

# **Title: A 70 year old with confusion - another delirium? Cerebral vasculitis as a rare cause of encephalitis**

**Category: Case Reports**

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## **Introduction**

We present a case of cerebral vasculitis as a rare cause of encephalitis in a patient with a history of autoimmune pathology.

## **Case Report**

A 70 year old female presented with a 4 day history of confusion, drowsiness and headaches. She has a history of autoimmune haemolytic anaemia, splenectomy, atrial fibrillation, stroke, hypertension and statin induced myopathy. She was pyrexial and confused without focal neurological deficits. Routine bloods including HIV/hepatitis serology were unremarkable other than a CRP of 26; septic screen was negative and CT head showed only old infarction. CSF showed WCC 25 100% lymphocytes, RCC 60, protein 1.7, glucose 3.9; negative gram stain and TB culture. Acyclovir was started pending CSF viral PCR's however her GCS fluctuated from 8-15 and her fevers continued. PCR was negative for HSV, VZV, CMV and EBV. MRI showed subcortical oedema affecting the white matter, deep nuclei, brainstem and cerebellum with several foci of microhaemorrhages; the working differential was an inflammatory process, atypical viral encephalitis or CNS lymphoma. Autoimmune serology showed an ANA titre 1/1280, dsDNA 61 with strongly positive anticardiolipin IgG suggesting a diagnosis of cerebral vasculitis likely lupus related. Methylprednisolone was given followed by tapering prednisolone course and GCS dramatically improved in days. She had no further fevers and walked out of hospital with follow up MRI demonstrating significant improvement of all abnormalities and repeat CSF showing no WCC and protein of 0.46.

## **Discussion**

Cerebral vasculitis is a rare cause of encephalitis but should be considered early in patients with known autoimmune pathology and CSF demonstrating a raised protein and lymphocytes. Poor clinical and radiological progress on antivirals, negative viral PCR's (sensitivity 80-98%<sup>1</sup>), a dramatic response to steroids and positive autoimmune serology supported this diagnosis. Systemic Lupus Erythematosus presents with neurological symptoms in 3% of cases<sup>2</sup> with neurological manifestations carrying a poor prognosis<sup>3</sup>. Treatment involves induction with cyclophosphamide or methylprednisolone prior to maintenance steroids or sparing agents.

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## **Title: A Hungry Heart**

### **Category: Case Reports**

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#### Introduction

Eating disorders are associated with the highest mortality rate of any psychiatric disorder<sup>1</sup>. The association between malnutrition, electrolyte disturbances and cardiovascular complications has been described<sup>2,3,4,5</sup>. It is for these reasons, that such patients are admitted to acute hospitals, to allow close monitoring. We present a case of Anorexia Nervosa that demonstrated alarming electrocardiographic (ECG) changes, which could not be explained by the serum electrolyte levels alone.

#### Case

An 18 year old female, with emotionally unstable personality disorder, was admitted with a six-week history of anorexia and >10kg weight loss. Due to the high risk of re-feeding syndrome she was admitted under the medical team for nasogastric (NG) feeding and monitoring. Examination was unremarkable.

Admission bloods revealed U&Es (including potassium, magnesium, phosphate and calcium), LFTs and FBC within normal range.

Admission ECG demonstrated significant changes – widespread ST-segment depression and T-wave inversion, with a prolonged QTc interval.

Echocardiography was normal, with no evidence of structural or valvular disease, or pulmonary hypertension.

NG feeding and vitamin and electrolyte supplementation were commenced, and as feeding was established, serial ECGs showed resolution of both the ST-segment and QT interval. However, T-wave inversion was persistent.

#### Discussion

ECG changes associated with electrolyte imbalance are common in the context of starvation, particularly QT prolongation and bradycardia<sup>1,2,4,6</sup>. However, in this case, electrolytes were essentially normal. This can be explained by starvation-derived anatomical remodelling of the heart<sup>3,6</sup>. Furthermore, there was no evidence of bradycardia; likely due to copious quantities of caffeinated drinks that the patient was ingesting, which was identified on further questioning.

#### Conclusion

Despite normal electrolytes, a routine ECG should be performed in those admitted with eating disorders and malnutrition. Young patients with recurrent and prolonged periods of starvation should have cardiology follow-up due to increased risk of cardiovascular disease in adult life.

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**Title: A not so common cause of adrenal insufficiency**

**Category: Case Reports**

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We present a case of adrenal insufficiency brought on by bilateral adrenal haemorrhage following warfarin therapy for a recent diagnosis of pulmonary thromboembolism. The patient in question was a 70 year old female who had had a total right knee replacement for osteoarthritis 2 weeks preceding her present admission. In spite of methodical adherence to prophylactic low molecular weight heparin she presented with a sudden onset of shortness of breath with associated chest pain which was then diagnosed by CTPA to be pulmonary embolism involving an isolated right upper lobe pulmonary artery for which she was duly commenced on warfarin. Four days post warfarin initiation she presented with a sudden onset of excruciating upper back pain followed by 3 collapses at home with syncope prompting her husband to call out for the ambulance. In the emergency department she was noted to be anaemic with haemoglobin of 69g/l and an urgent CT scan of her abdomen revealed evidence of large retroperitoneal bleed with additional adrenal haemorrhage more on the right side. Biochemistry also revealed a new hyponatremia though with normal potassium with hypotension. She was resuscitated with blood transfusion but continued to exhibit persistent hyponatremia with now also a new symptom of extreme lethargy and tiredness prompting the short synacthen test which revealed poor adrenal reserve with a baseline cortisol of 106nmol/l with a 30 minute response of only 122 nmol/l. She has since being commenced on hydrocortisone with a follow up endocrine appointment. The importance of reviewing patient symptoms with the biochemistry cannot be understated given the complexity of her presenting symptoms and the need to keep an open mind to a potentially life threatening endocrine disorder which can be easily treated, such treatment being lifesaving cannot be over emphasised.

# **Title: A Rash Decision: Understanding the Side Effects of Novel Immunotherapy**

**Category: Case Reports**

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With a 5 year survival of less than 10% and median survival of 6-12 months, metastatic melanoma is associated with poor survival rates.<sup>1</sup> Ipilimumab has been associated with increased survival in patients with previously treated or unresected melanoma<sup>2</sup>. Here, we report a case of multi-organ vasculitis and subsequent organ failure secondary to Ipilimumab therapy.

A 68 year-old man, with known metastatic malignant melanoma treated with Ipilimumab immunotherapy, presented to hospital with a short history of fever and malaise. Initial investigations revealed a mildly elevated C-reactive protein (CRP). There was no obvious source of sepsis with normal chest x-ray and urinalysis. The patient was started on intravenous antibiotics for sepsis of unknown origin. Unfortunately, inflammatory markers continued to rise and his renal function started to decline. In view of the pyrexia of unknown cause, a CT (computed tomography) scan of his chest, abdomen, and pelvis was carried out but showed no evidence of melanoma progression. The patient subsequently developed a vasculitis rash, suspected paralytic ileus and worsening kidney injury. Vasculitic screen revealed c-ANCA positivity. Advice from specialist renal and oncology teams was sought, but the patient unfortunately died due to multi-organ failure and post-mortem confirmed the presence of multi-organ vasculitis.

Ipilimumab is a monoclonal antibody antagonist to cytotoxic T-lymphocyte associated antigen (CTLA-4) and is associated with a high risk of adverse events due to unrestrained proliferation of T-lymphocytes. Clinical trials report adverse events in upto 80% of subjects.<sup>4</sup>

This case highlights that greater awareness of potential serious side effects of immunotherapeutic agents is required as more become available in clinical practice. Patients will often have their treatment managed in specialist tertiary centres, but will present to local district general hospitals when acutely unwell. Therefore, physicians in district general hospitals need to be aware of the serious adverse effects of newer immunotherapy agents

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3. Fellne C.; Ipilimumab (Yervoy) prolongs survival in advanced melanoma: Serious side effects and a hefty price tag may limit its use; P T. 2012 Sep; 37(9): 503-511, 53

## **Title: A Review of a Diving Emergency**

**Category: Case Reports**

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Medical Physicians should consider barotrauma and decompression illness (DCI) in any patient presenting after a recent SCUBA dive, even apparently shallow dives and act without delay to transfer the patient to a recompression facility.

### **Learning points:**

- 1-Not to rule out diving injuries even if incident occurred at shallow depth.
- 2-If you suspect DCI contact nearest hyperbaric chamber urgently.
- 3-Asthmatics are more prone to barotrauma in view of their increased positive airway pressure.

### **Case Presentation:**

33 year old woman was ascended rapidly from no more than 10 metres depth. she appeared extremely distressed and short of breath, but stated she had asthma and medication was available at her car and made a swim for shore. She swam approximately 50 metres before showing signs of distress and loss consciousness, On arrival to shore an off-duty paramedic performed immediate CPR and ROSC was obtained after 2 cycles.

She was intubated and ventilated. Arterial blood gases showed type 1 respiratory failure. CT head and chest demonstrated cerebral oedema and no pneumothorax or pulmonary embolism. Post mortem reported the cause of death as hyperbaric injury.

### **Discussion:**

Decompression sickness is caused by the formation of bubbles of inert gas coming out of solution in blood and tissues on depressurisation. They commonly materialise in venous circulation, but can cross to arterial circulation via a right-to-left cardiac shunt, most commonly a patent foramen ovale. It often presents with numbness, joint and muscle pain, dizziness, headache and weakness.[1,2,3]

Barotrauma occurs when ambient pressure changes cause expansion or contraction of gas within a closed body compartment. Common sites for such injury are the middle and inner ear, the sinuses, and lungs.[4,5]

The management of DCI involves giving 100% oxygen, rehydration, lying the patient supine to increase nitrogen washout, and transferring to a decompression chamber for definitive management. If this transfer must take place by air, this should occur below 1,000ft or within a pressurised cabin. Cardiopulmonary resuscitation should be performed if necessary.[2,4,6]

### **References**

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**Title: ABCD-E for EXPOSURE: A good reason to strip and flip**

**Category: Case Reports**

**Main Author: Lucy Connor**

**Co-Authors: Micayla Telfer**

**Case Report:**

A 25 year-old female type 1 diabetic managed by insulin pump presented with a 1 week history of malaise, fever and lethargy associated with back pain. No other focal symptoms.

On examination, her abdomen was described as 'diffusely tender' and a urine dipstick was positive for leucocytes. She was diagnosed and treated for UTI. The degree of sepsis was underestimated, and with hindsight a comprehensive, systematic exposure and examination had not been completed. The next morning she had deteriorated. Sepsis and pain were now disproportionate to clinical signs.

On performing personal care, nursing staff noticed an odd area on her buttock. Clinician review confirmed an area of necrotic tissue, surrounded by spreading cellulitis but no crepitus.

We suspected necrotising fasciitis and instigated appropriate emergency treatment, investigation and referrals. CT findings reinforced the suspicion of necrotising fasciitis (see images). She was taken to theatre for immediate laparotomy, retroperitoneal debridement, appendectomy, de-functioning colostomy and debridement of perineum. She required ICU care for organ support and ultimately survived to be discharged home.

**Discussion:**

In some cases of sepsis, identification and surgical control of the source is vital to prevent death<sup>1, 2</sup>.

Necrotising fasciitis is a rare, rapidly progressive soft tissue infection with high lethality. There are some clinical findings traditionally associated with necrotising fasciitis, several of which were present in this case, none are particularly sensitive<sup>3</sup>. Diagnosis requires a high index of suspicion.

**Learning points:**

This case highlights the importance of full exposure on clinical examination. This is particularly important if the patient is acutely unwell or the presentation is atypical. This patient mentioned her back/buttock pain on initial presentation but the area was not formally examined at that time.

The acute physician must remain vigilant to serious surgical pathology presenting to their unit.

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# **Title: Absent Intrahepatic Inferior Vena Cava Presenting as Deep Vein Thrombosis - A Previously Undocumented Variant of 'KILT Syndrome'**

**Category: Case Reports**

**Main Author: Tom Cibulskas**

## **AIM**

This report aims to illustrate a previously-undescribed variant of inferior vena cava abnormality, which presented on the acute medical take in a young and previously well patient as lower limb DVT. The patient also showed signs of chronic liver disease related to his vascular problem.

## **METHODS**

This patient's history and initial examination clearly pointed towards a left leg DVT. However, his other abnormal examination findings (hepatosplenomegaly and abdominal varices) indicated previously undiagnosed portal venous hypertension. The aetiology of this was not immediately apparent, but the differential included pre-hepatic causes (e.g. congenital portal vein abnormalities or chronic portal vein thrombosis), intrinsic hepatic causes (e.g. cirrhosis due to alcohol, infective hepatitis, metabolic disorder or granulomatous disease) and post-hepatic causes (e.g. Budd Chiari syndrome or space occupying lesion). Extensive investigations demonstrated an absent intrahepatic Inferior Vena Cava as the cause of his problems.

## **OUTCOMES/RESULTS**

The patient's calf swelling improved and he was discharged after 9 days, with anticoagulation clinic follow-up. Unfortunately he later represented with ulceration of left lower limb, which was attributed to impaired venous drainage to the limb and was treated conservatively. An outpatient screening endoscopy for oesophageal varices was planned, but was not performed as the patient returned to Pakistan. A liver biopsy to confirm the presence of cirrhosis was also considered, but was ruled out due to bleeding risk and absence of potential change to the patient's immediate management.

## **CONCLUSION**

Vascular abnormalities are overall a rare cause of unprovoked DVT, but make up a significant proportion of DVT presentations in young adults. This case combines a typical presentation of DVT with an unusual underlying vascular problem, including a vascular malformation which has not previously been associated with DVT. The case highlights the need to be vigilant for signs of unusual causes in common presentations.

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+ 4 other references not included due to word limit

**Title: Acute confusion - are CT head findings absolute or are there differential diagnoses to be considered?**

**Category: Case Reports**

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Patients commonly present with acute confusion to acute medical units.

A 74 year old female, previously very independent despite a background of dementia, previous TIA/CVA, hypertension and breast cancer (cured), was admitted after an episode of collapse. This was followed by confusion, vomiting and becoming bedbound.

She was initially treated for sepsis of unknown origin, and a confusion screen was completed.

The patient developed a seizure, however, the initial CT head showed no acute pathology. A diagnosis of post-stroke seizure was now considered, yet the seizures continued in spite of treatment, making MRI head scanning impractical.

The patient became increasingly agitated with fever and therefore the diagnosis was reviewed. A CT head was re-performed and a lumbar puncture carried out. The CT head was reported as a likely thrombus along the course of the left middle cerebral artery, whilst CSF protein levels were raised and showed pleocytosis. The patient was therefore treated for an acute cerebral infarct with the addition of IV antibiotics and antivirals.

PCR performed on the CSF - tested positive for Herpes simplex virus (HSV) type-1.

Given the non-specific signs and symptoms - herpes simplex viral encephalitis is commonly delayed in diagnosis and consequently has significant morbidity and mortality. Therefore, this case presentation aims to consider an overview of differential diagnoses for acute confusion and increase awareness of this specific condition. Simultaneously, this report will discuss limitations with common radiological studies in such a case and as seen in this patient, exemplify how the repeat CT head findings, reported above, can overlap with similar findings also commonly found in patients with HSV encephalitis. Thereby, ultimately, increase the index of suspicion amongst acute physicians and prevent a delay in diagnosis.

**Title: Acute Isolated Respiratory Failure Unmasking a Diagnosis of Myasthenia Gravis – A Rare and Unusual Presentation**

**Category: Case Reports**

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Myasthenia Gravis (MG) is often complicated by respiratory failure during a myasthenic crisis, however for most patients respiratory manifestations usually occur during the late stages of the disease in conjunction with pre-existing neurological signs and symptoms. Isolated respiratory failure as the initial presentation is a rare phenomenon. We present an unusual case of a patient with previously undiagnosed MG presenting with respiratory failure as her initial presenting complaint.

A 74 year-old-female with a background of hypertension presented with a two day history of dyspnoea, orthopnoea and chest heaviness. She denied any other systemic symptoms.

Clinical examination revealed a respiratory rate of 18 breaths-per-minute and SpO<sub>2</sub> of 94% on air with shallow breathing which worsened upon lying flat. Cardiorespiratory, gastrointestinal and neurological examinations were otherwise unremarkable. Initial bloods including Troponin-I and chest radiographs were normal. Arterial blood gas sampling demonstrated a fully compensated respiratory acidosis.

She was initially treated for presumed cardiac failure with diuretics, however continued to deteriorate over 24-48hours. CTPA excluded a pulmonary embolism and echocardiogram revealed a normal left ventricular systolic function. Repeat blood gas sampling demonstrated type two respiratory failure with a reduced forced vital capacity. She was subsequently transferred to ITU where she was eventually ventilated and a neurology review was sought. Serology for Anti-AChR and Anti-MuSK antibodies was negative however electromyography confirmed a diagnosis of MG. She was treated with steroids and immunoglobulins and later discharged home on Pyridostigmine.

MG is a chronic autoimmune disease most commonly presenting with muscle weakness, bulbar and ocular signs. In the later stages of disease respiratory failure affects approximately 3-8% of patients<sup>1</sup>. Isolated respiratory failure without neurological manifestations as the initial presentation has been reported in only a handful of cases, thus highlighting the importance of considering a neuromuscular disorder as a cause of unexplained respiratory failure to ensure specific treatment is instigated.

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## **Title: Acute upper gastrointestinal bleed – refer to gastroenterology? Think again!**

**Category: Case Reports**

**Main Author: Antony Aziz**

### **Aim**

Acute upper gastrointestinal (GI) bleeds are responsible for approximately 25,000 admissions each year.<sup>1</sup> The clotting screen is an essential and inexpensive investigation in such patients. This case highlights the importance of acting upon an abnormal clotting screen in a patient with recurrent GI bleeds without a clear identifiable source despite extensive investigation, in order to expedite the diagnosis of a rare condition.

### **Case**

An 80 year old gentleman presents with his fourth GI bleed in eight months. In the three admissions prior, he had been extensively investigated with multiple endoscopies and CT angiograms. The only positive finding was oozing blood in the proximal jejunum on push enteroscopy assumed to be from angiodysplasia, and this was treated with adrenaline injection and argon plasma coagulation (APC). He was discharged each time after being stabilised with blood transfusions but trials of tranexamic acid and octreotide were unsuccessful in preventing relapses of bleeding in the community. On the fourth admission, it was noted that his activated partial thromboplastin time (aPTT) was elevated on routine clotting screen, and review of previous clotting screens revealed 16 normal prothrombin times but 15 prolonged aPTTs. Haematology opinion was sought and his von Willebrand factor and factor VIII levels were found to be low. He was diagnosed with acquired von Willebrand syndrome and started on intravenous immunoglobulin and eventually thalidomide. His bleeding settled. He underwent twelve endoscopies and four CT scans and was transfused twenty two units of blood before the diagnosis was made.

### **Discussion**

Acquired von Willebrand syndrome is a rare cause of mucocutaneous bleeding in the elderly, with less than 700 cases described worldwide prior to 2012.<sup>2</sup> A prolonged aPTT must be investigated further and early involvement of the haematologist in liaison with the gastroenterologist enables prompt diagnosis, appropriate investigation and targeted management of this condition.

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2. Tiede, A., 2012, Diagnosis and treatment of acquired von Willebrand syndrome: Thrombosis research, v. 130, p. S2-S6.

## **Title: Airway Obstruction Secondary to a Hemi-goitre**

### **Category: Case Reports**

**Main Author: Nosheen Iqbal**

**Co-Authors: Rajeev Advani  
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### **Summary**

An 82 year-old female presented acutely with shortness of breath. She had a background of asthma and diabetes. Four years previously, she had a left hemithyroidectomy for a large left multinodular goitre causing compressive symptoms.

On this acute presentation, her initial management was that for suspected asthma exacerbation – with poor response to therapy. Clinical assessment revealed a large right-sided goitre, bi-phasic stridor, and the use of accessory muscles of respiration.

The patient was suspected to have airway compromise secondary to extrinsic tracheal compression from the goitre. Urgent ENT input was sought. Management included intravenous dexamethasone, adrenaline nebulisers and the use of heliox. The patient was transferred to ICU where she was intubated and ventilated. Computerized Tomography (CT) scan confirmed the presence of a right-sided multinodular goitre with retroclavicular extension causing left sided tracheal deviation and compression.

The patient underwent an urgent right completion-thyroidectomy. Intraoperatively, adhesions and scar tissue from her previous surgery tethered the trachea and hence allowed tracheal compression from the remaining right thyroid goitre. Histology was benign in nature. The patient had an uneventful post-operative recovery and at 3-weeks follow up was free from compressive symptoms.

### **Learning Points**

1. Differentiating between upper airway noises (stridor) and lower airway noises (wheeze) indicates the level of airway obstruction. Furthermore, in patients with stridor, sub-classification into inspiratory and bi-phasic stridor allows greater localisation to the level of airway obstruction.
1. Any suspicion of airway compromise requires urgent escalation and multi-disciplinary involvement including ENT surgeons, anaesthetists, and critical care. In suspected cases of tracheal compromise and lower airway compromise, thoracic surgeons and respiratory physicians may be required.
1. It is rare for a single-sided goitre to cause airway compromise resulting in urgent thyroidectomy. As such, differential diagnosis must be kept broad and doctors' intuition and experience are invaluable.

**Title: An unusual case of abdominal pain**

**Category: Case Reports**

**Main Author: Krishna Shah**

Case Report: A 20 year old previously fit and well female presents with a short history of abdominal pain, localised to the right iliac fossa, accompanied by pyrexia and loose stools. Investigations showed raised inflammatory markers and an ultrasound that was suggestive of a ruptured appendix. The patient was taken to theatre for an urgent laparoscopy; the findings of which were a normal appendix with an inflamed caecum and terminal ileum. The abdominal cavity was washed out and a drain left in situ.

Post operatively, the patient was slow to recover with increased lethargy. She developed an acute kidney injury with a rapidly declining glomerular filtration rate. She was hydrated aggressively with no improvement in her renal function. Blood results then showed a steady decline in haemoglobin and platelet levels with a profound anaemia of Hb. The patient was taken over by the nephrologists and pulsed with high dose methylprednisolone. There was no response to this treatment and the patient had developed end stage renal failure with anuria within 72 hours of the laparoscopy. She required urgent haemodialysis and plasma exchange.

A renal biopsy showed thrombotic microangiopathy, and a presumptive diagnosis of haemolytic uraemic syndrome was made. She was started on eculizumab and showed good response with complete recovery of her renal function, and no further relapses reported at 9 months.

Discussion: Haemolytic uraemic syndrome presents as a triad of an acute kidney injury, haemolytic anaemia and thrombocytopenia.

This case highlights the importance of recognising and responding to an abnormal recovery of a young, previously well patient. Early involvement of specialist teams can provide key input and facilitate the rapid escalation of a patient to a high dependency unit.

**Title: An unusual case of meningitis**

**Category: Case Reports**

**Main Author: Hannah Richardson**

**Co-Authors: Phillip Unsworth  
Haris Rathur**

A 39 year old, alcohol dependent male was admitted with fluctuating GCS and pyrexia. He was initially managed as decompensated alcoholic liver disease. There was rapid deterioration in his GCS within the first 24 hours of admission.

At 36 hours following admission, it was identified that the patient was demonstrating signs of sepsis; further assessment identified the likely cause to be neurological and a lumbar puncture was performed. CSF showed 185 white cells (80% lymphocytes/20% polymorphs) and the patient was commenced on Acyclovir and Ceftriaxone. Results were in keeping with a diagnosis of viral meningitis and the patient was transferred to a high dependency bed but GCS continued to deteriorate. The patient was intubated and transferred to ITU towards the end of day 3.

The patient continued to deteriorate during his ITU admission. On day 5, repeat CT head showed evidence of gross cerebral oedema. His case was discussed with the regional neurosurgical team who recommended a repeat lumbar puncture. CSF pressure was very high and showed 600 white cells (92% polymorphs). The patient was continued on acyclovir and ceftriaxone following discussion with the microbiology team.

On day 8, brain stem testing was performed. The patient was declared Brain Stem Dead and discussions regarding organ donation were done. The family agreed and the patient's kidneys were harvested for donation. The case had been discussed with the coroner and no post-mortem was required.

Following organ donation, the two recipients of the donated kidneys began with the same neurological symptoms, and were both deceased, with the same clinical picture within two weeks of renal transplant. Post-mortem examination of the recipients revealed evidence of a nematode infection of the CNS by *Halicephalobus gingivalis* – a very rare cause of eosinophilic meningitis.

# **Title: An Unusual Case of Sepsis on the Acute Medicine Ward**

## **Category: Case Reports**

**Main Author: Charlotte Peal**

### Clinical Presentation

A 46yr old obese female, with poorly controlled diabetes, presented with headaches, fevers and tachycardia. She was drowsy, with neck stiffness and photophobia. She was tender in the right upper quadrant, which correlated with an obstructive picture on her liver function tests. With two potential sources of sepsis (CNS and abdominal), she was treated empirically. Blood cultures grew *Klebsiella pneumoniae* and treatment continued with intravenous meropenem as per microbiology advice.

Abnormal LFTs and RUQ tenderness prompted abdominal imaging. A round, predominately low-attenuation, multiseptated lesion within the liver, suggestive of an abscess was identified on both US and CT. Of note, there was no sigmoid diverticula disease.

During admission, the patient complained of blurred vision in her right eye. A vitreous tap and corneal scrapings were taken. She was treated for endogenous endophthalmitis, with intravitreal and topical antibiotics.

Inflammatory markers settled and the patient showed good clinical response to treatment, and she was discharged with a prolonged course of oral antibiotics. Unfortunately, she lost sight in her right eye.

### Discussion

This is a case of metastatic sepsis, presenting in a young female with poorly controlled diabetes. She was treated for *Klebsiella* bacteraemia, pyogenic liver abscess and endophthalmitis. The triggering site of infection remains unknown.

*Klebsiella* has become the predominant isolate from patients with pyogenic liver abscess, and diabetes (poor glycaemic control in particular) is a well-documented predisposing factor and endophthalmitis a common phenomenon of metastatic infection from *Klebsiella* Pyogenic Liver Abscess.

A liver abscess may present with vague symptoms and is a source of sepsis to consider, particularly in susceptible adults. They commonly occur from gut bacteria translocation via the portal venous system. Ultrasound or CT imaging is essential to diagnosis, and broad-spectrum antibiotics for treatment. If medical management fails, or the abscess is >6cm, percutaneous drainage may be required. If drainage is unsuccessful, or there are multiple abscesses, surgery may be required.

### Conclusion

Sepsis presents commonly in Acute Medicine, occurs more frequently than myocardial infarction and has a higher rate of mortality than any cancer. The elderly, co-morbid and immunocompromised are most susceptible, and it is a leading cause of death and morbidity, even in the developing world.

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# **Title: An unusual cause of Syndrome of inappropriate ADH secretion resulting in a serious complication**

## **Category: Case Reports**

**Main Author: Rangaprasad Karadi**

**Case details:** A 79 year old lady presented with acute confusion with serum sodium level of 119 mmol/l and elevated inflammatory markers with CRP of 169 mg/l. Initially she reported head pulsing sensation. She was treated with Intravenous acyclovir and intravenous antibiotics. Investigations for suspected Viral Encephalitis including CT head and Lumbar puncture were within normal limits. Her urine sodium was significantly elevated, with low serum osmolality and clinical euvolemia suggesting Syndrome of inappropriate ADH secretion (SIADH). There was no obvious culprit medication. She had a normal short synacthen test, Thyroid function test. CT thorax and abdomen were within normal limits. She underwent MRI brain that did not show obvious abnormality. Meanwhile serum sodium level improved to 126 mmol/l with cautious intravenous hydration. Her inflammatory markers improved with CRP decreasing to 59 mg/l and the confusion resolved. She was discharged following satisfactory assessment by the physiotherapy team with a presumptive diagnosis of 'Sepsis from unknown source'. A week later she presented with classical symptoms of global headache, jaw claudication and sudden loss of vision. Her CRP remained elevated at 49 mg/l. Giant cell arteritis was suspected and treated with intravenous methylprednisolone. The headache and jaw claudication resolved but unfortunately, she endured complete blindness in left eye and very limited vision in the right eye due to central retinal artery occlusion. Temporal artery biopsy confirmed diagnosis of giant cell arteritis.

**Discussion:** SIADH has wide range of differentials including acute neurological diseases, malignancy, acute pulmonary disease and medication/drugs<sup>1</sup>. Literature review subsequent to this revealed a case series where SIADH was the presenting feature of Giant cell arteritis<sup>3</sup>. Nearly 40% of patients with Giant cell arteritis do not report the classical symptom of headache<sup>2</sup>.

This case highlights the importance of considering Giant cell arteritis as a potential differential in elderly patients presenting with SIADH even in the absence of classical pattern with headache. A high index of suspicion will avert the dreaded complication of blindness that unfortunately could not be avoided in our patient.

### References:

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2. Dasgupta, Bhaskar, et al. "BSR and BHPR guidelines for the management of giant cell arteritis." *Rheumatology* (2010)
3. Luzar, M. J., R. L. Whisler, and G. G. Hunder. "Syndrome of inappropriate antidiuretic hormone secretion in association with temporal arteritis." *The Journal of rheumatology* 9.6 (1982): 957.

**Title: An Unusual Presentation of Chest Pain in a Thai Kickboxer**

**Category: Case Reports**

**Main Author: Kyriaki Paroutoglou**

**Co-Authors: Kyriaki Paroutoglou**

**Case Report:**

A 17-year-old male Thai kick boxer presented to A&E complaining of chest pain for one day. He had no significant past medical history or regular medications. The pain started during a fight, although he denied any blunt chest trauma or other significant injury during the contest. The pain was worse on inspiration, coughing, yawning, swallowing and lying down. He denied palpitations, shortness of breath, haemoptysis, presyncope or syncope. There was no significant family history of cardiac disease or sudden death and no risk factors for pulmonary embolism. He denied smoking, taking illicit drugs or performance enhancers. Incidentally, he had had a throat infection a month previously treated with antibiotics.

On examination, there was no focal tenderness on palpation of the chest wall. His respiratory and cardiovascular systems were unremarkable and there was no sign of surgical emphysema.

His blood tests were normal with the exception of a D-dimer of 767. His ECG was also normal. His chest x-ray revealed pneumomediastinum. He had a CT scan of his chest, which showed extensive mediastinal air tracking down and around the outside of the oesophagus. Appearances suggested a pulmonary source for the air and a barium swallow showed no contrast leaking into the mediastinum. He remained clinically well and was discharged home the day after. At one month follow-up in the cardiothoracic clinic he was completely asymptomatic and was discharged back to his GP.

**Conclusion:**

Pneumomediastinum is an uncommon condition. The literature mainly describes case reports or small case series. It can be spontaneous or associated with a blunt force, penetrating chest trauma, and endobronchial or oesophageal procedures. It can rarely lead to clinically significant complications and therefore it is important to investigate the source and the extent of the underlying damage, with appropriate imaging and expert review by cardiothoracic surgeons if available. In our case, the cardiothoracic team felt that sudden, rapid movement of the patient's torso during the fight was the cause of the pneumomediastinum through possible covert rupture of small airways.

## **Title: An unusual presentation of stroke in a young patient**

**Category: Case Reports**

**Main Author: Ratna Aumeer**

**Co-Authors: Mishti Oberoi**

An unusual presentation of stroke in a young patient

Mishti Oberoi, Ratna Aumeer.

Case

We present the case of a carotid dissection, which led to Horner's syndrome and cranial nerve XII palsy. A 50 years old male attended with a week history of headache, which started suddenly. He developed weakness of tongue, with deviation to left side over the next 3 days. On examination he had left miosis and ptosis and left tongue deviation. The rest of neurological examination was normal.

His initial CT brain was normal. MRI angiogram of head and neck was organised and this showed occlusion of the distal part of the left internal carotid artery extending intra-cranially, suggesting a thrombosed dissection.

He was diagnosed with left Horner's syndrome and hypoglossal palsy secondary to left internal carotid artery dissection. His case was discussed with the Vascular team who advised that surgery was not required. He was then started on aspirin.

Discussion

The incidence of carotid dissection is about 3 per 100000/year.

The features include ipsilateral headache, Horner's syndrome, nerve palsies and potentially devastating cerebral ischaemic events.

Horner's syndrome is caused by compression of the ascending sympathetic supply within the carotid sheath resulting in the triad of ptosis, miosis and anhidrosis. Extracranial dissection can cause cranial nerve palsy with cranial nerve XII being the most commonly affected.

If initial CT yields negative results, this should be followed up by more definitive imaging, such as MRA or CTA.

The goal of management is to prevent progressive neurological deficits. Antiplatelet therapy and anticoagulation have been used both individually and in combination, with antiplatelet therapy being recommended in patients with dissection.

Learning point

It is important to recognise that internal carotid artery dissection is one of the common causes of acute ischaemic stroke in young adults. It should always be considered in patients presenting with headache with Horner's syndrome and nerve palsies.

References

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2. Kasravi M, Leung A, Silver I. Dissection of the internal carotid artery causing Horner syndrome and palsy of cranial nerve XII. CMAJ June 2010 Vol 182.

# **Title: Aortic Dissection in the Context of Alcoholic Cardiomyopathy**

**Category: Case Reports**

**Main Author: Rachel Knight**

**Co-Authors: Deepak Nama  
Harriet Hurrell**

## **Introduction**

This is an individual case of Acute Aortic Dissection in the context of alcoholic cardiomyopathy – something previously unreported within medical case report literature.

## **Case Presentation**

A 40 year old gentleman admitted with a collapse after drinking vodka with a background of alcoholic cardiomyopathy (Ejection Fraction 40% recovered from 10%) and complications of a Left Mural thrombus currently anti-coagulated. He complained of epigastric pain on admission and underwent a CT scan of the thorax, abdomen and pelvis with contrast to exclude ischaemic bowel which showed no focal abnormalities.

He was referred to medicine via the emergency department with possible de-compensation of his underlying heart failure and acute kidney injury secondary to his relapse into alcoholism. Shortly after arrival to the acute Medicine ward the patient had a cardiorespiratory arrest.

He was taken to the intensive care department, intubated and ventilated. The ITU Speciality Trainee completed a trans-oesophageal ECHO which showed an aortic dissection. His second CT was completed almost 24 hours exactly after the first arrest and showed an extensive Type A dissection with significant extension into the right common carotid artery and minor extensions into the left common carotid and subclavian and a likely left renal infarct (Figure 2 &3).

## **Discussion**

Aortic Dissection has a well documented risk associated with hypertension and connective tissue disorders (Elhers Danlos & Marfans). The medical literature does not relate any current risk in underlying cardiomyopathy of any aetiology. This patient had developed a severe Heart Failure with an ejection fraction of 10% believed to be secondary to alcohol excess. On cessation of alcohol this gentleman's ejection fraction recovered to 40% with a clinical improvement. It is proposed that a relapse of his alcoholism and the associated improvement in his ejection fraction created the appropriate intimal fragility and pressure changes to cause Aortic Dissection.

## **Title: Bleeding Limb- Acquired Haemophilia A (AHA)**

**Category: Case Reports**

**Main Author: Thu Ra Tun**

**Co-Authors: Sanjeev Srivastava**

### **Bleeding Limb**

#### **Acquired haemophilia A (AHA)**

**Aim:** to increase awareness of acquired coagulation disorder, obtain prompt diagnosis and treatment

**Method:** this case was presented in Medical meeting for education

**Outcome:** interactive discussion, transferred to tertiary centre

#### **Case History**

68 years old lady was admitted following multiple bruises, pain and swelling of the left leg without injury. On examination, extensive haematoma with intact neurovascular bundle on left leg.

Three days before, she came to A&E due to mild bruise and pain due to minor scotch. APTT (63.1) Hb 99. discharged for outpatient haematology review.

**Past Medical history:** Crohn's disease, type 2 DM, Glaucoma, Hypertension, non-smoker

**Medications:** Lisinopril, Rosuvastatin, Metformin SR (not on warfarin or NOAC)

#### **Investigations**

**Hb 70**, wbc 14, plt 407, INR 0.9, **APTT 71.9**, APTT ratio **2.7**, Fibrinogen 4.9,

Mixing studies: equivocal,

**Factor VIII <1** (50-150), normal Factor IX, XI and XII

UE, LFT, CRP, Ca normal.

CT angiogram left lower limbs: extensive haematoma.

Haematologist reviewed, blood transfusion, prednisolone 1mg/kg, transferred to tertiary centre, received bypassing agents

#### **Acquired Haemophilia A (Factor VIII inhibitors)**

#### **Learning Points:**

Acquired coagulation disorder should be considered for recent onset of abnormal bleeding with abnormal APTT/PT without anticoagulants

**Delay in diagnosis and treatment** is common putting patients at unnecessary risk of severe bleeding as in this case

**Avoid iatrogenic bleeding:** avoid IM injections, minimize IVs

understanding **Coagulation algorithms**

#### **Discussion**

AHA results from deficiency of factors or formation of inhibitors

**Incidence:** 1.5 case/**million**/year, most common elderly, median age of 75–80 years

**Associations:** PMR, RA, SLE & autoimmune d/o, malignancy, SLE, pemphigoid in 50%

**Mortality:** 8- 42%, are related to treatment, infection bleeding.

### **Investigations**

In this case as PT is normal and therefore patient has intact extrinsic pathway.

As APTT is prolonged, intrinsic factor abnormalities due to factor VIII, IX, XI and XII need to be excluded. Inhibitors against factor VIII or vWF are most common in contrast to other coagulation factors.

### **Treatment**

Haematologist experienced in the treatment of patients with inhibitors because bleeding may be very severe and prompt haemostatic control is required to reduce morbidity and mortality.

By passing agents:(rFVIIa), (aPCC) and (FEIBA).

### **Eradication of the inhibitor in AHA**

First, exclude malignancy

### **Immunosuppression**

**Prednisolone** 1 mg/kg/d either alone or combined with **cyclophosphamide** 1–2 mg/d orally. **Rituximab and calcineurin inhibitors**, multiple immunosuppressive agents can be used

### **References**

BCSH guideline:

Acquired coagulation factor inhibitor,

Diagnosis and management of acquired coagulation factor inhibitor: A guideline from UKHCDO

## **Title: Case report of patient with acute anterior spinal artery syndrome**

**Category: Case Reports**

**Main Author: James Fox**

### **Acute spinal artery syndrome in a 78 year old woman**

This case report regards a 78 year old woman who presented to AMU with collapse. Prior to her admission she had lived alone and was fully independent. Her PMH included hypertension, osteoarthritis, hypothyroidism and polymyalgia.

She presented with collapse upon arriving home from a holiday in Greece, and described her legs suddenly 'giving way', after which she was unable to stand. She had experienced vomiting for 2 days prior to this, but denied any other infective or neurological symptoms.

On examination, she was found to have a bilateral flaccid paraplegia, with power 0/5 in both lower limbs, reduced reflexes and down-going plantars. Initial examination revealed intact fine touch sensation. The remainder of her examination was normal.

Clinically a diagnosis of Guillain barre syndrome was suspected. Initial investigations including MRI spine and lumbar puncture were normal, yet despite this treatment was commenced with IV immunoglobulin.

After 2 weeks, she had shown no improvement neurologically and a further MRI spine was undertaken. This revealed a thoracic spinal cord infarction in the anterior spinal artery territory. Subsequent, detailed examination revealed a loss of pain and temperature sensation from the T10 level downwards, and a diagnosis of anterior spinal artery(ASA) syndrome was made.

ASA syndrome refers to infarction of the anterior spinal cord, and is characterised by loss of motor function and spinothalamic sensation below the spinal level with preservation of the dorsal columns<sup>1</sup>. Non-traumatic spinal cord infarction may result from a number of aetiologies, including aortic disease, vasculitis, embolism and spinal artery aneurysm<sup>2</sup>. ASIA scores are used to grade the severity of spinal cord damage, with A/B classes associated with a poor prognosis<sup>3</sup>.

This case highlights the importance of a detailed neurological examination in anyone presenting with an acute onset focal neurological deficit.

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## **Title: Case Report: Designer benzodiazepine; Flubromazolam**

### **Category: Case Reports**

**Main Author: Emma Keohane**

**Co-Authors: Tom Dickinson**

#### **Background:**

A 25 year old man presented to ED with delirium. His family reported he had been taking 'legal highs' bought off the internet, as well as oxycodone, ketamine and smoking cannabis. On admission he was mute and appeared to be having visual hallucinations. He had a mental health history and was taking prescribed analgesia for chronic back pain.

Over the two days after admission he had increasing seizure activity, was treated with lorazepam and loaded with phenytoin, following an episode of status.

He then spiked a temperature, however no source of infection was identified and extensive investigations including a CT head and LP were all normal.

We later found out he had taken Flubromazolam; a benzodiazepine derivative research chemical or new 'designer benzo'.

#### **Treatment:**

Due to the increasing severity of symptoms, it was suggested this was more in keeping with withdrawal than overdose, therefore we treated with a weaning course of lorazepam over 3 weeks, adjusting for the potential longer half-life with a prolonged course.

#### **Discussion:**

Whilst researching Flubromazolam, we found there was no TOXBASE entry, multiple online user forums, but sparse clinical information. A literature review found only one publication which details the possible chemical structure, but little information about clinical characteristics (Huppertz et al 2015).

In another publication the author took a similar drug and demonstrated a half-life of 100 hours, with traces in his urine 28 days after ingestion (Moosmann et al 2013). Traditional benzodiazepines have half-lives ranging from 2 hours for Midazolam to 24 hours for Diazepam (TOXBASE 2014).

#### **Learning Points:**

- The rapid evolution of 'legal highs', now freely available online, means doctors face new challenges in treatment of this patient population.
- When faced with a clinical situation for which there has been no precedent, we must be pragmatic in our approach.
- Mental illness can lead to patient's self-medicating in order to help control symptoms.

#### **References:**

**Huppertz LM, Bisel P, Westphal F, Franz F, Auwärter V, Moosmann B.** *Characterization of the four designer benzodiazepines clonazolam, deschloroetizolam, flubromazolam, and meclonazepam, and identification of their in vitro metabolites.* Forensic Toxicology. April 2015

**Moosmann B, Huppertz LM, Hutter M, Buchwald A, Ferlino S, Auwärter V.** *Detection and identification of the designer benzodiazepine flubromazepam and preliminary data on its metabolism and pharmacokinetics.* Journal of Mass Spectrometry November 2013; 48(11):1150-9

**TOXBASE: Diazepam, updated November 2014.**

<http://www.toxbase.org/Poisons-Index-A-Z/D-Products/Diazepam/>

**Title: Central Pontine Myelinolysis following rapid correction of hyponatraemia**

**Category: Case Reports**

**Main Author: Karl Jackson**

**Central Pontine Myelinolysis following rapid correction of hyponatraemia**

**Dr Karl Jackson F2**

Sunderland Royal Hospital

Central Pontine Myelinolysis is a demyelinating disorder of the pons often associated with rapid correction of hyponatraemia.

A 26 year old woman was brought to A+E following an unwitnessed collapse. She reported a long history of alcohol excess – consuming 9L of cider a day until the day before admission. Blood tests revealed severe hyponatraemia and hypokalaemia. Examination was unremarkable. She was admitted to ICCU for electrolyte replacement via a central line and later that day had a generalised tonic clonic seizure. She was then intubated before self-extubating a day later whilst agitated. Prior to step-down to the ward, she self discharged after being deemed to have capacity. She was reluctantly brought back to A+E a week later due to increasing confusion, odd behaviour, ataxia and dysarthria. Electrolytes were normal and examination revealed nystagmus at the extremes of gaze. She was initially treated as Wernicke's encephalopathy and managed on a short stay ward whilst receiving intravenous Pabrinex. She again attempted to self discharge before having a DOLS initiated. Although initially non-compliant with MRI a further attempt was successful. Prior to the report becoming available she was reviewed by gastroenterology and deemed medically fit for discharge after some improvement during the admission. The MRI report revealed central and extra pontine myelinolysis and she was requested to return to hospital. Her symptoms continued to improve and she was discharged with outpatient neurology follow up a week later.

## **Title: Clozapine induced bowel obstruction: An under-estimated severe side effect**

### **Category: Case Reports**

**Main Author: Mauro Culasso**

**Co-Authors: Divya Ramadasan  
Nigel Langford**

*Clozapine induced bowel obstruction: An under-estimated severe side effect.*

*ACUTE MEDICINE DEPARTMENT, LEICESTER ROYAL INFIRMARY, UNIVERSITY HOSPITALS OF LEICESTER*

Clozapine is an atypical anti-psychotic that is used in patients with treatment resistant schizophrenia and those with severe treatment related extrapyramidal symptoms<sup>1</sup>. A common side effect is constipation due to gastrointestinal hypomotility. The prevalence is between 14-60%<sup>2</sup>. This is thought to be mainly due to its anti-cholinergic effects. In the UK there have been a few reports of Clozapine's severe gastrointestinal side effects.

We report a case of severe Clozapine induced constipation that led to bowel obstruction. A 56- year-old gentleman was admitted to the Acute Medical Unit with severe abdominal pain, distension and vomiting. Past medical history included schizophrenia, type 2 diabetes mellitus and depression. He was on Clozapine 500 mg once daily and Pirenzepine 50 mg three times daily. Patient reported chronic constipation previously. Imaging showed gross faecal loading as well as mild right hydronephrosis and hydroureter secondary to faecal compression. The patient was managed with enemas and regular laxatives with good effect.

The case highlighted the importance of recognising Clozapine's less identified side effects as patients on atypical antipsychotics commonly present to acute medicine. There is a documented risk of life-threatening conditions for patients on Clozapine. These include adynamic ileus, gastric outlet obstruction, bowel obstruction, ischaemia, necrosis, perforation and peritonitis<sup>1,3</sup>.

The risk of constipation in patients on Clozapine is increased by a sedentary lifestyle, dietary habits, inability to verbalise their symptoms or provide a history. In addition, health care professionals may underestimate the problem. Previous case reports describe a risk of rapid decompensation once abdominal symptoms have been reported so admitting teams should consider imaging where appropriate. Medication needs to be reviewed and Psychiatry need to be involved<sup>4,5</sup>. Clozapine induced constipation remains an underappreciated side effect and there is no formal guidance on how to treat it.

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## **Title: Diagnostic Challenges of Image-Enhancing Drugs**

**Category: Case Reports**

**Main Author: Ruth Doolin**

**Co-Authors: Emma Crowe**

A 23-year-old Asian male, normally fit and well, presented to ED with a 3-hour history of chest tightness, palpitations and feeling breathless. He denied smoking, drinking alcohol or drug use. On examination the only findings were tachycardia (130bpm) and tachypnoea (28 breaths/min). VBG revealed lactate 6.1mmol/L, glucose 15mmol/L and potassium 2.2mmol/L. ECG showed non-specific inverted T-waves in II and aVL.

Diagnostic uncertainty resulted in treatment for sepsis, pulmonary embolism and ischaemic heart disease (IHD) pending further investigations. Despite this he deteriorated, with persisting hypotension and tachycardia, elevated creatinine kinase (6420ng/m) and rising lactate (to 8.8mmol/L). Care was transferred to ITU and Time-of-Flight (TOF) screen was sent to the poisons laboratory. This revealed presence of Clenbuterol, an illegal  $\beta$ 2-agonist with anabolic properties. He subsequently admitted using 'shakes' from his gym. He was managed supportively and monitored closely after discharge.

### Discussion

Steroids and image enhancing drugs (SIED's) have long been recognised for misuse in the bodybuilding culture, however recreational use amongst image conscious young men is rapidly increasing<sup>(1,2)</sup>. Typically SIED use starts in males aged 18-29,<sup>(1)</sup> however disclosure is limited,<sup>(2)</sup> contributing to difficulties estimating prevalence and diagnostic uncertainty.

Clenbuterol, a  $\beta$ 2-agonist with anabolic properties, is used in this setting for effects on weight loss and muscle mass. Its high and prolonged serum level makes it a desired agent for bodybuilding, and explains the delay in clinical improvement.<sup>(3)</sup> Other  $\beta$ 2-agonist actions increase lactate production and result in sympathomimetic effects causing rhabdomyolysis, hypokalaemia and IHD.<sup>(4)</sup>

As a profession we need to familiarise ourselves with the physiological profiles of common SIED's in order to identify toxicity and prevent over-investigation and over-treatment. Given that rhabdomyolysis is associated with numerous SIEDs<sup>(5)</sup> in acutely unwell young men with unusual symptoms, early testing of creatinine kinase could help guide use of specialist drug screening.

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**Title: Dysphagia - A DISH too far?**

**Category: Case Reports**

**Main Author: Wasim Mir**

**Co-Authors: Ozerah Choudhry  
Helen Sharman  
Syeda Naqvi**

Diffuse idiopathic skeletal hyperostosis (DISH), or Forestier's Disease, is a rheumatological abnormality characterised by widespread bony ossification. Recognised increasingly in the elderly population, we report a case of DISH and its associated complications.

An 81-year-old gentleman was admitted with a fractured neck of femur and underwent hemiarthroplasty. Past medical history included ischaemic heart disease and severe left ventricular systolic dysfunction. During admission he developed urosepsis and was noted to have poor oral intake and dysphagia. Collateral history revealed progressively worsening dysphagia with previous normal OGD. Speech and Language Therapy deemed his swallow unsafe and he was kept nil by mouth. NG insertion proved to be unsuccessful following multiple attempts. After C spine X-ray and discussion with the GP, a diagnosis of DISH was confirmed, with dysphagia secondary to massive osteophytes. Unsuitable for surgery, medical management was deemed appropriate with a 'feed at risk' regimen. Unfortunately, 1 month following discharge the patient was admitted with aspiration pneumonia and died.

DISH is characterized by progressive calcification and ossification of contiguous vertebrae, producing syndesmophytes. Aetiology is unknown and epidemiological data suggests an incidence of 6 – 12%, most common in males between 60-70 years<sup>1</sup>. Presentation includes pain, stiffness, dysphagia, stridor, myelopathy and spinal cord compression. X ray and CT/MRI is the mainstay of diagnosis. The incidence of dysphagia secondary to cervical hyperostosis is estimated at 20%<sup>2</sup>. Dysphagia typically occurs with solids progressing to liquids. The most common sites for osteophytes causing dysphagia are C5-6, C4-5 and C2-3.

Dysphagia secondary to DISH is an under-recognized condition and can result in aspiration pneumonia, severe nutritional deficiency and serious impact on quality of life in the elderly. Management should be of a multi-disciplinary approach, including analgesia, SALT and Dietician review, and consideration for surgical excision. All patients should have discussion of long-term feeding options to prevent complications.

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# **Title: Emphysematous Pyelonephritis - A Case Report**

**Category: Case Reports**

**Main Author: Paarul Prinja**

**Co-Authors: Emma Crowe  
Ozerah Choudhry**

## Introduction

Emphysematous pyelonephritis (EPN) is an acute necrotising infection of the renal system caused by anaerobic uropathogens. The majority of patients have diabetes mellitus. Clinical presentation includes fever, abdominal or flank pain, acute kidney injury, dysuria, nausea and vomiting. Subcutaneous emphysema and crepitus on the flank area may occur in very severe cases.

Organisms most commonly associated with EPN are Klebsiella species, Proteus, Pseudomonas, Streptococcus and as in this case Escherichia Coli. Traditional management has been surgical, however there is increasing evidence basis for good outcomes following medical management.

## Case

A 34 year old lady with poorly controlled diabetes presented to the Emergency Department with Sepsis, Acute Kidney Injury and Diabetic Ketoacidosis. Despite aggressive fluid resuscitation, antibiotics and resolution of her DKA, she remained unwell. She was tachycardic and had a reduced frothy urine output. Urine cultures grew Escherichia Coli and Gentamycin was added to Co-Amoxiclav.

On reassessment, she was found to have a tender left flank, CT abdomen showed an enlarged left kidney, with air present, little remaining normal parenchyma, consistent with a diagnosis of severe Emphysematous Pyelonephritis.

She was reviewed by the urology team and underwent percutaneous stenting with good success.

## Learning points

- Control of diabetes, resuscitative management and minimally invasive treatment have improved outcomes in EPN.
- Where nephrectomy has traditionally been performed, a trial of medical management with drainage should be employed
- There is ongoing need for classification of severity of EPN in order to guide management decisions.
- Whilst CT scanning is the Gold standard imaging modality, the role of bedside US scanning early in admission has yet to be explored.
- Given the increasing evidence supporting equally successful outcomes in medically managed EPN compared to surgical intervention, randomized controlled studies are greatly needed to guide future practice.

## References:

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# **Title: Flecainide intoxication in a patient with Wolff-Parkinson-White syndrome**

## **Category: Case Reports**

**Main Author: Amrita Randhawa**

**Co-Authors: Eilidh McGinnigle  
John Gemmill**

Flecainide is a class 1c-antiarrhythmic drug. It blocks sodium channels in cardiac myocytes.<sup>1</sup> It is indicated for the use of supraventricular re-entrant tachycardias and non-sustained VT in a structurally normal heart. Overdose of this drug is uncommon and often associated with life threatening cardiotoxicity, the management of which is unfamiliar among doctors.

We report a 63 year old female with a history of Wolff-Parkinson-White(WPW) syndrome, who presented after non-accidental ingestion of 1600mg of flecainide with an unknown quantity of alcohol. Six hours post ingestion, she became haemodynamically compromised, complicated by a metabolic acidosis. Her initial electrocardiogram (ECG) showed prolongation of PR, QRS and QTc intervals, Figure 1. This continued to worsen as plasma flecainide peaked, Figure 2. ECG changes demonstrated are more severe than those reported in post mortem case studies. She was resuscitated with normal saline, intravenous magnesium, sodium bicarbonate and lipid emulsion therapy was administered. She showed no obvious response to treatment and her ECGs improved in a time dependant manner consistent with flecainide pharmacokinetics. ECG returned to baseline 30 hours post ingestion (Figure 3). Our patient was discharged after psychiatry consult and referred for accessory pathway ablation.

The half-life of flecainide is 16 hours and it is metabolised in the liver and predominantly excreted by the kidneys.<sup>2</sup> In overdose, haemodynamic compromise affects renal clearance and subsequently propagates toxicity and arrhythmias. The mainstay of treatment is supportive management, administering a sodium load, correcting acidosis and encouraging excretion.<sup>3,4</sup>

There is no antidote for flecainide intoxication and no literature for the management of flecainide toxicity in WPW. This case highlights that even with evidence based treatment, it only led to initial stabilisation while we awaited a fall in plasma levels for clinical improvement. It is difficult to know how she survived these ECG changes given the published cases in the literature.

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**Title: Legal Highs: A Fatal Case of Deliberate Ingestion**

**Category: Case Reports**

**Main Author: Neelsuraj Patel**

**Co-Authors: Alexandra Freeman  
Robert Golding**

A 38-year-old female presented to the Emergency Department at 0200 following a collapse shortly after a deliberate witnessed ingestion of the 'legal high' Columbian. On arrival she was intubated, with oxygen saturations of 85% on high flow oxygen, blood pressure 70/50, heart rate 120 sinus rhythm and GCS 3/15 since the call to the Emergency Services. She was admitted to the intensive care unit for stabilization. A CT brain scan was performed. This showed early evidence of right hemispheric ischaemia. She was managed supportively for three days but showed no signs of neurological improvement. A repeat CT brain scan showed evidence of global ischaemia. The decision was therefore made to withdraw care.

No information was available about this drug. The packet revealed no information about the contents. The National Poisons Information Service was contacted for further information, and supportive therapy was advised.

Urine testing revealed the presence of ethylphenidate, a novel psychoactive substance, acting as both a dopamine reuptake inhibitor and a norepinephrine reuptake inhibitor, with clinical features similar to that of amphetamines and other stimulants.

Altering the molecular structure of drugs while preserving the psychoactive effects allows existing drug laws to be bypassed, producing Novel Psychoactive Substances (NPS), commonly known as '*legal highs*'. There is no testing of these substances or quality control resulting in a wide variation of drug concentration and therefore effects. Substances are often sold as 'bath salts' or plant food. Identification of substances ingested can be difficult.

A recent temporary class order has prohibited the sale and distribution of ethylphenidate, making it a Schedule 1 drug. However cases continue to present to the Emergency Department.

We will describe the mechanism of toxicity, effects, management and methods for detection of ethylphenidate.

Final presentation will include CT images and laboratory data on ethylphenidate testing.

# Title: Meningococcal bacteraemia - not your usual tonsillitis

## Category: Case Reports

Main Author: Marcus Simmgen

### Background

*Neisseria meningitidis*, a gram-negative diplococcus, is much feared for its potential to rapidly cause devastating meningitis and/or septicaemia. Both conditions are associated with high morbidity and substantial mortality and with causing outbreaks in close-living communities. Here we describe an unusual benign course of meningococcal bacteraemia due to tonsillitis, the diagnostic process, management and public health implications.

### Case Report

A 27-year-old male shopkeeper of Turkish origin presented twice within a month with several episodes of fever, malaise and variable headaches. The main clinical finding was tonsillar enlargement, erythema and mild ulceration. Throat swabs showed commensal flora only. He was treated for bacterial tonsillitis and discharged from AMU on both occasions with oral Co-Amoxiclav.

A blood culture subsequently yielded *N. meningitidis* and the patient was called back. He had remained clinically well; meningeal infection was nevertheless actively excluded. All other results from several standard septic screens returned as normal/negative. Intense tonsillar and cervical lymphatic uptake was the only significant finding on a PET-CT scan. Public Health England was notified and proceeded to trace close contacts. As no invasive respiratory procedure had been carried out, no healthcare professional required post-exposure antibiotic prophylaxis.

### Discussion

The detection of *N. meningitidis* in an AMU patient usually requires immediate action to minimise the risk for serious sequelae. It represents a notifiable disease due to its potential virulence; spread occurs via aerosol droplets and secretions<sup>1</sup>. The organism is present in the nasopharynx of 10% of the human population, its only natural host<sup>2</sup>. Meningococcal tonsillitis appears rare<sup>3</sup> and associated mild bacteraemia has been described only once before<sup>4</sup>.

### Conclusion

Repeat microbiological sampling led to the detection of an unusually docile form of meningococcaemia. This consistent approach to fever is essential on an AMU, where more severe presentations of *N. meningitidis* must be suspected and confirmed at the earliest opportunity.

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## **Title: More than just a tight back!**

### **Category: Case Reports**

**Main Author: Ibrahim Bhatti**

**Co-Authors: Alexander Smith  
Toby Graves  
Gaurav Agarwal**

### **Introduction**

Lower back pain probably affects around one-third of the UK adult population each year.<sup>1</sup>

For patients presenting with a new episode of low back pain consideration needs to be given to the possibility that there is a specific cause for their pain.<sup>2</sup>

We describe an unusual cause of severe back pain in a young gentlemen.

### **Case summary**

A 30 year old male admitted with severe acute lumbar back pain which had awoken him from sleep. The pain was localised to the left paravertebral region. Urinalysis was positive for a large amount of blood. A diagnosis of renal colic was made.

CT KUB was reported as normal and no clear cause for patient's pain identified

Patient's pain continued to worsen requiring increasing analgesia.

AST on liver testing was elevated at 378 and a corresponding Creatinine Kinase (CK) was 66,000. The patient denied any illicit drug use or alcohol excess and was not taking any prescribed medications.

Six hours prior to admission the patient had been to the gym after a period of absence and had undertaken a series of dead lifts.

Examination revealed that the left lower paraspinal muscles were tense, very tender and sensation was altered over the area.

Re-evaluation of CT scans revealed an oedematous swollen left erector spinae muscle (fig. 1) which was confirmed on MRI (fig. 2).

A diagnosis of **acute paravertebral compartment syndrome** (PVCS) was made with evidence of rhabdomyolysis.

### **Discussion**

Acute Compartment Syndrome (ACS) has rarely been reported in the paraspinal muscles.

ACS was first described in 1881 by Volkman.<sup>3</sup> ACS is a surgical emergency warranting prompt evaluation and treatment to avoid complications and to preserve muscle function.<sup>4</sup>

Acute exertional paravertebral compartment syndrome (PVCS) has only been reported 15 times previously; notably 11 of these cases involved patients having partaken in lumbar specific weight lifting exercises, such as dead lifts as occurred in our case.

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**Title: Muscle Weakness – A rare initial presentation of Vasculitis.**

**Category: Case Reports**

**Main Author: Sriramprasad Akula**

**Co-Authors: Subhash Rana**

**Introduction:** Vasculitic Neuropathy is associated with C-ANCA vasculitis like microscopic Polyangitis. Muscle weakness is reported but not usually the first presenting symptom.

**Case History:** 49 year old female, who was fit and active presented with 6 day history of muscle aches which she first noticed while decorating her house. The following day she noticed weakness of her limbs which progressed quite rapidly to an extent that she was unable to get out of bed by herself.

Examination revealed diffuse muscle tenderness in both upper and lower limbs. She had power of 4/5 in her upper limb muscle groups and 2/5 at hip, knee and 1/5 at ankle with normal reflexes and no obvious sensory level.

Laboratory investigations showed raised Creatinine Kinase of 2615, troponin 1429 and ESR of 78. ECG showed sinus rhythm with no ischaemic changes. Myositis screen was negative.

Initial impression was polymyositis even though her muscle involvement was atypical and was started on Prednisolone. On day 3 she developed foot drop with worsening weakness and was transferred to tertiary neurology center. Autoimmune screen was positive for C-ANCA with raised PR3 >200 and nerve biopsy showed vasculitic changes.

**Diagnosis:** Mononeuritis multiplex secondary to C-ANCA vasculitis. Her raised CK and troponin were due to muscle infarction rather than myositis and was started on Cyclophosphamide with good effect and was transferred for neuro rehabilitation.

Nerve biopsy: Normal and Vasculitic neuropathy.

Nerve\_biopsy.jpg

**Discussion:** Vasculitic neuropathy is due to inflammation of Vasa nervorum of blood vessels and is commonly seen in C-ANCA associated vasculitis. Isolated involvement of peripheral nervous system is rare and is referred as non systemic vasculitic neuropathy. Mononeuritis multiplex is one of the three presentations of vasculitic neuropathy and is more specific for diagnosis of vasculitis. Nerve biopsy with muscle biopsy is the gold standard for diagnosis.

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**Title: Occlusion of uncommon anatomic variant of thalamic blood supply so called (Artery of Percheron) presenting as bilateral thalamic infarcts**

**Category: Case Reports**

**Main Author: Bayan Mahmood**

**Co-Authors: Suleman Kanani  
Alex Lupu  
Jelena Sergentani  
Daniel Varga**

**Background:**

Two cases of bilateral thalamic infarction are discussed. Both patients presented similarly with decreased consciousness and visual disturbance within a five month period of a stroke rotation.

**Summary:**

Although infarcts restricted to the thalamus were reported for the first time more than 100 years ago by Dejerine and Roussy, they remain an uncommon presentation of stroke and account for only 11% of all vertebrobasilar infarcts. Bilateral thalamic infarction is rare, accounting for only 0.6% of all cerebral infarctions, and results from a combination of predisposing factors and anatomic variations. The pattern of bilateral thalamic stroke is usually seen after paramedian artery infarct of the so called artery of Percheron (AOP); this is a rare anatomical variant of thalamic blood supply arising from the posterior cerebral artery, providing bilateral arterial supply to the paramedian thalami and the rostral midbrain. AOP occlusion accounts for 4-18% of all thalamic strokes and 0.1-2% of all ischaemic strokes.

The thalamus is involved in several functions of the body including regulation of sleep and wakefulness, motor control, receiving auditory, somatosensory and visual sensory signals, and relaying sensory signals to the cerebral cortex. Thalamo-cortico-thalamic circuits are involved in consciousness, arousal, level of awareness, and activity. Patients may take days to weeks to recover from a thalamic infarct and seem to be in a sleep-like state. In these cases, both patients were noted to be sleeping for the entire day and as somnolence cleared, vertical gaze palsy became apparent.

**Conclusion:**

Clinical correlation and appropriate imaging are essential to diagnose bilateral thalamic infarctions. Although it has been reported in literature that it is rare to see this type of infarction, a review of recent case reports and our experience based on these case presentations raise the question of whether bilateral thalamic infarcts are really rare or simply underdiagnosed.

**Title: Oh... My tummy hurts!**

**Category: Case Reports**

**Main Author: Harjinder Kaur Kainth**

**Co-Authors: Vishnu Machineni  
Jagtar Pooni**

Introduction

Abdominal pain is the single most common reason for emergency admission to hospital<sup>1</sup>. Up to 41% of cases cannot be diagnosed in the Emergency Department without further investigations<sup>2</sup>. We present a case of fatal abdominal pain in a young adult.

Case

A 26-year-old, previously fit and well, Asian male was admitted to the Emergency Department having been found on the street with abdominal pain. Due to language barriers, there was very little history and conflicting information with regards to alcohol consumption. There was a presumed two-day history of intermittent abdominal pain and vomiting. Examination revealed marked tachycardia with a normal blood pressure, dehydration and tender upper abdomen with hepato-splenomegaly. Blood tests revealed severe lactic acidosis, grossly deranged liver function and coagulopathy. Renal function and chest x-ray showed no abnormalities.

Subsequent urgent CT abdomen revealed

1. Superior mesenteric vein thrombosis (SMV)
2. Hypoperfusion of the liver (with patent portal vein)
3. Ischaemic small and large bowel, consistent with venous infarction in the SMV territory.

Despite aggressive intensive care management and extensive bowel resection, the patient rapidly deteriorated, developing multi-organ failure (hepatic, renal, metabolic and respiratory) and abdominal compartment syndrome, leading to cardiovascular collapse and subsequent unresponsive cardiac arrest.

Discussion

Mesenteric vein thrombosis accounts for less than 10% of all cases of mesenteric ischaemia; the remainder are arterial<sup>3</sup>. It affects a younger population and the onset of symptoms is usually insidious (e.g. more than 30 days), with complete bowel infarction being rare. Mortality in these cases is usually due to multi-organ failure.

Conclusion

Given the non-specific presentation of mesenteric vein thrombosis, diagnosis is often delayed<sup>3</sup>. In a patient with no obvious cause for abdominal pain and biochemical derangement, urgent investigations should be performed to reach a diagnosis and commence appropriate management before the onset of multiple organ failure and almost-certain fatality.

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**Title: Ovarian Hyperstimulation Syndrome Causes Internal Jugular Vein Thrombosis and Pulmonary Emboli**

**Category: Case Reports**

**Main Author: Kyle Petrie**

**Co-Authors: Rebeca Carter  
Andrew Choong  
Alun Davies  
Audrey Wong**

Ovarian hyperstimulation syndrome (OHSS) is a recognised complication of the induction of ovulation during in vitro fertilisation (IVF). OHSS is the result of a change in vascular permeability that results in a fluid-shift that manifests clinically as ascites and pleural effusion (1). Consequently, a loss of intravascular volume confers a 100-fold increase in venous thrombosis risk (2).

Today, 2% of all births in the UK are a result of IVF (3). As the use of assisted conception increases, the incidence of OHSS continues to rise with an increased rate of presentation to primary and acute care services.

We present the case of a 33-year-old female who was referred to our Acute Admissions Unit with unilateral neck swelling and pleuritic chest pain that ultimately required a superior vena cava filter for multiple pulmonary emboli from an internal jugular vein thrombosis. She had a previous diagnosis of OHSS early in her pregnancy.

This case highlights the need for further education of acute care services to reduce maternal and foetal morbidity and expedite treatment. Further study is required for consensus on the most appropriate management regimens to ensure safe and effective care.

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# **Title: Pyrexia of Unknown Origin as an Atypical Presentation of Large Vessel Vasculitis: The Role of FDG-PET-CT**

**Category: Case Reports**

**Main Author: Rachel Smith**

**Co-Authors: Arun Ramappa  
Mahmood Memon**

## **Introduction**

Pyrexia of unknown origin (PUO) has a wide differential diagnosis, resulting from a spectrum of diseases including infection, malignancy and connective tissue disorders.<sup>1</sup> We present the case of a middle-aged female with a history of persistent pyrexia in the absence of infective symptoms, and the role of FDG-PET-CT in her ultimate diagnosis.

## **Case Report**

A 53 year old Caucasian female was referred to the Acute Medicine Unit from her GP with pyrexia and persistently raised inflammatory markers (C-reactive protein (CRP), 291mg/L) in the absence of obvious infection.

On admission she disclosed a three week history of evening pyrexia, lethargy, and weight loss. She had occasional night sweats but no shortness of breath, cough or haemoptysis, and no recent travel history. Physical examination was unremarkable and routine observations within normal limits. Blood tests revealed anaemia (Hb 99g/dL; 130g/dL six weeks previously), persistently elevated CRP (276mg/L) and erythrocyte sedimentation rate (135mm/h).

Initial investigations were negative; septic screen did not yield source of infection. Throughout admission she had episodes of pyrexia > 38.3°C. Immunology screen was negative, blood cultures were negative; the CT chest, abdomen and pelvis was negative, and trans-thoracic echo was normal. FDG-PET-CT scan was organised to rule out malignancy; instead she was found to have inflammation of the ascending and descending thoracic aortic wall extending into the large aortic arch, compatible with a large vessel vasculitis picture. She was referred to rheumatology and received a course of prednisolone with good response.

## **Discussion**

FDG-PET has been shown to be of diagnostic value in PUO in a review of studies of a combined population of 302 patients.<sup>2</sup> This case reinforces the requirement to systematically investigate PUO and utilise advanced imaging techniques such as FDG-PET-CT to inform diagnosis, particularly in patients with raised inflammatory markers and seemingly no potentially diagnostic clues.

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**Title: Severe case of methaemoglobinaemia. Survival against the odds**

**Category: Case Reports**

**Main Author: Richard Yardley**

**Co-Authors: Ashraf Kamour  
Mark Longshaw  
James Stuart**

**Context:** Methaemoglobin is an altered form of hemoglobin in which ferrous (Fe<sup>++</sup>) ions are oxidized to the ferric (Fe<sup>+++</sup>) state, which is unable to bind oxygen. The remaining ferrous heme binds oxygen more easily, but is less able to off-load it to tissues. Acquired methaemoglobinaemia occurs as a result of drugs or substances that, due to oxidative stress, cause an increase in the production of methaemoglobin. [1]

This particular case is noteworthy in that it represents one of the highest reported methaemoglobin levels (MetHb 90.6%) that anyone has survived. It is also of interest that the patient survived with no long term sequelae, and that only one dose of methylene blue was required to resolve the methaemoglobinaemia.

**Case report:** A 26- year-old female drank a bottle of amyl nitrate labelled as "Liquid Gold" which was allegedly sold at a music festival. The patient believed that the 'liquid gold' was in fact an alcohol shot.

On arrival, she had a blue-grey appearance, with peripheral oxygen saturation reading at 77% on 15L oxygen, heart rate was 122bpm, blood pressure 78/50 mmHg, and GCS 3/15. ECG showed severe global ST segment depression. Arterial blood gas showed pH 7.11, pCO<sub>2</sub> 2.8 kPa, pO<sub>2</sub> 60.1 kPa, Lactate 16.3 mmol/L, Methaemoglobin 90.6%, base excess -21.0. Methylene blue was administered intravenously in a dose of 2mg/kg. Patient clinical condition steadily improved, and the methaemoglobin level progressively declined over the next few hours. She was discharged from the high dependency unit next day.

**Conclusion:** Methemoglobinemia is commonly documented following amyl nitrate usage [2]. Recreationally, though amyl nitrate "poppers" is traditionally insufflated, ingestion cases are also common [3]. Young festival goers in particular need to be aware of the dangers of drinking 'poppers', and physicians need to be aware of the clinical hallmark of methaemoglobinaemia and management options available.

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## **Title: Splenic Injury as a Complication of Colonoscopy: More common than we think?**

**Category: Case Reports**

**Main Author: Peter Ng**

Splenic injury during colonoscopy is thought to be rare. The case reported here would not have been diagnosed if the patient had not presented with a pleural effusion. It is likely that this complication may present with differing severities of clinical features with a significant proportion thought to be due to expected post-colonoscopy insufflation discomfort. This complication is under-recognised and under-reported and therefore is unlikely to be thought of as a diagnosis.

A 63 year old lady had an elective day case colonoscopy but did not seek medical attention for the abdominal pain following the procedure. She presented to hospital 6 weeks after the procedure with dyspnoea and hypoxia and was investigated for a PE. She was diagnosed with a parapneumonic pleural effusion but the CTPA also discovered a splenic haematoma for which there was no other cause in her history other than the colonoscopy 6 weeks prior. By this time, she had no clinical features from her abdomen and she was therefore managed conservatively.

This complication is well documented in the literature and is primarily thought to be due to excessive traction on the splenocolic ligament. Several risk factors for developing this complication have been proposed. However, with the increasing use of colonoscopy for cancer surveillance it is likely that this will be seen more frequently and begs the question about whether this potentially fatal complication should be discussed when obtaining consent.

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**Title: Spontaneous Pneumocephalus – A Rare & Unusual Presentation Mimicking a Subarachnoid Haemorrhage on the Acute Medical Take**

**Category: Case Reports**

**Main Author: Dayle Terrington**

**Co-Authors: Elena Purcaru  
Chirag Patel  
Nicky Ueckermann  
Zaheer Babar  
Harith Altemimi**

Presentation with a headache and suspected subarachnoid haemorrhage (SAH) is a common scenario in the acute medical assessment unit. As a result such patients often undergo a CT scan followed by a diagnostic lumbar puncture to assess for the presence of xanthochromia if the scan excludes an obvious SAH. We present a rare and unusual cause for a patient presenting with a sudden onset headache secondary to spontaneous pneumocephalus which mimicked a SAH.

A 62-year-old gentleman with a background of Barrett's oesophagus, bronchiectasis and previous sinusitis was referred by his GP to the medical assessment unit with a suspected SAH. He initially presented with a severe sudden onset frontal headache which developed three days previously following an episode of sneezing whilst leaning forward. The headache persisted over the course of three days and worsened with coughing and straining. Apart from this he remained well and denied any further symptoms or history of trauma. A full clinical examination was unremarkable and routine bloods all remained within normal parameters. In light of his history an urgent CT head was organised and demonstrated extensive pneumocephalus with pneumatisation of the occipital bones and C1 vertebral body with surgical emphysema noted over the posterior neck. There was no radiological evidence of a SAH or space occupying lesion.

Neurosurgical advice was sought from a tertiary centre and he was subsequently managed conservatively with analgesia and follow-up imaging arranged.

Pneumocephalus is a rare entity and is defined as the presence of air within the cranial cavity which maybe idiopathic in aetiology or secondary to craniofacial trauma, base-of-skull tumors or post-operatively following neurosurgery<sup>1</sup>. Clinically, the condition often mimics a SAH presenting with a thunder-clap headache after coughing, sneezing or valsalva maneuvers, and is therefore an unexpected radiological finding which can be challenging to diagnose and manage due to its rarity.

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<sup>1</sup> Santiago G. Abbati et al. Spontaneous intraparenchymal otogenic pneumocephalus: A case report and review of literature. Surg Neurol Int. 2012; 3: 32.

**Title: The Management of Acute Airway Obstruction- Assessment and management of a rare cause of acute airway obstruction**

**Category: Case Reports**

**Main Author: Kate Skinner**

**Co-Authors: Natalie Walker**

Objective: To describe the approach to managing acute airway obstruction in adults, forming a key part of the AIM curriculum<sup>1</sup>

Design: Case report and review of the literature

#### Case Report

A 52 year old male with known alcoholic liver disease presented with confusion and vomiting. On admission bloods showed acute kidney injury and raised calcium of 5.16 with no clear cause. This was treated with fluids and pamidronate and investigations commenced. 24 hours after admission he developed sudden acute airway obstruction with trismus. This was unresponsive to initial treatment including adrenaline IV, hydrocortisone IV and magnesium IV with simple airway manoeuvres in place and resulted in an emergency tracheostomy being performed on the Acute Medical Unit (AMU). CT later demonstrated a large squamous cell carcinoma of the tongue base with lymphatic spread explaining the physical trismus and initial presentation. Unsuitable for active treatment he was discharged to a hospice.

#### Discussion

This extremely rare case demonstrates why airway management is a key part of the acute medical curriculum. It illustrates the fundamental skills a doctor requires to recognise and manage acute airway obstruction in order to prevent mortality and morbidity. Acute airway obstruction is a serious and life threatening presentation more commonly presenting in children. In adults causes include vomit, intra-luminal tumours, head injury, alcohol and external compression.<sup>2</sup> Management can range from simple airway adjuncts and oxygen therapy to the complete spectrum of difficult airway management including a surgical airway.<sup>3</sup> The management is largely algorithm led although as with this case provisions are made to move off algorithm depending on local skills.<sup>4</sup>

#### Conclusion

Acute airway obstruction is a time critical medical emergency which must be managed effectively and escalated both through the algorithms and to senior staff. Recognising acute airway obstruction is a vital skill for acute physicians and trainees.

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**Title: Think Twice Before Burn Out**

**Category: Case Reports**

**Main Author: Ana Catana**

**Co-Authors: Vinnamala Sudheer  
Elena Craescu**

**INTRODUCTION**

Dinitrophenol-DNP-is a cellular metabolic poison that is a precursor for TNT and shares a similar structure,it was previously used as an explosive. Available as an unlicensed weight loss and body building agent, easily obtained via the internet and highly toxic. Is absorbed by ingestion,inhalation and via skin.

**CASE**

A 28 year old female of Eastern European background self presented to the admission unit with SOB for two days,profuse sweating and vomiting.She gave no history of iv drugs but was going to a gym where she had obtained diet pills.Despite of BMI 23 she gradually increased dosage of the pills with 3 more daily,not being aware of the consequences,with an intention to lose weight.She gave no previous history of psychiatry issues. On examination she was drowsy but orientated in time and place. Had profuse sweatiness and tachypnea. Her HR was 125, BP 115/70 and Sats 98%.Had a clear CXR and ECG showed sinus tachycardia. Her bloods were normal including CRP and DDimer but had mild renal impairment.Over the next few hours she gradually deteriorated and became hypotensive 80/50 with RR45. At this stage she was transferred to ITU for arterial line and aggressive fluid resuscitation.Appropriate advise was taken from ToxBase regarding decontamination and decided that no sideroom is required. In day three she developed rhabdomyolysis with CK of 7000 which improved with fluid management and didn't required haemodiafiltration. With close monitoring she improved in 24 hours and was ready for discharge. She is now a happily pregnant lady with her first child.

**DISCUSSIONS**

-This is a case of a normal female with BMI 23 who was not aware of potentially life threatening consequences of the unlicensed diet pills

-She developed rhabdomyolysis, hypotension and profuse sweatiness which can lead to death if treatment is not initiated in time

-High level of clinical suspicion with close monitoring is necessary in order to treat these cases

-Need for changing and monitoring the early purchase of these pills over the internet

**References**

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## **Title: Thinking about eosinophilia in Acute Medicine**

### **Category: Case Reports**

**Main Author: Iona Gilmour**

**Co-Authors: Elisa Gonzalez Garcia  
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- 67 year old female
- PMH: Asthma under antileukotriene treatment, sinusitis. Otherwise fit and well, non-smoker.
- HPC: 2 week history of intermittent chest and back pain. Progressive shortness of breath on exertion over past 6 months, dry cough. Recent transient loss of vision lasting 3-4 minutes.
- Examination findings: pan-systolic murmur loudest over mitral area with radiation into axilla. Reduced air entry at both bases.
- CXR: Globular heart, prominent hilar markings.
- Blood tests: Eosinophils:  $4.28 \times 10^9/L$  ( $0.0-0.8 \times 10^9/L$ ) = 40% of WCC, Troponin I: 126ng/L (
- Differential diagnosis: 1) Allergic bronchopulmonary aspergillosis 2) Hypereosinophilic syndrome 3) Autoimmune/vasculitis.
- Further investigations: 1) CT chest: bilateral pleural effusions, pericardial effusion, widespread ground glass opacities. 2) Echocardiogram: Severe mitral regurgitation. 3) Full autoimmune/vasculitis screen: p-ANCA positive.

### **Eosinophilic granulomatosis with polyangiitis [EGPA]**

- Vasculitis of small and medium sized arteries.
- Cardinal features: asthma, chronic rhinosinusitis and prominent eosinophilia.
- Typically three phases over a 20 year period: Prodromal, Eosinophilic, Vasculitic.
- Multi-system disorder, most commonly affecting the lung, followed by skin.
- Cardiovascular manifestations cause approximately one half of deaths.
- Valvular insufficiency related to myocardial fibrosis is well recognised.
- ANCA found in 40-60% of patients, of these 70-75% are p-ANCA positive.

### **Diagnostic criteria – 1 example**

#### *American Rheumatology Association criteria*

1. Asthma
2. Eosinophilia >10% of differential
3. Neuropathy
4. Pulmonary opacities
5. Paranasal sinus abnormality
6. Extravascular eosinophils found on biopsy

4 out of 6 criteria to be met (sensitivity 85%, specificity 99.7%)

Stabilize the patient and make a proper differential diagnosis from minute one based on an acute systemic presentation with marked eosinophilia, was the challenge. Patient was admitted in Cardiology; Rheumatology referral made one month following discharge when p-ANCA returned positive, despite meeting 5 out of 6 of the criteria (suspected transient ischaemic optic neuropathy). Currently being referred for mitral valve replacement. Learning point: early recognition of vasculitis presentation is crucial, particularly with regards to multi-organ involvement. Referral to rheumatology should not be delayed if suspected on clinical grounds.

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**Title: Toxic Shock Syndrome**

**Category: Case Reports**

**Main Author: Tamer Shalaby**

**Co-Authors: Alexander Keough**

**Learning Points:**

1-High index of Suspicion of S. Aureus TSS in menstruating ladies with tampons who present with febrile illness with no obvious source of infection.

2-Hypotension and MOF occur within hours

3-TSS may be resistant to aggressive fluid and antibiotic therapy.

4-Blood (C+S) is usually negative in TSS.

5-The role for clindamycin and IVIG

**Case Presentation:**

45 year old female presented to A&E been generally unwell for the past 12 hours. On prednisolone and mycophenolate mofetil for an autoimmune disease.

Normal examination save temperature of 39.7°C and tachycardia at 110. Tazocin and fluid resuscitation given. However, Two hours later her systolic BP reduced to 60. Nursing staff reported that there was a tampon with green discharge – which grew s. aureus later but negative blood C+S.

In ICU she was started on meropenem, clindamycin and IVIG. Required high doses of noradrenaline and Vasopressin after aggressive fluid resuscitation; subsequently haemofiltration after hours of oliguria and worsening renal function. Good recovery after 7 days.

**Discussion:**

Strains of s. aureus producing TSST-1 which is a superantigen that interact with T Cells to induce massive cytokine production including TNF-alpha, TNF- Beta, (IL-1, IL-2, IL-6) <sup>1-3</sup>.

Vaginal colonisation followed by penetration of a sufficient concentration of TSST-1 across the epithelium; abrasion from tampon use.

Fever >38.9, GIT disturbance, macular erythema involving palms and soles, mucous membrane hyperaemia<sup>4</sup>. Hypotension with MOF occurs usually within 72 hours with significant mortality rate<sup>5</sup>.

Clindamycin is superior to penicillin because of its potency in suppressing bacterial toxin synthesis<sup>1,6</sup>. (IVIG) therapy has showed anti TSST-1 effect<sup>6, 7</sup>.

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**Title: When being all right is not alright**

**Category: Case Reports**

**Main Author: Harriet Nerva**

**Co-Authors: Phillip Copley**

A 36 year old Caucasian gentleman presented in shock, with a 2 day history of malaise, and sudden severe epigastric pain and haematemesis on the morning of admission. Born with a univentricular heart, he had undergone childhood cardiac surgery to fashion a Fontan circulation, necessitating lifelong warfarin. He described no other past medical or surgical history. This hospital did not hold his medical records, as had returned to the country only briefly to visit family here.

On examination, a facial petechial rash was noted. Haematological investigations revealed leucopenia, thrombocytopenia and severe disseminated intravascular coagulation. After commencement of broad spectrum antibiotics, fluids and blood products, a surgical review was requested and a CT chest, abdomen and pelvis was performed. This showed no pathology, but revealed right isomerism situs abnormality and absence of a spleen. Howell-Jowell bodies were present on his blood film and he was diagnosed with overwhelming sepsis of unknown cause secondary to asplenia. He deteriorated and, despite intensive care, died 20 hours after admission. After death, polymerase chain reaction confirmed a pneumococcal septicaemia.

Congenital asplenia is rare, but is a well-known risk factor for overwhelming sepsis. Asplenic patients should carry an emergency supply of appropriate antibiotics for emergency use and require regular vaccinations against *Pneumococcus* and *Haemophilus influenzae*. Unusually, to the best of our knowledge, this patient was unaware that he was asplenic, leaving him highly vulnerable to infection. This case highlights the many diagnostic challenges faced in acute medicine when limited information is available. We briefly review the mechanisms underlying the development of situs abnormalities during the gastrulation period of development, and their association with asplenia. The recommended investigations and management of situs abnormalities and asplenia are outlined, with emphasis on prompt identification of these patients in the acute setting.

**Title: When is a stroke not a stroke? An unusual mimic presenting to AMU**

**Category: Case Reports**

**Main Author: Shyamal Patel**

**Co-Authors: Nicholas Smallwood  
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Creutzfeldt-Jakob disease (CJD) is a rare prion disease which classically manifests with rapidly progressive dementia, abnormal movements (myoclonus) and typical electroencephalographic (EEG) changes.<sup>1</sup> It is a challenging diagnosis to make due to variability of initial symptoms and rapid progression leading to patients often being misdiagnosed or identified in the end stages.<sup>2,3</sup>

We describe the case of a 74 year old Caucasian man with a background of hypertension and hypothyroidism. Prior to admission he was recently discharged from a district general hospital with a diagnosis of stroke, based on a history of unsteadiness, dysphasia, new onset right arm tremor and consistent diffusion-weighted MRI changes. He re-presented to our acute medical unit seven days later with worsening dysphasia and general reduction in his mobility, and stroke remained the working diagnosis. Further history revealed a progressive cognitive decline and myoclonic movements of the right arm which were first noted a month prior to his admission, and this raised the possibility of an alternative pathology. He underwent a further MRI scan which revealed abnormal high signal on DWI and FLAIR in the caudate head and anterior putamen (figure 1). An EEG was performed which showed periodic sharp wave complexes over the left fronto-centro-temporal regions, and CSF analysis revealed the presence of protein 14-3-3 and protein S100. Put together, these investigations and history supported the diagnosis of CJD, confirmed by the national surveillance unit in Edinburgh. Unfortunately, the patient declined rapidly and died while an inpatient. On review, there was no evidence of previous stroke.

**Conclusions/learning points:** Even with an apparently diagnostic MRI, symptoms of stroke with a history of progressive cognitive decline and particularly myoclonic movements should raise the possibility of prion disease. Although rare, early diagnosis prevents unnecessary investigations and treatments and allows appropriate palliative care input early, where appropriate.

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