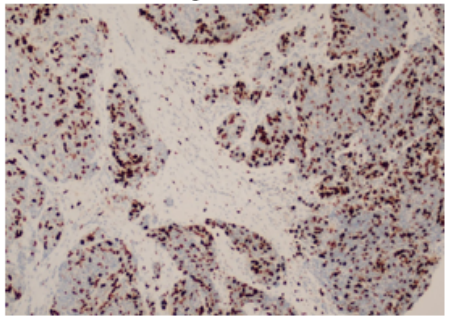


Figure – 4
Ki-67 index 30%

Investigation

- CT Scan: multiple lobulated soft tissue enhancing lesions involving the subcutaneous tissue of the **neck, chest, abdominal wall and retroperitoneum**. And deep organs such as the **brain, lungs, liver and spleen**.
- Biopsy: metastatic neuroendocrine tumour. Immunohistochemical staining was consistent with a **carcinoid tumour**.

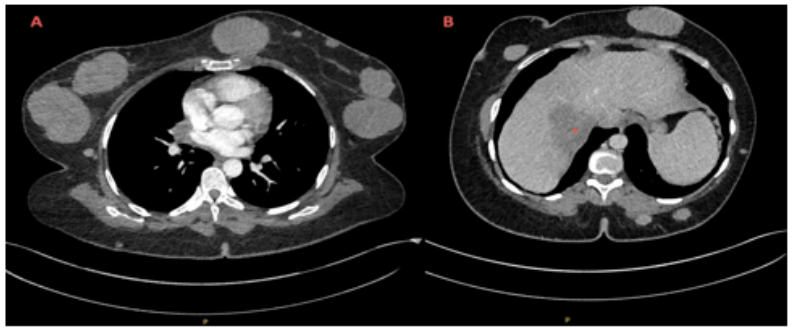


Figure – 3 (A) Multiple lobulated soft tissue density deposits in the chest wall (B) lobulated hypodense lesion in the liver

Conclusion

- Cutaneous metastases from carcinoid tumours can mimic benign dermatological conditions, leading to delays in diagnosis.
- The varied presentation of the cutaneous nodules in this case: from painless to ulcerated and from fixed to mobile illustrates the diverse morphological spectrum of such metastases.

References

1. Zuetenhorst JM, Taal BG. Metastatic carcinoid tumors: a clinical review. The oncologist. 2005 Feb 1;10(2):123-31.
2. Amorim GM, Quintella D, Cuzzi T, Rodrigues R, Ramos-e-Silva M. Cutaneous metastasis of neuroendocrine carcinoma

Hypercalcaemia as the initial manifestation of Addison's Disease

Authors: Dr Orlaith Fogarty, Dr Nyein Nge Nge, Dr. Taofeek Ojewuyi, Dr Ali Rathore, Dr Ayanbola Adepoju
Southend University Hospital, Mid and South Essex NHS Foundation Trust, Southend-On-Sea, United Kingdom

Background:

- Hypercalcaemia is a common biochemical finding, which is often asymptomatic, with causes broadly classified into PTH-dependent and PTH-independent.
- This case report describes a patient with undiagnosed primary adrenal insufficiency presenting with PTH independent hypercalcaemia, hyponatremia and acute kidney injury.

Case:

- A 39-year-old man presented with dizziness, low mood, anorexia, nausea, vomiting, and weight loss.
- He was found to be hypotensive and had generalised skin hyperpigmentation.
- The biochemistry results on admission are seen in Table 1.
- The initial working diagnosis was that of PTH-independent hypercalcaemia-induced dehydration with AKI.
- Malignancy was ruled out with a CT chest, abdomen and pelvis scan.

	Value	Reference
Sodium (mmol/L)	126	133-146
Potassium (mmol/L)	5	3.5 - 5.3
Urea (mmol/L)	13.7	2.5 - 7.8
Creatinine(µmol/L)	148	59 - 124
Adjusted calcium (mmol/L)	3.05	2.2 - 2.6
Parathyroid hormone (pmol/L)	0.3	1.3 - 9.3

Table 1: Urea and electrolytes

	Value	Reference
ACTH (ng/L)	1301	<50
Cortisol (nmol/L)	<11	185-624
TSH (mU/L)	11.36	0.3 - 5.0
Free T4 (pmol/L)	8.9	7.9 - 16.0
Renin (nmol/L/hr)	1.4	0.3-3.5
Aldosterone (pmol/L)	<60	90-700
Adrenal autoantibodies	Positive	

Table 2: Subsequent endocrine testing

- Adrenal insufficiency was confirmed (Table 2). The patient was commenced on hydrocortisone replacement and subsequently levothyroxine.
- Following steroid replacement, the patient and their electrolytes improved, with the hypercalcaemia and hyponatremia resolving.
- Whilst aldosterone was suppressed, **uncommonly in Addison's disease, renin was normal.**
- The patient was initially managed without fludrocortisone; however, this was introduced at three months when renin levels began to rise (Table 3).

	On admission	Follow-up	Reference
Renin (nmol/L/hr)	1.4	13.8	0.3-3.5
Aldosterone (pmol/L)	<60	<60	90-700

Table 3: Renin and Aldosterone

Conclusion:

- Addison's disease can present atypically with PTH-independent hypercalcaemia, hyponatremia, hypotension and acute kidney injury.
- A high index of suspicion of adrenal insufficiency will enable swift diagnosis in patients with unexplained electrolyte disturbance.

Disclaimer: This case is due to be presented at the Society for Endocrinology Clinical Update 2025 (3-5 November 2025)

Repeated presentation with exercise induced asthma exacerbation- Have you considered EILO?

Lwin Paing, Saquib Siddiqui

Introduction

We present a case of exercise-induced laryngeal obstruction (EILO) masquerading as asthma exacerbation.

Case Presentation

28-years old female presented to A&E with breathlessness and wheeze with multiple previous admissions with asthma exacerbation. Further exploration revealed onset during marathon training. Symptoms triggered at peak exercise and no other exposure to trigger factors. She had wheeze on auscultation.

Poor response to asthma treatment prompted investigation for asthma mimics.

Asthma screening blood panels were done which showed unremarkable. Most recent spirometry was unremarkable.

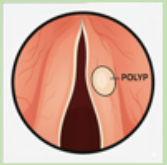
Continuous laryngoscopy during exercise confirmed swelling of the left vocal fold causing obstruction. Biopsy showed benign pathology, surgically removed by ENT.

Postoperatively, no recurrence of symptoms.

NHS South Tees Hospitals NHS Foundation Trust

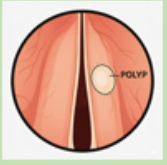
Discussion

- EILO is temporary, reversible, and inappropriate paradoxical movement of the vocal cords or supraglottic structures during inspiration, triggered by exercise.
- More common in young females due to narrower laryngeal anatomy.
- Manifest as dyspnoea, high-pitch inspiratory breathing sounds to clear-cut stridor, respiratory distress, tachypnoea and or panic reactions. Symptoms remain inactive until intense exercise pursues.
- Symptoms typically appear during maximal intense physical activities and resolve within 2-3min of exercise cessation unless ongoing hyperventilation
- EILO often mimics asthma but differs in timing and no response to treatment.
- Continuous laryngoscopy during exercise is the diagnostic gold standard.
- Management requires a multidisciplinary approach with respiratory physicians, ENT specialists, speech therapists, and psychologists.
- If non-surgical therapy is refractory, supraglottoplasty can be considered. Any vocal cord lesion can be surgically resected for resolution of symptoms as was done in our patient.



Vocal cord at rest

Fig 1. Illustration on changes of vocal cords



Vocal cord on exertion

Key Learning Points

1. EILO can mimic exercise-induced asthma and lead to misdiagnosis and failed treatment.
2. Always consider asthma mimics when symptoms are exercise-related and unresponsive to standard therapy.
3. Multidisciplinary management provides the best outcomes

References

1) Sayad E, Das S. Exercise-Induced Laryngeal Obstruction. https://www.ncbi.nlm.nih.gov/books/NBK556148/
2) Arne Coussement , Pieter Goeminne , Nico De Crem et al. A 63-Year-Old Man With Persistent Chest Constriction on exercise, DOI:10.1016/j.chest.2025.03.004
3) Makiko Ohtani, Masamitsu Kono, Shunji Tamagawa et al. Exercise-induced laryngeal obstruction in a competitive swimmer associated with the use of a snorkel, DOI: 10.1016/j.jsams.2025.04.001
4) Hege Havstad Clemm, J Tod Olin, Cameron McIntosh et al. Exercise-induced laryngeal obstruction (EILO) in athletes: https://doi.org/10.1136/bjsports-2021-104704
5) Thomas Halvorsen, Emil Schwarz Walsted, Caterina Bucca et al. Inducible laryngeal obstruction: an official joint European Respiratory Society and European Laryngological Society statement,

RECURRENT IECOPD WITH T2RF

NHS
Nottingham
University Hospitals
NHS Trust

Background IECOPD with decompensated T2RF is associated with high mortality rate (≈20% within 30 days). Medical treatment & NIV are established therapy, yet post-discharge management remains critical in determining outcomes.

Case report A 69-year-old woman, with a background of COPD, mild obesity, limited mobility and smoking, had 3 hospital admissions of IECOPD with decompensated T2RF within 1 month.

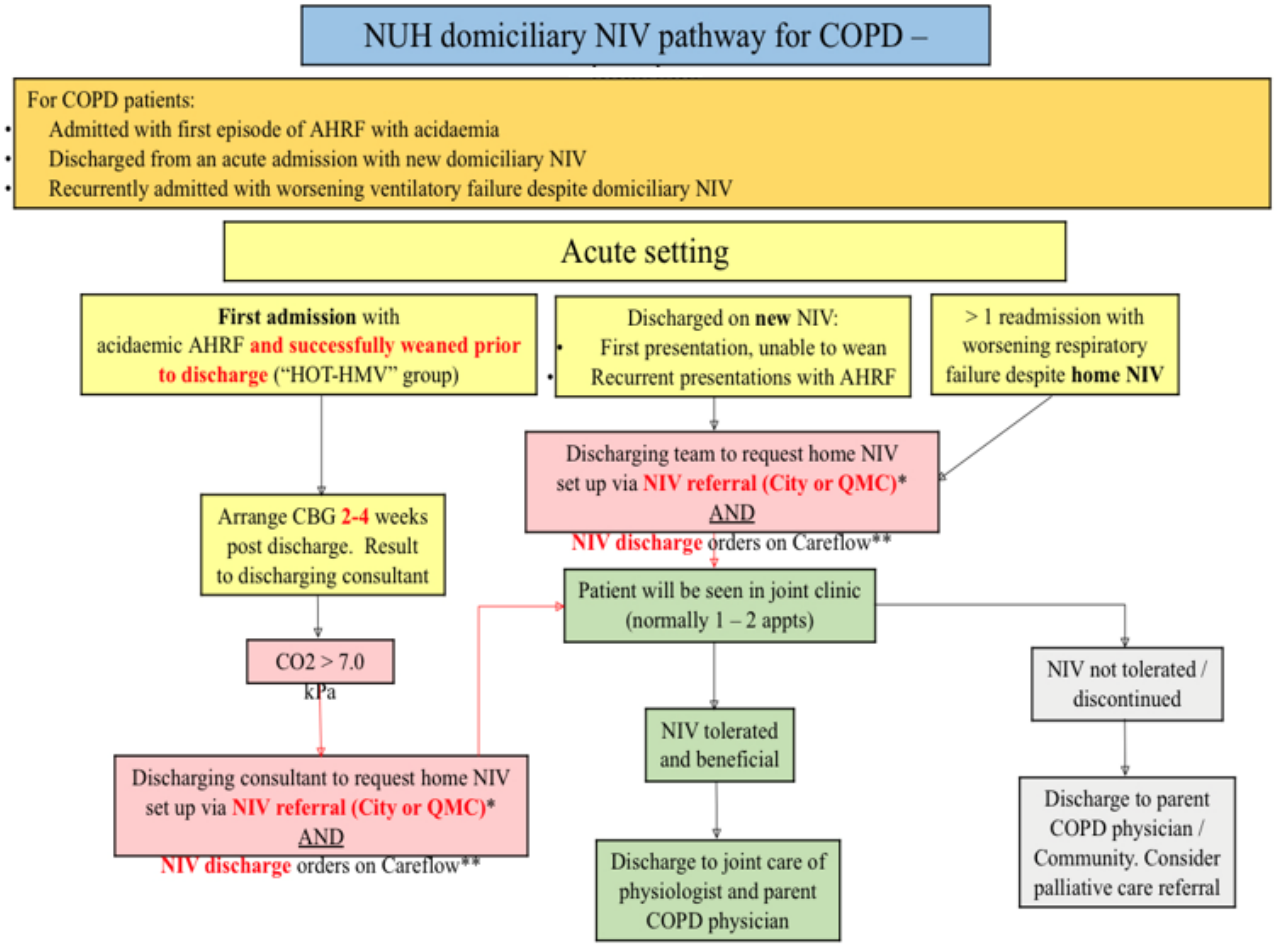
1st admission: She was admitted with IECOPD with decompensated T2RF (Ph 7.25, PCo2 9.35), reversed to normal Ph/PCo2 with Abx, steroid & nebs. CXR showed emphysematous changes. She was discharged with respiratory nurse OPA.

2nd admission: Few days later, she was re-admitted with the same presentation of IECOPD with decompensated T2RF (Ph 7.33, PCo2 8.9), persisting despite standard medical therapy. CXR showed RLZ opacification. She improved with NIV, which was gradually weaned over 5 days. In the following 6 days, daily monitoring of blood gases assured no further hypercapnia after stopping NIV (Ph 7.4, PCo2 5.4). she was monitored for 48 hours after successful weaning off oxygen, then discharged with OP lung function tests and domiciliary NIV assessment in 4 weeks as per HOT-HMV.

3rd admission: Few days post-discharge, symptoms recurred and IECOPD medical treatment was initiated at home by ambulance. However, she rapidly desaturated on 15L O2, leading to two successive cardiac arrests (PEA). ROSC was achieved in hospital, followed by trial of thoracocentesis for suspicion of Pneumothorax but showed minimal effect. CXR showed no pneumothorax, and severe T2RF was noted (Ph 7.0, PCo2 10.9). Given her low physiological and functional reserve, end-of-life care was initiated and she passed away in hours.

Discussion Despite clinical improvement, NIV weaning over 5 days, maintaining normocapnia for 6 days Post NIV, maintaining oxygen saturation on room air for 48 hours post O2 therapy, a rapid deterioration happened only 3 days post-discharge.

Conclusion Recurrent admission with IECOPD with T2RF in a short span of time should be considered as a higher risk of rapid clinical deterioration.



Learning Points

On top of following trust guidelines, initiation of domiciliary NIV pre-discharge should be considered based on clinical judgement in patients who had recurrent IECOPD with T2RF admissions in a short span of time once NIV is required.

Author & Co-authors

Eslam Shehab, CT1
Millind Sovani, Respiratory consultant
Sygiin Chong, Respiratory consultant
Celestia Singh, CT1
Annabel Birchall, FY1

Starved for immunity: A Rare Case of Disseminated Mycobacterium szulgai

Dr Tanushree Dewan*, **Dr Mansha Bhirvani*** Dr Divina Morais*, Dr Niladri Dutta, Dr Sana Ahmer, Dr Daniel Menzies
Glan Clwyd Hospital

* Denotes joint first authorship

GIG CYMRU NHS WALES
Bwrdd Iechyd Prifysgol Betsi Cadwaladr University Health Board

Introduction

- Nontuberculous mycobacteria (NTM) are environmental pathogens.
- Mycobacterium szulgazi is rare (<0.2% isolates) but pathogenic.
- This case highlights the link between malnutrition, psychiatric illness, and infection.

Presentation

- 42F with bulimia, anxiety & depression
- Weakness, polydipsia, Na 116 mmol, BMI 13kg/m²

Investigation

- CT Chest: Upper lope cavitary changes
  Blood culture: Mycobacterium szulgai

Treatment

- Macrolide, Rifampicin, Fluoroquinolone

Outcome

- Deterioration, multi-organ failure and death

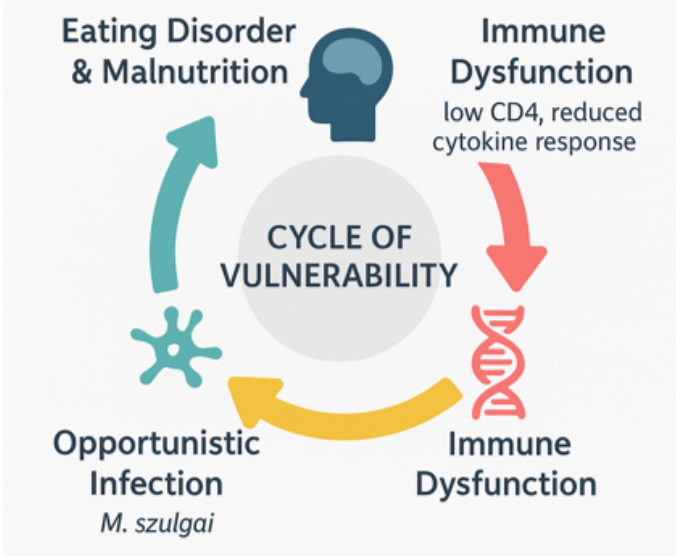


Image 1; Cycle of vulnerability

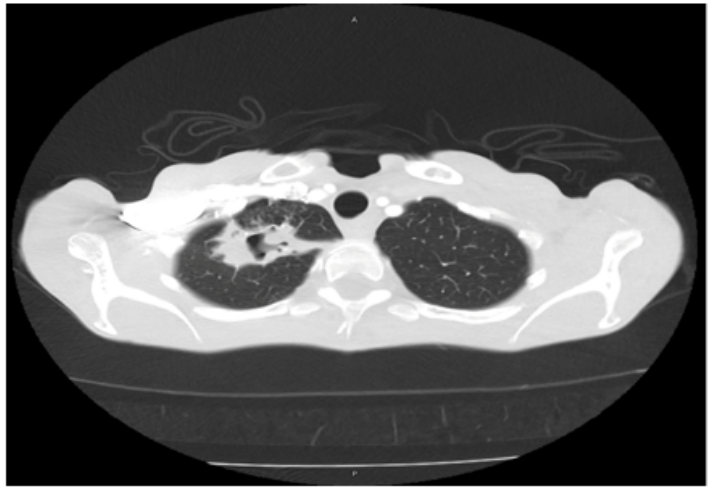


Image 2: CT chest: Cavitary changes

Discussion

- Severe malnutrition → functional immunodeficiency.
- Disseminated NTM infection is possible without HIV/immunosuppression.
- Diagnostic delay common due to subtle imaging/culture requirements.
- Requires prolonged multidrug therapy (>12 months).
- Multidisciplinary input vital.

Conclusion

- This case highlights the role of psychiatric disorders and nutritional neglect in predisposing to opportunistic infections
- M. szulgai infection may occur in malnourished psychiatric patients.
- Early multidisciplinary care (psychiatry, ID, nutrition) is essential.
- Mental health and nutrition underpin infection outcomes.

References

1. Prevots DR, Marras TK. Clin Chest Med. 2015;36(1):1–13.
2. Henry M, et al. Clin Infect Dis. 2004;39(11):1709–12.
3. Cederholm T, Jensen GL. Curr Opin Clin Nutr Metab Care. 2017;20(2):161–8.
4. Shin S, et al. Tuberc Respir Dis. 2013;75(1):1–8.
5. Daley CL, et al. Am J Respir Crit Care Med. 2020;202(5):e62–122.

VARICELLA-INDUCED HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN AN IMMUNOCOMPETENT YOUNG MALE: A RARE CLINICAL CHALLENGE

Nehal Varghese[1], Smitha Muraletharan[2] Department of Internal Medicine, Aster Medcity, India

INTRODUCTION

Hemophagocytic lymphohistiocytosis (HLH) is a rare but life-threatening hyperinflammatory syndrome triggered by infections, malignancy, or autoimmune conditions.
Varicella zoster virus (VZV) is an uncommon precipitant, with only a few adult cases reported.

CASE SUMMARY

A 23-year-old male with recent history of road traffic accident presented with acute low back pain of one day duration
On examination, he had fever - 101°F, tachycardia -115/min, tenderness over the L3–L5 region. Systemic examination was unremarkable.
Initially, local causes were ruled out (CT spine was normal)
He subsequently developed fever and headache—started on IV antibiotics, analgesics, and supportive care.
Lab findings: Deranged liver function tests —SGOT 431 U/L , SGPT 484 U/L → USG abdomen normal; Hepatitis A, B, C, E & HIV negative.

-Day 2: Severe chest pain requiring ICU monitoring—cardiac enzymes, ECG, ECHO normal; CT aortogram was done (aortic dissection was ruled out).

-Day 3: Developed vesiculopapular rashes over face, neck, torso → clinically diagnosed with varicella zoster infection—started on IV antivirals.

-Day 4: Persisting fever. Lab findings: Elevated liver enzymes (SGOT-3109 U/L and SGPT-1932 U/L), Severe thrombocytopenia (20K/uL), elevated ferritin (41260ng/mL), low fibrinogen (135mg/dl) → suggestive of early HLH. Urgent Hematology consult → Started on Intravenous Immunoglobulin (2gm/kg over 48 hours) and IV steroids.

-Day 6: Lab parameters showed improving trend (SGOT-1908 U/L and SGPT-1082U/L), Platelet count (45K/uL), Ferritin (23371ng/mL. Fever spikes associated with headache and vomiting persisted → blood/urine cultures repeated, IV antibiotics escalated.CT Brain with venogram → cerebral venous thrombosis ruled out.

-Day 8: Patient clinically better., Platelet count (70K/uL) → continued antivirals & antibiotics and supportive care.

-Day 16: 14-days of IV acyclovir completed.Symptomatically improved and discharged.

DISCUSSION

Varicella is rarely associated with HLH in immunocompetent young adults.Our case underscores the importance of early recognition and intervention with antivirals and immunomodulatory therapy. Recent adult HLH guidelines highlight the need for prompt initiation of therapy, even before full diagnostic criteria are met, due to the high risk of mortality.This case highlights the diagnostic challenges, the importance of a multidisciplinary approach, and the need to consider HLH in severe or atypical varicella presentations.

DIAGNOSTIC CRITERIA OF HLH

The diagnosis of HLH can be established if either A or B is fulfilled:
A.A molecular diagnosis consistent with HLH
B. Any 5 of the 8 following clinical and laboratory criteria for HLH:
1.Fever >38.5° C
2.Splenomegaly
3.Cytopenia (affecting ≥2 of 3 lineages in peripheral blood):
—Hemoglobin <9 g/dL (in infants <4 weeks: Hb <100 g/L)
—Platelets <100×109/L
—Neutrophils <1.0×109/L
4.Hypertriglyceridemia and/or hypofibrinogenemia: fasting triglycerides >3.0 mmol/L (>265 mg/dL) or fibrinogen ≤1.5 g/L
5.Hemophagocytosis in bone marrow, spleen, liver, lymph nodes, or other tissues
6.Low or absent natural killer (NK) cell activity
7.Serum ferritin concentration ≥500 µg/L
8.Soluble CD25 (soluble IL-2 receptor) ≥2400 U/mL

CONCLUSION

Varicella-induced HLH is rare but potentially fatal. Prompt diagnosis and combined antiviral and immunomodulatory therapy can significantly improve outcomes.

ECTOPIC HYPERPARATHYROIDISM: DIAGNOSTIC CHALLENGES AND MULTIDISCIPLINARY MANAGEMENT

ZARA SHARIF, RYANNA JACOB, ABEL SINI, COSMINA SCHITENAU, ANTONY GEORGE
UNIVERSITY HOSPITALS NORTH MIDLANDS, ROYAL STOKE UNIVERSITY HOSPITAL

INTRODUCTION

•Ectopic hyperparathyroidism occurs when parathyroid tissue is located outside its normal anatomical site — commonly in the thymus, mediastinum, or retroesophageal space.

•It arises from abnormal embryological migration and affects approximately 16% of patients with primary hyperparathyroidism.[1]

•Thorough investigation of patients presenting with symptoms is vital in ensuring they are treated appropriately, as incomplete excision can lead to persistent disease.

OBJECTIVES

•We are presenting three cases which demonstrate the different manifestation, investigative findings and management of ectopic hyperparathyroidism.

•We aim to highlight the diagnostic diversity and multidisciplinary management of ectopic hyperparathyroidism, in order to optimise patient care.

CASE 1: EARLY LOCALISATION

• 41 year old female suffering from night sweats, brain fog and mood swings, as well as hypercalcaemia identified by her GP.

•Further investigations revealed primary hyperparathyroidism. A nuclear medicine parathyroid scan was carried out to identify whether there was a focal localised parathyroid adenoma present.

•A radioiodine uptake scan found homogenous uptake by the thyroid gland, as well as additional uptake in the superior mediastinum by a 12mm ectopic parathyroid gland.

•A single, curative procedure using Video-Assisted Thoracoscopic Surgery (VATS) was carried out.

•This patient was fortunate as unlike many reported cases where ectopic parathyroid tissue is only discovered after failed initial surgery, this lesion was localised at the outset.

CASE 2: MALIGNANT ECTOPIC

• 61 year old asymptomatic male with hypercalcaemia on routine blood tests by GP. Further investigations confirmed primary hyperparathyroidism.

• Initial ultrasound of parathyroid gland showed no evidence of an adenoma.

• A nuclear medicine scan which showed an ectopic parathyroid adenoma in his thorax.

• Consequent surgical intervention occurred through the collaboration of endocrinologists, ENT and cardiothoracic surgeons.

• Surgical excision confirmed a malignant lesion.

• Studies show that the likelihood of carcinoma in ectopic parathyroid tumours is extremely low, one report highlighting that of 84 mediastinal parathyroid tumours, only 1.2% were malignant.[2]

• This demonstrates the importance of considering the risk of malignancy when an ectopic gland has been identified.

CASE 3: ALTERNATIVE MANAGEMENT

• 65 year old female identified as having hypercalcaemia with elevated parathyroid hormone levels.

• Nuclear medicine imaging identified an ectopic parathyroid gland left of the aortic arch.

• Despite explaining the curative potential of surgical management to this patient, she expressed concerns about potential surgical complications, and preferred to proceed with cinacalcet.

• After further discussion in the endocrinology clinic, the patient agreed to re-evaluate her management options once input from the cardiothoracic surgeons was received.

• This case demonstrate the importance of shared decision making with patients, but also the importance of a multidisciplinary approach to ensure patients are informed about all of their management options.

DISCUSSION: WHY IDENTIFYING AN ECTOPIC PARATHYROID GLAND MATTERS

• Allows for a single, curative procedure

• Helps to determine the surgical approach to management

• Detection of rare malignancy

• Facilitates structured multidisciplinary care

FUTURE DIRECTIONS

How can we improve detection of ectopic parathyroid glands?

• Develop regional or national database of ectopic parathyroid cases.

•Foster a collaborative approach with various other specialties depending on the site of the ectopic parathyroid.

CONCLUSION

Ectopic hyperparathyroidism presents unique diagnostic and therapeutic dilemmas. These cases highlight the significance of efficient multidisciplinary team work, and how effective communication and collaboration between endocrinology, radiology and surgery can avoid unnecessary procedures for patients, and enable rapid diagnostic and curative success.

REFERENCES

1. Noussios G, Anagnostis P, Natsis K. Ectopic Parathyroid Glands and their Anatomical, Clinical and Surgical Implications. Experimental and Clinical Endocrinology & Diabetes. 2012 Nov 22;120(10):604–10.

2. Ruizhi Jiajue, Song A, Wang O, Li W. Persistent Hypercalcemia Crisis and Recurrent Acute Pancreatitis Due to Multiple Ectopic Parathyroid Carcinomas: Case Report and Literature Review of Mediastinal Parathyroid Carcinoma. Frontiers in Endocrinology. 2020 Sep 10;11.

Severe SLE complicated by HLH, PRES, and CMV: diagnostic and therapeutic challenges

Authors: Dr Sumaiya Khalil[1], Dr Maria Ashwini[1], Dr Mohammad Ehsan Siddiqui[1], Dr Fahim Dilbar[1], Dr Fiona Hayes[1]
[1] Barking Havering and Redbridge University Hospitals NHS Trust, London, UK

Background

SLE is a multisystem inflammatory disease. Secondary HLH is rare but life-threatening. Immunosuppression is essential yet increases infection risk. PRES may arise from hypertension, therapy, or infection and can mimic neuropsychiatric lupus.

Initial Diagnosis

A 36-year-old woman presented with fever, pancytopenia, and rash, meeting SLICC criteria for SLE. Persistent fevers, cytopenias and hyperferritenemia, despite corticosteroids suggested secondary HLH.

HLH

HLH confirmed by H-score and bone marrow haemophagocytosis. Managed with methylprednisolone and cyclophosphamide. Refractory inflammation required Anakinra, leading to improvement in cytopenias and ferritin

CMV Infection

Developed fever and cytopenias during immunosuppression. CMVPCR positive; ganciclovir led to viral clearance. This underscored the infection-immunosuppression balance.

PRES

She developed visual disturbance and seizures. MRI showed parieto-occipital PRES. Likely due to hypertension and immunotherapy. Resolved with BP control and supportive care.

Recovery

Multidisciplinary care achieved full recovery with normal counts, resolved inflammation, and restored neurological function.

Conclusion

SLE with HLH poses diagnostic and management challenges. Early HLH recognition and balanced immunomodulation are key to prevent mortality.

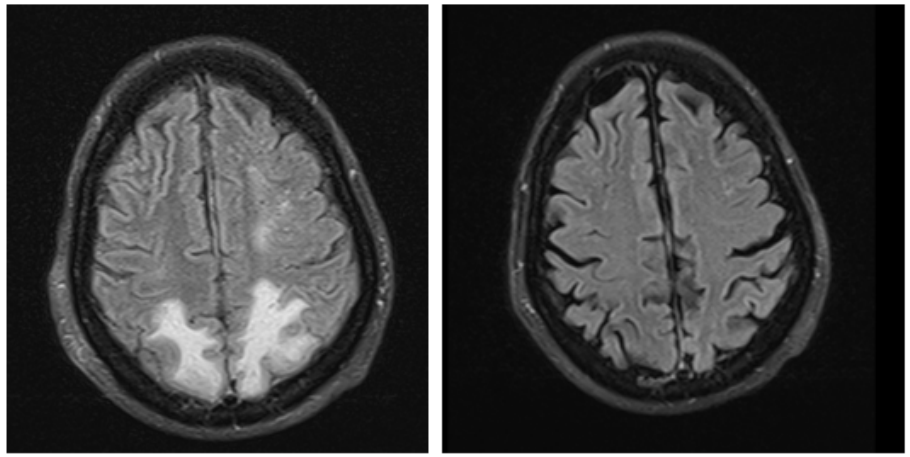


Fig 1 : Posterior reversible encephalopathy syndrome (PRES)
Fig 2: Resolution of parieto-occipital oedematous changes secondary to PRES after 1 month

Learning Points

- Recognize HLH early in uncontrolled SLE.
- Balance immunosuppression against infection risk.
- PRES can mimic neuropsychiatric lupus but is reversible .

Hoarseness due to recurrent laryngeal nerve palsy secondary to functional mitral regurgitation in ischemic cardiomyopathy: a variant of Ortner's syndrome

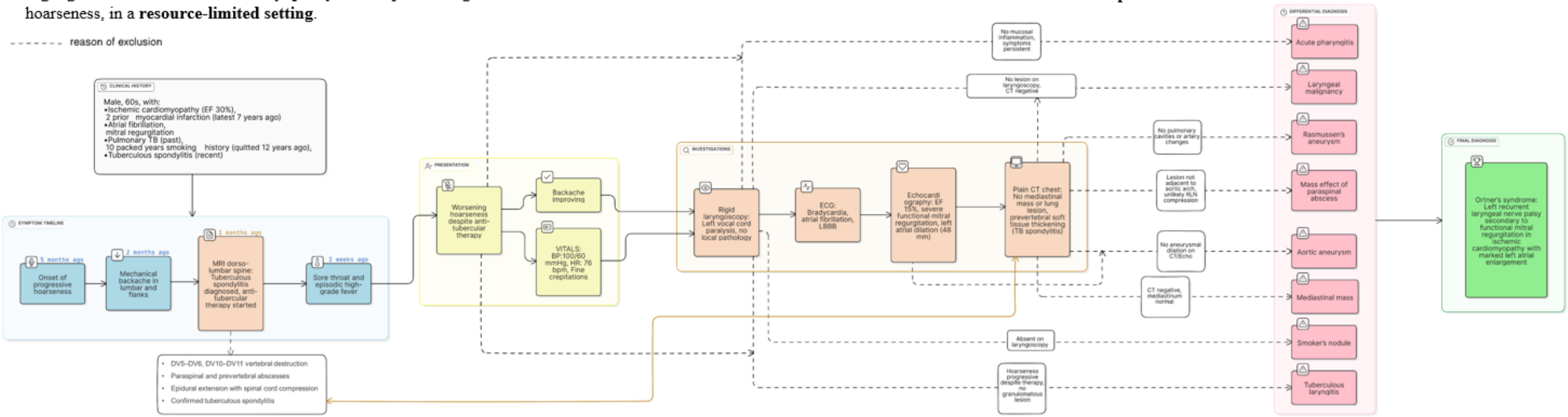
Shahid, Furqan[1]; Shahzadi, Sobia[1]
Mohi ud Din Islamic Medical college – AJK, Pakistan

Background

- **Ortner's syndrome** is a rare cause of unilateral **recurrent laryngeal nerve (RLN) palsy** due to **cardiovascular pathology**.
- Traditionally linked to **mitral stenosis**, novel research has implicated that it arises from **RLN compression** between the **aorta or ligamentum arteriosum** and **dilated pulmonary artery** within the **aortopulmonary window**.
- In this report, we describe what we believe is one of the few reported cases of **Ortner's Syndrome due to RLN palsy secondary to functional mitral regurgitation** in **ischemic cardiomyopathy** as a suspected diagnosis of hoarseness, in a **resource-limited setting**.

Case Presentation / Differential Diagnosis / Investigations

Figure 1: Diagnostic and investigative pathway
(Our own figure)



Outcome and Follow-Up

- **Further evaluation** with **contrast-enhanced CT (contrast was avoided due to renal impairment)**, **transoesophageal echocardiography**, and **biopsy** of the soft tissue lesion was recommended, along with consideration of **surgical mitral valve intervention** or **vocal cord medialization**.
- However, due to **financial constraints**, **comorbidities**, and **resource limitations**, the patient **declined further interventions** and was **lost to follow-up**.

Discussion

- Several **cardiovascular causes of RLN palsy** have been reported; its association with **mitral regurgitation** remains rare, with only a few cases described in the literature.
- In our patient, **functional mitral regurgitation due to ischemic cardiomyopathy** resulted in **marked left atrial dilatation**, leading to **RLN compression and hoarseness**.

Learning Points

- This case underscores the importance of considering **Ortner's syndrome** in patients with **unexplained hoarseness** and **significant cardiac disease**.
- **Echocardiography** plays a pivotal role in diagnosing **underlying cardiac causes**, while **chest imaging** assists in excluding **noncardiac aetiologies**.
- In **resource-limited settings**, awareness of this **rare entity** is crucial, as **diagnostic and therapeutic options** may be constrained.
- **Early detection** and **optimisation of cardiac status** may help prevent **progression of cardiac remodelling** and **secondary complications** such as **neuropathic hoarseness**.

Keywords: Ortner's syndrome, Functional ischemic mitral regurgitation, hoarseness .

Carotid Sinus Syndrome Unmasked by Eating: A case report and review of literature

Aza Abdulla (AA) Tayyab Mahmood (TM), Julianna Raghu (JR), Ahmad Khwanda (AK)

Introduction

Symptomatic hypotension following a meal in older people is common but poorly recognised and frequently underdiagnosed. Mildly symptomatic cases are often overlooked, while those with more severe symptoms may present after they have resolved. Post-prandial hypotension (PPH) may also potentiate an underlying asymptomatic hypersensitive carotid sinus, amplifying cardiovascular changes and leading to syncope or collapse. .We describe an older patient in whom dizziness was ultimately diagnosed as carotid sinus syndrome (CSS) only after post-prandial tilt-testing and carotid massage.

Case Presentation

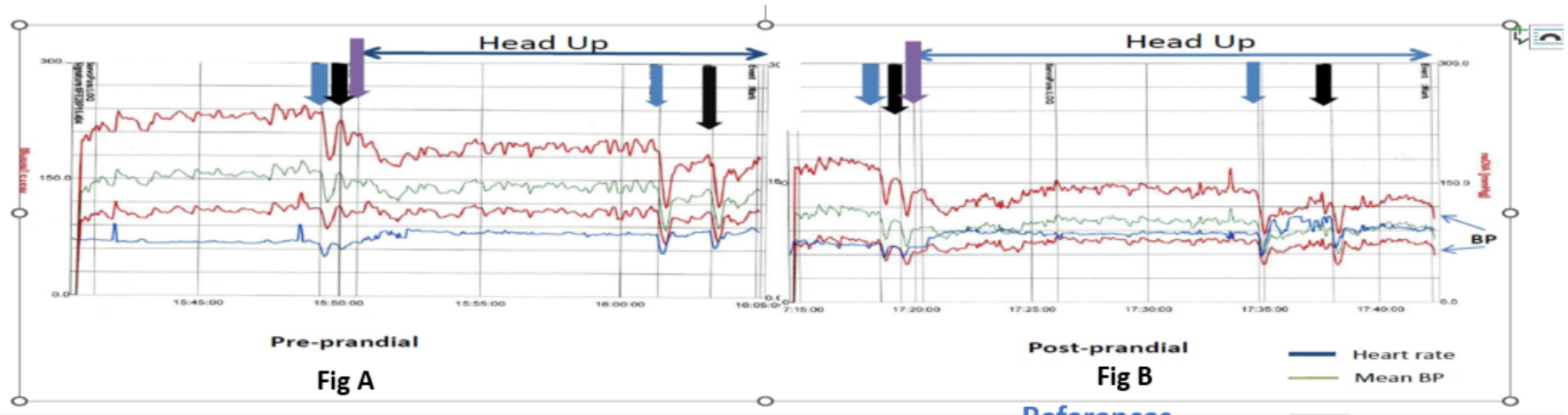
An 88-year-old woman was referred to our geriatric's clinic with a 7-month history of a "muzzy" head and dizziness. Symptoms were unrelated to posture, activity, or rising from bed, and differed from her earlier benign positional vertigo. Examination revealed no orthostatic hypotension, and blood tests, 24-hour ECG, CT, and MRI brain were largely unremarkable aside from small vessel disease and an old thalamic infarct.

Ambulatory BP monitoring demonstrated mild systolic drops coinciding with meals, raising suspicion of PPH. Tilt-testing and carotid sinus massage were therefore undertaken pre- and post-prandially. The pre-prandial tilt-test showed **(Fig A)** only mild asymptomatic BP fall with carotid massage in the upright position producing a vasodepressor response but no symptoms.

After eating **(Fig B)** , baseline BP and heart rate were lower, and carotid massage provoked marked hypotension and bradycardia with dizziness. On head-up tilt, these changes were magnified, with BP falling to 85/48 mm Hg, significant dizziness, and near collapse

Discussion

PPH, orthostatic hypotension (OH), and CSS are recognised causes of dizziness, falls, and blackouts in older adults. Though distinct, they often overlap and augment symptoms. PPH is defined as a systolic BP fall ≥20 mmHg within two hours of eating, or to <90 mmHg. OH is defined as a systolic drop ≥20 mmHg or diastolic drop ≥10 mmHg within three minutes of standing. CSS is characterised by symptomatic BP fall or asystole >3 seconds on carotid massage, with cardioinhibitory and vasodepressor forms. PPH occurs in up to a third of healthy older adults and is particularly common in frailty, Parkinson's disease, heart failure, and geriatric inpatients. OH may affect up to 81% of older patients, and CSS is reported in around 40% of those over 80 referred to syncope clinics. Despite this, PPH underdiagnosed. Our case highlights that asymptomatic CSS may be unmasked by eating, producing syncope or falls



Pre-prandial

Fig A

Post-prandial

Fig B

Conclusion

This is the first reported case of CSS triggered by meals. It demonstrates that asymptomatic hypersensitive carotid sinus may become symptomatic following eating. Clinicians should enquire about post-meal symptoms and consider post-prandial tilt-testing with carotid massage when standard evaluation is unrevealing. Awareness of this association is essential, as timely recognition may guide management. Treatment remains speculative, and further studies are needed to explore effective interventions

References

1. Jansen RWAMM & Lipsitz LA. Postprandial hypotension : epidemiology , pathophysiology , and clinical management . Ann Intern Med. 1995;(March).

2. Jansen RWMM. Postprandial hypotension: Simple treatment but difficulties with the diagnosis. Journals Gerontol - Ser A Biol Sci Med Sci. 2005;60(10):1268–70.

3. Vloet LCM, Pel-Little RE, Jansen PAF, Jansen RWMM. High prevalence of postprandial and orthostatic hypotension among geriatric patients admitted to Dutch hospitals. Journals Gerontol - Ser A Biol Sci Med Sci [Internet]. 2005 [cited 2020 Nov 30];60(10):1271–7. Available from https://academic.oup.com/biomedgerontology/article/60/10/1271/553135

When Elevated D-Dimer isn't VTE

Isa Alshamsan[1], Reem Alameri[2], Hari Nair[1]
Betsi Cadwaladr University Health Board[1], Royal college of Surgeons in Ireland[2]

Background

D-dimer is a fibrin degradation product routinely measured to exclude venous thromboembolism (VTE). Although highly sensitive, it lacks specificity and may be raised in infection, inflammation, malignancy, trauma, or after surgery and anticoagulation. Markedly elevated levels without evidence of VTE can pose a diagnostic challenge. This case highlights spontaneous splenic rupture as a rare but serious cause of extreme D-dimer elevation.

Case Presentation

An 83-year-old woman presented with a two-week history of intermittent, non-exertional, left-sided pleuritic chest pain. She denied breathlessness, leg swelling, or abdominal pain. Past medical history included paroxysmal atrial fibrillation, varicose veins, and recent shingles. Regular medications were Edoxaban and Amlodipine.

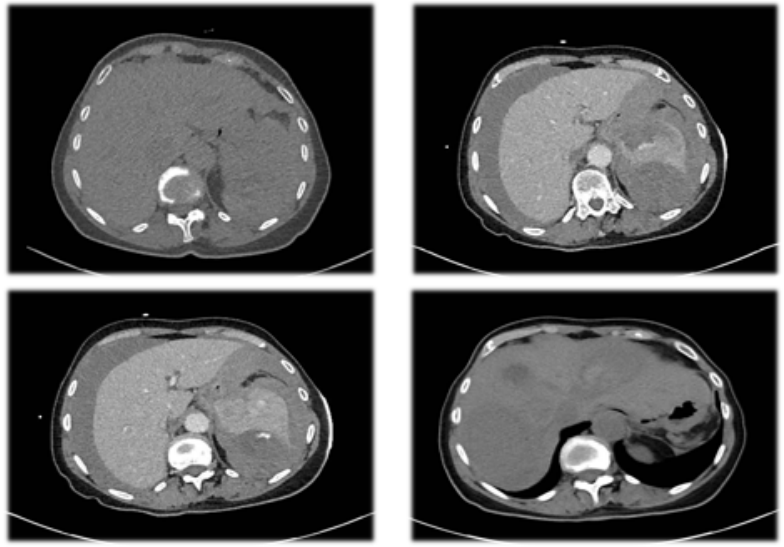
On examination, she was haemodynamically stable and afebrile, with normal cardiovascular and abdominal findings. Initial investigations showed a markedly elevated D-dimer of 10,000 ng/mL, rising to >14,000 ng/mL the same day. Other blood tests were normal, ECG confirmed atrial fibrillation, and CT pulmonary angiogram excluded pulmonary embolism. She was admitted for observation and intravenous antibiotics.

Investigations & Outcome

During the first night of admission, the patient experienced an acute collapse with transient loss of consciousness and hypotension. Despite an initial fluid challenge, she remained hypotensive and appeared clammy and peripherally shut down. Venous blood gas revealed a haemoglobin drop from 119 g/L to 77 g/L, raising suspicion for internal bleeding. Examination showed a distended, tender abdomen, with no evidence of gastrointestinal bleeding.

She received fluid resuscitation, high-dose intravenous proton pump inhibitors, and a blood transfusion. Once stabilised, an urgent CT abdomen and pelvis demonstrated a large-volume haemoperitoneum with suspected splenic rupture and active bleeding. Edoxaban was reversed, and she underwent emergency laparotomy confirming splenic rupture, with surgical haemostasis achieved.

Postoperatively, she required 48 hours of ICU monitoring, followed by transfer to the surgical ward. She recovered well and was discharged home after three additional days, with appropriate follow-up arranged.



Discussion

D-dimer is widely used in acute medicine to help exclude venous thromboembolism (VTE) because of its high sensitivity. However, it lacks specificity and may be elevated in infection, inflammation, malignancy, trauma, surgery, or with advancing age. Markedly raised D-dimer in the absence of thromboembolism presents a significant diagnostic challenge.

In this case, the patient presented with pleuritic chest pain and markedly elevated D-dimer but had no pulmonary embolism on CT pulmonary angiogram. Her subsequent haemodynamic collapse and abdominal distension revealed a spontaneous splenic rupture - a rare but life-threatening cause of internal bleeding. This underscores the importance of maintaining a broad differential diagnosis when faced with unexplained biochemical abnormalities.

Spontaneous splenic rupture, though uncommon, is recognised in patients on anticoagulation and in those with splenic pathology or infection. In this instance, Edoxaban may have contributed to the severity of bleeding. Early recognition, prompt reversal of anticoagulation, and timely surgical intervention were crucial for a favourable outcome.

Ultimately, elevated D-dimer should never be interpreted in isolation. Clinicians must integrate results within the clinical picture and remain alert to atypical causes, particularly when the patient's condition deteriorates unexpectedly. Vigilance and broad diagnostic thinking are essential to ensure timely diagnosis and appropriate management.

Learning Points

- D-dimer is highly sensitive but non-specific; interpretation requires careful clinical correlation.
- Spontaneous splenic rupture is a rare but life-threatening cause of acute abdomen, especially in anticoagulated patients.
- Markedly elevated D-dimer without VTE should prompt investigation for alternative diagnoses.
- Early recognition and prompt surgical management are vital to improve outcomes.

Isolated Coronary Arterial IgG4-Related Disease

When the Diagnosis Doesn't Fit the Criteria

Dr Jasper Mogg, Dr Clare Chown, Dr Edward Wheatley, Dr Sophie Langdon, Dr Christopher Goode, Dr Hannah Sinclair, Dr Kirstin Laverick

1 Patient Background

65-year-old male plumber

- Idiopathic left optic neuritis (40)
- Peyronie's disease (50)
- Hypercholesterolaemia (51)
- Atypical chest pain (52)
- Bilateral sensorineural hearing loss (58)
- Hypertension (63)
- Family history of ischaemic heart disease, hip fracture, and unspecified arthritis
- Never smoker, 20 units alcohol/week. 177cm, 100kg, BMI 32

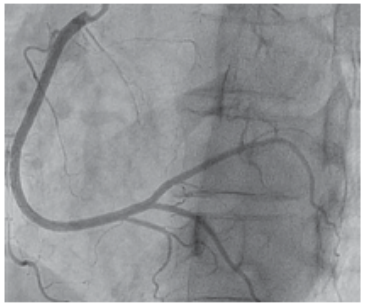
2 Lead-up

2021

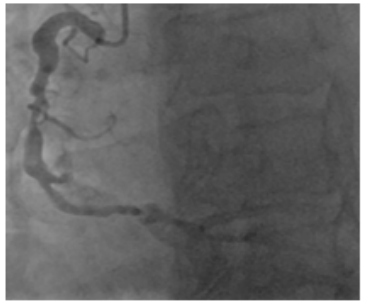
- Ischaemic chest pain and raised serum troponin.
- Flow-limiting atheromatous lesions in LAD and LCx with ectatic, dominant RCA.
- Drug-eluting stents, DAPT, statins, and holistic secondary prevention.

July 2023

- Further troponin-positive chest pain.
- Invasive angiography showed LAD and RCA aneurysms and stenoses.
- CT coronary angiogram (CTCA) showed markedly concentrically and eccentrically thickened segments of all coronary artery walls and perfusion defects in inferior segments suggested RCA flow restriction.



Normal right anterior oblique view of RCA



Aneurysmal and stenosed RCA seen in this patient

3 Rheumatology Review - December 2023

- Stable angina, occipital headaches, fatigue, and myalgia for 1 year.
- Arthralgia of hands, feet, elbows, knees, and neck with 1 hour morning stiffness.
- Red, maculopapular rash occurring on the trunk once a week.
- No other CTD features, pathergy, testicular pain, fever, VTE history, weakness, muscle wasting, synovitis, or effusion.
- Normal inflammatory markers (CRP <0.6mg/L, ESR 22mm/hr) and CK.
- Raised IgG4 (5.7g/L, normal <1.1g/L) and mild eosinophilia ($0.51x10^9$/L).
- Negative ANCA, CTD, and infection screening (see QR code for full list).
- PET-CT showed coronary FDG avidity and reactive mediastinal lymph nodes.

Differential Diagnosis

- Fibromuscular dysplasia (FMD), polyarteritis nodosa, Takayasu's, or IgG4-RD.
- CT angiography demonstrated no evidence of FMD.

IgG4-RD Diagnosis

- Substantially raised IgG4 levels and mild eosinophilia
- Lack of extracardiac findings on PET-CT
- Typical demographic
- Typical pattern of coronary involvement[3]

4 IgG4-Related Disease

- Autoimmune condition causing fibroinflammatory lesions in nearly any organ.
- Morbidity and mortality driven by obstructive or compressive pathology indirectly from organomegaly or directly by cellular infiltration and fibrosis.[1]
- Requires clinical, serological, radiological, and histological correlation.[2]
- Coronary IgG4-RD vasculitis is a recognised but uncommon manifestation of the condition (1-3%)[5], but appears to be very rare in isolation.[3]

2019 ACR/EULAR Classification Critera[4] and 2020 Japanese Revised Comprehensive Diagnostic Criteria[1]

- Characteristic histopathology of lymphoplasmacytic infiltrate and storiform fibrosis (most diagnostic).
- Serum IgG4 level (5x upper limit of normal highly indicative).
- Clinical or radiological involvement of characteristic organ: glands/thorax/hepatopancreatobiliary/kidney/retroperitoneum.

5 Management

Monthly prednisolone taper commenced (40-30-20-15-10-9-8-7-6-5-4-3-2-1-0).

One-Month Review

Much improved angina and energy levels, excellent response to steroid therapy.

Six-Month Review

CTCA on 9mg prednisolone OD showed marked improvement. IgG4 had fallen to 0.91g/L. Clinical, radiological, and serological response to steroid strengthened diagnostic confidence. Maintenance rituximab treatment initiated.

6 Discussion

- The two main systems of IgG4-RD classification and diagnostic criteria are centred around histology and non-cardiac presentations, so this case of isolated coronary involvement does not reach their thresholds.[4]
- Confirmatory biopsy not possible due to procedural risk, but the diagnosis appears secure based on the clinical, radiological, and serological profile.
- Review of the patient's past medical history reveals conditions that could be linked to IgG4-RD, but PET-CT has demonstrated no other ongoing inflammation. The relevance of these findings is currently unknown.
- This case represents a rare but important presentation of IgG4-related disease.

7 Learning Points

- IgG4-RD can present with isolated coronary involvement.
- The coronaries are rarely (1-3%) involved.
- Typical findings are aneurysms and/or periarteritis.
- The criteria for IgG4-RD do not cover all instances of the condition.
- Rheumatologists and cardiologists should be aware of this pattern of disease.
- Future diagnostic or classification criteria should take into account this possibility.

References, Results & Feedback

HIV and a sore ankle:

A rare case of calcium pyrophosphate deposition in a young patient living with HIV

Dr Sophie Langdon, Dr Ed Wheatley, Dr Clare Chown, Dr Jasper Mogg

INTRODUCTION

Atraumatic arthritis is frequently seen in the UTC. Some cases can be life threatening and significant morbidity can result. The presence of an HIV diagnosis leads to additional considerations in this patient group, which are illustrated here. This case also highlights a rare cause of joint pain in younger people and suggests a possible unrecognised association with HIV or its treatment.

CLINICAL CASE

A 36-year-old male presented to the urgent treatment centre.

Chief complaint: Nine-day history of pain and swelling of the left ankle.

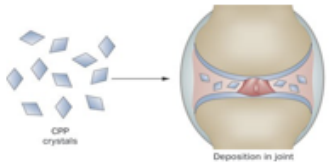
History of present illness: He had been self-medicating with ibuprofen and paracetamol, but his symptoms persisted.

- No similar previous episodes
- No preceding illness.
- No trauma
- No new or high-risk sexual partners
- Nil other symptoms

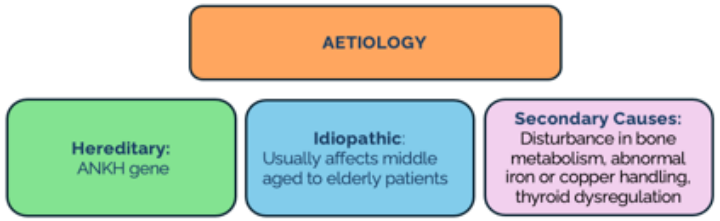
Past medical Hx: HIV infection, stably undetectable.
Social Hx: non-smoker, occasional alcohol use, no substance use. BMI 30.
Medication: Stribild OD (elvitegravir, tenofovir-DF & emtricitabine).
Family Hx: nil

WORKUP/ MANAGEMENT

Examination: ankle effusion
Investigation: microscopy revealed calcium pyrophosphate crystals.
Management: prednisolone 20mg OD 1/52



DISCUSSION



Studies have shown a link between HIV, or ART, and secondary causes of CPPD. It could be expected, given this overlap with the risk factors for CPPD, that increased incidence would be seen in HIV patients, but this is not a recognised association at present. However HIV patients are at risk of developing gout, which is felt likely to be related to ART, in particular ritonavir. Furthermore, 30-40% of HIV patients commonly experience arthralgia,

CONCLUSION

Given that crystal arthritis diagnoses are often made without the gold standard test of crystal microscopy, it begs the question as to whether CPPD being underdiagnosed and could be responsible for more morbidity in this patient population than is recognised.

LEARNING POINTS

- Young people presenting with CPPD should be investigated for secondary causes.
- HIV or its treatment may be associated with secondary CPPD
- Despite its rarity, CPPD should be considered as a cause of arthritis in those younger than the typical demographic.
- Over the counter medication such as ibuprofen can interact with ART
- People with HIV need not be considered as immunocompromised if they are known to have an undetectable viral load.

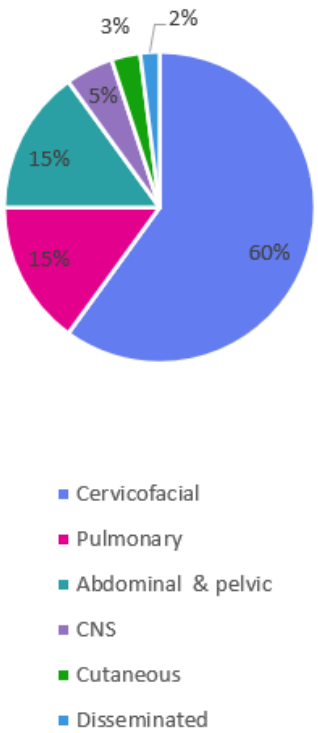
References: . Hammoudeh M et al. 1998. Pseudogout in a young patient. *Clin Rheumatol* 17:242–5. doi:10.1007/BF01451057. 2. Kleiber Balderrama C et al. 2017. CPPD and comorbidities in US veterans. *Arthritis Care Res* 69:1400–6. doi:10.1002/acr.23160
3. Dala-Ali BM et al. 2010. Pseudogout hip pain in HIV. *Case Rep Med* 2010:842814. doi:10.1155/2010/842814 4. Rho YH et al. 2012. Risk factors for pseudogout. *Rheumatology* 51:2070–4. doi:10.1093/rheumatology/kes204

Pulmonary Actinomycosis, The Great Masquerade: Two Distinct Presentations

Author: Dr Mohammed Akif Bin Halim[1], Dr Nilakanta Sumanam[2], Dr Poe Phyu[3], Dr Hein Zaw[4], Dr Vadsala Baskaran[5]

NHS
Nottingham University Hospitals
NHS Trust

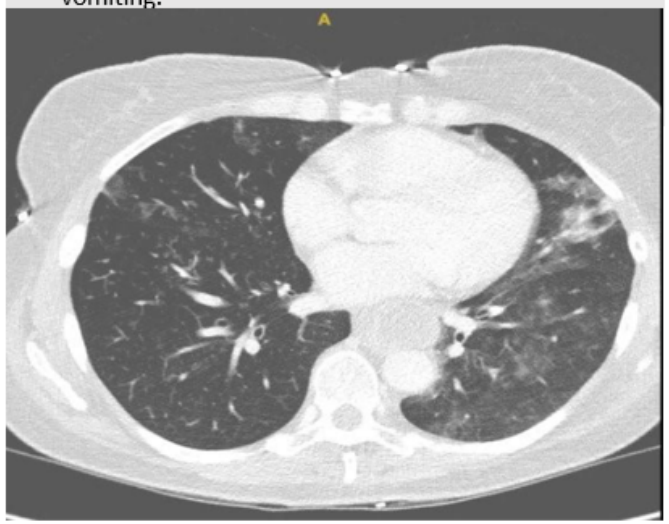
Background:
Actinomycosis is a rare, chronic bacterial infection known to be associated with suppurative swellings and formation of sinus tracts. **Only 15% is pulmonary but has the highest chance of becoming disseminated. Commonly mistaken for malignancy or M. TB**



Case 1

A 52-year-old Caucasian woman with a history of gastric band slippage and esophageal dysmotility presented with.

- **Symptoms**: 6months history of nocturnal cough, weight loss (3 kg), fever, abdominal pain, nausea, and vomiting.



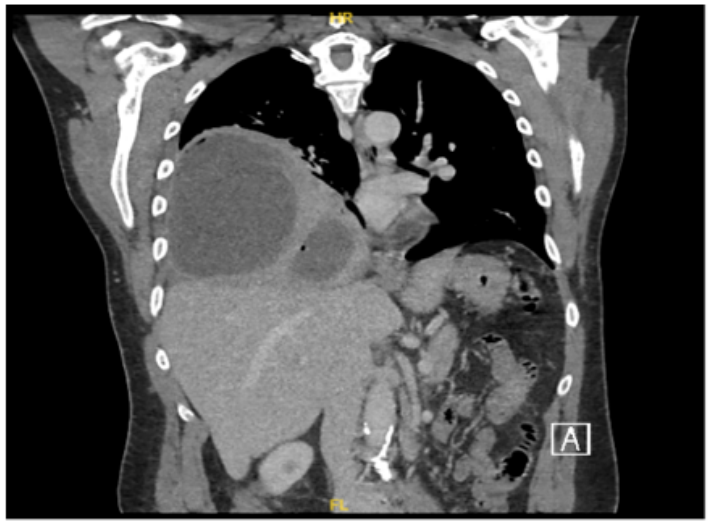
CT thorax: *showing a dilated esophagus and bilateral ground-glass opacities with tree-in-bud appearance.*

- **Lab results**: Mildly elevated CRP and normocytic anemia.
- **Microbiology**: Bronchoalveolar lavage isolated *Actinomyces graevenitzii.*
- **Treatment**: 8 weeks of Amoxicillin and Metronidazole for the first 2 weeks.
- **Follow-up** imaging showed resolution of the infection but persistent esophageal dilation; surgical removal of the gastric band was planned to lessen aspiration risk.

Case 2

A 53-year-old man with hyperlipidaemia, cannabis use, impacted molar and lead poisoning presented with.

- **Symptoms**: 3 weeks of fever, night sweats, cough, and significant weight loss (13 kg). No response to a course of Doxycycline.



CT thorax: *showing a right lower zone effusion, encysted empyema, and lung abscess.*

- **Lab results**: marked leukocytosis (WBC 34 x10^9/L) and elevated CRP (393 mg/L).
- **Microbiology**: Pleural fluid culture- *Actinomyces Schaalii, Fusobacterium nucleatum.*
- **Treatment**: 4 weeks of intravenous Co-amoxiclav + 6 weeks of oral Metronidazole, then switched to Doxycycline due to a rash, completing 6 months of therapy.
- **Follow-up** imaging confirmed resolution. Dental assessment identified an infected impacted molar requiring removal.

Risk factors: Poor dentation, Gastric outlet Obstruction, Malnutrition, Chronic lung disease, immunosuppression, Vomiting, Aspiration

Conclusion: These two cases highlight a high index of suspicion is required, especially in patients with the relevant risk factors.
Case 1 demonstrated an insidious onset of presentation, whilst **Case 2** demonstrated an acute presentation with pleural empyema, a complication of pulmonary actinomycosis.
Early diagnosis + microbiological confirmation and appropriate prolonged duration of antibiotic therapy leads to successful outcomes.

References:

- Alsamawi M, Al-Mashdali AF, Eldeeb Y. Pulmonary actinomycosis as a cause of chronic productive cough in a heavy smoker male with poor dental hygiene: A case report. BMJ Case Rep. 2022;2022: e256320. doi:10.1136/bcr-2023-256320.
- Sims D, Kerry A, Billingham K Actinomycosis mimicking metastatic lung malignancy *Thorax* 2024;**79:**694-695.
- Allan L, Said H, Shanks A Pulmonary actinomycosis presenting with empyema *BMJ Case Reports CP* 2023;**16:** e256320.

An Unusual Manifestation of a Common Disease

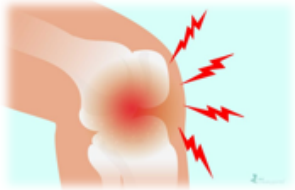
A 27-Year-Old Man with Tophaceous Gout

Dr Clare Chown, Dr Sophie Langdon, Dr Ed Wheatley, Dr Jasper Mogg

Clinical Case

Presentation

27-year-old with a large, tender effusion to left knee



Background

Left tibial plateau fracture 13 years prior

Initial Investigations/Management

- Apyrexial with normal observations
- Unremarkable inflammatory markers
- X-ray: large effusion, no evidence of fracture
- Managed as a soft tissue injury with orthopaedic follow-up

Orthopaedic Clinic

- MRI: well-demarcated bone erosion to lateral margin of patella
- 22 x 15 x 12mm mass of indeterminate soft tissue material
- Referred to the sarcoma MDT

Differentials

- Giant cell tumour of the patella
- Pigmented villonodular synovitis
- Gout
- Synovial chondromatosis

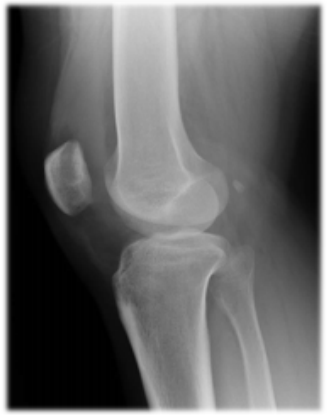
Further Investigations

- Core biopsy: variably-sized deposits of pale, acellular, crystalline material surrounded by fibrous tissue consistent with tophus
- Serum urate: 595µmol/L

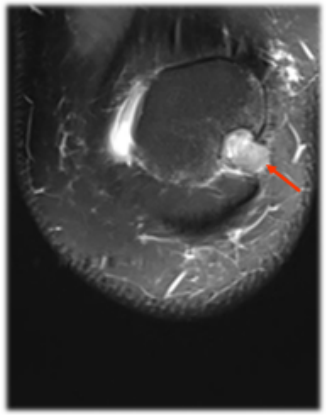
Diagnosis

Tophaceous gout, commenced on urate lowering therapy

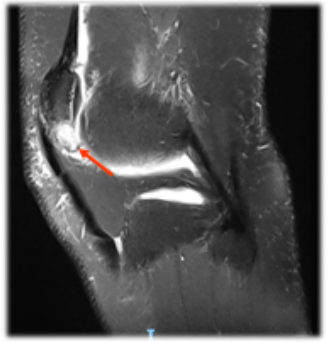
Imaging



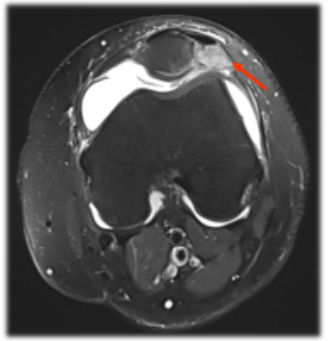
Plain lateral XR L knee – large effusion, small calcified body, no other abnormalities



MRI L knee T2 FS coronal – well-demarcated lateral patellar lesion



MRI L knee T2 FS sagittal – well-demarcated lateral patellar lesion



MRI L knee T2 FS transverse – well-demarcated lateral patellar lesion

Discussion

- Gout is rare under the age of 30 with an estimated prevalence of 0.4% [Li 2019]
- Gouty tophi typically emerge after at least 10 years of uncontrolled gout [Rana 2021]
- Tophi as a presenting complaint of gout is unusual [Salavastru 2020]
- The patella is a rare location for tophi [Clark 2016]

Learning Points

1) **Think gout in young patients**

   Gout should be actively considered in the differential diagnosis of atraumatic, or mildly traumatic, joint pain even in young adults

2) **Consider cardiovascular risk**

   Early-onset gout is associated with increased cardiovascular risk and effective treatment may reduce this risk

3) **Tophi can be the first presentation of gout**

   These can affect the patella, and can go unnoticed on plain imaging

4) **Tophi are not always easily identified**

   Radiologically, gouty tophi can be indistinguishable from several inflammatory and neoplastic Conditions, so biopsy may be needed

5) **MDT is important in diagnostic uncertainty**

   Gout does not necessarily follow a straightforward diagnostic pathway.

References & Feedback

Takotsubo Syndrome Triggered by Atypical Pneumonia: A Case of Legionella Infection

Authors Dr I M Mohamud, Dr Hasnain Kanchwalla

Affiliations: Craigavon Area Hospital – HSCNI

Background

Takotsubo cardiomyopathy (TTC), also termed stress-induced cardiomyopathy, is a transient form of left ventricular systolic dysfunction occurring without obstructive coronary artery disease[3]. It is often misdiagnosed as acute coronary syndrome (ACS) due to overlapping features such as chest pain, ST-segment elevation, and elevated troponin levels. While emotional and physical stressors are common triggers, infections are increasingly recognized as precipitants[5,6]. Legionella pneumophila, a cause of severe community-acquired pneumonia, is rarely linked with TTC. Its cardiac complications usually include myocarditis and arrhythmias[4], with TTC only occasionally reported[1,2]. The overlap of pneumonia, sepsis, and myocardial dysfunction poses a diagnostic challenge.

Case presentation

A 68-year-old woman was admitted after an unwitnessed fall with prolonged immobility. She was confused, febrile, and had elevated inflammatory markers, acute kidney injury, and rhabdomyolysis. Chest radiography showed left basal consolidation, and intravenous antibiotics were started. A distal radius fracture was also identified. Despite initial improvement, she developed worsening dyspnoea requiring increased oxygen therapy. Repeat chest X-ray revealed progression of consolidation. ECG showed sinus rhythm with STsegment elevation in V1–V2, and serum troponin was elevated.

Investigation and management

Investigations:

Echocardiography demonstrated impaired systolic function with apical akinesis and an ejection fraction of ~40%. The left ventricular apex appeared globular, consistent with TTC. Mild aortic stenosis and trace valvular regurgitation were noted, with normal right ventricular function. Coronary angiography showed minor coronary artery disease, a patent circumflex stent, and no evidence of thrombus or plaque rupture, excluding ACS. Respiratory work-up confirmed Legionella pneumophila infection by urinary antigen testing.

Management and Outcome:

Antibiotics were escalated to aztreonam and metronidazole, later rationalized to oral levofloxacin. Supportive management for TTC was continued. Renal function and infection markers improved, oxygen was weaned, and the patient was discharged with further OP Cardiology follow-up and Rehabilitation

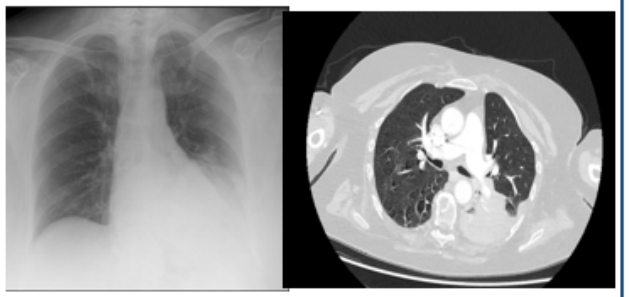


Figure 1: X-ray and CT scan results showing left lower lobe consolidation consistention with legionella pneumonia

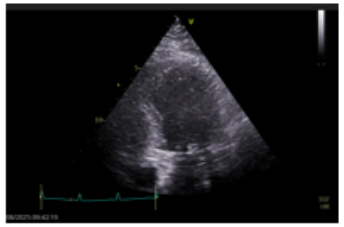


Figure 2: ECHO showing apical ballooning and hypokinesis

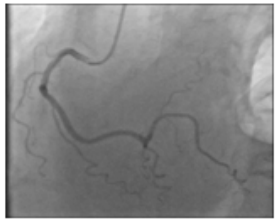


Figure 3: Angiogram showing patent vessels

Discussion

TTC accounts for 1–2% of patients presenting with suspected ACS[8]. It predominantly affects post-menopausal women and may be triggered by severe infections, particularly pneumonia[5]. Although Legionella pneumonia is usually associated with myocarditis or arrhythmias[4], rare cases of TTC have been documented[1,2]. Diagnosis relies on identifying the characteristic echocardiographic findings, reduced ejection fraction, unobstructed coronary arteries, and an identifiable stressor. This case highlights the need to consider atypical pathogens in severe or non-resolving pneumonia. Early detection of Legionella enables targeted therapy with fluoroquinolones such as levofloxacin, improving outcomes. Multidisciplinary collaboration is essential for optimal recovery

Conclusions

Conclusion:

We present a rare case of TTC complicating Legionella pneumophila pneumonia in an elderly woman. Clinicians should maintain suspicion for stress-induced cardiomyopathy in septic patients with ECG changes and elevated troponins, while also considering atypical pathogens in pneumonia. Prompt diagnosis, targeted antibiotics, and appropriate cardiac care facilitated full recovery and added to the growing evidence linking infection with Takotsubo syndrome.

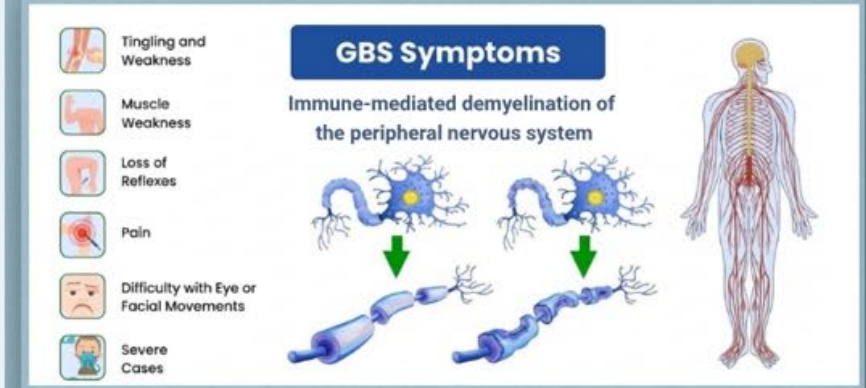
References

- Elikowski W, Mašek-Elikowska M, et al. Severe transient LV dysfunction in Legionella pneumonia. Pol Merkur Lekarski. 2019;47(277):19–24.
- Ahmed N, et al. My lung broke my heart! TTC due to pneumonia. F1000Research. 2018.
- Li P, Li C, Mishra AK, et al. TTC and respiratory diseases. Eur Heart J Open. 2022;2(2):oeac009.
- Gowani SA, Kumar A, Arora S, Lahiri B. Legionella pneumonia with myocarditis. Conn Med. 2013;77(6):331–4.
- Zalewska-Adamiec M, et al. TTC: early complications and 2-year mortality. Neth Heart J. 2016;24(9):511–9.
- Lyon AR, et al. Current state of knowledge on TTC: ESC HFA Position Statement. Eur J Heart Fail. 2016;18(1):8–27.

When spasticity Conceals Paralysis: A case of vaccine-Associated Guillain-Barré syndrome in HSP

Introduction

Guillain-barré syndrome is an acute immune-mediated polyneuropathy. It often follows infection as Campylobacter, CMV, EBV, or Zika. Rarely, it occurs following vaccination. Hereditary spastic paraplegia is a neurogenerative disorder causing progressive spasticity and weakness. The coexistence of HSP and GBS is exceptionally rare, often leading to diagnostic delay.



Case Summary

A 71-year-old man with X-linked HSP developed progressive weakness, stiffness, and paraesthesia two days after receving Moderna (JN.1 Spikevax) and influenza vaccines. He experienced transient facial droop and slurred speech.
Examination: initial 3/5 global weakness, diminished sensation.
Progression: UL 1/5-2/5, LL 1/5, areflexia, impaired proprioception.
MRI: normal; CSF: albuminocytologic dissociation; antiganglioside antibodies: negative. Despite IVIG therapy, he deteriorated and died from respiratory failure.

Aim

To highlight the diagnostic challenge of differentiating GBS from baseline HSP progression following COVID -19 vaccination.

Key Findings, Results

- Diagnostic delay due to overlap with chronic HSP weakness.
- Vaccine-associated GBS may present atypically with more cranial nerve involvement and lower antibody positivity.
- Negative antiganglioside antibodies do not exclude GBS.
- Vigilance needed in neurological comorbidities.

Recommendations

- Maintain high suspicion for GBS in neurological patients.
- Early neurology input and CSF analysis are vital.
- Recognise atypical post-vaccine presentations and treat promptly with IVIG.
- Encourage reporting of post-vaccine neurological events.

Conclusion

GBS can be masked by HSP. GBS may occur following COVID-19 vaccination, though causality is uncertain. Prompt recognition and treatment are crucial.

Thel Su Hlaing, Mary Ejiro, Khant Nyi Zaya,
Consultant Supervisor Adel Benyamin
Nevill Hall Hospital, Aneurin Bevan University Health Board, Wales, UK

References

Carbamazepine-Induced Agranulocytosis in a Trigeminal Neuralgia Patient

1. Nusrat Fatima 2. Rabindra Katwal

BACKGROUND

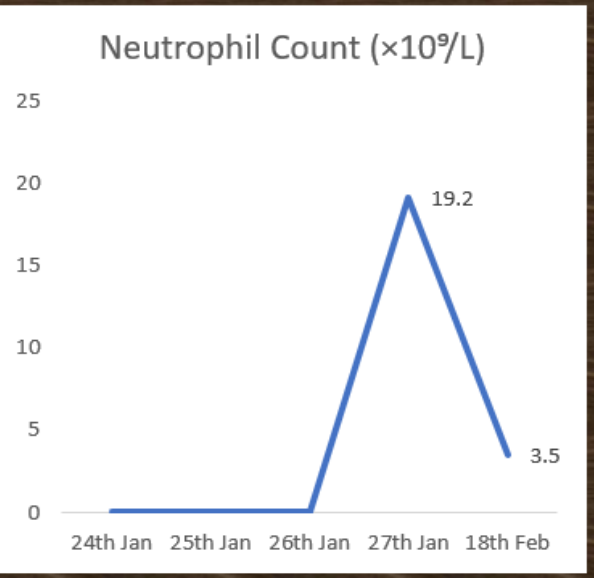
Carbamazepine is commonly used for the treatment of trigeminal neuralgia. It is associated with rare but potentially fatal adverse effects, including agranulocytosis and cutaneous eruptions.
This case report aims to highlight the **clinical features**, **early diagnosis**, and **management** of carbamazepine-induced agranulocytosis and cutaneous drug eruption.

CASE PRESENTATION

A 69-year-old male with a history of trigeminal neuralgia was prescribed **carbamazepine** on **December 24, 2024**, for symptom management.
Initially, he experienced **generalized malaise** by *January 9, 2025*, followed by **fever** on *January 13* and the onset of a **morbilliform rash** on *January 24*. The rash was accompanied by recurrent fevers reaching **39°C**, **desquamation**, **progression of erythema**, **oral soreness**, and **hand edema**.

- Blanching erythematous rash covering ~80% of the body
- No mucosal involvement or purpura
- Except FBC all other investigations were unremarkable

Day	Date	Clinical Event	Total WBC ($\times 10^9$/L)	Neutrophil Count $\times 10^9$/L	Remarks / Management
Day 1	Dec 24, 2024	Started Carbamazepine	normal	normal	Normal baseline counts
Day 31	Jan 24, 2025	Rash appears, high fever (39° C)	1.7	0.0	Severe neutropenia. Carbamezapine discontinued
Day 32	Jan 25, 2025	Further workup started	5.2	0.0	Clobetasol Mometasone furoate Cetraben lotion Fexofenadine
Day 33	Jan 26, 2025	started GM-CSF	3.4	0.0	Counts started improving
Day 34	Jan 27, 2025	Clinical improvement, rash resolving	26.3	19.2	Significant recovery
Day 35	Jan 28, 2025	Discharged	6.5	3.5	Neutrophil count normalized



DIFFERENTIAL DIAGONOSIS

- Carbamazepine-induced agranulocytosis
- Carbamazepine-induced cutaneous drug eruption
- The clinical presentation, along with hematologic findings, supported a diagnosis of carbamazepine-induced agranulocytosis with cutaneous drug eruption.

MANAGEMENT

Carbamazepine was immediately discontinued.
Treatment included:
Granulocyte-monocyte colony-stimulating factor (GM-CSF)
Clobetasol
Mometasone furoate
Cetraben lotion
Fexofenadine
The patient showed marked improvement and was discharged on the **5th day of admission**.

CONCLUSION

This case highlights the **importance of early recognition** and **immediate discontinuation** of carbamazepine in the event of hematologic and dermatologic adverse reactions. **Timely intervention** and **supportive care**, including GM-CSF and topical corticosteroids, can prevent further complications and **reduce morbidity** in such cases.

REFERENCES

1. Gupta, D., Bhardwaj, A., Prakash, R., et al.. Pancytopenia in Carbamazepine Therapy: A Rare and Serious Entity with Simple Prevention. *International Journal of Applied Basic Medical Research*.
2. Ran, D., Wang, N., Bao, F., et al.. Carbamazepine-induced follicular mucinosis-like drug eruption. *Clinical and Experimental Dermatology*.
3. Wu, S., Huang, L., Chen, J., et al.. Non-chemotherapy drugs inducing agranulocytosis: a disproportionality analysis based on the FAERS database. *Computational and Structural Biotechnology Journal*.

A Rare Case of Massive Fluid Overload: Diagnostic and Therapeutic Challenges of Pseudomyxoma Peritonei

Aye Thandar Aung[1], Aye Thuzar Kyaw[1], Myat Lin Aung[2], Kyaw Tint[1]

1.University Hospitals Plymouth NHS Trust 2. Torbay and South Devon NHS Foundation Trust

INTRODUCTION

Fluid overload is a common presentation to acute medical take. It has a wide range of differential diagnoses. We would like to present an interesting case of fluid overload which needed complex investigations for definitive diagnosis and ongoing management.

CASE REPORT

A 63-year-old male with a history of diabetes, no significant smoking or alcohol use, presented with a six-week history of severe anorexia, weight loss, abdominal discomfort, diarrhoea, anemia, and dyspnoea after a trip to Gambia.

Clinical examination revealed bilateral pleural effusions, massive ascites, and bilateral leg edema, suggesting significant fluid overload.

Initial investigations, including a comprehensive infectious disease screening, liver and renal function tests, albumin level, and echocardiogram, were unremarkable. CXR showed bilateral pleural effusion. Despite empiric treatment with diuretics, there was no clinical improvement.

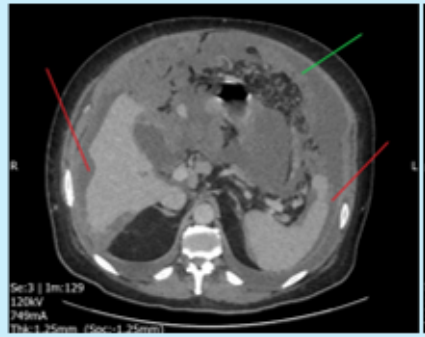


Fig.1 showing **scalloping of liver and splenic edges** (red lines) and **omental caking** (green line)

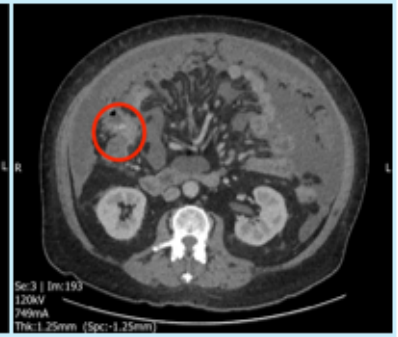


Fig.2 showing distended abnormal appendix

INVESTIGATIONS

A CT scan revealed bilateral pleural effusions, a **large omental cake** (green arrow), and extensive ascites with **scalloping of the liver and spleen** (red arrows), raising concern for pseudomyxoma peritonei (PMP). (Fig.1) CT report noted a distended, abnormal appendix (red circle), suggesting it was the likely primary tumor. (Fig.2) Colonoscopy showed diverticulosis with a normal appendiceal orifice. Tumour markers (CEA, CA19-9, CA125) were markedly elevated. Fluid cytology from both ascites and pleural effusion did not show evidence of malignancy. Ultrasound-guided omental core biopsy showed cystically dilated mucinous glands lined by rather bland epithelium within a pool of stromal mucin.

He was discussed in local Colorectal MDT and was referred to the Peritoneal Malignancy Institute. As the patient developed worsening symptoms, a 10-liter ascitic drain and a 2-liter pleural aspiration were performed. A repeat CT scan revealed moderate left pleural effusion and small ascites. However the patient experienced recurrent massive ascites, a tunnelled ascitic drain was placed under ultrasound guidance.

DISCUSSION

Pseudomyxoma peritonei (PMP) is a rare clinical entity characterized by diffuse intra-abdominal gelatinous ascites with mucinous implants on peritoneal surfaces.

Unlike typical cases that present with more straightforward abdominal symptoms, this case demonstrated an unusual picture - with extensive fluid overload.

Most reports in the literature do not describe such extreme fluid overload, underscoring the atypical nature of this presentation.

Early tissue biopsy proved more reliable than cytology for diagnosis.

Standard ascitic drain (14-gauge Rocket, or 18-gauge Bonanno) could be ineffective due to the mucinous fluid. More definitive drainage solutions included an 8-French (12.3-gauge, pale blue) pigtail catheter for acute drainage and a permanent tunnelled 15.5 French (<10 gauge) drain for long-term management.

The case emphasizes the importance of considering rare diagnoses like PMP in patients with unexplained fluid overload, weight loss, and anemia, when common etiologies have been ruled out.

REFERENCES

1) Al-Azzawi A, Zayoud H, Al-Asadi Z, et al. Aggressive Pseudomyxoma Peritonei: A Case Report with an Unusual Clinical Presentation. Int J Surg Case Rep. 2013;4(12):1128-1131. doi:10.1016/j.ijscr.2013.09.020.

2) Sioshansi S, Amini A. Pseudomyxoma Peritonei. In: StatPearls [Internet]. StatPearls Publishing; 2023. Accessed September 1, 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK541116/.

3) Al-Sabbagh M, Al-Sabouni T, Al-Nasser T, et al. Incidental diagnosis of pseudomyxoma peritonei by laparoscopy: a rare case from Syria. J Surg Case Rep. 2022;2022(12):rjac558. doi:10.1093/jscr/rjac558.

4) Jabbour P, El Khoury M, Maatouk M, et al. Pseudomyxoma Peritonei: A Case Report of a 62-Year-Old Female With Unexplained Abdominal Enlargement and Appendiceal Origin. Cureus. 2022;14(3):e23298. doi:10.7759/cureus.23298.

5) El Hadj T, Taha M, Mouad H, et al. Pseudomyxoma Peritonei: A Case Report Diagnosed in a 47-Year-Old Woman with Chronic Pelvic Abdominal Pain and Appendicular Origin: Review of the Literature and Management. J Gastrointest Cancer. 2018;49(1):109-113.

This case will be presented as a PowerPoint presentation on 7 November 2025 at the South West Regional Acute Medicine Conference.

Pulmonary Vascular Malformation and Hyperthyroidism

Authors : Dr Greeshma Joseph, Dr Mohit Inani, Dr Thimmegowda L. Govindagowda, Dr Arpitha Jayaramegowda

Royal College of Physicians

The Shrewsbury and Telford Hospital NHS Trust

Background

Pulmonary arteriovenous malformations (PAVMs) are abnormal connections between pulmonary arteries and veins bypassing capillary network and therefore causing pathological intrapulmonary right to left shunt.

- Orthodeoxia was observed in the case, which led to suspicion of PAVM.
- PAVM was confirmed by CT Pulmonary Angiogram.
- Due to concurrent tachycardia, thyroid function tests were performed, confirming hyperthyroidism.

Objectives

- To consider PAVMs as a differential diagnosis in cases of orthodeoxia.
- To recommend embolization even in asymptomatic patients.
- To consider screening for HHT in cases of PAVM.

Presentation

A young female was noted to have low oxygen saturation during a routine examination in primary care. She appeared comfortable at rest. During observation, she was noted to be tachycardic. A loud second heart sound was audible on auscultation. No telangiectasia was observed. The patient's high intensity sports activity despite hypoxaemia made this case noteworthy.

Figure 1

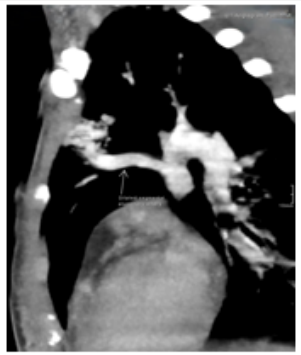


Figure 2

Investigations

- CT Pulmonary Angiography revealed vascular malformations in the anterior segment of the left upper lobe and posterior segment of the left lower lobe, which demonstrate dilated draining veins into the left pulmonary vein.
- Echocardiography revealed no abnormalities.
- TSH receptor antibody was elevated, with a value of 8.3 IU/L, confirming Grave's disease.

Arterial Blood Gas

	pH	pCO2 (kPa)	pO2 (kPa)	sO2 (%)
Initial	7.481	2.94	8.22	93.7
Lying	7.452	3.31	10.20	95.7
Standing	7.485	2.84	6.86	88.5

Table 1

Treatment

Hyperthyroidism may have increased the shunt , making its treatment essential. Carbimazole and Propranolol were initiated with Endocrine input. Input from specialists regarding embolization is currently awaited.

Thyroid Function Tests

	Initial value	Post treatment
Free T4 (pmol/L)	69.8	10.0
Free T3 (pmol/L)	33.4	-
TSH (mu/L)	<0.01	1.50

Table 2

References

- Santhirapala V *et al.* Orthodeoxia and postural tachycardia in pulmonary AVMs. *Thorax* 2014; 69:1046–7.
- hovlin CL *et al.* BTS Clinical Statement on pulmonary AVMs. *Thorax* 2017; 72:1154–63.
- Danyalian A, Sankari A, Hernandez F. Pulmonary arteriovenous malformation. *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.

Pacemaker Lead Endocarditis Secondary to Staphylococcus aureus Infection at a Flash Glucose Monitoring Site in Type 1 Diabetes: A Case Report

Linn Kyaw[1]; Al-Hayek Hamza[1]; Parsian Zahra[1]; Dalpathadu Sanjalee[1]
,Diabetes and Endocrine Department,[1]York General Hospital

Background

- Flash and continuous glucose monitoring (FGM/CGM) devices are Increasingly used to improve glycaemic control[1 2].
- Although generally safe, they may cause local or systemic infections[3], especially in poorly controlled diabetes.
- Such infections can rarely lead to severe complications , including device-related endocarditis.
- We present a case of *S. aureus* infection originating from an FGM site that progressed to pacemaker lead endocarditis.

Case Presentation

- A 45-year-old man with poorly controlled type 1 diabetes mellitus and multiple comorbidities who presented with fever, confusion, and pain at the site of his FGM sensor insertion.
  Clinical examination revealed signs of cellulitis with abscess formation (Figure 1).
- Cultures from both the wound and blood grew Staphylococcus aureus sensitive to flucloxacillin.
  Imaging identified a deep-seated shoulder infection, requiring repeated surgical washouts.
- Persistent bacteraemia raised suspicion for device-related infective endocarditis, which was confirmed by transoesophageal echocardiography, showing a vegetation on the pacemaker lead (Figure 2).
- The patient required pacemaker lead extraction and prolonged intravenous flucloxacillin therapy to achieve a full recovery.

Images

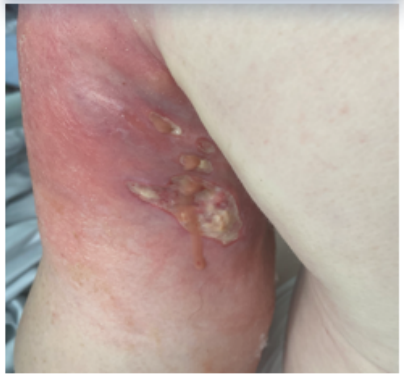


Figure 1. Examination on arrival showed erythema, swelling, and ulceration with discharge at the FGM site.

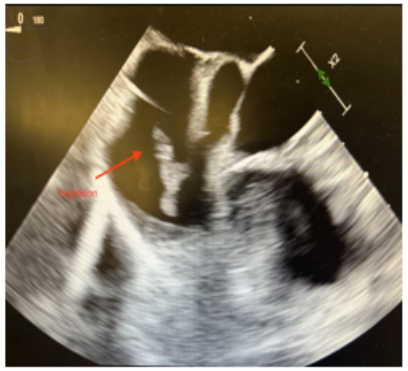


Figure 2. Transoesophageal echocardiogram demonstrating vegetations on pacing leads in the right atrium.

Discussion

- This case demonstrates how infections originating from CGM or FGM insertion sites can progress from local to severe systemic disease[1 2]
- Poor glycaemic control and comorbidities increase the risk to S. aureus infection and device-related endocarditis[3].
- Reported cases vary from local cellulitis to necrotising infection and sepsis[4 5].
- Early recognition, appropriate antibiotic therapy, and device removal with surgical intervention were essential for favourable outcomes.

Conclusion

- Infections related to glucose monitoring devices, though uncommon, can progress to serious and life-threatening complications.
  We should maintain a high suspicion for systemic infection in diabetic patients with implanted devices.
  Awareness of this rare but severe complication is essential to improve patient safety and outcomes.

References

1. Mian Z, Hermayer KL, Jenkins A. Am J Med Sci. 2019;358(5):332-339.
2. Asarani NAM et al. J Diabetes Sci Technol. 2019;14(2):328-337.
3. Attalla J, Maletkovic J. Proc UCLA Health. 2024;28(1).
4. Von Tokarski F et al. QJM. 2021;114(1):58-59.
5. Herrod SS et al. Clin Med. 2022;22(5):482-484.

A rare case of Carbimazole induced Lupus

Ibrahim Basar, Arooj Zulfiqar, Paulina Gronczewska, Hafiz Javed, Jayamalee Jayaweera, Haris Marath, Amna Zeeshan, Joohi Majeed

Introduction

Grave's Disease and Systemic Lupus Erythematosus (SLE) are autoimmune disorders that predominantly affect young females. While Grave's Disease has its manifestation in the thyroid, its systemic effects are well-recognized; SLE is a classic multisystem disease[1-4]. Drug-induced lupus is a lupus-like syndrome typically occurring months to years after exposure to certain medications. Classic culprits include hydralazine, procainamide, and isoniazid[1] . We present a case of a 45-year-old female who developed drug-induced lupus secondary to carbimazole.

Case presentation

A 45-year-old female presented to the hospital with new-onset rapid atrial fibrillation that proved resistant to standard rate control measures. She was also experiencing fever and exhibited classic symptoms of thyrotoxicosis, including a two-month history of palpitations, unintentional weight loss of 3 kg over one month, and a four-month history of a fine tremor.

Initial investigations revealed profoundly suppressed thyroid-stimulating hormone (TSH 0.02 mIU/L) and markedly elevated free thyroxine (fT4 69.6 pmol/L). Her Burch-Wartofsky score of 55 was highly suggestive of Thyroid Storm. She was subsequently started on intravenous metoprolol, oral propylthiouracil (PTU), Lugol's iodine and hydrocortisone. This treatment resulted in significant clinical improvement, allowing discharge with a diagnosis of Graves' disease and treatment consisting of carbimazole, a tapering course of prednisolone, and a proton pump inhibitor (PPI). Four weeks following discharge, she re-presented with flu-like symptoms and was diagnosed with Influenza A infection.

Despite appropriate antiviral treatment with oseltamivir and broad-spectrum antibiotics to cover for super-added bacterial infection, she continued to experience persistent fever and had an elevated C-reactive protein (CRP). This prompted an extensive workup for pyrexia of unknown origin (PUO). Extensive infection screens and radiological investigations yielded no definitive source. Rheumatological evaluation revealed positive antinuclear antibodies (ANA), anti-double-stranded DNA (dsDNA), and anti-Ro antibodies, leading to a diagnosis of drug-induced systemic lupus erythematosus (SLE)

This was supported by positive anti-histone antibodies, a characteristic serological marker of drug-induced lupus.

Management

Management involved immediate discontinuation of carbimazole (replaced with PTU) and initiation of prednisolone 20mg daily, which resulted in complete resolution of her fever and arthralgia. At follow-up in the rheumatology clinic, hydroxychloroquine was introduced as long-term immunomodulatory therapy for SLE, with good symptomatic control achieved.

Later rheumatology clinic review demonstrated significant proteinuria prompting review by the renal team, who organized a renal biopsy. The biopsy showed findings consistent with class 3 lupus nephritis. She was subsequently commenced on mycophenolate.

Discussion

Systemic lupus erythematosus (SLE) is a multi-system autoimmune disease predominantly affecting women aged 15–45. Common symptoms include fever, weight loss, fatigue, lymphadenopathy, oral/nasal ulcers, arthritis, serositis, and cytopaenias. Complications may involve lupus nephritis (presenting as nephrotic or nephritic syndrome) and rare cases of lupus cerebritis. Whilst commonly it is idiopathic in aetiology, it is crucial to remember that medications may lead to a presentation that mimics idiopathic lupus[1-3].

There are less than 10 case reports highlighting carbimazole as a potential trigger for drug-induced lupus, emphasizing the importance of considering autoimmune aetiologies in patients with persistent unexplained symptoms following medication initiation. Early recognition and appropriate management, including discontinuation of the offending drug and immunosuppressive therapy, can lead to favourable outcomes.

References

1.Haq I, Sosin MD, Wharton S, Gupta A. Carbimazole-induced lupus. BMJ Case Reports. 2013 Feb 7;2013:bcr2012007596–6.
2.Dyall-Smith DD. Drug-induced lupus erythematosus | DermNet [Internet]. dermnetnz.org. 2010. Available from: https://dermnetnz.org/topics/drug-induced-lupus-erythematosus
3.Solhjoo M, Bansal P, Goyal A, Chauhan K. Drug-Induced Lupus Erythematosus [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2020. Available from: https://www.ncbi.nlm.nih.gov/books/NBK441889/
4.Lane LC, Wood CL, Cheetham T. Graves' disease: moving forwards. Archives of Disease in Childhood. 2022 Jul 13;108(4):archdischild2022-323905.

McKittrick-Wheelock Syndrome Presenting With Severe Electrolyte Imbalance and Acute Kidney Injury

Authors: Dr Jun Kai Terry Tan*, Dr Richard Lloyd*

***Department of Critical Care, Ipswich Hospital**

NHS
East Suffolk and North Essex
NHS Foundation Trust

- 50M presented with acute-on-chronic diarrhoea, worsening lethargy, and poor oral intake
- Severe hyponatraemia (Na^+ 106 mmol/L), hypokalaemia (K^+ 2.2 mmol/L), hypochloraemia (Cl^- 71 mmol/L) and low ionised calcium (0.95 mmol/L). AKI with metabolic compensation.
- Serum osmolality 256 mOsm/kg, 9am cortisol normal
- Gut hormone panel negative, stool MCS negative
- Urinary Na^+ and Cl^- <20 mmol/L, trialled octreotide
- PMH: chronic diarrhoea secondary to pancreatic insufficiency.
- Third admission in 3 months for similar episodes.
- Previous CT: Rectosigmoid thickening [Figure 1] and flexi sig: 10 cm laterally spreading tumour [Figure 2]. Histology: tubulovillous adenoma with low-grade dysplasia.
- Repeat flexible sigmoidoscopy showed extensive circumferential carpet adenoma. A pelvic MRI Large mucinous rectal lesion; no extra-mural invasion or lymphadenopathy
- Underwent robotic ultra-low anterior resection with a loop ileostomy

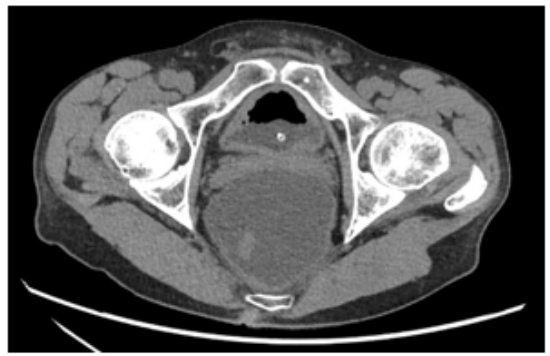


Figure 1

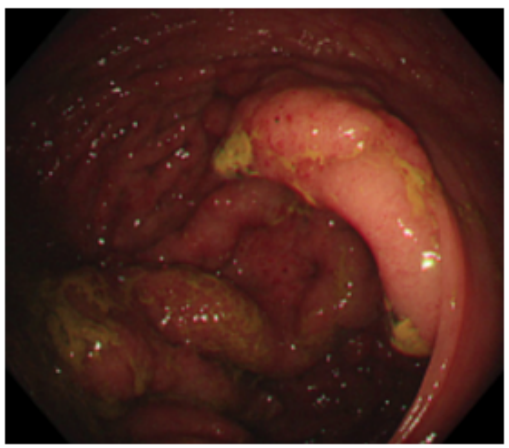


Figure 2

- **McKittrick-Wheelock Syndrome** is rare but causes severe chronic fluid and electrolyte loss. Diagnosis is often delayed due to non-specific symptoms resembling common gastrointestinal disorders.[1]
- **Pathophysiology**: Overexpression of COX-2 in adenomatous tissue → ↑ prostaglandin E2 production → Cl^- secretion & inhibition of Na^+ reabsorption → mucin production → secretory diarrhoea and electrolyte depletion.[2]
- **Management** focuses on aggressive fluid and electrolyte correction, followed by surgical resection of the adenoma. Surgical excision is curative and minimises risk of malignant transformation and symptom recurrence.

Take-Home Messages

- Consider MWS in patients with chronic diarrhoea, AKI, and severe electrolyte imbalance.
- Delayed diagnosis of MWS is common due to overlap with more common gastrointestinal conditions; timely imaging and endoscopy are essential for accurate diagnosis.
- Benign-appearing lesions can still cause profound systemic effects.

References

1. *de Sousa Miranda I, Ferreira JR, Rocha S, Monteiro M, Guillerme J, Domingos R. McKittrick-Wheelock Syndrome: A Neoplastic Cause of Electrolyte Imbalance. DOAJ (DOAJ: Directory of Open Access Journals). 2022 Mar 24;9(3):003231–1.*
2. *Einspahr JG, Krouse RS, Yochim JM, Danenberg PV, Danenberg KD, Bhattacharyya AK, et al. Association between Cyclooxygenase expression and colorectal adenoma characteristics. PubMed. 2003 Jul 15;63(14):3891–3.*

Rare Case of Bilateral Adrenal Haemorrhage Associated with HIV and Intravenous Stimulant Drug Use

Authors: Aya Eladl, Haris Khan, Salah Kouta
Department of Diabetes and Endocrinology, Northern Care Alliance NHS Foundation Trust Bury Care Organisation, Greater Manchester, UK

11 – 12 November
Med+ 2025

Background

Adrenal haemorrhage (AH) is a rare, life-threatening condition that can lead to acute adrenal insufficiency [1,2].

HIV infection and **stimulant drug use** have both been associated with adrenal dysfunction through mechanisms such as opportunistic infection, vascular injury, and stress-induced coagulopathy [3-7].

To our knowledge, this is the **first case** of non-traumatic AH in the context of HIV infection and illicit stimulant drug use.

Case Presentation

Demographics: 33-year-old, Caucasian man
Presentation: 5-day history of abdominal pain, fever, diarrhea, and vomiting.

Past medical history: HIV, illicit drug use [methamphetamine and cocaine], current smoker
Drug history: Truvada 200/245 mg once daily and Raltegravir 600 mg once daily

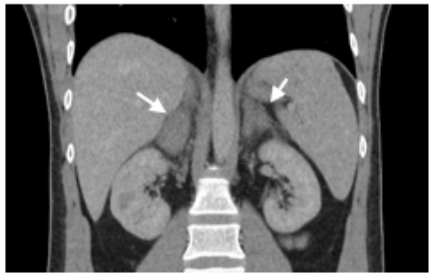
Full case images and reference list available via QR code

Management

- Imaging confirmed bilateral AH (Figure 1).
- Administered IV fluids, IV antibiotics, analgesia, and high-dose intravenous steroids (50 mg four times daily).
- Discharged on oral hydrocortisone 10 mg, 5 mg, and 5 mg.

Follow up

- Interval CT scan after 14 months showed complete resolution of the adrenal haemorrhages (Figure 2).
- Hydrocortisone day curve showed partial recovery of adrenal function.



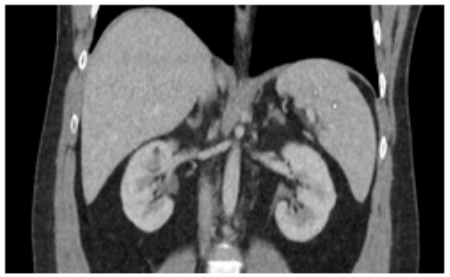


Figure 1 & 2. Contrast-enhanced CT of the abdomen and pelvis showing bilateral adrenal haemorrhage on admission (top) and complete resolution at 14 months (bottom).

Test	Result/Interpretation
Cortisol (9 AM)	95 nmol/L *(low)*
Synacthen Stimulation Test	95 → 150 → 201 *(inadequate)*
Aldosterone	Undetectable
Renin	0.1 pmol/hr *(low)*
Prolactin	187 mU/L *(normal)*
Urine & plasma metanephrines	Unremarkable
HIV-1 viral load	Undetectable
CD4 count	699 cells/µL *(normal)*

Table 1. Summary of Hormonal and Biochemical Investigations.

Discussion

Pathophysiology: In sepsis, bacterial endotoxins trigger pro-inflammatory cytokines causing adrenal venous injury and parenchymal haemorrhage[1,2,8].
Serial imaging is recommended to monitor resolution of the haematoma [8,9] .
Prognosis depends on early recognition and prompt treatment [1].

Key points:

Consider AH in **HIV-positive** or **IV stimulant drug–using** patients with **clinical features of adrenal insufficiency**.

Early recognition and **prompt steroid replacement** are crucial to prevent adrenal crisis and mortality.

HIV infection and **stimulant drug use** may increase susceptibility to **adrenal destruction and haemorrhage**.

Further research is needed on the **direct effects of HIV, antiretroviral therapy, and methamphetamine** on the adrenal gland.

Isolated ACTH deficiency due to Presumed Postpartum Lymphocytic Hypophysitis and Consequent Empty sella Syndrome, presenting with Severe Symptomatic Adrenal insufficiency- A Case Report

Dinath Perera[1], Naina Skariah[1], Maria Maridaki[1], Sath Nag[1]
[1]South Tees Hospitals NHS Foundation Trust, Middlesbrough, United Kingdom

NHS South Tees Hospitals NHS Foundation Trust

INTRODUCTION

- Isolated adrenocorticotropic hormone (ACTH) deficiency is a rare cause of adrenal insufficiency, characterized by low cortisol levels in the presence of suppressed ACTH, while other anterior pituitary hormones remain unaffected.
- Diagnosis is often delayed due to its nonspecific and insidious presentation.[1]

CASE DETAILS

- A 36-year-old woman with no significant past medical history and an uncomplicated pregnancy presented one year postpartum with severe fatigue and ~20 kg unintentional weight loss starting 3–4 months after delivery.
- Breastfed for 6 months- Stopped due to breast fibroadenomas
- Denies headaches, visual symptoms, or any major complications during or after delivery
- General appearance: markedly thin and pale.
- No buccal or gingival hyperpigmentation, vitiligo, or goiter
- Blood pressure: 118/82 mmHg, no postural drop
- Capillary blood glucose: 6.0 mmol/L

INVESTIGATIONS

Primary Care Workup:

- Subclinical hypothyroidism:
  - TSH: 12.58 mIU/L *(Ref: 0.27–4.2)*
  - FT4: 10.4 pmol/L *(Ref: 10.0–21.0)*
- Started on levothyroxine therapy

Referral to Rapid Diagnostic Centre (RDC):

- Due to unexplained weight loss
- CT TAP (Thorax, Abdomen, Pelvis): No evidence of malignancy

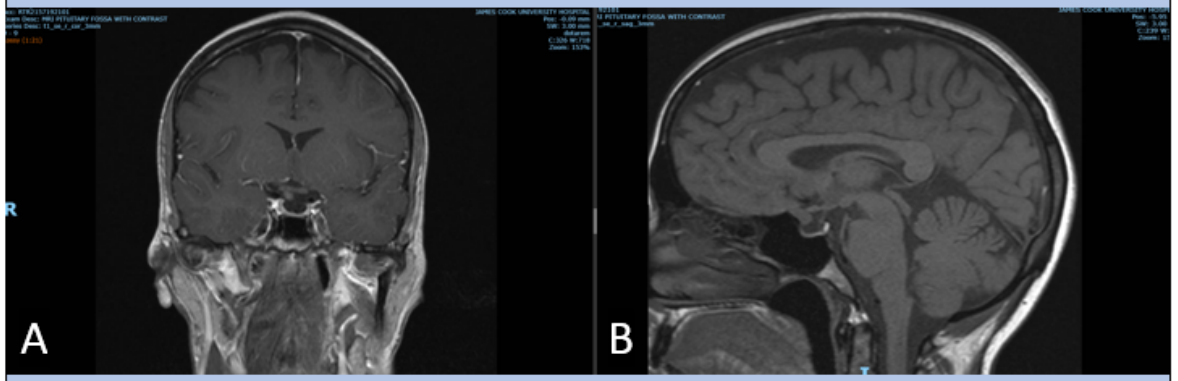
Further Evaluation by GP:

- Noted persistent lethargy and low weight
- Random serum cortisol:
  - Result: Undetectable cortisol <14 nmol/L

SDEC assessment

- Sodium 138 mmol/L(135-145); Potassium 4.2 mmol/L(3.5-4.5)
- Routine biochemistry otherwise normal
- **9 am cortisol <14 nmol/L**
- ACTH stimulation test : Fail (baseline cortisol <14 nmol/L;60 - minute increment 16 nmol/L)
- Anterior pituitary hormone profile
  - FSH 9 u/L, LH 8.3 u/L
  - Oestradiol 202 pmol/l(regular menstrual cycle post partum)
  - Prolactin 555 mU/L (59-619)
  - TSH 6 mU/L; FT4 15.2 pmol/L(10-21); On Levothyroxine
  - **Serum ACTH <2 ng/L (7.0-63.0)**
- Aldosterone 84.0 pmol/L (<630)
- Renin :0.5 nmol/h (0.3-2.2)
- IGF-1 14.3 nmol/L (9-31)

MRI PITUITARY



A. Empty sella with marked pituitary gland thinning (<1 mm); no evidence of pituitary adenoma
B. Tuberculum sellae meningioma on the right side, measuring 5 × 9 × 7 mm, with close abutment of the right communicating segment of the internal carotid artery.

DISCUSSION

- Isolated ACTH deficiency, though rare, often presents with nonspecific symptoms that require a high index of suspicion for timely diagnosis.
- Early recognition and prompt glucocorticoid replacement are crucial to improving outcomes and preventing potentially life threatening adrenal crises.[1]
- This case emphasizes that while postpartum fatigue is frequently attributed to psychosocial stressors related to newborn care, clinicians should remain vigilant for organic causes when symptoms are persistent, severe, or accompanied by unexplained signs such as weight loss or hypotension.[2]

REFERENCES

1) Charmandari E, Nicolaides NC, Chrousos GP. Adrenal insufficiency. Lancet. 2014 Jun 21;383(9935):2152-67.
2) Baral B, Sharma M, Khadka R, Naveed O, Bekele A. Postpartum Hypophysitis: A Case Report and a Literature Review. Cureus. 2024 Apr;16(4):e59396.

WHEN MANIA MASKS LUPUS: A RARE NEUROPSYCHIATRIC MANIFESTATION OF SYSTEMIC LUPUS ERYTHREMATOSUS

Authors: Dr. Shibin Thamban, Dr. Smitha Muraleedharan, Dr. Sanjana Nair (Department of Internal Medicine, Aster Medcity Kochi, India)

INTRODUCTION

Neuropsychiatric systemic lupus erythematosus (NPSLE) is one of the most challenging manifestations of lupus, presenting with diverse neurological and psychiatric syndromes. Organic mania is an exceptionally rare presentation and is frequently misdiagnosed as a primary psychiatric disorder, often delaying appropriate immunomodulatory therapy

CASE

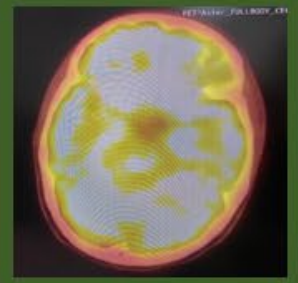
A 28-year-old female known case of psoriasis, psoriatic arthritis, hypothyroidism, PCOS and endometriosis presented with **acute confusion, agitation, psychosis, and tonic posturing of all limbs.** On examination, she had tachycardia, conscious with irrelevant talk and excess word output, tonic posturing of hands with diffuse wating of muscles of both hands.

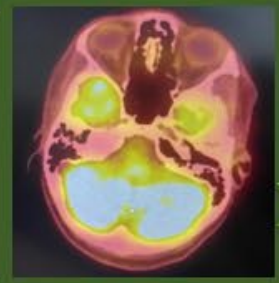
PAST MEDICAL HISTORY

Psoriatic arthritis- treated with sulfasalazine and low-dose prednisolone, later switched to methotrexate (10-15 mg/wk) and subsequently to Secukinumab (150 mg weekly. She had been on Ayurvedic medications for 6 m.

WORKUP

PS: microcytic hypochromic anaemia with neutrophilic leucocytosis; CRP,ESR- Negative
URE: no proteinuria, pus cells- nil
RFT, LFT- Normal, K+:3.1, Ca: 8.2, Phos: 3, TSH 6.0
Infective work up- PCT- 0.06, Blood , urine culture-sterile
→Infection was excluded





MRI brain with contrast: symmetric mild FLAIR hyperintensity in the basal ganglia, insular cortex, hippocampus and amygdala
CSF study: TC – 1;sugar normal 7; protein normal 33.2; KOH- no fungal elements; CSF TB panel: Negative and CBNAAT- Negative; Autoimmune encephalitis panel-negative **NCS:** axonal neuropathy
Autoimmune workup: ANA profile- RIB 1+ Ro 52 3+ SSA 3+ RNP/SM 3+ SM-ve ;
Anti-dsDNA 101.46 (borderline)
APLA work up: Negative
Complement: Normal
GAD65 antibody positive; NMDA/VGKC –Neg.
PET scan: Increased metabolic uptake in cerebellum/basal ganglia/medial temporal lobe . Diffuse muscle uptake. Moderate Pericardial effusion noted.

MANAGEMENT

She was initiated on IVIG (2 g/kg over 5 days; total 100 g) after an MDT and continued on steroids.

Based on the clinical, laboratory, and radiological findings, she was diagnosed as a possible evolving lupus with neurological involvement and Rituximab was initiated.

DISCUSSION

This case illustrates organic mania as a rare neuropsychiatric manifestation of evolving SLE, initially mimicking primary psychiatric illness. Diagnosis was challenging due to multiple autoimmune comorbidities, multisystem involvement, and inconclusive CSF findings. Positive autoimmune markers, PET-CT changes, and response to immunotherapy supported neuropsychiatric lupus with autoimmune encephalitis overlap. Multidisciplinary management and timely immunotherapy were crucial, emphasizing the need for high suspicion in young patients with acute psychiatric presentations and systemic features

CONCLUSION

Organic mania is an uncommon but important presentation of neuropsychiatric lupus. This case demonstrates the critical importance of considering SLE in the differential diagnosis of acute psychosis or mania, particularly in patients with autoimmune background and systemic involvement. Early MD management with immunotherapy can be life-saving.

Neuromyelitis Optica Spectrum Disorder presenting with Area Postrema Syndrome

Dr La Win Phyu Phyu
East Surrey Hospital, Surrey and Sussex Healthcare NHS Trust

Introduction

Neuromyelitis optica spectrum disorder (NMOSD) is an autoimmune astrocytopathy mediated by aquaporin-4 (AQP4) antibodies. The 2015 International Consensus Criteria recognise area postrema syndrome (persistent nausea and vomiting) as a core clinical feature.[1] Delayed recognition increases the risks of relapse, treatment resistance, and permanent disability.

Case summary

Presentation & Initial Assessment

- 28-year-old woman
- 3-week history: intractable vomiting, dizziness, unsteadiness
- Similar past episode treated as reflux disease

- **Initial investigations**
  - OGD, barium study, CT abdomen - **normal**
  - CT head -**normal**

- Despite fluid and electrolyte correction , unsteadiness and dizziness persisted, vomiting refractory to antiemetics

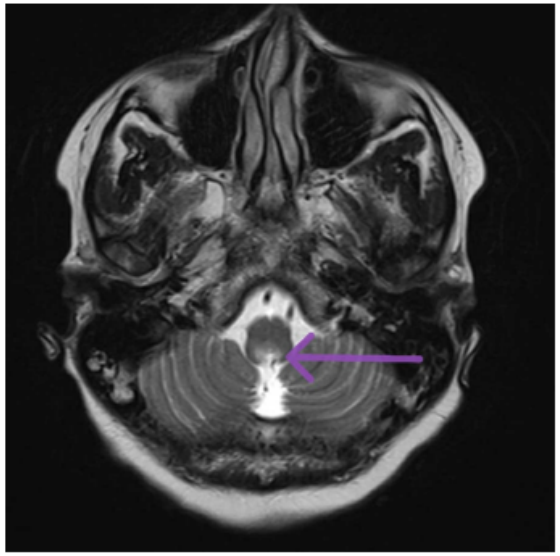
- **Progression of symptoms**: ascending paraesthesia, tongue numbness, dysarthria

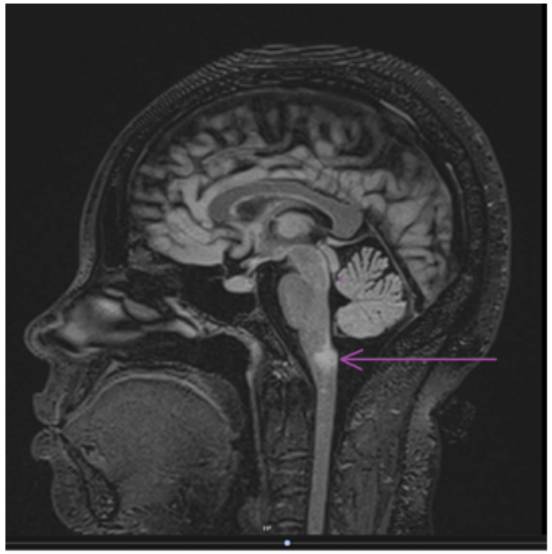
Further Assessment, Management & Outcome

- **MRI brain**: asymmetric T2/FLAIR hyperintensity in dorsal medulla (right area postrema)
- **CSF/serology**: AQP4 antibodies detected in CSF & serum
- **MRI spine** – normal

- **Treatment**
  - IV methylprednisolone x 5 days - limited response
  - (Persistent dizziness, oscillopsia, upbeat nystagmus)
  - Referred to tertiary NMOSD centre for **plasma exchange**

- **Outcome**: Clinical improvement, discharged with outpatient neurology follow-up





Discussion

NMOSD can mimic gastrointestinal disease, delaying diagnosis.2,3 Area postrema lesions explain intractable vomiting, with dorsal medullary spread causing oscillopsia, unsteadiness, and upbeat nystagmus.4 Myelitis-like symptoms may occur despite a normal spinal MRI. Steroid-refractory cases require rapid escalation to plasma exchange to prevent long-term disability.

Conclusion: Persistent vomiting with vestibular or ocular motor signs should raise suspicion for NMOSD with area postrema syndrome and prompt urgent brain MRI and antibody testing. Early recognition and timely escalation of treatment, including plasma exchange when steroid response is limited, are critical to prevent irreversible neurological damage. Reporting this case highlights that appropriate escalation can lead to recovery and safe discharge with neurological follow-up.

References
1. Wingerchuk DM, Banwell B, Bennett JL, Cabre P, Carroll W, Chitnis T, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology. 2015;85(2):177-89.
2. Apiwattanakul M, Popescu BF, Matiello M, Weinshenker BG, Lucchinetti CF, Lennon VA, et al. Intractable vomiting as the initial presentation of neuromyelitis optica. Ann Neurol. 2010;68(5):757-61.
3. Raj A, Valappil A. Area postrema syndrome: an unusual presentation of neuromyelitis optica spectrum disorder. Cureus.2023;15(4):e37925.
4. Oh SY, Kim JS, Dieterich M. Nystagmus and oscillopsia in medullary lesions. Neurology. 2011;76(11):918-24.

The Sepsis Impostor: Myocardial Failure Triggering Multiorgan Ischemia

University Hospitals Bristol and Weston NHS Foundation Trust

Author: Dr. Akbar Khan

Affiliations: University Hospitals Bristol and Weston NHS Foundation Trust (UHBW)

Dr. Abir Aijaz, Dr. Akbar Khan, Dr. Bhavna Murugesh, Dr. Abdul Bhat, Dr. Amit Badshah, Dr. Muhammad Badar Khalid

INTRODUCTION

St-elevation with new-onset left bundle branch block (LBBB) typically triggers an urgent STEMI pathway. However, not all cases reflect coronary occlusion. We describe a rare presentation where acute cardiac dysfunction, masquerading as sepsis, drove systemic ischaemia across multiple organs. This case highlights the diagnostic challenge of distinguishing true STEMI from myocarditis and stress cardiomyopathy, and the downstream impact of hypoperfusion on liver and kidney function.

UNIQUE NATURE

This case is notable for three reasons:

1.STEMI mimicry: Despite classic ECG changes, the absence of chest pain, modest troponin rise, and global hypokinesia pointed away from ACS.

2.Sepsis-like profile: The patient presented with encephalopathy, multiorgan dysfunction, and raised lactate ,features easily misattributed to infection or vasculitis.

3.Autoimmune distraction: A positive p-ANCA nearly led to a misdiagnosis, illustrating the danger of anchoring on single abnormal results.

CASE PRESENTATION

64-year-old woman

Medical History
1. Seronegative rheumatoid arthritis
2. Hypothyroidism
3. Migraine

Presenting Symptoms
- Acute occipital headach
- Vomiting Confusion
- No chest pain

Initial Observations
- Hypotension: 95/50 mmHg
- Preserved oxygen saturation
- Mild tachypnoea

ECG Findings
- New Left Bundle Branch Block (LBBB)
- Anterior ST elevation (V2–V4)

LABORATORY INVESTIGATIONS

Hepatic Injury (Ischaemic Hepatitis)	Renal Injury	Cardiac Markers(Troponin)	Metabolic (Lactate)	Immunology (p-ANCA)	Cardiac Imaging (Echocardiography)	Other Investigations
ALT >1079 U/L, AST >1800 U/L	Creatinine 136 µmol/L (acute kidney injury)	Peak 53 ng/L (modest elevation)	4.5 mmol/L	Positive	Global LV hypokinesia, Ejection Fraction 32%, **No regional wall motion abnormality**	Neuroimaging: Unremarkable, CSF studies: Unremarkable

After multidisciplinary review, this was reinterpreted not as acute coronary syndrome or systemic vasculitis, but as acute cardiac dysfunction likely myocarditis or stress cardiomyopathy driving multiorgan hypoperfusion. Supportive therapy was provided, methotrexate/ withheld, and immunosuppression avoided. The patient improved without PCI or steroids.

LEARNING POINTS

1.Cardiac Failure as Sepsis MimicAcute cardiac failure can masquerade as sepsis when hypoperfusion drives liver, kidney, and brain injury.

2. STEMI mimics such as myocarditis and Takotsubo cardiomyopathy must remain central in the differential when ST elevation occurs with normal coronaries.

3.Autoimmune Markers CautionAutoimmune markers may mislead; clinicopathological context is paramount.

4.Role of Early ImagingEarly echocardiography and multidisciplinary collaboration are critical in preventing unnecessary invasive interventions or immunosuppression.

CONCLUSION

This case represents a rare form of ischaemia-driven multiorgan insult mimicking sepsis, where acute left ventricular dysfunction created a systemic shock state. The lesson lies in recognising the heart not only as the source of symptoms but also as the failing pump precipitating distant organ injury. Careful interpretation of investigations and collaborative decision-making ensured correct diagnosis and recovery

REFERENCES

- Caforio ALP, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis. *Eur Heart J.* 2013;34(33):2636–2648.
- Lyon AR, et al. Stress (Takotsubo) cardiomyopathy—\a novel pathophysiological hypothesis to explain catecholamine-induced acute myocardial stunning. *Nat Clin Pract Cardiovasc Med.* 2008;5(1):22–29.
- Nishimura RA, et al. ST elevation with normal coronaries: The dilemma of myocardial infarction mimics. *Circulation.* 2017;135(14):1231–1235.
- NICE CKS. Chest pain - acute. 2023.
- RCEM Guidelines. STEMI – Acute Management. 2023.

Facial Diplegia with Paresthesias Variant of Guillain–Barré Syndrome Associated with Autoimmune Haemolytic Anaemia: A Case Report

Dr Shanaz Koyam Madath[1], Dr Bijoy Jose[1], Dr Deepak Charles[1], Dr Mathew Abraham[1] | [1]Aster Medcity, Kochi, India

Introduction

• Facial Diplegia with Paresthesias (FDP) is an uncommon Guillain–Barré syndrome (GBS) variant characterized by **bilateral lower-motor-neuron facial weakness** and **distal sensory disturbance** with preserved limb power.

• The concurrent presence of **Autoimmune Haemolytic Anaemia (AIHA)** is exceptionally rare and may significantly **alter therapeutic** decision-making, particularly when using intravenous immunoglobulin (IVIG).

•Awareness of such immune overlap syndromes is vital to avoid treatment-related morbidity.

Case Presentation

•**29-year-old female** presented with ascending numbness and subsequent **bilateral LMN facial palsy** within 48 hours.

•**Motor power preserved; reflexes diminished.**

•**MRI brain/spine – normal; NCS – absent F-waves → demyelinating neuropathy.**

•**CSF:** albuminocytologic dissociation.

•**Initial Hb 10.2 g/dL.**

Results

•Commenced on **IVIG (2 g/kg over 5 days)** for GBS.

•On Day 4 → developed **icterus** and laboratory evidence of **hemolysis** (↑ LDH, indirect hyperbilirubinemia, Coombs IgG positive).

•**Autoimmune screen:** ANA +, Anti-Ro52 +, Anti-Ro +, AMA-M2 +.

•**IVIG discontinued; high-dose methylprednisolone initiated.**

•**Mycophenolate mofetil** added for steroid-refractory anemia.

•**Neurological recovery** achieved within 1 week; **hematological recovery** over 4 weeks.

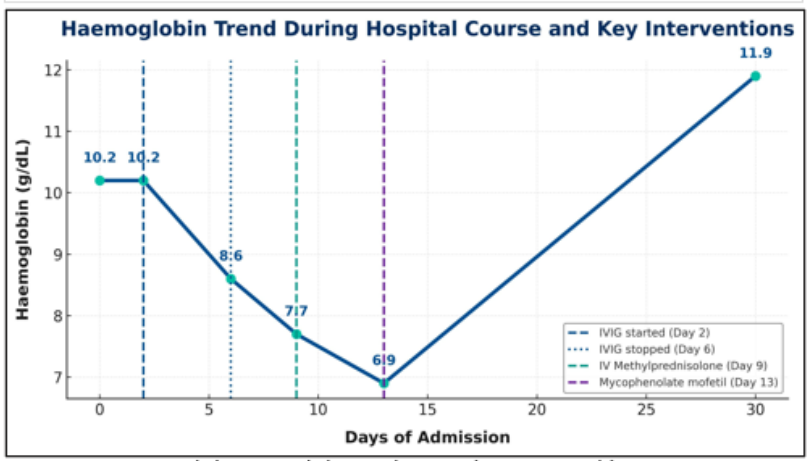


Figure 1. Hemoglobin trend during hospital course and key interventions.

Discussion and Conclusion

•**Facial Diplegia with Paresthesias (FDP)** is a rare Guillain–Barré variant; coexistence with **Autoimmune Haemolytic Anaemia (AIHA)** represents an exceptionally uncommon immune overlap syndrome.

•**IVIG-induced hemolysis** is a recognized but under-reported adverse effect, particularly in **autoantibody-positive patients**.

•Literature shows onset typically occurs **within 12 hours to 10 days** of infusion, with hemoglobin nadirs appearing **1 to 14 days post-treatment** and resolution over **several days to a week** in mild cases.

•This case's hemolysis on **Day 4** falls squarely within that expected window, emphasizing the importance of **post-IVIG vigilance,** although had a **delayed hematological recovery requiring steroids and immunosuppression** indicating a pre-existing associated **autoimmune condition,** unmasked by **atypical GBS.**

•Recommended practice**:** monitor hemoglobin **36–96 hours post-infusion** and continue periodic checks for up to **10 days**, as delayed hemolysis can occur.

•**Early identification, prompt discontinuation of IVIG, and tailored immunosuppression** (steroids ± MMF) are crucial for achieving both neurological and hematological recovery.

•This case highlights the need for **individualized, multidisciplinary management** and continuous hematologic surveillance in atypical GBS variants with autoimmune overlap.

•Maintain a high index of suspicion for autoimmune overlap in atypical GBS, monitor hematological parameters closely during IVG therapy, timely immunosuppressive modulation ensures dual neurological and hematological recovery.

Presented at RCP Med+ 2025, Royal College of Physicians, London, UK – November 2025

CENTRAL PONTINE MYELINOSIS WITH NORMONATREMIA IN A CHRONIC ALCOHOLIC

Dr Zuha Khan | Dr Y Joel Suvarna Raju | Dr Samia Dilrus Syeda | Dr Ashwini Kumar

George Eliot Hospital NHS Trust

Introduction

Central pontine myelinosis (CPM) is usually linked with rapid correction of sodium.[1] In Chronic Alcoholics CPM may occur even with normonatremia although the cause and pathogenesis remains unclear.[1] Here we describe one such case.

Case Summary:

- 48-year-old male presented with acute confusion (2-day history); background of heavy alcohol use (12–14 units/day), 5 pack-year smoking, and occasional cannabis use.
- On examination: Disoriented but no focal deficits. Bloods and CT head were unremarkable.
- Admitted for observation; started on vitamin B and C replacement and referred to the alcohol liaison team.
- Despite treatment, confusion persisted. Mental health team found poor Mini-ACE score, confabulation, and lack of capacity.
- MRI brain showed T2/FLAIR hyperintensity in the central pons, suggestive of early CPM.
- Continued on vitamin therapy and supportive care; discharged to a care home. Over months, memory improved, confabulation resolved, but short-term recall remained impaired.

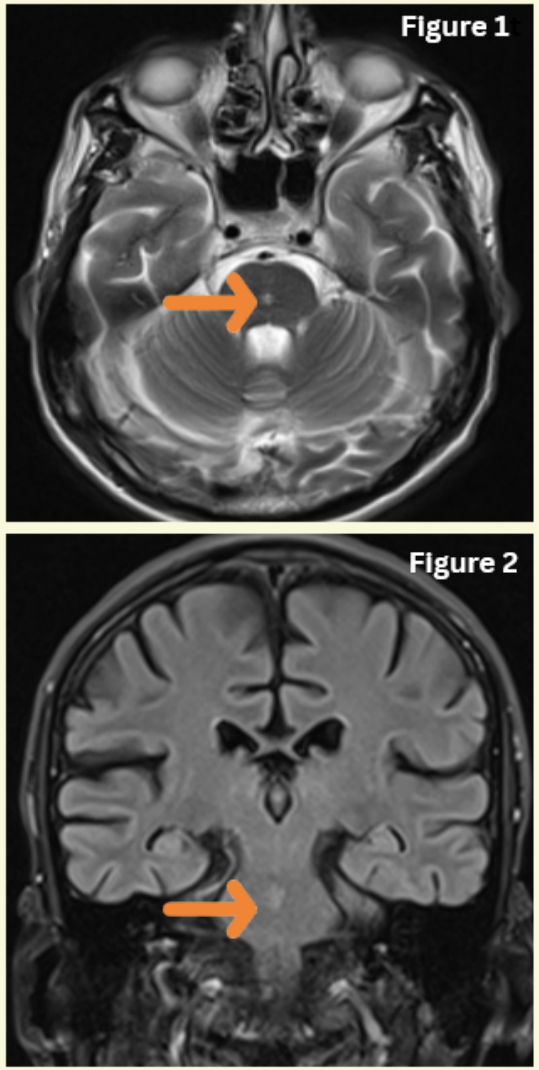


Figure 1 and 2 show images of MRI brain showing a hyperintensity in the pontine region s/o CPM.

Discussion:

Similar to published cases, this case highlights the atypical presentation of CPM in patients with chronic alcoholism.[2] The proposed hypothesis include osmotic changes during withdrawal, dysfunction of the liver, cerebral atrophy and chronic malnutrition, which may make the brain susceptible to demyelination despite stable electrolytes.[3-6]

Conclusion:

This is a rare case of CPM in a patient of chronic alcoholism which can manifest with subtle neurological deficits such as confusion. Physicians should pay attention to the development of CPM in chronic alcoholics and MRI is the key for early diagnosis.[3-4] CPM should be considered as a differential diagnosis in such patients and prompt neuroimaging should be performed to avoid delaying the diagnosis.

References

The Silent Stanford A

NHS
Wirral University Teaching Hospital
NHS Foundation Trust

Introduction:

Aortic dissection represents one of the most catastrophic emergencies in cardiovascular medicine, carrying a mortality rate that increases by 1-2% per hour without surgical intervention.

While classic teaching emphasises abrupt, severe pain as the hallmark presentation, we present an extraordinary case that defies this paradigm — a completely asymptomatic dissection discovered incidentally in a patient with a strong family history of connective tissue disorder.

This case not only challenges fundamental clinical assumptions but also powerfully underscores the critical importance of proactive screening.

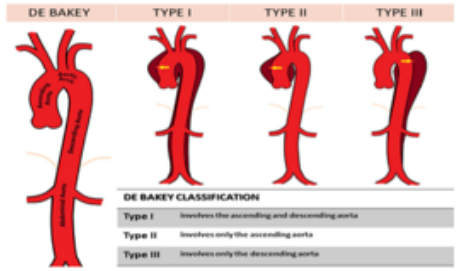
Case Presentation:

A 56-year-old **asymptomatic** female was referred for outpatient transthoracic echocardiography to investigate a **newly discovered diastolic murmur** noted during a routine follow-up for hypertension management.

Her history was significant for a strong **family history of Marfan syndrome** in her mother and sister, though she had never undergone formal genetic testing or dedicated aortic imaging. Despite thorough and repeated questioning, she explicitly denied any pain, dyspnoea, syncope, or neurological symptoms.

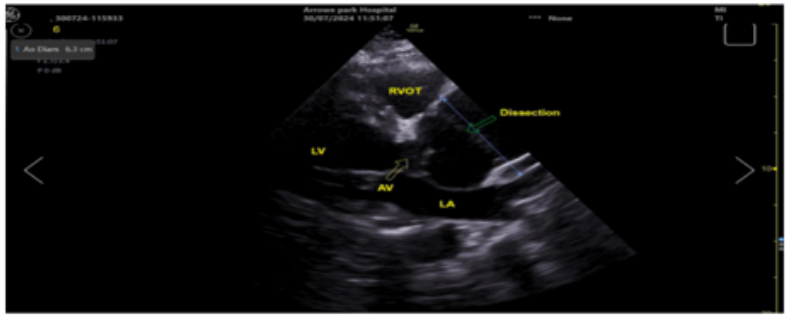
Cardiovascular auscultation revealed a soft, early diastolic murmur. She was haemodynamically stable (BP 111/69 mmHg, HR 64 bpm). Remarkably, lab results, including troponin-I and D-dimer, returned well within normal limits.

Bedside echocardiography demonstrated a severely dilated aortic root of 5.9 cm with a highly mobile, oscillating intimal flap and moderate-to-severe aortic regurgitation. Emergency CT angiography confirmed an **Acute Stanford A (DeBakey type II) Dissection** originating from a gigantic 72mm root aneurysm, extending to the brachiocephalic artery origin, with no evidence of mural thrombus, suggesting an acute event.

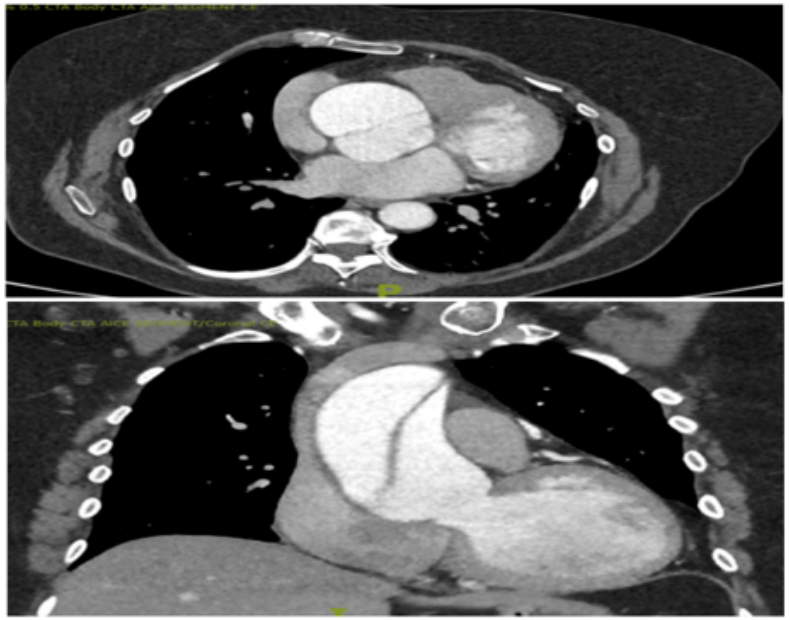


The patient was immediately started on a titrated labetalol infusion for strict haemodynamic control, targeting systolic BP <130 mmHg and heart rate <60 bpm in preparation for emergency surgical repair.

Bedside ECHO:



CT Aortogram:



Clinical pearl:

Sometimes, the most critical warnings come not with a scream, but with a whisper. In high-risk patients, a single new murmur can be the only clue to a silent, lethal dissection

- A clue that, when heeded, grants the power to alter a fatal outcome.

Discussion:

This case is exceptional for its triad of unusual features:

1. The silent clinical presentation

2. The extreme dimensions of the aortic root aneurysm at discovery.

3. The completely normal biomarkers despite an acute dissection.

In patients with high risk of connective tissue disorders, chronic aortic remodelling and neural adaptation may attenuate or eliminate the typical pain response, rendering the dissection clinically silent until terminal rupture.

The murmur of acute aortic regurgitation, therefore, becomes a paramount— and potentially solitary—sentinel sign, mandating immediate and definitive vascular imaging.

The patient was transferred to tertiary care for surgical intervention on the same day, exponentially increasing her chance of survival.

This case proves that relying solely on classic symptoms like severe pain may miss life-threatening aortic dissections. Therefore, proactive imaging and monitoring in patients with connective tissue disorders are essential.

Dr M Hamad - Consultant Acute Medicine
Mrs S Dasuqi - Critical care Clinical Pharmacist
Dr A Sancaran - Senior Clinical Fellow Acute Medicine
Dr H Suliman - FY2
Dr L Malik - FY1

References:

1. Association of painless acute aortic dissection with increased mortality. Park SW, Hutchison S, Mehta RH, et al. Mayo Clin Proc. 2004;79:1252–1257. doi: 10.4065/79.10.1252.
2. 2. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. Hagan PG, Nienaber CA, Isselbacher EM, et al. JAMA. 2000;283:897–903.
3. 3. Asymptomatic aortic dissection in Marfan syndrome. Wong T, Kilner PJ, Gatzoulis MA. Heart. 2002;87:66. doi: 10.1136/heart.87.1.66

Declaration - No conflict of interest

Battling Persistent COVID-19 Pneumonitis in Immunocompromise: A Remdesivir Success Story

Dr J Collins, Dr W Butterfield, Dr R Sheridan
Royal Devon University Healthcare NHS Foundation Trust

Introduction

Patients with chronic lymphocytic leukaemia (CLL) have impaired immunity, making them highly vulnerable to severe COVID-19, particularly when treated with anti-CD20 therapies like Rituximab. Their weakened innate and adaptive immune responses lead to poor viral clearance, prolonged infection, and high mortality [1-3]. Our case demonstrates successful off-label Remdesivir use in a CLL patient with late-stage COVID-19, resulting in rapid recovery where treatment options are otherwise limited.This case demonstrates successful off-label Remdesivir use with rapid clinical and virological improvement.

Case Presentation

64-year-old man with CLL (on Venetoclax maintenance, prior Rituximab), COPD, T2DM, AF on Rivaroxaban, and prior CABG, presenting with fever, cough, and breathlessness. Vaccinated with 4 COVID-19 doses (2× AstraZeneca, 2× Pfizer mRNA).

Day	Event / Intervention
1	Admission → Bronchodilators, IV Dexamethasone (RECOVERY TRIAL [4])
2-9	IV → Oral steroids. Amoxicillin/Doxycycline → Piperacillin with Tazobactam → Meropenem under microbiology guidance based on sensitivities
10	8L High-Flow Nasal Oxygen initiated (Figure 1)
19	Unable to wean <5 L O_2; CTPA: Bilateral Ground-Glass Opacities → COVID pneumonitis (based on CT (Figure 2), lack of antibiotic response + persistently low Cycle threshold (Ct) of 23)
20	Remdesivir started → Marked improvement
24	Weaned off oxygen therapy
30	Ct 37 → Viral clearance (Figure 3); Discharged

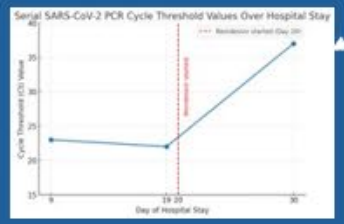


Figure 3. Serial SARS-CoV-2 PCR Ct values over hospital stay

Figure 1. Timeline of oxygen requirement pre- and post-Remdesivir.

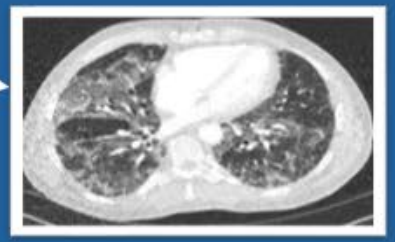


Figure 2. CT pulmonary angiogram on day 19 demonstrating COVID-19 Pneumonitis

COVID Antiviral Considerations

- Sotrovimab withheld (Ab >500 U/mL – Local Protocol)
- Paxlovid contraindicated (Rivaroxaban interaction – Liverpool COVID-19 Interaction Checker [5])

Discussion

- Patients with CLL treated with venetoclax and anti-CD20 therapy have a higher risk of SARS-CoV-2 infection (OR 1.75) with infection rates up to 62.9% [6].
- Low RT-PCR Ct values, indicating high viral load, correlate with greater COVID-19 severity and mortality, especially Ct <25 versus >30 (OR 2.31) [7–9]. In this case, persistently low Ct values and minimal clinical improvement supported a diagnosis of persistent COVID pneumonitis.
- Remdesivir, an RNA polymerase inhibitor, reduces mortality, ICU admissions, and hospital stay length [10,11]. Our case suggests Remdesivir may benefit immunocompromised patients even when started late (>Day 7).
- Further research is needed to optimise antiviral timing in prolonged COVID-19 cases.

Conclusions

In immunocompromised patients, persistent COVID pneumonitis should be considered with ongoing O_2 need and rising inflammatory markers.
Ct trends and imaging aid in assessing viral persistence.
Remdesivir may be effective even >7 days after symptom onset, offering a key option when other antivirals are unsuitable.
This case underscores the need for updated treatment pathways and specific guidance for high-risk haematology patients.

(1) Scarfò *et al.* (2020) reported on COVID-19 severity and mortality in CLL through the ERIC and CLL Campus collaboration (*Leukemia*), (2) Akbarzadeh *et al.* (2024) conducted a systematic review and meta-analysis of COVID-19 outcomes in CLL patients (*Rev Assoc Med Bras*), (3) Arellano-Llamas *et al.* (2022) discussed the impact of the SARS-CoV-2 pandemic on CLL management (*Curr Oncol Rep*), (4) the RECOVERY Collaborative Group (2021) demonstrated the benefit of dexamethasone in hospitalized COVID-19 patients (*N Engl J Med*), (5) the University of Liverpool COVID-19 Drug Interactions resource provided guidance on antiviral and drug interaction management, (6) Autore *et al.* (2023) examined COVID-19 outcomes in CLL patients treated with Venetoclax with or without anti-CD20 therapy (*Blood*), (7) Bustin and Mueller (2005) described the diagnostic utility of qRT-PCR (*Clin Sci*), (8) Rao *et al.* (2020) reviewed the clinical utility of SARS-CoV-2 cycle threshold (Ct) values (*Infect Dis Ther*), (9) Shah *et al.* (2021) conducted a meta-analysis linking Ct values with COVID-19 outcomes (*Open Forum Infect Dis*), (10) Libra *et al.* (2023) evaluated the effects of Remdesivir on hypoxia and inflammation in COVID-19 pneumonia (*Viruses*), and (11) Bigman-Peer *et al.*(2022) described biphasic COVID-19 courses in anti-CD20–treated patients (*EJCRIM*).

Swimmer's Itch in a returning traveler : an interesting case of rashes

Grace Thomas, Trust Grade Registrar, General Medicine
Croydon University Hospital

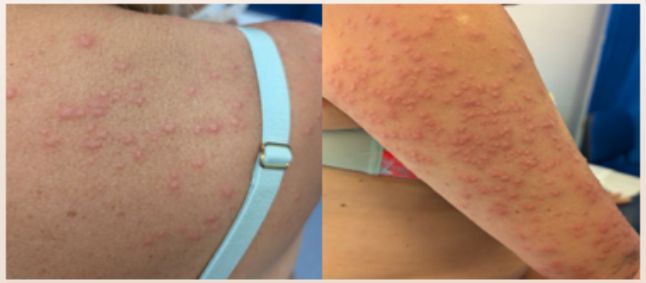
Introduction

Swimmer's itch, also known as cercarial dermatitis, is a cutaneous hypersensitivity reaction triggered by the penetration of non-human larvae into the skin, typically contracted through contact with freshwater. While it often resolves on its own, its diverse symptoms can resemble other sun-related skin conditions, especially in travelers who experience simultaneous sun exposure.

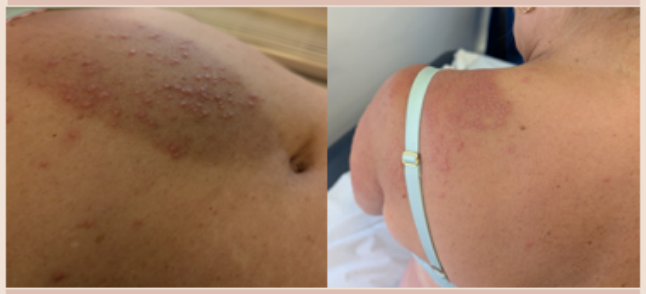
Also known as 'duck itch' (New Zealand), 'duckworms' (US), 'hoi con' (Thailand), 'kobanyo' (Japan), 'sawah' (Malaysia), and 'rice paddy itch' or 'clam diggers itch.

Case Presentation

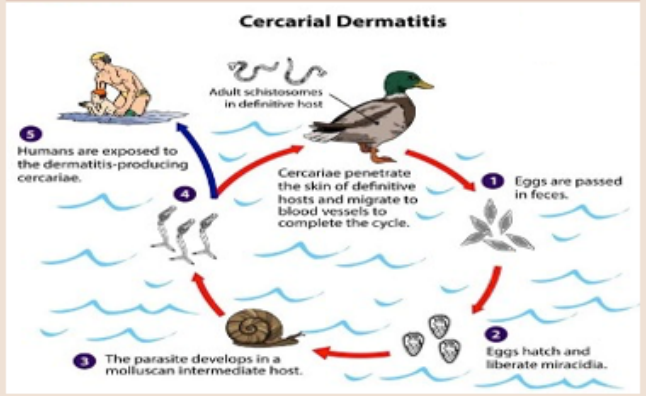
- Eastern European woman in her early 60s, residing in the UK.
- 5-day history of a pruritic erythematous rash on the arms, shoulders, and back.
- Travel history to Kenya.
- Exposure to both fresh and salt water.
- No significant past medical history except for Herpes simplex in the past.
- No significant drug history, however, took anti-Malarial prophylaxis - Malarone (proguanil/atovaquone) during her trip.
- No significant family history.
- No systemic symptoms.
- Examination: revealed monomorphic, papulo-vesicular lesions predominantly on sun-exposed areas, with a distinct oval distribution on the abdomen matching the swimwear cutout.
- Labs: normal blood counts, renal and liver function, mildly raised CRP of 25 mg/L
- Negative for HSV-1, HSV-2, and VZV.
- Skin biopsy was declined.
- Based on the exposure history, lesion morphology, and self-limiting course, swimmer's itch was considered the most likely diagnosis.



Photos depicting erythematous, monomorphic, papulo-vesicular rash



Picture above showing a tanned oval area in the abdomen corresponding to the lady's swimwear cutout.



Management

She was managed with fucibet cream and topical emollient. On follow-up via telephone, the patient reported significant improvement in her symptoms, with near-complete resolution of the rash.

She did not require any further medical intervention beyond supportive care and sun avoidance.

Discussion

Swimmer's itch typically presents within hours of freshwater contact but may have a delayed onset, particularly in first exposures or mild infestations. Distribution is often limited to exposed skin, and lesions may overlap with sun-exposed patterns, complicating diagnosis. Awareness of aquatic exposure history is essential to differentiate it from photodermatoses such as polymorphic light eruption.

A broad differential was considered - Polymorphic light eruption, sea bather's eruption, actinic prurigo to name a few.
This case underscores the importance of a thorough travel history, drug review, and dermatologic assessment in evaluating post-travel rashes. Management focuses on supportive care and sun avoidance. The patient's significant improvement on follow-up without the need for intensive intervention supports a self-limited process. A focused exposure history can guide diagnosis and avoid unnecessary investigations.

References

DermNet NZ: Swimmer's itch—aetiology, clinical features. https://dermnetnz.org/topics/swimmers-itch.

Verbrugge LM, Rainey JJ, Reimink RL, Blankespoor HD: Swimmer's Itch: Incidence and Risk Factors. Am J Public Health. 2004 May;94(5):738-741. doi: . 10.2105/ajph.94.5.738.

Masquerading of Mesenteric Ischemia – A Case Report

Yousef Khorma MBBCH
Royal Liverpool University Hospital – Liverpool, England

Introduction

Inflammatory bowel disease (IBD) is a chronic gastrointestinal condition characterized by inflammation of the intestinal tract, leading to non specific symptoms such as abdominal pain, diarrhoea, and weight loss.

This case presents a unique diagnostic challenge involving a 46-year-old female with IBD-like symptoms who developed a significant ischaemic complication, ultimately leading to a revision of the initial diagnosis.

Case presentation

- **6 mo history:** Abdominal pain and intermittent diarrhoea → referred to colorectal clinic.
- **Investigations (OP) :** FIT 79 µg/g ↑, fecal calprotectin >1700 µg/g ↑, normal FBC/U&E, ferritin 198 ↑ → suspected IBD.
- **Colonoscopy (OP):** Erythema, diffuse ulcerations (caecum → rectum); histology: mild–moderate chronic inflammation → provisional IBD.
- **Few days later:** Admitted with Acute worsening with bilious vomiting.
- **CT (Day 1):** Mild splenic changes; started IV steroids for presumed flare.
- **Day 3:** Pain refractory; CXR → pneumoperitoneum, lactate 2.18
- **Repeat CT:** Occlusive **SMA thrombus**, hepatic-flexure perforation, bowel ischaemia (caecum–20 cm TI), necrotic gallbladder.
- **Surgery:** Right hemicolectomy, SMA stent, cholecystectomy.
- **Recovery:** 11-day ICU stay → discharged on apixaban + aspirin.
- **Outcome:** IBD diagnosis retracted → **final: SMA thrombosis with ischaemic bowel (IBD mimic).**

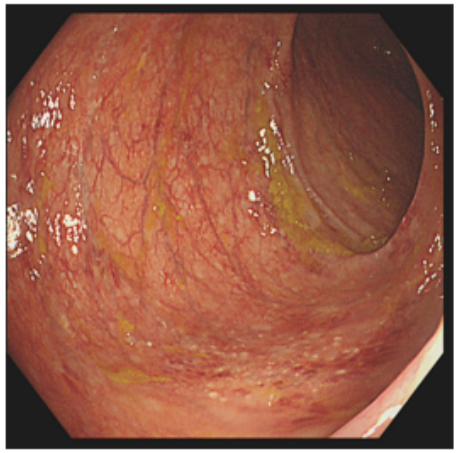


Image 1: Inflamed and congested Ascending colon.

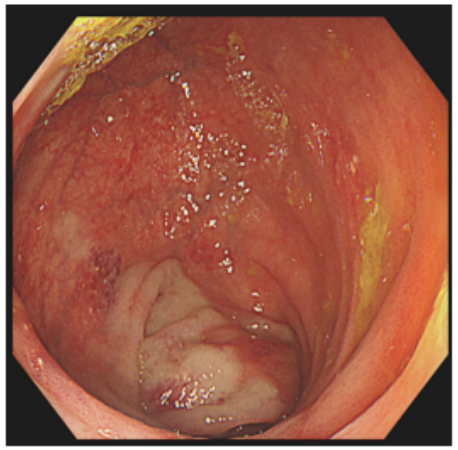


Image 2: Inflamed and congested cecum

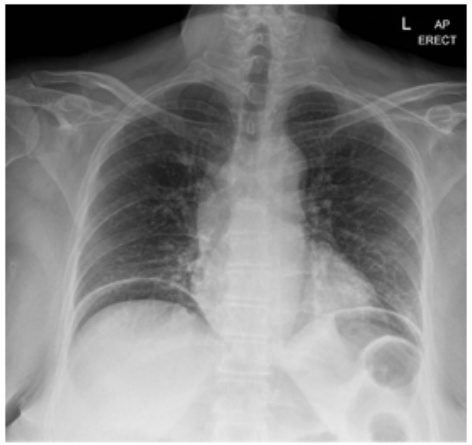


Image 3: Air under diaphragm from pneumoperitoneum

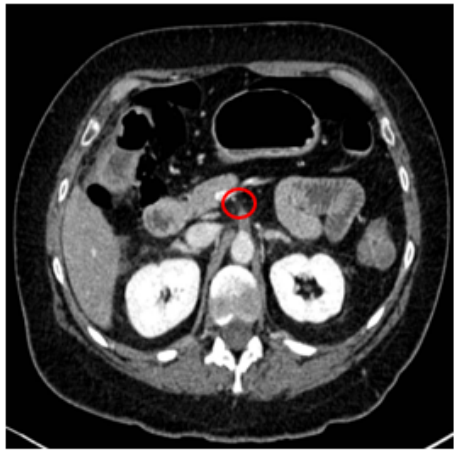


Image 4: SMA Thrombus on CTA

Discussion & conclusion

- A prior diagnosis of IBS may have obscured the development of evolving mesenteric ischaemia.
- Retrospective imaging revealed a superior mesenteric artery (SMA) thrombus that was initially missed.
- The presumed IBD diagnosis was later overturned following identification of ischaemic bowel and vascular pathology.
- Highlights the need for diagnostic vigilance when gastrointestinal symptoms are atypical or disease progression is unexpected.
- SMA thrombosis can mimic IBD and demands early recognition and multidisciplinary management to prevent complications.

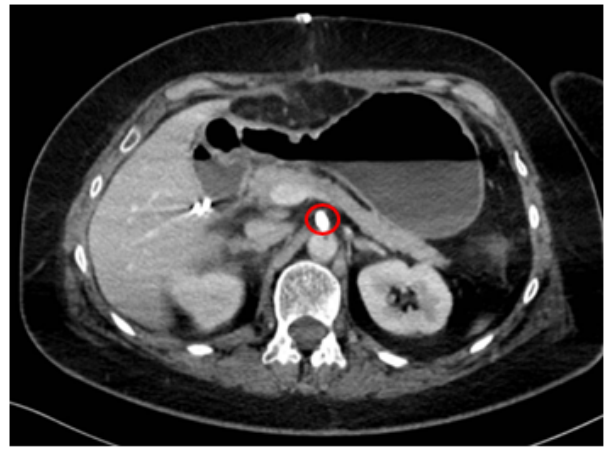


Image 5: Stenting of SMA

DERMATOMYOSITIS TRIGGERED BY CHIKUNGUNYA VIRUS INFECTION

Authors: Arzoo Javed, Nehal Yemula, Aimen Ayaz, Onn Shaun Thein, Megan Rutter

Key points in history

- 40-year-old female with background of coeliac disease presented 4-week history of progressive shortness of breath on exertion, periorbital swelling, and a sandpaper-like rash along with recent travel to Miami and the Caribbean, where she recalled an infected mosquito bite.

Clinical events and work up

- Type 1 respiratory failure requiring ICU admission for HFNO - initially managed as ARDS
- Imaging showed bilateral ground-glass opacities, and inflammatory markers were significantly raised (Figure 1)
- Microbiological tests were negative except for a positive chikungunya virus IgM. Autoimmune screening revealed a strongly positive ANA & anti-Jo-1, weakly positive anti-Ro-52
- Signs of inflammatory myositis affecting thigh and gluteal compartments on MRI (Figure 3)
- CK peaked at 1566 U/L

Management

- 3 days of IV Methylprednisolone, followed by mycophenolate mofetil
- Clinical and radiological improvement (Figure 2) noted with CK levels improving to 399 U/L
- Discharged with ongoing rheumatology and respiratory follow up

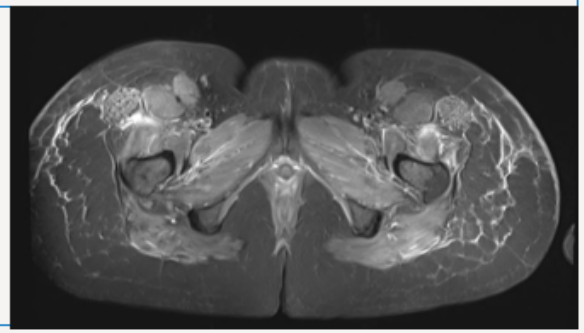


Figure 3: MRI pelvis axial view demonstrating changes consistent with myositis

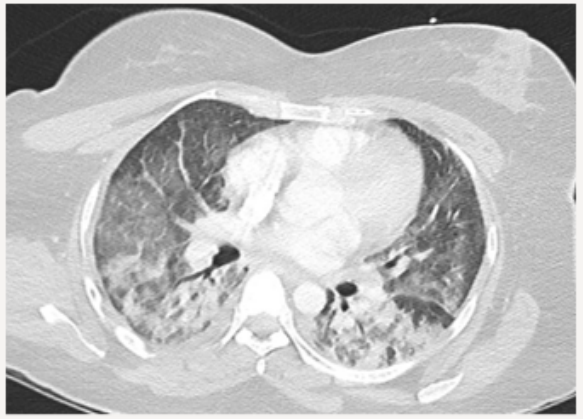


Figure 1: CECT scan chest axial view demonstrating bilateral ground glass changes pre-steroids

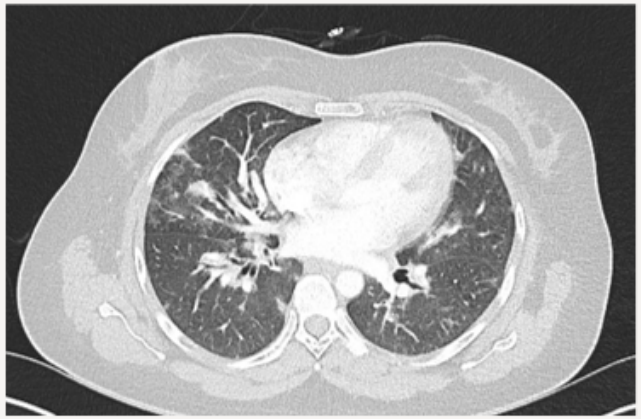


Figure 2: CECT scan chest axial view demonstrating clearer lung fields post-steroids

Discussion

Dermatomyositis following chikungunya virus (CHIKV) infection is exceptionally rare. To our knowledge, this is only the second reported case in the literature (1), and the first case occurring in the subacute phase(2,3).

Our patient's presentation was distinctive for its subacute onset, anti-Jo-1 antibody positivity, and severe lung involvement resembling ARDS. CHIKV, transmitted by Aedes mosquitoes, classically causes an acute febrile illness with rash and polyarthralgia (4).

This case highlights the importance of recognising post-viral autoimmune myopathies in patients with recent arboviral exposure, particularly when respiratory symptoms and myositis coexist. Early recognition and immunosuppressive therapy are key to improving outcomes and preventing long-term disability.

References

1.Raiza Cansian Tuão, Mariana, Paula, Isac Ribeiro Moulaz, Dalmaso BF, Cristo VV, et al. Antisynthetase Syndrome after chikungunya infection: A case report. Clinical Case Reports. 2022 Sep 1;10(9).
2. Dev N, Kumar R, Gogna A, Sharma S. Chikungunya-induced inflammatory myositis: a case report in India. Tropical doctor [Internet]. 2019 Jul;49(3):241–3. Available from: https://pubmed.ncbi.nlm.nih.gov/31018774/
3. Martins HA. Chikungunya and Myositis: A Case Report in Brazil. JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH. 2016
4. Vairo F, Haider N, Kock R, Ntoumi F, Ippolito G, Zumla A. Chikungunya. Infectious Disease Clinics of North America. 2019 Dec;33(4):1003–25.

An Interesting Case Of An Unusual Triad: Case Report On A Forgotten Syndrome; Milk-Alkali Syndrome

Introduction & Case Presentation

- A 54-year-old male presented with confusion and vomiting.
- **Past medical history:** Treated tonsillar carcinoma.
- **Initial findings:** Severe Acute Kidney Injury (AKI), hypercalcaemia, hypokalaemia, and metabolic alkalosis.
- **Treatment:** He was treated with intravenous fluids and potassium replacement, then discharged with outpatient follow-up. However, three months later, he re-presented with dysphagia and similar biochemical abnormalities (AKI, hypercalcaemia, metabolic alkalosis).

Diagnostic Workup

- **Hypercalcaemia workup:** Normal PTH, vitamin D, and myeloma screen. Elevated phosphate. Low magnesium.
- **Renal workup:** Significant proteinuria and anaemia. Renal ultrasound suggested intrinsic renal disease.
- **Key Finding:** Renal biopsy revealed chronic tubulointerstitial damage with abundant calcium phosphate deposits.
- **Crucial History:** Uncovered a daily intake of >2 pints of milk mixed with baking soda (for indigestion) and high-dose Vitamin D3 (3200 IU/day).

Diagnosis

"Milk-Alkali Syndrome"
Diagnosed based on the classic triad:

1- Renal Failure
2- Hypercalcaemia
3- Metabolic Alkalosis

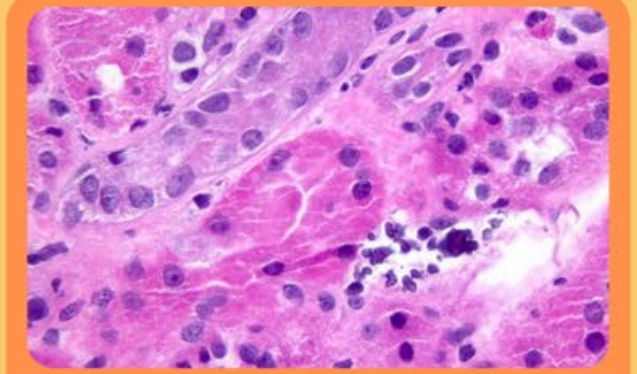
...alongside the supportive history and biopsy findings.

Management & Outcome

- **Intervention:** Dietary modification, reduced milk intake, increased non-dairy fluids, and discontinued baking soda/Vitamin D3.
- **Outcome:** Gradual improvement in calcium and renal function.
- **Final Status:** Patient's renal function & hypercalcaemia improved. Education provided on potential kidney transplant/haemodialysis.

Key Take aways:

- Milk-alkali syndrome is a reversible cause of hypercalcaemia and AKI, but delayed diagnosis can lead to irreversible renal failure.
- It is the third most common cause of hypercalcaemia.
- Clinical Pearl: Suspect this syndrome in any patient with the classic triad and meticulously take a dietary and over-the-counter supplement history.
- Prompt recognition and withdrawal of the inciting agents are crucial to prevent chronic kidney damage.

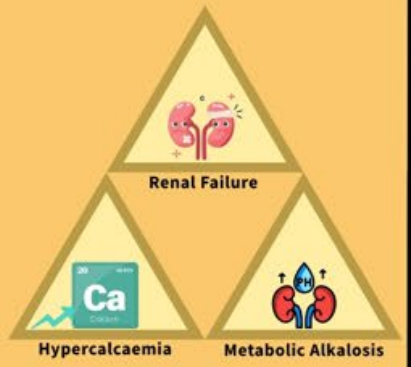


Courtesy of Alexei Mikhailov, M.D., Ph.D & PathologyOutlines.com see URL
www.pathologyoutlines.com/topic/kidneynephrocalcinosis

Royal College of Physicians

THE UHS WAY

NHS
University Hospital Southampton
NHS Foundation Trust



References:

1. Patel AM, Goldfarb S. Milk-Alkali Syndrome. StatPearls. Treasure Island (FL): StatPearls Publishing; 2024. https://www.ncbi.nlm.nih.gov/books/NBK557500/ [Accessed 15 August 2025]
2. Medarov BI. Milk-alkali syndrome. Clin J Am Soc Nephrol. 2010;5(4):691–9

Authors:
Dr. Jeshwin Thamburaj
Dr. Hamza Naveed Virk
Dr. Hamna Manan
Dr. Kirsten Armstrong
Dr. Helena Edwards
Dr. Shreya Kandel

Mollaret's Meningitis

NHS Wirral University Teaching Hospital NHS Foundation Trust

Dr M. Hamad (Consultant in Acute Medicine) • Mrs S. Dasuqi (Critical Care Clinical Pharmacist) •
Dr A. Sancaran (Senior Clinical Fellow Acute Medicine) • Dr J. Coulthurst (FY-1)

Introduction:- Mollaret's meningitis (recurrent benign lymphocytic meningitis) is a rare neurological syndrome characterised by:

- ≥3 recurrent aseptic episodes
- Self-limiting course
- Strong association with HSV-2

First described by Pierre Mollaret (1944).

Case Presentation

35-year-old man presented with acute severe headache, photophobia, vomiting, and neck stiffness.
History: three identical self-resolving episodes over the past two years, each requiring admission (7–10 days recovery).
No significant past medical or drug history.
Exam: meningism without focal deficits, rash, or systemic findings.

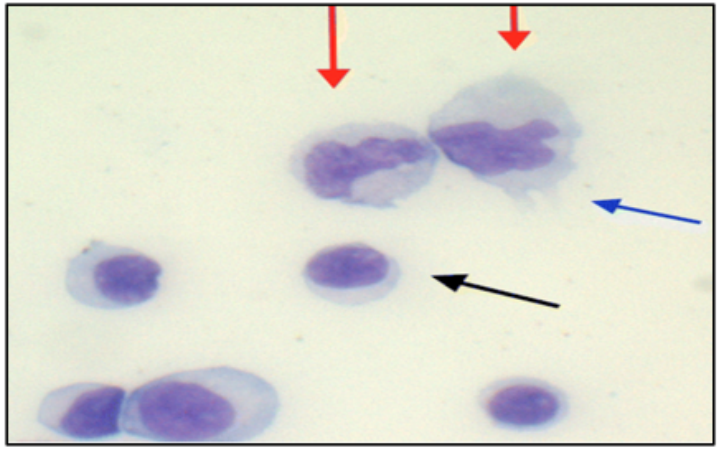
Management

Investigations: Bloods, CXR, ECG, and CT brain were normal.
MRI: mild meningeal enhancement.
CSF: lymphocytic pleocytosis, raised protein, normal glucose.
HSV-2 DNA detected on PCR, consistent with prior results.
Empirical antibiotics stopped once viral cause confirmed.
IV Acyclovir (10 mg/kg q8h) continued → marked improvement within 72 hours. Discharged with neurology follow-up and consideration for long-term antiviral prophylaxis.

Relevance to Acute Medicine

Patients often present acutely with meningitis symptoms mimicking bacterial meningitis. Recognition of recurrent, self-limiting pattern prevents unnecessary broad-spectrum antibiotic use.
Detailed history (previous identical episodes) is diagnostic gold. Targeted HSV PCR testing and early microbiology/neurology input streamline management. Highlights the importance of antimicrobial stewardship and pattern recognition in acute settings.

Fig 1: Mollaret Cells (endothelial polymorphonuclear cells)



Aseptic meningitis mimics:-
SLE, Behcet's disease, Sarcoidosis, Dermoid cyst, Neoplastic meningitis, Lymphoma.

Discussion :-

Mollaret's meningitis should be considered in any patient with recurrent aseptic meningitis.
-**CSF findings:** transient pleocytosis, Mollaret cells, and positive HSV-2 PCR (though PCR sensitivity may fluctuate).
-**Diagnosis:** a negative PCR does not exclude the condition — clinical context and recurrence pattern are key.
-**Treatment:** typically antiviral therapy (e.g. acyclovir). Spontaneous recovery can occur even without antivirals.
-Long-term antiviral suppression may reduce recurrences, though evidence remains limited.

References:

1. Mascia RA, Smith CW. Am J Med Sci 1984; 287:
2. Galdi AP. Arch Neurol. 1979; 36:657–658.
3. Sendi P, Graber P. CMAJ 2006; 174 (12) 1710-1710-a

Declaration : No conflict of interest

University Hospitals Birmingham NHS Foundation Trust

Approach to primary differentiated thyroid cancer identified during evaluation of primary lung cancer.

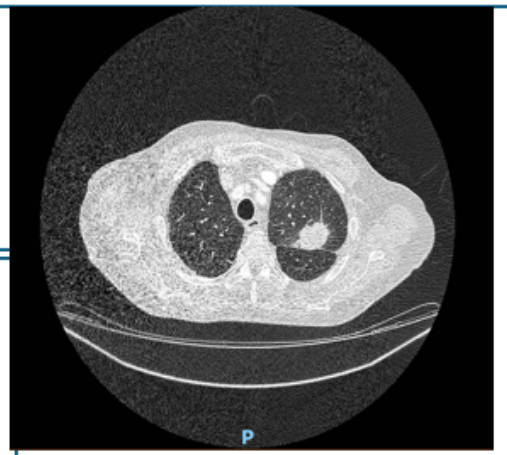
Porwal, Vishma; Tahsin, Saara; Kalaiah, Yogesh
University hospitals Birmingham NHS Trust

Introduction

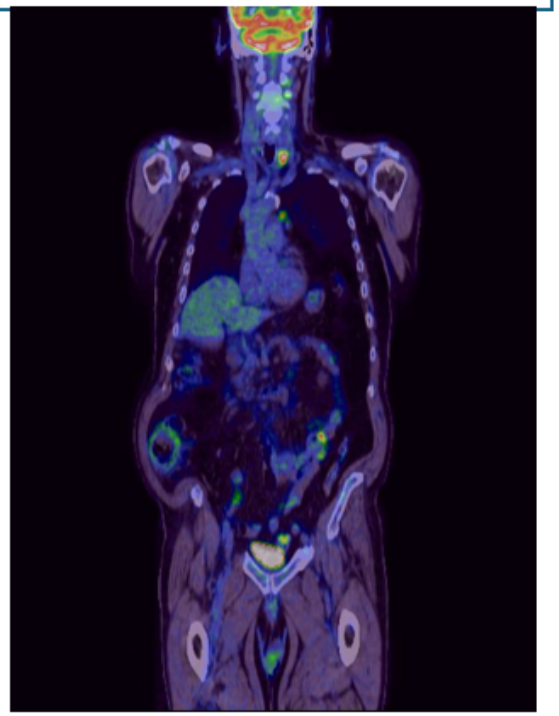
Incidental FDG-avid thyroid nodules on PET-CT scans may represent either metastasis or a synchronous primary malignancy. Accurate distinction is vital, as management and prognosis differ significantly. We present a rare case of concurrent primary lung adenocarcinoma and primary papillary thyroid carcinoma.

Case Report

An 80-year-old man presented with a persistent chest infection, and imaging revealed a left upper lobe mass. Histology confirmed lung adenocarcinoma with a KRAS G12C mutation and high PD-L1 expression. PET-CT showed an additional 2 cm FDG-avid thyroid nodule, which ultrasound and FNA confirmed as papillary thyroid carcinoma with normal thyroid function.



4.2 x 3.1 cm soft tissue mass: T2aN1Mx.



This confirmed left primary lung adenocarcinoma with PET/CT staging of T2bN2M0 and with a focal uptake seen within a part calcified 20mm nodule at the inferior pole of the left thyroid lobe.

Discussion

The patient was managed through a multidisciplinary team involving oncology, endocrinology, radiology, and palliative care. Given the aggressive nature of his lung adenocarcinoma, lung-directed chemoradiotherapy was prioritised, followed by durvalumab maintenance. The thyroid lesion remained stable, and surgery was deferred as his condition declined. This case highlights the importance of recognising synchronous primaries, thorough evaluation of FDG-avid thyroid nodules, and multidisciplinary decision-making to avoid overtreatment in patients with limited prognosis.

Conclusion

The case highlights the need to carefully evaluate incidental PET-CT findings and distinguish between metastasis and synchronous primaries. Management should prioritise the more aggressive malignancy, while indolent tumours may be safely observed. Early multidisciplinary and palliative input ensures treatment remains balanced, appropriate, and patient-centred.

References

1.Albany C et al. *J Thorac Dis.* 2011;3:271–3.
2.Ferra J et al. *Eur J Case Rep Intern Med.* 2021;8.
3.Haugen BR et al. *Thyroid.* 2016;26:1–133.
4.de Leijer JF et al. *Eur J Case Rep Intern Med.* 2021.

Revealing the Intersection: Scleroderma Renal Crisis Complicating Membranous Nephropathy

Turna Zaman, Monowara Rahman Shipi, Rafid Mustafa, Saifuddin Mohammad Kibria, Ashar Uddin Kazi

Introduction

Systemic sclerosis (SSc) is a chronic connective tissue disease that can cause progressive skin and organ fibrosis. A severe renal complication, scleroderma renal crisis (SRC), presents with abrupt hypertension and acute kidney injury, carrying high mortality [1]. Recent reports note increasing cases without skin involvement, making diagnosis difficult in the absence of classic features or autoantibodies [2]. This case highlights the diagnostic and management challenges of atypical SRC and emphasizes the importance of early recognition and intervention.

Case Presentation

An 82-year-old woman with a history of systemic sclerosis (anti-Scl-70 positive) and remote membranous nephropathy presented with a prolonged feeling of unwellness, progressive lower-limb swelling extending to her abdomen, and reduced urine output over several months. She denied urinary or respiratory symptoms. On admission, her blood pressure was 205/111 mmHg, and examination revealed 3+ pitting oedema, sclerodactyly, and dilated nailfold capillaries.

Investigations showed acute kidney injury (creatinine 144 µmol/L, eGFR 29 mL/min), hypoalbuminaemia (22 g/L), and nephrotic-range proteinuria. Imaging demonstrated widespread oedema, pleural and pericardial effusions, and ascites. She was treated with IV diuretics, fluid restriction, and antihypertensives (amlodipine and captopril).

Despite these interventions, her renal function deteriorated, and she developed flash pulmonary oedema, requiring emergency haemodialysis.

A renal biopsy revealed primary membranous nephropathy (PLA2R-positive) with acute vascular injury showing endothelial swelling, fibrinoid necrosis, and “onion-skin” arteriolar changes, consistent with scleroderma renal crisis (SRC). She was continued on dialysis and discharged on enalapril and darbepoetin alfa, though she later missed follow-up.

This case highlights the diagnostic challenge of SRC without new skin changes and the coexistence of dual renal pathology in systemic sclerosis. Early recognition, prompt ACE inhibitor therapy, and a multidisciplinary approach are crucial to improving outcomes and preventing irreversible renal failure.

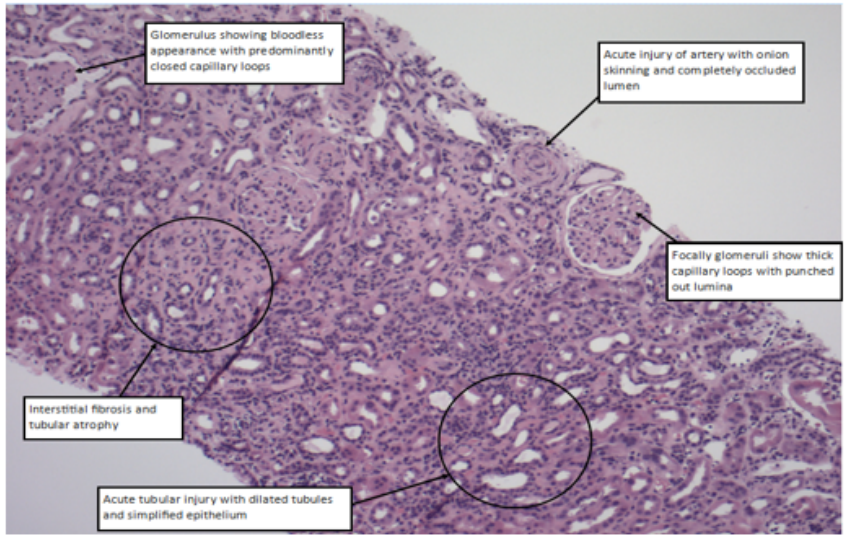


Figure 1: Hematoxylin and eosin-stained section of the renal biopsy

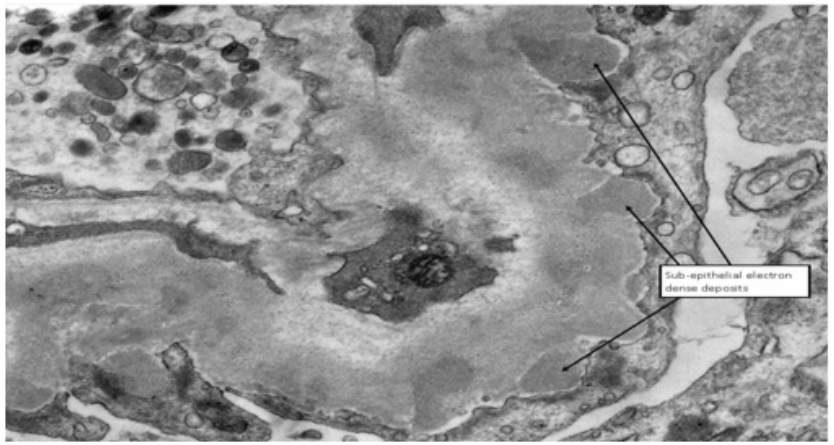


Figure 2: Electron microscopy image of the renal biopsy

Conclusion

This case highlights the challenges of diagnosing scleroderma renal crisis (SRC) in systemic sclerosis (SSc) without typical skin changes. The coexistence of membranous nephropathy complicates management. Early recognition and prompt ACE inhibitor therapy are essential, as delayed intervention can lead to irreversible renal failure and dialysis dependence.

References

1. Shimizu T, Iwamoto N, Okamoto M, et al.: Scleroderma renal crisis complicated with thrombotic microangiopathy triggered by influenza B virus infection. Intern Med. 2019, 58:441-5. 10.2169/internalmedicine.1441-18

2. Hansriviijit P, Omeonu KF, Lawal HO, Gangireddy M, Gadhiya KP, Dhatt RS: A 45-year-old man with scleroderma renal crisis associated with a history of systemic sclerosis sine scleroderma. Am J Case Rep. 2020, 21:e927030. 10.12659/AJCR.927030

A Rare Complication of Type 1 Diabetes: Mauriac Syndrome Presenting with Diabetic Ketoacidosis in a Young Adult

Authors: Dr Zoha Iftikhar [1], Dr Mir Umar Farooq [2]
Affiliations: East Kent Hospital and University Foundation Trust- William Harvey Hospital

NHS
East Kent Hospitals University
NHS Foundation Trust

Introduction

Mauriac syndrome, also known as **glycogenic hepatopathy,** is a rare complication of poorly controlled Type 1 Diabetes Mellitus.

Pathophysiology:

- Fluctuating insulin and glucose levels cause excessive hepatic glycogen deposition, hepatomegaly and metabolic imbalance.

Presentation

A 22-year-old white British female with a history of poorly controlled T1DM and recurrent DKA.

- Admitted to the emergency department with confusion, agitation, hypotension, and a Glasgow Coma Scale (GCS) of 8/15.
- Examination findings included cold, mottled extremities and abdominal tenderness without peritonism.

Investigations

Initial Laboratory Findings:

Revealed a severe metabolic acidosis: pH 6.80, bicarbonate 0.9 mmol/L, base excess -30.6.
DKA confirmed with blood glucose 40.1 mmol/L and ketones 5 mmol/L. Initial lactate was 1.4 mmol/L, CRP 1 mg/L, WBC 24.5
Sepsis was ruled out on clinical exam & biochemistry

Further Biochemistry:

Liver function tests showed **mildly elevated transaminases with preserved synthetic function.**
Autoimmune and chronic liver disease screens were negative. Renal functions normal

Imaging:

A CT scan of the abdomen excluded bowel ischemia but showed gross hepatomegaly with fatty changes.

Management & Clinical Course

Managed in critical care for DKA with insulin, IV fluids, potassium, bicarbonate, and CVVHDF for severe acidemia. Despite correction of glucose, ketones, and pH, lactate paradoxically rose to 11 mmol/L. CVVHDF corrected acidosis but not lactatemia; hypoperfusion causes were excluded.
A variable-rate insulin infusion with glucose was found to worsen lactate levels.

Additional findings

- Transition to basal–bolus insulin achieved euglycemia and partial lactate improvement (5–7 mmol/L).
- Further history revealed **delayed menarche (age 17), short stature (<3rd percentile),** and **truncal obesity**.

These features, in conjunction with the normal clinical and biochemical picture, led to a **diagnosis of Mauriac syndrome.** (*Written informed patient consent obtained).*

Conclusion

- **Mauriac syndrome can present in young adults** with poorly controlled, long-standing T1DM, not just in children
- **Persistent lactate** elevation despite clinical improvement in DKA associated with **hepatomegaly with raised transaminases and short stature** in a Type 1 Diabetic should raise high index of suspicion for Mauriac syndrome.

References

1.*Deemer KS, et al., Case Rep Crit Care. 2016;2016:6072909. doi: 10.1155/2016/6072909. PMID: 27699071; PMCID: PMC5035668.*
2. Fitzpatrick E et al., a tertiary liver centre Archives of Disease in Childhood 2014;99:354-357

A Case Series: Fast, Broad and Irregular Tachycardia – A Treatable Life-Threatening Arrhythmia

Gan, Yi Lung; Win, Kyaw Zaw; Marshall, Howard; de Bono, Joseph; Lencioni, Mauro; Ensam, Bode; Kalla, Manish

Royal College of Physicians

NHS University Hospitals Birmingham NHS Foundation Trust

Introduction:

A broad complex tachycardia is defined by a QRS duration >120 ms on ECG. When rapid and irregular, it typically indicates pre-excited atrial fibrillation (AF) with rapid ventricular response — a life-threatening rhythm requiring immediate cardioversion. [1] We present two young patients with pre-excited AF who underwent successful accessory pathway ablation.

Case Summaries:

Case 1: 28-year-old male with out-of-hospital cardiac arrest. ECG showed broad, irregular tachycardia consistent with pre-excited AF. EPS demonstrated a left-sided accessory pathway successfully ablated at the 4 o'clock position of the mitral annulus.

Case 2: 19-year-old male with palpitations and near-syncope during exercise. ECG revealed short PR and pre-excitation. EPS and electro-anatomical mapping confirmed a left-sided pathway which is successfully ablated.

Both patient had adenosine challenge test post ablation showing AV block without pre-excitation which confirmed pathway elimination.

Case 1 Figures:

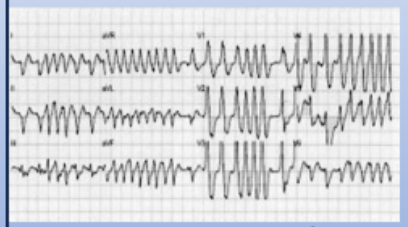


Figure 1A. Presenting ECG showing Pre-excited AF with positive QRS in V1 suggestive of left sided WPW.

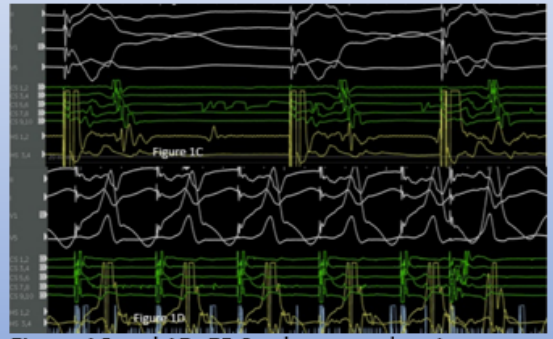


Figure 1C and 1D. EP Study traces showing eccentric CS activation and pre-excitation.

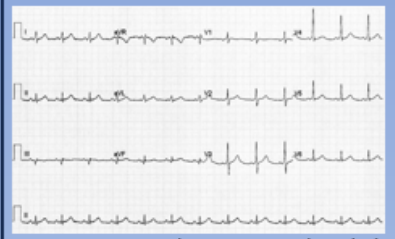


Figure 1B. Baseline ECG with subtle Delta waves.

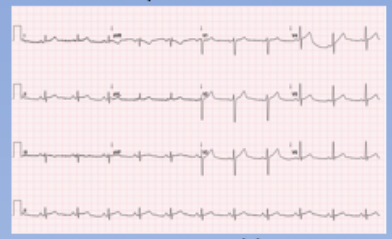


Figure 1E. ECG post ablation.

Case 2 Figures:

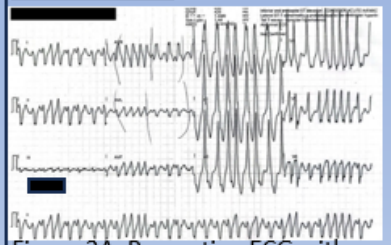


Figure 2A. Presenting ECG with pre-excited AF.

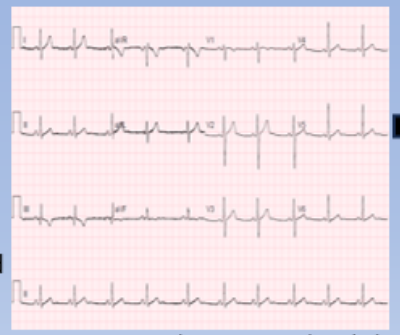


Figure 2B. Baseline ECG with subtle pre-excitation.

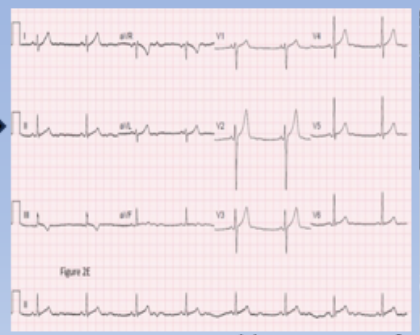


Figure 2E. ECG post ablation. Loss of Pre-excitation (Lead II).

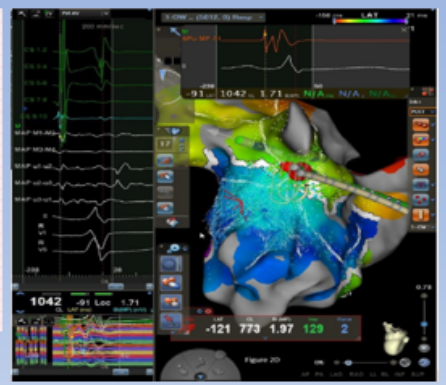


Figure 2D. Electro-anatomical mapping showing left sided accessory pathway.

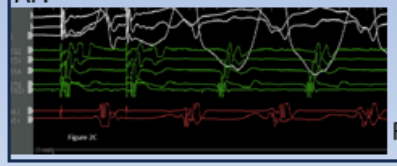


Figure 2C. EP traces showing initiation of antidromic AVRT.

Discussion:

These cases highlight important diagnostic and management considerations in young patients with accessory pathways. Subtle pre-excitation may be missed on surface ECG, particularly with left-sided pathways due to their distant location on the mitral annulus.

This highlights the importance of careful ECG analysis in patients with unexplained palpitations, even when initial investigations appear normal. Pre-excited AF carries a high risk of sudden cardiac death but is highly amenable to catheter ablation, with excellent long- term outcomes. [2]
EPS is therefore essential for both diagnosis and definitive management.

Adenosine challenge testing is an inexpensive and invaluable bedside test in evaluating suspected accessory pathways, as AV nodal blockade may unmask latent pre-excitation.

Following ablation, adenosine testing provides a simple bedside method to confirm pathway elimination by demonstrating AV block without pre-excitation.

Conclusion:

Pre-excited AF is a rare, life-threatening arrhythmia requiring urgent recognition. Catheter ablation offers a definitive cure with excellent outcomes.

References:

1. Richter S, Brugada P. Circulation. 2006; 114: e638-9.
2. Ibrahim Ali Sherdia AF et al. Indian Heart J. 2023; 75(2):98–107.

The B12 Illusion with Whippets Abuse

Dr Saleem **SHEIKH**, Dr Aarthi **SANCARAN**, Dr Ahmar **IFTIKHAR TALIB**,
Dr Paulson **SAMUEL**, Dr Sanjana **SRINIVAS**

NHS
Wirral University Teaching Hospital
NHS Foundation Trust

BACKGROUND: **Nitrous oxide** is a colourless gas which was **initially used as anaesthetic for dental procedures** [1]. It is now easily accessible due it's **sale in the catering industry** as "whippets" - aerosol chargers used in canisters of whipped cream. It is the **second most used drug between 16-24 yr olds** [2]. It is inhaled via balloons and leads to a feeling of **euphoria which can last from seconds to a few minutes**, depending on the individual's rate of metabolism.

CASE PRESENTATION

18-year-old male presenting with a **3-week history** of **progressive bilateral lower limb numbness, weakness, and gait unsteadiness**.

No past medical history, no recent viral infections. No family history of neurological diseases.

No history of vegetarianism or risky sexual behaviours. Later disclosed that he used **approximately six whippet canisters weekly for six weeks**.

EXAMINATION

Neurological examination revealed **proximal (4/5) and distal (3/5)** MRC power scale **weakness**, **glove-and-stocking sensory loss** (right>left), and **absent lower limb ankle and knee reflexes**.

INVESTIGATIONS

Normal serum B12 and **folate** but **hyper-homocysteinaemia** (145 µmol/L). **MRI spine ruled out structural lesions** but was limited by motion artifact.

PATHOPHYSIOLOGY

Nitrous oxide causes **inactivation of B12**, leading to **disruption in the homocysteine metabolism** and the maintenance of the myelin sheaths. This in turn leads to oxidative stress, endothelial damage and **subacute degeneration of the spinal cord**. It classically affects the **corticospinal tract** and **dorsal column** due to their high myelin demands.

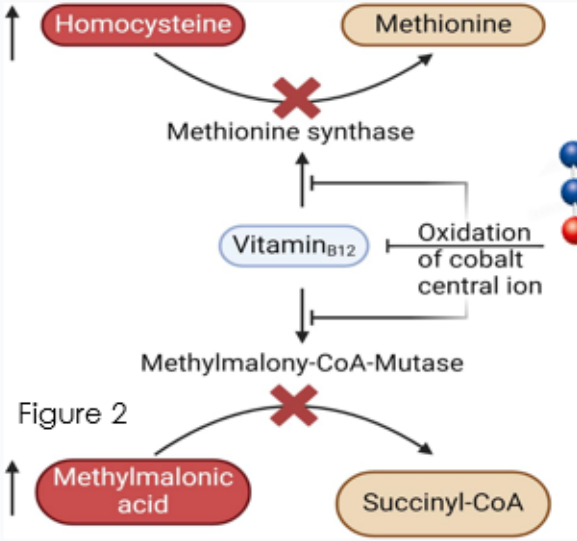


Figure 2

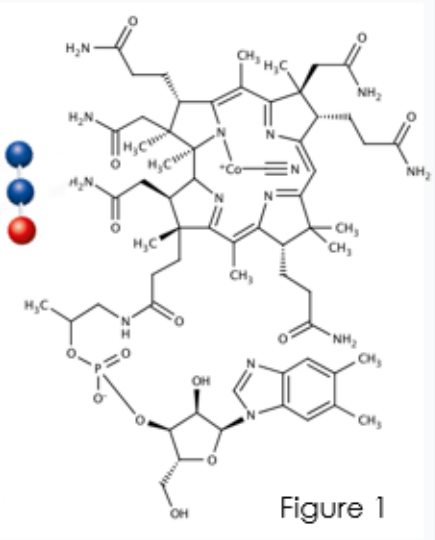


Figure 1

REFERENCES

https://www.ncbi.nlm.nih.gov/books/NBK532922/
https://www.ons.gov.uk/peoplepopulationandcommunity/crimeandjustice/articles/drugmisuseinenglandandwales/yearendingmarch2020/pdf
https://pn.bmj.com/content/23/3/222#F1

TREATMENT

MEDICAL: **Immediate intramuscular vitamin B12** (1 mg on alternative days) helps to **bypass nitrous oxide–inactivated enzymes** and **folate supplementation supports homocysteine reduction**. Treat for 2 weeks and then assess neurological response. Continue until symptoms resolve. Can treat up to 8 weeks [3].
REHABILITATION: Ongoing intensive physiotherapy and rehab targets gait instability and motor weakness.
OUTCOME: Under Neurology follow-up at Walton CNN; homocysteine levels have normalized.
DIFFERENTIALS: **GBS**, **transverse myelitis**, **SCD from true B12 deficiency**, or **HIV associated vacuolar myelopathy**.

CONCLUSIONS

- Acute physicians should remember chronic use of N2O causes a **neuro-psychiatric presentation** and a normal B12 level does not exclude it as the underlying pathology is that of a **functional B12 deficiency**, **homocysteine and methylmalonic acid levels should be assessed early on**.
- **Early SCD may lack MRI abnormalities**, emphasizing the role of examination and biochemistry.
- **Prompt treatment helps reverse symptoms**.

The authors declare that they have no conflict of interest.

May-Thurner Syndrome in a Young Woman with Newly Diagnosed Ulcerative Colitis: A Contributing Factor in Extensive Deep Vein Thrombosis

Dr Meha Sanghi[1] , Dr Amr Youssef [1] , Dr Leah Ifeoluwa Ajayi[1] , Dr Qasim Muhammad[1]

[1] Manchester University NHS Foundation Trust, Manchester, United Kingdom

Introduction:

Venous thromboembolism (VTE) is a major cause of morbidity and mortality. Patients with inflammatory bowel disease (IBD) have a 2–3× higher risk of VTE due to systemic inflammation, platelet activation, and endothelial dysfunction.[1,2] May-Thurner Syndrome (MTS) is an underdiagnosed anatomical compression of the left common iliac vein by the right common iliac artery, predisposing to iliofemoral deep vein thrombosis (DVT).[3] Coexistence of MTS and active IBD creates a compounded thrombotic risk.

Case Summary:

A woman in her mid-20s presented with painful left leg swelling. Ultrasound confirmed extensive iliofemoral DVT. CT venography revealed features consistent with MTS (Figure 1). Flexible sigmoidoscopy demonstrated severe mucosal inflammation with histological confirmation of ulcerative colitis (UC) (Figure 2). The extensive DVT and identification of MTS prompted early vascular input. Thrombolysis was discussed to reduce swelling and preserve venous function, but given haemodynamic stability, absence of phlegmasia, and patient preference, she was commenced on rivaroxaban, systemic steroids & mesalazine.

Discussion:

This case highlights the synergistic interaction of systemic (IBD) and anatomical (MTS) risk factors in precipitating severe DVT. While IBD alone increases thromboembolic risk, concomitant MTS may accelerate propagation, explaining the left-sided distribution and extensive iliofemoral involvement, where clot burden and risk of post-thrombotic syndrome are greater. [4] Few reports describe concurrent UC and MTS, underscoring the rarity and clinical importance of this presentation. [5,6]

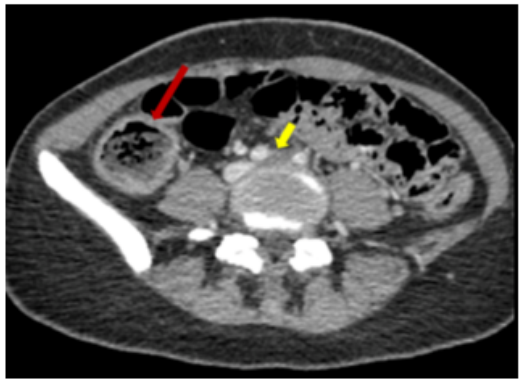


Figure 1: CT venogram showing compression of the left common iliac vein (yellow arrow – MTS) and bowel wall thickening (red arrow).

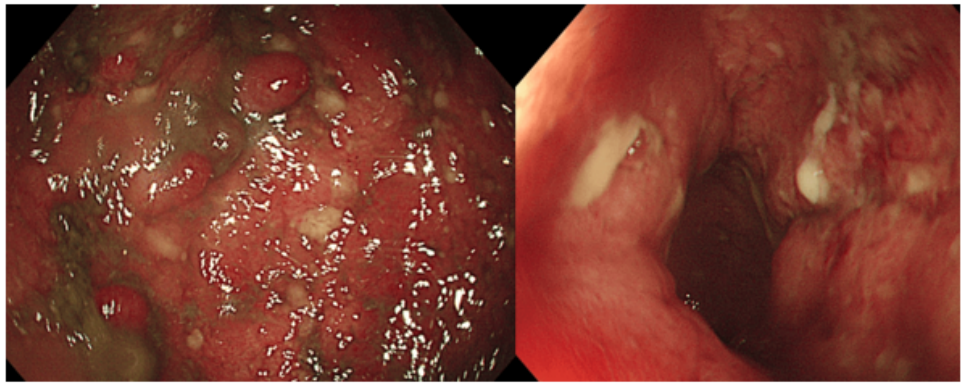


Figure 2: Endoscopy showing severe continuous ulceration and friability consistent with severe UC

Conclusion:

- Coexistence of UC and MTS compounded the risk for extensive iliofemoral DVT
- Consider potential synergy between systemic inflammatory conditions and venous anomalies in atypical or left-sided DVT
- Early multidisciplinary involvement & comprehensive workup are vital to optimise outcomes

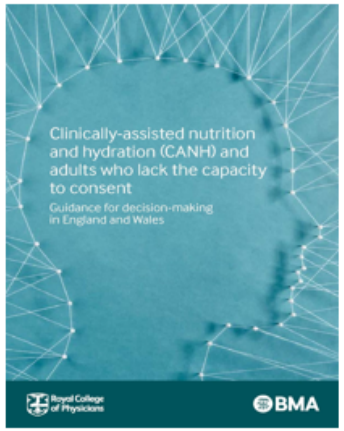
1. Grainge MJ et al. Lancet. 2010;375:657–63. 2. Nguyen GC, Sam J. Am J Gastroenterol. 2008;103:2272–80. 3. O'Sullivan GJ et al. J Vasc Interv Radiol. 2000;11:823–36. 4. Cockett FB, Thomas ML. Br J Surg. 1965;52:816–21. 5. Hamada K et al. Clin J Gastroenterol. 2022;15:971–6. 6. Kalu SU et al. Vasc Endovasc Surg. 2013;47:301–7.

Successful withdrawal of Clinically-assisted Nutrition and Hydration (CANH) for a patient in a persistent vegetative state at a District General Hospital in the UK. Reflection on the UK law, 2024 BMA/RCP guidelines and the Islamic law.

Divakar Prabhu, Sugna[1], IMT2, Wexham Park Hospital; Menzies, Sarah[2], Respiratory Consultant, Wexham Park Hospital

Background

The withdrawal of CANH is a topic that has been reviewed by the UK legal system several times. Starting in 1993, it was established that CANH is a treatment and not routine care[1]. Since then, there have been several notable cases defining when this law can be applied and who fits the criteria[2]. In 2018, the Supreme Court concluded that there was no legal requirement for cases to be brought to the court provided there was agreement upon what was in the patient's best interests. The BMA/RCP CANH guidelines were initially written in 2018, with a recent update in 2024[3].



Case history

Mrs. K, a fit 60-year-old woman with no comorbidities.

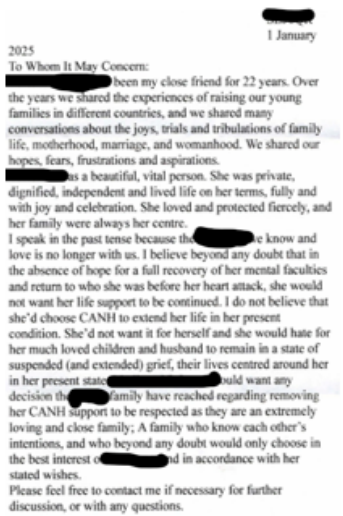
- She was visiting Qatar in early November 2024 where she had an out-of-hospital cardiac arrest with immediate cardiopulmonary resuscitation (downtime 17 minutes).
- She had an anterior STEMI on arrival at the hospital and underwent PCI. She remained intubated until mid-November; after which a Tracheostomy was performed. Brain imaging demonstrated a hypoxic ischaemic encephalopathy.
- Her family requested withdrawal of care, citing her wishes to avoid living with a disability. The hospital consulted its Ethical and Legal departments and concluded that they could not withdraw nutrition without an explicit directive from the patient. She had not made a living will previously.
- She was repatriated to her local hospital in the UK after 8 weeks. She remained in a vegetative state with a GCS of 8/15 (E4,V1,M3) throughout, with no acute concerns from a cardiology standpoint.
- Blood investigations were unremarkable. Her repeat imaging remained unchanged, and an EEG showed no defined electrocortical activity.
- In accordance with her family's statements of her likely intentions, her consultant followed the BMA/RCP 2024 CANH guideline, obtaining opinions from specialists in neurology, neurorehabilitation, palliative care and the Trust's legal team, and secured letters of support from family and friends. Nutrition was withdrawn 8 days after repatriation, with palliative care involvement. She passed away peacefully after 3 weeks.

Discussion

Our patient was in an Islamic country when she went into a Prolonged Disorder of Consciousness. In Islamic law, nutritional support is generally considered basic care and not medical treatment[4]. As she had not previously documented her wish to be allowed to die in the event of her suffering an event leading to significant disability, the Ethical and Legal departments in Qatar were unable to sanction the removal of her nutrition and hydration.

When she returned to the UK, we could apply the BMA/RCP CANH guidelines as she met the criteria. It was a challenging process emotionally and practically, and this had not been done previously in our hospital as far as we were aware. We had to work through the clinical steps and ensured we sought the opinions of all of her close family and friends and had evidence of this, at a very difficult time.

Overall, our experience of the process was positive, and we hope that the UK CANH guidelines benefit other clinicians, patients, and their families in the future.



[redacted]

1 January 2025

To Whom It May Concern:

[redacted] been my close friend for 22 years. Over the years we shared the experiences of raising our young families in different countries, and we shared many conversations about the joys, trials and tribulations of family life, motherhood, marriage, and womanhood. We shared our hopes, fears, frustrations and aspirations.

[redacted] as a beautiful, vital person. She was private, dignified, independent and lived life on her terms, fully and with joy and celebration. She loved and protected fiercely, and her family were always her centre.

I speak in the past tense because th[redacted]e know and love is no longer with us. I believe beyond any doubt that in the absence of hope for a full recovery of her mental faculties and return to who she was before her heart attack, she would not want her life support to be continued. I do not believe that she'd choose CANH to extend her life in her present condition. She'd not want it for herself and she would hate for her much loved children and husband to remain in a state of suspended (and extended) grief, their lives centred around her in her present state[redacted]uld want any decision th[redacted]family have reached regarding removing her CANH support to be respected as they are an extremely loving and close family; A family who know each other's intentions, and who beyond any doubt would only choose in the best interest o[redacted]d in accordance with her stated wishes.

Please feel free to contact me if necessary for further discussion, or with any questions.

Figure 1 –letter of support

References

1. Great Britain. House of Lords. Airedale NHS Trust v. Bland. All Engl Law Rep. 1993 Feb 4;[1993]1:821-96. PMID: 11648606.
2. McHale J. A right to die or a right to live? Discontinuing medical treatment. Br J Nurs. 2011 Nov 10-23;20(20):1308-9. doi: 10.12968/bjon.2011.20.20.1308. PMID: 22068006.
3. Clinically assisted nutrition and hydration toolkit [Internet]. The British Medical Association is the trade union and professional body for doctors in the UK. Available from: https://www.bma.org.uk/advice-and-support/ethics/adults-who-lack-capacity/clinicallyassisted-nutrition-and-hydration
4. Alsolamy S. Islamic views on artificial nutrition and hydration in terminally ill patients. Bioethics. 2014 Feb;28(2):96-9. doi: 10.1111/j.1467-8519.2012.01996.x. Epub 2012 Jul 29. PMID: 22845721.
5. Szawarski, Piotr & Kakar, Vivek. (2012). Classic Cases Revisited: Anthony Bland and Withdrawal of Artificial Nutrition and Hydration in the UK. Journal of the Intensive Care Society. 13. 126-129. 10.1177/175114371201300209

A Rare Intersection of Systemic and Ocular Disease: Purtscher's Retinopathy Secondary to Alcoholic Pancreatitis with Spontaneous Recovery

Jessica Holmes, *Diana Princess of Wales Hospital Grimsby* jessica.holmes21@nhs.net

NHS Humber Health Partnership

References

Background

Purtscher's retinopathy is an **occlusive microangiopathy causing retinal ischemia and haemorrhages** [1]. It presents as sudden painless loss of vision unilaterally or bilaterally and is rare (0.24 cases per million) [2].

Case report

Initial presentation

- 50s male presented with vomiting and abdominal pain for 3/7 and **sudden bilateral blurred vision**
- Past medical history:

Alcohol excess (500-1000ml vodka/day), controlled asthma, no previous visual problems or prescription

- Assessment:

Severe generalised tenderness on abdominal palpation. Witnessed generalised tonic-clonic seizure which self-terminated without head injury

Initial Investigations

Test	Result
CRP	238mg/L
Amylase	387u/l
LFTs	Isolated rise in GGT
CT abdomen	Pancreatic fat stranding

Fig 1. Table showing significant initial investigations and results

Initial Management

- He was treated for acute pancreatitis and alcohol withdrawal
- Pancreatitis managed with IV 0.9%NaCl and IV co-amoxiclav for 5 days
- Pain managed with IV paracetamol and oral liquid morphine
- Alcohol withdrawal managed with a reducing regime of chlordiazepoxide

Ophthalmology Assessment

Despite treatment of pancreatitis and alcohol withdrawal he continued to **experience bilateral visual blurring, dark spots on central vision, impaired light and colour differentiation.**

On examination he had significantly reduced visual acuity bilaterally (Fig 2) which improved over time. He was referred for ophthalmology examination (fundoscopy, fluorescein angiography and OCT)

	VA Rt eye	VA Lt eye
Presentation	6/60 (6/48)	Finger counting
At 1 week	6/24 (6/9.5)	6/60 (6/19)
At 12 weeks	6/4.8	6/6
At 9 months	6/4.8	6/4.8

Fig 2: visual acuity over time (VA) ()=pinhole correction

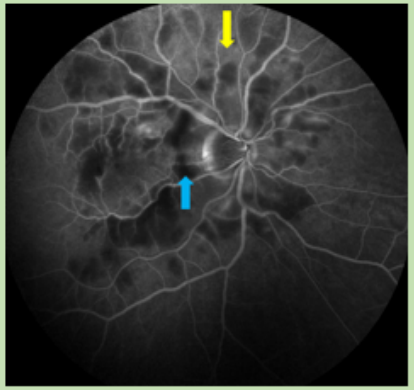


Fig 3. FA of Rt eye in the late venous phase. Yellow: hyper-fluorescence likely arteriolar leakage, Blue: masking of choroidal fluorescence likely ischemia

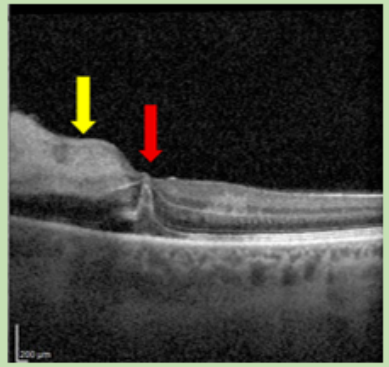


Fig 4. OCT Lt eye. Red arrow: Fovea obliteration. Yellow arrow: papillo-macular bundle swelling with hyper-reflective lesion likely cotton wool spot

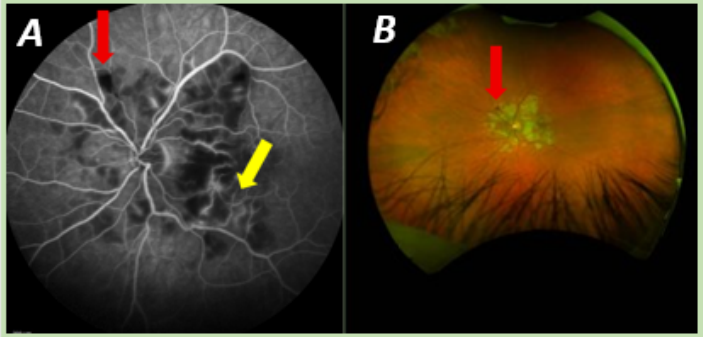


Fig 5: A: Lt eye FA, late venous phase. Red: hypofluorescence representing blot haemorrhage. Yellow: arteriolar vessel leakage. Background choroidal vessel drop out likely ischaemia. B: Lt eye Optos on initial assessment. Red: corresponding blot haemorrhage

Summary of findings

Fundoscopy: patchy whitish areas surrounding discs with blot haemorrhages
Optical Coherence Tomography (OCT): retinal oedema bilaterally
Fluorescein Angiography/Optos: bilateral macular oedema, cotton wool spots (CWS)

Diagnosis: Purtscher's Retinopathy

Management and follow up

- Patient counselled on **unclear prognosis**
- **Conservative management** due to unclear benefit of steroids/anti-VEGF and lack of high-quality data
- Subjective experience was that vision had **improved gradually**, left eye remains worse than right. He still suffers with cloudy spots. Overall, he **reports negligible affect on quality of life**
- His visual acuity has returned to above average baseline by 9 months, but he has a significant nasal steppe on formal visual fields assessment bilaterally

Purtscher's Retinopathy

- Rare and poorly reported (2024 review found only 114 cases) [3]
- Diagnosis: 3/5 of Purtscher's flecken (polygonal retinal whitening), Retinal haemorrhages, Cotton wool spots, Probable aetiology, Complementary investigations (leaky vessels, oedema) [4,5]
- Classically related to trauma (RTAs, CPR, Valsalva manoeuvre) or systemic diseases (Purtscher's-like retinopathy) [6] (pancreatitis, SLE, DIC, sepsis)

Pathogenesis:

- Occlusion of terminal arterioles causing infarct of the capillary bed caused by emboli such as cholesterol, fibrin, leukocyte aggregates and fat [1].

Prognosis:

- No clear prognosis or prognostic indicators identified
- No significant difference in outcomes from cases managed with steroids vs without [7]

Lessons

This case adds to the limited literature by documenting **significant spontaneous recovery without intervention.** Consideration of this rare differential with early referral to ophthalmology should be considered in patients with acute pancreatitis who report visual loss.

A Saddle Pulmonary Embolus with Subsequent Embolic Stroke from an Intracardiac Thrombus In-transit

Andrew Morrish[1], Fadwa Al-Qadi[1], Kanwal Tariq[2], Claudette Phillips[3] 1,2: Respiratory Medicine, 3: Radiology; Hull University Teaching Hospitals NHS trust, Hull, HU3 2JZ

NHS Humber Health Partnership

Introduction

Saddle pulmonary embolism (SPE) refers to a thrombus located at the bifurcation of the main pulmonary artery, and is often associated with hemodynamic instability, increased complication rates, and poorer outcomes if not managed actively [1] . Intracardiac thrombi (ICT), particularly within the left atrium (LA), left atrial appendage, or left ventricle, significantly increase the risk of systemic thromboembolism, including ischemic stroke and acute ischemia involving abdominal organs or lower limbs [2]. While echocardiography remains the most used modality for diagnosing ICT, CTPA is also valuable, particularly in certain clinical contexts.

Case Presentation

Initial Presentation, Investigations and Management

Middle-aged obese male, reduced mobility post right foot debulking surgery presents with hypoxic respiratory failure

CTPA: Extensive submassive PE + right heart strain

US: No DVT (right leg cannot be scanned due to cast)

Started on weight-adjusted therapeutic dose LMWH

Clinical Course

At presentation, required high flow nasal cannula oxygen

Weaned successfully to room air over next 24 hours.

Acute Neurological Episode

Sudden, transient episode of right homonymous inferior quadrantanopia and expressive dysphasia, lasting approximately 15 minutes. Neurological examination was unremarkable following resolution of symptoms

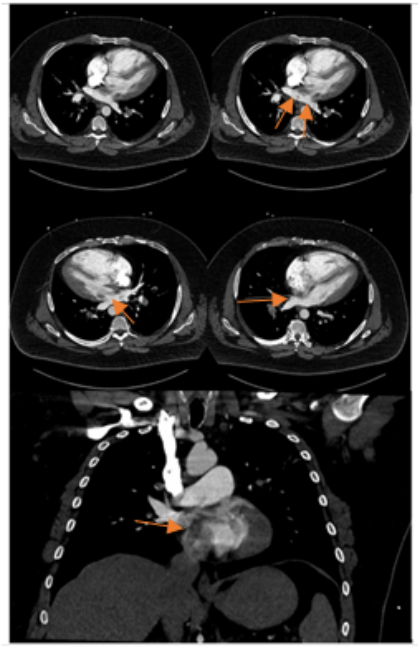


Fig 1. CTPA Images capture intracardiac thrombus in transit early in patients' admission.

Neuroimaging

MRI Brain: Multiple acute supratentorial infarcts, consistent with proximal embolic source

Cardiac Assessment

TTE inconclusive

TOE deferred (risk of interruption to anticoagulation during acute SPE)

Re-evaluation of Initial CTPA

Demonstrates a filling defect within the right atrium adjacent to the interatrial septum (IAS), extending across the IAS into the LA. These findings suggest an ICT in transit (ICTIT), straddling the IAS

Ongoing Management

Continued therapeutic LMWH, switched to DOAC. Significant clinical improvement. Discharged

Further Evaluation

Repeat TTE (with bubble study): inconclusive

TOE: scheduled to assess for IAS defect with plan for closure

Three-Month Follow-up

Repeat CTPA: Complete resolution of thrombus

Bilateral US leg: persistent (or new) right leg DVT. Switched to warfarin

Discussion

PE with right heart thrombi have higher mortality than PE alone (21% vs 11%) [3], with right-sided thrombi seen in up to 18% of massive PE [4]. Bi-atrial thrombi are rare, with 30-day mortality around 18% [5]. Earlier recognition of ICTIT on imaging may have altered management.

No consensus exists for treating PE with ICTIT. Options include thrombolysis, anticoagulation, or embolectomy. Meta-analyses show thrombolysis improves survival versus other approaches [6,7], though surgery offers lowest mortality when thrombi cross a PFO (10.8%) [5]. In our case, ICTIT was found post-stroke, making thrombolysis contraindicated. No RCTs confirm LMWH or DOAC efficacy in this setting. Case reports of similar presentations describe mixed outcomes: PE with ICTIT treated in the first instance with fibrinolysis [8] and LMWH converted to DOAC [9] were also followed by embolic strokes; PE with ICTIT and embolic stroke on presentation reacted well to LMWH-to-warfarin therapy, with complete resolution of ICTIT straddling PFO [10].

These reports highlight the need to routinely identify ICTIT on CTPA and refine treatment strategies.

Echocardiography remains key for detecting intracardiac thrombi.

TOE is recommended when TTE is inconclusive [11], though image quality can limit accuracy. In this case, TOE was contraindicated, underscoring the value of detailed CTPA review when echo findings are uncertain.

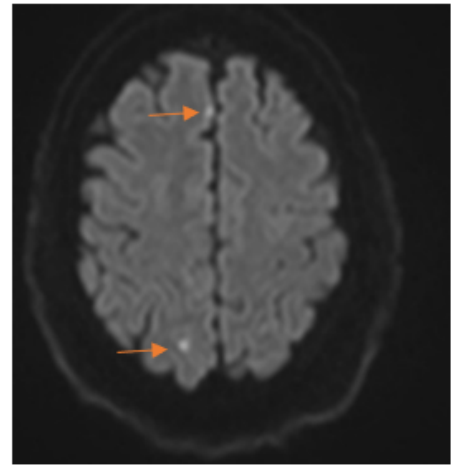


Fig 2. MRI showing multiple regions of restricted diffusion in different vascular territories. Suggestive of embolic infarcts

Conclusion

This case of high-burden PE underscores the crucial need for thorough evaluation of CTPA images with attention to the presence of ICTIT. Early identification of this could prompt more aggressive treatment. Reassessing imaging is particularly valuable in patients who develop neurological symptoms or suffer a cerebrovascular event, especially when echocardiographic results are inconclusive. Complex cases like this benefit greatly from a multidisciplinary team approach to ensure accurate diagnosis and optimal management.

References

Transverse Myelitis Revealing Relapsing-Remitting Multiple Sclerosis in a Patient with Crohn's Disease on Anti-TNF Therapy: A Rare but High-Impact Clinical Intersection

Dr. Sara Gawargeous

NHS Gloucestershire Hospitals NHS Foundation Trust

INTRODUCTION

Multiple sclerosis (MS) and Crohn's disease (CD) are both immune-mediated but rarely occur together (1). Anti-tumour necrosis factor (anti-TNF) drugs are widely used for CD but have been linked to demyelinating events (2-4), with an estimated incidence of less than 1 in 1,000 patients (5). These neurological complications can develop at any stage during therapy and may mimic other acute neurological conditions, delaying recognition. We present a case of transverse myelitis as the first presentation of relapsing-remitting MS in a patient on adalimumab for CD, highlighting the importance of vigilance for neurological symptoms in IBD patients.

MATERIALS AND METHODS

Clinical examination, neuroimaging, cerebrospinal fluid (CSF) analysis, and multidisciplinary review were performed. MRI was conducted using T2-weighted and FLAIR sequences, with both brain and full spinal imaging obtained to assess dissemination in space. Laboratory testing included CSF oligoclonal band analysis. Drug history, including timing of anti-TNF therapy initiation, was reviewed in detail to explore temporal relationships with symptom onset.

RESULTS AND DISCUSSION

A 40-year-old woman with a 7-year history of CD on azathioprine and adalimumab presented with acute right leg weakness and difficulty walking. Examination revealed pyramidal weakness, hyperreflexia, and extensor plantar response, raising suspicion for an upper motor neuron lesion. MRI revealed multiple cervical cord T2 hyperintense lesions and a new supratentorial periventricular white matter lesion, both typical of demyelinating disease. CSF analysis showed oligoclonal bands, confirming relapsing-remitting MS. Adalimumab was stopped and high-dose intravenous methylprednisolone given, resulting in partial recovery and improvement in gait.

This case highlights the diagnostic challenge of isolated spinal cord relapses, especially in patients with complex autoimmune backgrounds. The temporal association between anti-TNF use and demyelination supports a possible drug-triggered MS onset (3,6-8). Similar cases in the literature emphasise the need for early MRI in any IBD patient with new focal neurological deficits. Clinicians should be alert to neurological symptoms in IBD patients on anti-TNF therapy, as early diagnosis may prevent irreversible disability (9).

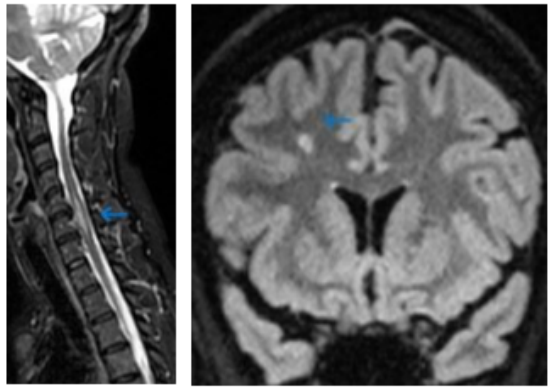


Figure 1. MRI demonstrating dissemination in space. (A) Sagittal T2-weighted cervical spine MRI showing hyperintense lesion at C2–C4 (arrow). (B) Coronal FLAIR brain MRI showing periventricular white matter lesion (arrow).

CONCLUSION

Prompt recognition of demyelination in IBD patients on anti-TNFs can guide treatment changes, avoid further neurological injury, and reduce long-term morbidity. This case adds to evidence linking anti-TNF therapy with demyelinating disease (2-4,6-9) and reinforces the need for multidisciplinary collaboration between gastroenterologists and neurologists in such scenarios.

REFERENCES& ACKNOWLEDGEMENTS

I would like to sincerely thank Dr. Taha Elsahy, Consultant in Acute Medicine at Peterborough Hospital, and the Neurology Department for their guidance, support, and valuable insights in the management of this case.

"Scan to access full references"

From Euphoria to ICU - Unraveling a Young Adult's Collapse

NHS Wirral University Teaching Hospital NHS Foundation Trust

CASE STUDY:

A 20-year-old male normally fit and well was sent to UMAC by the GP due to hyperkalemia, severe abdominal pain, dehydration, and malnutrition. He had metabolic acidosis, acute renal failure and had not opened his bowels for 8 days; guarding was noted, without peritonism.

Social History: Chronic intranasal ketamine use (>12 months, ≥8g/week); no significant sexual history.The combination of renal and hepatic abnormalities, along with chronic ketamine use, was the key in diagnosis.

Investigations: Key lab findings: Hyperkalemia 8.3 mmol/L, Hyponatremia 105 mmol/L, Urea 48 mmol/L, Creatinine 434 µmol/L, Bicarbonate 15 mmol/L, pH 7.25, Lactate 2.2. LFTs Markedly elevated Transaminases with normal Bilirubin.

CT AP: Bilateral hydroureteronephrosis, contracted bladder with no anatomical obstruction. Diffuse intrahepatic biliary ductal dilatation without obstructive cause.

Management:

ICU admission with bilateral nephrostomies and slow correction of electrolyte abnormalities resulting in significant improvement and patient's recovery.

Diagnosis:

Ketamine-induced uropathy, cystopathy, and cholangiopathy.

Non-obstructive uropathy due to chronic urothelial inflammation, reduced bladder compliance and functional outflow resistance. Intrahepatic biliary dilatation with normal bilirubin suggests ketamine-related bile duct toxicity.

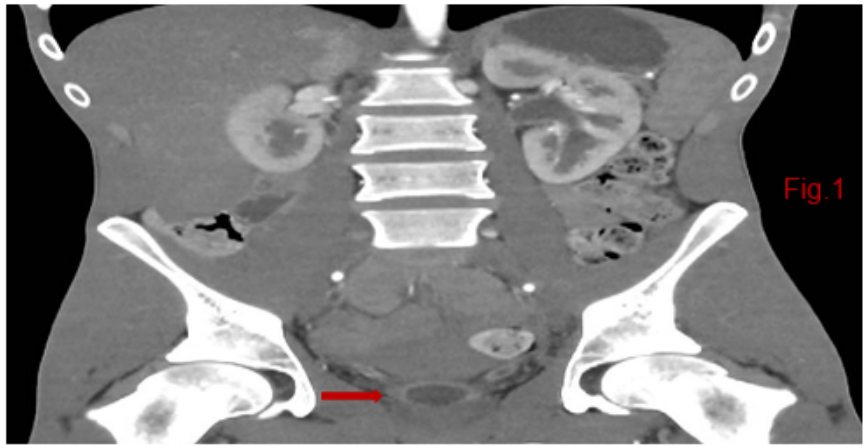


Fig.1

Discussion: Ketamine, an NMDA receptor antagonist, is commonly used in anesthesia and pain relief. Its hallucinogenic and dissociative effects have fueled recreational use especially in young adults. This case highlights irreversible bladder fibrosis and hepatobiliary injury from chronic use, figure 2 illustrates a possible mechanism of pathophysiology but little is currently understood. Clinicians should be alert to these atypical, multisystem effects. Non-obstructive uropathy and cholangiopathy may mimic surgical emergencies but reflect drug toxicity. Early recognition and cessation are key to preventing lasting damage.

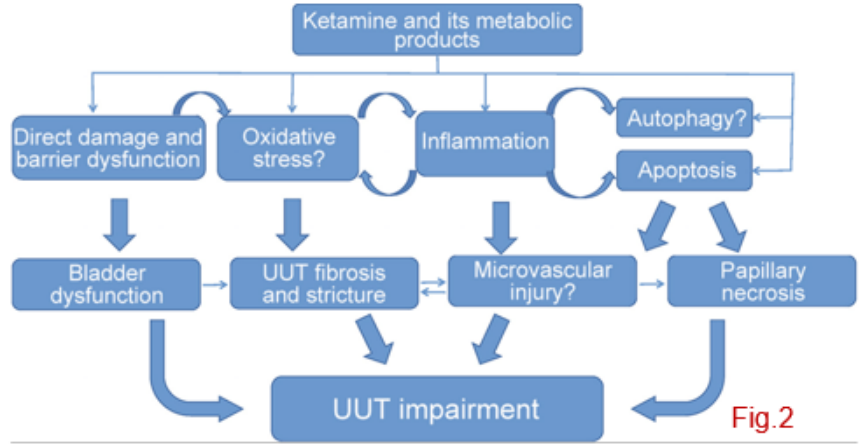


Fig.2

Differentials:

Obstructive uropathy (e.g. prostatic, urethral stricture), Neurogenic bladder (spinal pathology, diabetes), Interstitial cystitis / radiation cystitis, Drug-induced cystitis (cyclophosphamide)

Clinical Pearl: In young patients with unexplained AKI or hyperkalemia, think ketamine cystitis — a silent cause of obstructive uropathy**. Check the bladder, stop the ketamine, save the kidneys.**

Authors

Dr. A. Gilmore – Consultant in Acute Medicine

Dr. A.Sancaran , Dr. A.Noble, Dr. D.Mcdaid, Dr. D.Mcgrath

References:

1.National Institute for Health and Care Excellence (NICE), Ketamine. British National Formulary (BNF).

2.Fenton, C. and Yousef, S. (2023) KetamineToxicity. StatPearls. Treasure Island (FL): StatPearls Publishing =

No conflict of interest

When Minds and Bodies Collide: Managing Complex Physical Illness in a Psychiatric Inpatient

Lead Author: Christina Regi F2 (County Durham and Darlington Foundation Trust). Co Authors: Adarsh Iyer SHO, Mathew Kunnel Jomon GPST1

1. INTRODUCTION

Individuals with mental illness exhibit a higher prevalence of physical comorbidities, with multi-morbidity expected to increase [1].
Managing physical health problems whilst maintaining psychiatric care has proven to be challenging.

Case Relevance:

- underscores the importance of integrating physical and psychiatric care
- addresses the clinical challenges involved
- proposes strategies to enhance collaboration between Medicine and Psychiatry to improve patient outcomes.

2. CASE BACKGROUND

Young male with **complex autism** and a **pathological demand avoidance** behavioural profile.
Childhood history: Ventricular septal defect (VSD) and tricuspid regurgitation.
Pre-admission: Long-term inpatient at an **acute male psychiatric ward**
Current admission: Presented with a 4-week history of **shoulder pain**, attributed to weightlifting.
Clinical findings: Features suggestive of **injury/ haematoma**, but **elevated infection markers** raises a concern for **infection**.
⚠️ Management complicated by **episodes of non-compliance** and **repeated absconding.**

3. CASE CONTENT

Day 0	🩸 Bloods: Raised CRP and WCC. 🦴 Left shoulder X-ray: Subluxation noted. 🧲 MRI: Haematoma identified, possible superimposed infection. 🧫 Blood cultures: Grew *Streptococcus anginosus*. ❤️ Echocardiogram: VSD with left-to-right shunt, otherwise normal. 🌡️ Clinical course: Developed fevers →commenced on antibiotics.
Day 2	**Arthroscopic washout** performed (bursa); rotator cuff intact. suspected infected haematoma but synovial fluid culture negative → alternative source sought.
Day 4	**CT TAP:** Multiple bilateral pulmonary septic emboli (no large PE on CTPA). Given background of congenital cardiac abnormality → concern for infective endocarditis (IE).
Day 11	**TTE/ TOE** (transthoracic/ transoesophageal echocardiogram): No evidence of vegetation. After IE MDM + Adult Congenital Heart Disease (ACHD) review → treated as **presumed infective endocarditis**. [Duke's Criteria]

4. CHALLENGES/ ADAPTATIONS

Clinical / Diagnostic	Patient Non-compliant with TOE procedure → • Patient returned to Psychiatry hospital in evenings; came back for day reviews & blood tests. • Admitted night before procedure; NBM maintained.
Medication	No clear medication list; missed doses meant poor continuity of Psychiatric care. Difficulty in rationalising antimicrobial therapy (patient reluctance, vascular access, out-of-hours dosing) → • IV ceftriaxone 2g OD (instead of TDS) • Single-dose IV dalbavancin 1500mg gave 14 days coverage.
Patient & Staff Safety	Patient threatening self-harm, violence & absconding; medical staff had not received restraint training → assistance was provided by two trained carers from the psychiatric unit.
Psychosocial	Distress for staff and family → maintained frequent communication between mental health team and medical team.

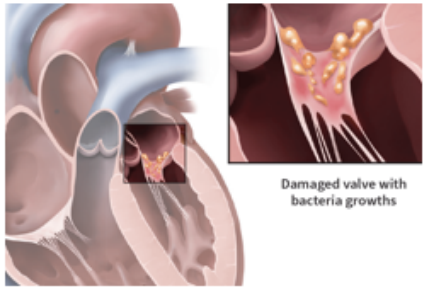


Image: Bacterial vegetations on a damaged heart valve in infective endocarditis.
Source: University of Heart Ottawa Heart Institute

5. OUTCOME

Coordinated care: Orthopaedics, Microbiology, OPAT, Psychiatry, Same Day Emergency Care, Cardiology & ACHD. Completed **4 weeks parenteral antibiotics.**

Organism, Streptococcus, was highly sensitive. Good clinical & biochemical improvement.
Orthopantomography (OPG) XR excluded oral source

6. LEARNING POINTS

- Collaborative medical + psychiatric care teams
- Cross-disciplinary training for staff
- Standardized shared-care protocols
- Shared electronic records & structured communication
- Regular multidisciplinary case

References

1. Pizzol D, et al. Relationship between severe mental illness and physical multimorbidity: a meta-analysis and call for action. BMJ Ment Health. 2023;26(1):e300870

The Hot Gallbladder: A Rare Cause Of Acalculous Cholecystitis In A Returning Traveller

Sorby, Serisha; Stoddart, Miranda; Daes, Gavin; Maddox, Nicola

Southmead Hospital, Southmead Road, Bristol, BS10 5NB

Background

Fever and diarrhoea in returning travellers is a common presentation with many differentials. It requires a thorough travel history alongside appropriate and targeted microbiological testing to ensure correct diagnosis and antimicrobial management.

Case Presentation

A man in his twenties presents with a 1-month history of fevers, diarrhoea, vomiting, upper abdominal pain and a headache.

- Returned 1 week before presentation after a month of travelling to Bangkok and Chiang Mai (*Thailand*), Bali (*Indonesia*) and Barcelona (*Spain*)
- Engaged in a range of activities whilst travelling including fresh water swimming, jungle tours and eating food in local restaurants and from food trucks
- Multiple mosquito bites whilst in South East Asia
- Symptoms first started after eating street food in Indonesia

- No past medical history
- No malarial prophylaxis taken.
- Typhoid vaccination 3 years prior to travel.

- On presentation, he was tachycardic and febrile (38.9°C). Observations were otherwise normal.
- Examination findings were notable for tenderness in the right upper quadrant.

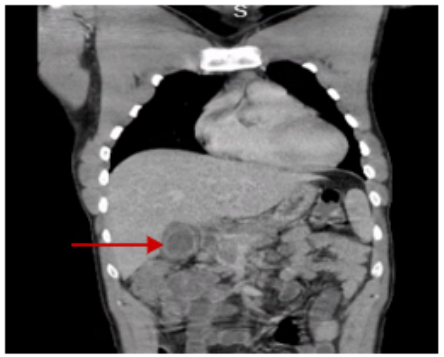
References

Investigations and Results

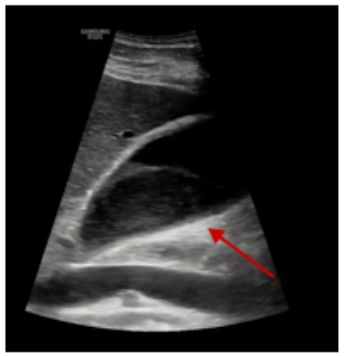
Blood Test Results

WCC	↑ **20.00** (4.0 - 11.0)
Neutrophils	↑ **17.66** (1.5 - 8.0)
CRP	↑ **124** (< 5.0)
ALT	↑ **64** (10-60)
LFTs otherwise normal. Renal function normal	
HIV, hepatitis B and hepatitis C negative	

Imaging Results



CT Chest-Abdomen-Pelvis: gallbladder oedema in the absence of biliary stones, dilatation or collection.



US Upper Abdomen: biliary sludge and a thickened gallbladder, in keeping with an acalculous cholecystitis.

Microbiology

Blood cultures: *Fusobacterium peridonticum* positive (negative on subsequent repeat)
Stool PCR: *Salmonella Newport* positive

Diagnosis and Management

This patient was diagnosed with **non-typhoidal salmonella (NTS) acalculous cholecystitis**.

The *F peridonticum* was felt to be a contaminant as it was out of keeping with the clinical syndrome.

Treatment comprised of supportive management and three days of IV ceftriaxone, followed by a single dose of IV co-amoxiclav. He was discharged with a 7 day course of oral co-amoxiclav and azithromycin. He made a full recovery.

Discussion

Salmonella enterica can be classified as typhoidal and non-typhoidal salmonella (NTS) serovars, both of which are a major source of foodborne illness worldwide. NTS typically causes an acute self-limiting gastroenteritis and in around 5% of cases extra-intestinal disease. (1)

S. enterica serotype Newport was isolated in this patient with acalculous cholecystitis. Whilst NTS rarely causes acute cholecystitis (2, 3), this is, as far as we are aware the first reported case of acute cholecystitis caused by the Newport serovar.

Salmonella Newport has previously been shown to have a lower mortality and less association with invasive disease compared to other common serotypes. (4) Invasive NTS is more common with certain serotypes, as well as immunocompromised patients and those at extremes of age, suggesting bacterial genetics may also play a role in disease manifestation.(1)

Clinicians should be vigilant for NTS in returning travellers with fever and diarrhoea and should be aware of extra-intestinal manifestations.

A thorough travel history in patients with acute cholecystitis is crucial for appropriate microbiological testing and selecting effective antimicrobial treatment, especially given rising antibiotic resistance in NTS. (5)

Out of the (orbital) box: A Case of IgG4-Related Disease Presenting with Lacrimal Gland Swelling

Ali Arfa (MD, MRCP, Medical Ophthalmology Registrar), Simin Arfa (MD, Radiology registrar)

NHS
Cambridge University Hospitals
NHS Foundation Trust

Introduction

IgG4-related disease (IgG4-RD) is a chronic, relapsing-remitting, multi-system fibroinflammatory disorder characterized by tumefactive lesions, dense lymphoplasmacytic infiltrates rich in IgG4-positive plasma cells, and often elevated serum IgG4 levels. It may affect multiple organs either synchronously or metachronously, with a tendency to mimic malignancy or infection.

Case Presentation

A 43-year-old Caucasian female with a background of systemic lupus erythematosus and antiphospholipid syndrome (diagnosed in 2015) presented with progressive bilateral lacrimal gland swelling over several months.

	Right eye	Left eye
Visual acuity	6/6	6/7.5
Intra Ocular Pressure	12	12
External eye examination	Enlarged lacrimal gland	Enlarged lacrimal gland
Proptosis	No exophthalmos	No exophthalmos
Ocular motility	Normal	Normal
RAPD	absent	absent
Ishihala	15/15	15/15
Anterior segment	NAD; Dry eye	NAD; Dry eye
Posterior segment	NAD	NAD

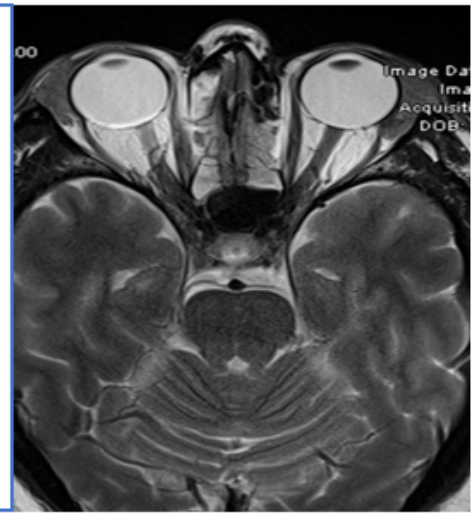
Her medical history included sensorineural hearing loss (2015), asthma, migraine, and previous cholecystectomy. She was taking hydroxychloroquine but previously did not tolerate methotrexate. She first noticed bilateral scleral icterus in January 2023. Liver function tests and hepatitis screen were normal. Multiple GP and optician consultations led to treatment for presumed dry eye disease without improvement. She reported progressive orbital swelling, dry eyes and mouth, and epigastric discomfort. Investigations for Sjogren syndrome and sarcoidosis were negative.
In January 2024, she presented to the Emergency Department with severe epigastric pain and was diagnosed with acute pancreatitis. She subsequently developed pancreatogenic diabetes (type 3c).

Investigations

- Raised serum IgG4 (4.85 g/L)
- MRI orbit: bilateral lacrimal gland enlargement (L>R)
- Biopsy of lacrimal gland: chronic inflammatory changes consistent with IgG4-related disease
- FNA of pancreas: IgG4 background staining
- ACE, calcium, myositis antibodies: normal/negative

Management and Outcome

Oral prednisolone 40 mg was commenced in March 2024 and tapered over eight weeks. There was a rapid clinical response with marked reduction of lacrimal gland swelling after 7 days. However, symptoms recurred after cessation of steroids. Mycophenolate mofetil (MMF) was initiated as a steroid-sparing agent but was not tolerated. Rituximab therapy was then started.



Discussion

IgG4-RD is an under-recognised systemic disease with diverse manifestations. Orbital involvement occurs in 17–60% of cases, most often affecting the lacrimal glands, trigeminal nerve, orbital fat, and extraocular muscles. Pancreatic disease (autoimmune pancreatitis) is frequently the first presentation. Early diagnosis and corticosteroid therapy usually lead to good short-term outcomes, though relapses are common. Diagnosis combines clinical features (tumefactive lesions), serology (elevated IgG4), and histopathology (IgG4-positive lymphoplasmacytic infiltrate with fibrosis). Differentials include Sjogren syndrome, sarcoidosis, granulomatosis with polyangiitis, thyroid eye disease, orbital lymphoma, and idiopathic orbital inflammation.

Conclusion

This case illustrates the importance of considering IgG4-related disease in patients with bilateral lacrimal gland enlargement and systemic features such as pancreatitis. Multisystem involvement, characteristic serology, and histopathology support the diagnosis. Early steroid therapy is usually effective, but many patients require long-term immunosuppression to maintain remission.

References

1. Wallace, Z.S. et al. (2024) 'Current and future advances in practice: IgG4-related disease', Rheumatol Adv Pract.
2. Perugino, C.A. et al. (2023) 'Evaluation of the safety, efficacy, and mechanism of action of obexelimab for the treatment of patients with IgG4-related disease: an open-label, single arm, single centre, phase 2 pilot trial' The Lancet Rheumatology.
3. Stone, J.H. et al. (2012) 'IgG4-related disease', N Engl J Med, 366, 539–551.
4. Lanzillotta, M. et al. (2020) 'Advances in the diagnosis and management of IgG4 related disease', BMJ, Vol 369.
5. McNab AA, McKelvie P. IgG4-¬ related ophthalmic disease. Ophthalmic Plast Reconstr Surg. 2015;31(2):83–88.