A case of delayed traumatic haemorrhage on AMU

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Superior gluteal artery injury is relatively rare, and is most frequently associated with fractures of the pelvis or penetrating trauma\(^1\). It can however lead to life-threatening haemorrhage.

We report the case of a patient who presented following a fall from the top to the bottom of a flight of stairs. A full trauma assessment, including computed tomographic (CT) imaging from the patient’s head to the pelvis, revealed no significant bony or soft tissue injury. A small left gluteal haematoma was noted without active bleeding. During her stay in the Emergency Department (ED) she experienced a transient hypotensive episode and subsequent investigation revealed bilateral pulmonary emboli. She was transferred to the Acute Medical Unit for therapeutic anticoagulation with low molecular weight heparin (LMWH) and further management.

Over the following 24 hours the patient experienced more hypotensive episodes, with her haemoglobin falling from 111 g/L to 73 g/L. Re-examination demonstrated significant expansion of the gluteal haematoma. Interventional Radiology input was requested regarding insertion of a temporary inferior vena cava (IVC) filter. The Consultant Radiologist additionally recommended a CT angiogram of her pelvis to identify any source of bleeding. This revealed a left superior gluteal artery false aneurysm on the periphery of the haematoma. Urgent coil embolisation of the false aneurysm was undertaken, and confirmation angiography displayed haemostasis. The IVC filter was then sited. The patient recommenced LMWH 2 days later, and had an uneventful recovery, before being discharged to a community rehabilitation hospital.

This case serves to remind Acute Physicians to remain vigilant about the risk of haemorrhage in trauma patients despite being fully assessed by our colleagues in ED, particularly if anticoagulation is commenced. Additionally, this case highlights the importance of seeking early advice from our colleagues in Interventional Radiology, whose intervention enabled the patient to restart LMWH with minimal delay.

A case of empyema complicating cannabis-induced bullous lung disease

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Cannabis is the most frequently used illegal drug in the United Kingdom\(^1\), although the health risks associated with cannabis smoking are often under-appreciated.

**Case**
A 54 year old male presented to the acute medical take with a three-week history of left-sided pleuritic chest pain associated with cough, fever, orthopnoea and worsening breathlessness. He had recently returned from holidaying in Jamaica. His past medical history included bullous emphysema, complicated by two episodes of right sided spontaneous pneumothorax, culminating in surgical bullectomy four years previously. He reported an extensive history of cannabis smoking.

The patient was tachypnoeic, with pulse 100 beats/minute, blood pressure 120/80 mmHg and oxygen saturations 80% on air, temperature 38\(^\circ\)C. Examination revealed decreased air entry throughout the left lung, with dullness to percussion over the left mid-zone and base. An arterial blood gas showed a PaO\(_2\) 7.62kPa, PaCO\(_2\) 5.1kPa. The admission chest radiograph revealed a left apical bulla containing an air fluid level, a pleural effusion at the left base and rightward mediastinal shift (Figure 1). Oxygen therapy and intravenous antibiotics were commenced.

CT thorax confirmed a large left apical bulla with an air-fluid level, and loculated effusion (Figure 2). Ultrasound-guided pleural tap confirmed an empyema, necessitating drainage. His symptoms improved, and he subsequently underwent a successful open resection of the infected bulla.

**Discussion**
A higher incidence of respiratory complications are noted amongst cannabis users\(^1\) including spontaneous pneumothorax\(^2\), emphysematous lung disease\(^3\) and respiratory infection\(^1\). This is at least partly attributable to differences in the method of smoking (larger inhalations, longer breath-holding, no filters), a higher carcinogen content compared with tobacco\(^4\), and tetrahydrocannabinol-mediated immune suppression\(^1\). Whilst high profile media campaigns press for cannabis legalization, often trivializing its side-effects, this case highlights the complications associated with long-term cannabis use.

**References**
A case of Euglycemic Diabetic Ketoacidosis in a Pregnant Type 2 Diabetic

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Introduction:
Diabetic Ketoacidosis (DKA) is a life threatening condition during pregnancy. It carries high mortality (up to 30%) for the fetus and the mother. Ketosis has been implicated in fetal distress and causes adverse neurological outcome. During pregnancy, euglycemic diabetic ketoacidosis is reported in patients with diabetes and warrants prompt treatment. This case report illustrates a patient with type 2 diabetes and euglycemic DKA.

Case Description:
A 34 year old Asian lady diagnosed with type 2 diabetes 3 years ago postnatally, following gestational diabetes during her last pregnancy. Preceding the current pregnancy, she was on Metformin only. She was treated with Metformin and Insulin during this pregnancy with excellent glycaemic control (HbA1c 37mmols/mol).
She self-presented at 36 +2 weeks with history of vomiting for 3 weeks. She had not taken her metformin and insulin for 2 days. She was clinically dehydrated.
Investigations revealed admission capillary blood glucose of 4.8mmol/l. Urine ketones were 4+ and Blood ketones 2.6. She was acidic (pH 7.28) with a HCO3 of 11, Lactate 0.9. FBC, U&E, LFT and cardiotocography for fetal monitoring were normal.
The patient was initially treated with intravenous fluid rehydration only and insulin infusion was deferred because blood glucose was normal. Her acidosis failed to improve. Hence diagnosis of Euglycemic Diabetic Ketoacidosis was made and intravenous insulin infusion was started. She recovered very well and was discharged home after 4 days with Insulin alone as her treatment.

Discussion:
Euglycemia does not rule out diabetes as a cause of ketoacidosis, especially during pregnancy. Therefore, treatment should be promptly started in accordance with the guidelines of management of diabetic ketoacidosis promptly.
DKA is a possibility in patients with type 2 diabetes and pregnancy and could present with non-specific symptoms and normal blood glucose. Prompt diagnosis and management with intravenous insulin along with dextrose will prevent significant morbidity for both the mother and the fetus.
A 22-year-old female with poorly-controlled type 1 diabetes mellitus (T1DM) for the past few years, presented with a history of right upper-quadrant abdominal pain, nausea and vomiting for 1 week. Physical findings included dehydration and tender hepatomegaly. Blood gas analysis confirmed diabetic ketoacidosis (DKA) and she was managed according to the local DKA protocol and the DKA resolved soon. Her Liver Function Tests (LFTs) were deranged, with moderate transaminitis for nearly 2 years. An Ultrasound scan of her abdomen showed coarse liver echo pattern suspicious of fatty liver. A liver disease screen (autoimmune, viral and metabolic) was negative. The pattern of her abnormal LFTs paralleled with elevated glycosylated haemoglobins and the abnormal lipid profile for the past few years (table).
With a presumptive diagnosis of glycogenic hepatopathy as a cause of her liver dysfunction, she was managed with strict glycaemic control with insulin and diet that improved her abdominal symptoms before discharge in 4 days.
On a subsequent outpatient clinic review, four weeks later, her glycaemic control, abnormal LFTs and lipid profiles, were remarkably improved (table) with the resolution of the right upper quadrant pain and hepatomegaly.
Glycogenic hepatopathy is an under-recognised complication of poorly-controlled T1DM. Persistently elevated blood glucose levels result in increased glycogen production and storage in liver that cause hepatocellular injury.1,2,3 Complete resolution of liver dysfunction occurs with prompt control of diabetes.1,2 Unlike non-alcoholic fatty liver disease, a hepatic complication of type 2 diabetes due to fat deposition that leads on to liver fibrosis in many affected patients,4 glycogenic hepatopathy does not cause fibrosis. Glycogenic hepatopathy can be histologically confirmed by liver biopsy, however limitations and morbidity associated with the invasive procedure often precludes biopsy. Good glycaemic control as demonstrated in this case, mostly resolves the abnormal LFTs.

References


* Kindly see attached file for tables.
Acute Acalculous Cholecystitis and Pericarditis as Rare Complications of Q Fever

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Q fever is a zoonotic disease caused by Coxiella Burnetii, typically associated with exposure to farm animals. According to Public Health England, Q fever is underdiagnosed and has outbreaks potential. We present the case of a 40 years old man admitted with acute acalculous cholecystitis (AAC) and pericarditis caused by Q fever. This, to our knowledge, is the first such case reported in the English literature.

The patient was an electromechanical engineer who repaired pumps for farmers and zoos. Apart from his dog, he had no direct contact with animals, also denied foreign travel.

He was admitted with a three weeks history of malaise, high fever, anorexia, weight loss, generalised myalgia and arthralgia, and upper abdominal pain.

He was pyrexial with a temperature of 40°C and upper abdominal tenderness on examination. Blood tests showed raised inflammatory markers with CRP 350, hyponatraemia 125, hypoalbuminaemia 24, mild anaemia 124, severe thrombocytopenia 11, initially normal WCC though subsequent neutrophilia and lymphocytosis, raised LFT with ALP 162, GGT 422, ALT 121, bilirubin 50. A CT scan of abdomen showed features compatible with AAC. Echocardiogram revealed a small pericardial effusion.

Initial treatment with broad spectrum antibiotics was not successful in resolving his illness. However, his extensive microbiological tests showed strongly positive serology IgM by CFT for acute Q fever. He was treated with a 21 day course of Doxicycline leading to restoration of health. Repeat interval serology will be undertaken to screen for chronicity.

In summary, we present a case of severe AAC and pericarditis associated with acute Q fever. Raised LFT and hyponatraemia are common, with thrombocytopenia occurring in about 25% of patients. His occupation of working with farmyard pumps may have led to exposure to this zoonosis caused by Coxiella Burnetii. Clinicians should be vigilant of Q fever in patients with such symptoms.
Pyomyositis is defined as a subacute purulent infection of skeletal muscles, which rarely occurs in non-temperate climates. Involvement of the piriformis muscle has rarely been reported in the literature. We present a case of pyomyositis affecting the piriformis muscle presenting as an emergency to a medical assessment unit.

A previously fit 18 year old female presented with a three day history of pyrexia, vomiting and right lower back pain radiating down the posterior thigh following a recent self-limiting throat infection. There was no significant past medical history or recent travel. Initial examination revealed a tachycardia of 118 beats-per-minute, pyrexia of 40.5°C, non-specific lower abdominal and right buttock tenderness. Neurological examination revealed reduced proximal power in the right leg as a result of pain and a hyporeflexic ankle jerk. Rectal examination was normal. Blood tests revealed a WCC of 23.0x10⁹/L and CRP of 312mg/L, and were otherwise unremarkable including creatine kinase. Streptococcus pyogenes was isolated from blood cultures. Autoantibody and HIV tests were negative. A CT scan of the abdomen and pelvis initially showed no abnormality, however when re-reviewed identified mild enlargement of the right piriformis muscle. An MRI scan demonstrated focal inflammation and oedema within the piriformis muscle with proximity to the sciatic nerve. Gynaecological assessment revealed no abnormalities; laparoscopy excluded a pelvic source of infection and normal appendix.

Full recovery occurred after completing seven days intravenous antibiotics therapy with penicillin and clindamycin followed by oral amoxicillin for two weeks, in combination with analgesia and physiotherapy.

Piriformis pyomyositis is a rare infection often proceeding to abscess formation and mimicking sciatica due to its close proximity to the sciatic nerve. In this case, haematogenous spread from a streptococcal throat infection seems the most likely source. Thus, a high index of suspicion is required in any patient presenting with sepsis and sciatica.
An unusual cause for white out on a chest x-ray.
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CASE REPORT
A 73 year old lady presented to hospital with a 3 month history of increasing shortness of breath. She reported no associated chest pain, cough or weight loss. She was known to suffer from atrial fibrillation, and hypertension. Her current medications were Warfarin, Atenolol, and Bendroflumethiazide. She was an ex-smoker having quit 40 years ago. Initial clinical examination showed a decreased air entry of her left lung field which was dull to percussion; the remainder of her clinical examination was unremarkable. Her chest x-ray (figure 1) was reported by radiology as a large left sided pleural effusion. However, a blind diagnostic tap was unsuccessful. Subsequent CT scan demonstrated a pleural based mass along the left lateral chest protruding into the pleural space causing passive collapse of the left lung and a right mediastinal shift. The mass showed homogeneous fat attenuation, with collateral vessels arising from the left internal thoracic vein within the mass (figure 2). The scan findings were consistent with large lipoma in the left hemithorax and small lipoma in the right posterior right hemithorax. She was referred to a tertiary centre with eventual surgery to remove her lipoma.

Discussion
Lipoma is a benign tumour of the subcutaneous tissues. Pleural lipomas are very rare, and grow very slowly. Patients usually present at 40-60 years of age. Lipomas are usually asymptomatic and detected incidentally with a chest x-ray. At later stages of disease patients present with dyspnoea and chest heaviness. Surgical resection, if possible, is the preferred mode of treatment for therapeutic and diagnostic purposes (1-4).
This case reminds the clinician that there are multiple causes for white outs on a chest X-ray and emphasises the importance of using ultrasound guidance for all pleural aspirations.

Consent
Written consent has been obtained from the patient.

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A 41 year old lady presented to ED with pins and needles and weakness radiating down her right arm. She had been indoor skydiving where she was supported by air with no harness. She was adamant that no injury or trauma was sustained during the indoor skydive. Upon leaving, she immediately noticed pins and needles in her right hand, particularly in her thumb, index, and middle finger. Her arm was ‘dead’; she was able to lift it slightly, but stated she could not really use it. She had no neck pain.

She had no headache and visual symptoms. She had no bowel or bladder symptoms. She had no significant past medical history and was not on any medications.

On examination she was unable to raise her right arm. Her C4 dermatome appeared intact; but she complained of numbness, particularly in the outer and medial aspects of the right arm. Sensation at the level of T2/T3 was intact, but there was significant numbness of the thumb and index finger. There was a reduced biceps and supinator reflex, but triceps reflex was adequate. There was reduced flexion of the right arm.

She was diagnosed as having a C5-C6 brachial plexus injury. She was managed through ambulatory care with physiotherapy and one week later her symptoms were improving.

C5-C6 injuries are rare manifestations of brachial plexus injuries. Patients may present with or without shoulder trauma, and the rapid diagnosis of the injury may lead to quicker treatment reducing the need for more invasive investigations. This is the first reported case of a patient sustaining a C5-C6 brachial plexus injury as a result of indoor skydiving. This form of peripheral neuropathy should be explored further in the context of indoor skydiving as it may form an additional basis for informed consent of this activity.
Cellulitis caused by Nocardia brasiliensis infection in an immunocompetent patient: Report of a rare case

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Case history:
We report a case of a 81-year-old woman who presented with five days history of skin erythema on her left calf after travelling from California to Yorkshire. Two days prior to the onset of symptoms she reported injuring her leg on an Agave cactus thorn while she was gardening in California, USA. Two days after the injury, she returned to the UK and was prescribed oral flucloxacillin in primary care. The inflammation in her leg continued to spread. Three days later she was admitted to the hospital. On examination, there was extensive cellulitis with blistering and swelling of the leg (image1). Blood tests showed C-reactive protein of 238 mg/l and white cell count of 11.1 x 10^9/L. The blisters were deroofed and swabbed, fluid and tissue sent for culture and the patient was started empirically on piperacillin/tazobactam, clindamycin and teicoplanin. Gram stain of the tissue and fluid showed no organisms. After 48 hours incubation all three samples grew small creamy colonies of filamentous, catalase positive, gram positive rods. These were found to be weakly acid fast by modified Ziehl-Nielsen staining. The organism was identified as a Nocardia sp by MALDI-TOF. 16S RNA PCR confirmed that the isolate was Nocardia brasiliensis. Her antimicrobial treatment was then switched to meropenem and cotrimoxazole 960mg tds intravenously for one week and she was discharged with a plan to complete three months of oral cotrimoxazole. The lesions on her legs are improving (image2).

Conclusion:
Cutaneous Nocardia infection following cactus thorn injury is well described in the American literature. The relative ease of global travel ensures that patients may import exotic infections into the UK and that a travel history should always be sought. Disease in immunocompetent patients is rare and primary cutaneous infection is uncommon. Clinical outcome is good though a prolonged treatment may be required. MALDI-TOF enables microbiological isolates to be identified more rapidly, accurately and cheaply than immunological and biochemical methods and is likely to become the standard technique for identifying microbes over the next few years.*

Chronic central venous access can be associated with acute complications that typically present to the acute medical service, especially out of hours. We present the case of hepatic pain and dysfunction due to an internal jugular venous dialysis line tip which had migrated into the liver parenchyma.

Case: A 35 year old lady with a background of IgA Nephropathy on haemodialysis four days a week, presented with worsening right upper quadrant pain and mild hepatic dysfunction. She had a previous failed cadaveric renal transplant and multiple failed arteriovenous fistulae. As a result she haemodialysed through a tunnelled central right internal jugular venous catheter. The catheter had been inserted 4 months earlier without any complications. The dual-lumen single sheath line was of a standard length.

Blood tests showed an ALT 450mmol/L and ALK phos of 190mmol/L. The remainder of the liver, renal and inflammatory profile were normal or as expected for a chronic dialysis patient. The toxicology and blood borne viral were also normal. Imaging revealed a normal ultrasound appearance of the liver and biliary tree. The chest xray was also normal. The patient had a CT abdomen which showed that the distal tip of her tunnelled dialysis catheter was situated in her right portal vein and causing a compromised hepatic vein blood flow (2 images available).

In retrospect, chest x-ray examination preformed for acute attendances with bronchopneumonia had shown the further internalisation of the dialysis catheter (3 images available).

The patient was not aware of the line migrating internally as the proximal exposed end had not become shorter. The dialysis line was subsequently removed and exchanged for a new dialysis catheter at the same site. Immediately following the procedure the patients settled and within days the liver function improved to normal limits.

Conclusion: The case highlights the need to consider the consequences of iatrogenic intervention as causes of acute pathology, and to thus have a wide differential diagnostic pool.

**Patient consent:** Written consent evidence can be provided on acceptance
A 46 year old female attended ambulatory emergency clinic (AEC) complaining of a non-resolving sore throat of two weeks duration with an associated left sided neck mass. Her GP diagnosed tonsillitis in the community and started oral antibiotics, but due to continuing symptoms the patient presented to hospital. On examination there was erythema of her tonsils with no purulent exudate. The mass on the left side of her neck was firm, warm and exquisitely tender on palpation.

Blood tests revealed a neutrophilia with associated elevated CRP. Initial chest X-ray showed bilateral opacities in her left upper and right lower lobes. She went on to have a CT thorax/neck which revealed two left upper lobe opacities with air bronchograms and a right lower lobe cavitating lesion. The scan also revealed multiple lymph nodes in the neck, a subcutaneous gelatinous abscess on the left side of her neck and a left internal jugular vein (IJV) thrombosis.

With the antecedent pharyngitis, persistent fever, IJV thrombosis and septic pulmonary emboli she was diagnosed with Lemierre’s syndrome. Lemierre’s syndrome characteristically describes acute oropharyngeal infection with subsequent development of septic thrombophlebitis of the IJV resulting in metastatic infection. Prompt diagnosis is vital for appropriate antibiotic therapy to cover the most common bacterial causes of Lemierre’s syndrome, oral commensal anaerobes most commonly fusobacterium necrophorum. Although common in the pre-antibiotics era the use of antibiotics to treat bacterial pharyngitis led it to be known as the forgotten disease in the 1980s, however it’s incidence has been increasing in recent years such that it has now become important to bear in mind. Our patient received 28 days of IV Ertapenem with oral Clindamycin and was warfarinised for three months. On last follow-up her symptoms had resolved and her blood tests and CXR had normalised.

References
The case
A 35 year old lady presented for induction of labour. She had no past medical history and no medications. Her pregnancy had been uneventful.
Four hours after induction she developed headache, left hemiplegia and a reduction in consciousness. CT brain revealed a right frontoparietal intracranial haemorrhage (ICH) and she was transferred to her nearest neurosciences centre. CT angiogram was normal and she underwent emergency caesarean section and evacuation of the ICH.
Digital subtraction angiography demonstrated widespread variability in the diameter of the intracranial vessels (figure 1) and Reversible Cerebral Vasocostruction Syndrome (RCVS) was diagnosed. She was treated with nimodipine, labetolol and methylprednisolone.

RCVS
RCVS is defined as severe headaches, with or without other neurological symptoms and diffuse segmental constriction of cerebral arteries that resolves spontaneously within three months [1]. Mean age at presentation is 45 years, with a female preponderance. The thunderclap headaches usually last 1-3 hours, have a trigger and occur over 1-4 weeks [1]. Patients can suffer vomiting, photo/phonophobia, neurological deficits, seizures and blood pressure surges [2]. Secondary precipitants of RCVS are listed in table 1. Our lady was peripartum and oxytocin used for induction may have contributed. Initial brain imaging and angiography can be normal, although various stroke types can occur. The exact pathophysiology is unknown and is probably multifactorial [3].

There are no randomised control trials to guide management. Nimodipine has been shown to reduce headache, but does not alter the course of cerebral vasocostruction [2]. RCVS is usually self-limiting and prognosis is determined by stroke occurrence.
After five months, our patient can mobilise with a stick and needs minimal assistance with activities of daily living. Her baby is well.

Conclusion
More advanced imaging has led to more cases of RCVS being diagnosed [4]. Acute physicians need an awareness of this syndrome as a differential diagnosis of thunderclap headache to ensure thorough work-up.

References
A 54 year old lady presented with few weeks of lethargy and frontal throbbing headache. Past history was insignificant except a trans-sphenoidal resection of a pituitary adenoma six months earlier. She was taking no regular medications and leading a healthy lifestyle.

On examination she was alert and orientated. Vital signs were normal, except one reading of pyrexia of 38 Celsius. There were no signs of meningism or photophobia. Neurological examination was normal apart from residual left superior quadrantanopia.

Blood works revealed normal inflammatory markers, with WCC 7.7 and CRP < 1, and low random cortisol. CT scan head showed higher attenuation tissue in an expanded pituitary gland but appeared unchanged from previous MRI three months before.

Lumbar Puncture revealed 25 lymphocytes x 10⁶/Litre with raised protein 0.82 g/L. She was diagnosed with viral meningitis and commenced on intravenous acyclovir, pending viral PCR.

A Short Synacthen test was arranged which showed hypoadrenalinism secondary to ACTH deficiency and she was commenced on steroid replacement with hydrocortisone. Other pituitary function markers were unremarkable (TSH 1.63 Prolactin 83 LH 0.1)

An MRI pituitary was arranged in a view of hypopituitarism which revealed a multi-loculated pituitary abscess. She was started on broad spectrum antibiotics and transferred to a Neurosurgical Unit for further management.

Pituitary abscesses can be primary or occur as a rare complication of trans-sphenoidal surgery (1, 2) where sinus disease can progress up the operative track. The most common presentations are headache and hypopituitarism (3). The patient rarely present with evidence of a serious infection, peripheral leucocytosis and meningism are uncommon features (4).

The case informs us that whilst hypopituitarism is a common complication of pituitary surgery, abscess formation should be considered in the differential diagnosis when the patient present with headache, even if the inflammatory markers were not raised. As in this case they were in no way indicative of the serious underlying diagnosis.

Scalp Necrosis presenting as a severe complication of Giant Cell Arteritis on a Medical Assessment Unit setting

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Giant cell arteritis (GCA) or temporal arteritis as it is commonly referred to, is a medium to large vessel vasculitis which tends to affect those aged greater than 50 with a peak onset at around 70 years. Patients typically present with headache, jaw claudication, scalp tenderness and amaurosis fugax. Upto 50 % of all patients suffer with Polymyalgia Rheumatica. Severe complications such as visual loss and stroke can occur. Rarely, it causes scalp necrosis 1.

Scalp necrosis usually results from chronic inflammation involving the extra-cranial branches of the aortic arch. This is commonly associated with visual loss. Since its first description in 1947 there have been approximately 100 cases of scalp necrosis in patients with GCA published in the literature1.

We report a case of a 77 year old man who presented with a 4 week history of bilateral scalp necrosis. He complained of a headache, scalp tenderness and jaw claudication. His inflammatory markers were significantly elevated. Interestingly, he denied any visual disturbances. A diagnosis of GCA was made and he was started on high dose steroids immediately with adequate PPI cover and bone protection. He was referred to plastics immediately. Thereafter he was managed as an outpatient with regular clinic follow up on a reducing regime of steroids.

This case highlights a rare complication of GCA but emphasises the importance of prompt diagnosis with a high index of clinical suspicion and treatment to prevent further devastating complications. Although from a GCA point of view he was making good progress he sadly died a few months later following complications resulting from urinary sepsis.

References
Serotonin Toxicity following PMA ingestion

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Introduction
Recreational use of Paramethoxyamphetamine (PMA) has been attributed to an increasing number of deaths, with 20 reported in 2012 (1). It is sold as ecstasy ‘Green Rolex’ or ‘Dr Death’. Its potent serotonergic effect is the most likely mechanism for its toxicity, and a slow onset of action can lead to unintentional overdose. (2)

Aim
This case report highlights common presenting features of PMA toxicity; illustrating immediate management and subsequent clinical course.

Case Report
A 21 year old man presented to ED peri-arrest following ingestion of eight tablets with alcohol. On admission he had a GCS of 3, temperature 42°C, bilateral dilated pupils, muscle rigidity and refractory VT despite 3 synchronised DC shocks.
Active cooling was started with axillae ice packing, intravenous cold saline & cold water infused into the bladder & stomach in ED.
A further 2 synchronised DC shocks were administered with calcium gluconate and magnesium to treat hyperkalaemia of 6.8mmol/L. Spontaneous circulation was restored but temperature remained at 40°C. Dantrolene was given and deep peritoneal lavage with 3 litres of ice cold saline & Hartmann’s solution started.
His temperature settled to 37.1°C and he was sedated and intubated in ED.
His ITU admission was complicated: requiring treatment for rhabdomyolysis with urinary alkalinization; CVVH for acute kidney injury; N-acetylcysteine for hepatic impairment; further dantrolene for active cooling and cyproheptadine for continuing serotonin toxicity. He also developed disseminated intravascular coagulation and ventilator associated pneumonia. Further serotonergic drugs were avoided. He was discharged home after 3 weeks with normal renal function and no long term neurological impairment following resolution of critical care neuropathy.
Laboratory analysis of tablets subsequently confirmed Paramethoxyamphetamine.

Conclusion
Hyperpyrexia associated with serotonin syndrome is associated with multi-organ failure and increased mortality. Rapid identification of this clinical syndrome and aggressive temperature control may improve mortality.

References
Solving intractable seizures with IV Alfacalcidol, a lesson learnt in calcium metabolism?
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Grantham and district hospital
Ian Sajong Hamilton Lyons
Shirine Boardman

AIM
This case highlights the need for a holistic approach to hypocalcaemia and the potential causes of this metabolic disturbance. Particular focus is required to identify the contributory factors and the best ‘point of attack’ to treat the underlying deficiency or dysfunction.

A 66 year old woman with a transplanted kidney with an eGFR of between 13 to 20 presented with hypocalcaemia-induced seizures 3 times in a year (adjusted calcium varying between 1.43 and 1.75). She had several co-morbidities including a total colectomy for Ulcerative Colitis, malabsorption secondary to a high stoma output, parathyroidectomy for Primary Hyperparathyroidism and a living-related renal allograft. Initially seizures were treated with intravenous calcium but a progressive drop in levels occurred following discharge. Her dose of Sandocal 1000 was progressively increased to 5 tablets QDS to counter hypocalcaemia. Her Vitamin D levels had been low for several years (25nmol/L-36nmol/L). Initial management with an increasing dose of oral activated Vitamin D3 (up to Alfacalcidol 4mcg QDS) and intramuscular vitamin D3 (cholecalciferol 300,000IU) was ineffective at maintaining normal calcium levels or preventing fits. Intravenous infusion of magnesium sulphate was also unsuccessful. In view of her renal impairment and malabsorption, intravenous Alfacalcidol was finally trialled and her calcium levels began to rise. She stabilised on IV Alfacalcidol 2mg 3 times per week which her husband administers through her Hickman line. Her Calcium levels are now maintained between 2.31 and 2.34 with no further fits.

CONCLUSION
Hypocalcaemia is common and can have a variety of underlying causes. Whilst initial management is to urgently correct the calcium deficiency, an approach to address the underlying defects in calcium regulation is also required. This complex patient had a number of different pathological defects in her calcium metabolism including severe malabsorption complicated by renal impairment and a parathyroidectomy.

References:
Steroids in hypercalcaemia: the ACE up your sleeve.

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James Rowan-Parry
Phillip Jacobs

Aim
This case study aims to increase awareness of sarcoidosis as a cause of hypercalcaemia.

Methodology
A 63 year-old Caucasian female presented with a three month history of malaise and weight loss. She had previously been treated for a left breast carcinoma and was otherwise well. Examination was unremarkable. Serum calcium was elevated at 3.85mmol/L (corrected; normal range 2.15-2.61). Parathyroid hormone (PTH) was suppressed. The level of 25-hydroxy-vitamin D was 51nmol/L.

The initial diagnosis was of hypercalcaemia secondary to malignancy. Intravenous normal saline and pamidronate disodium were given to lower the serum calcium. Treatment response was marginal, and a trial of calcitonin was initiated to poor effect. A malignancy work-up revealed right axillary lymphadenopathy on breast ultrasound. CT of the chest, abdomen and pelvis highlighted mediastinal, paraaortic, axillary and pelvic lymph nodes, but no evidence of metastases.

Histological lymph node analysis reported non-necrotising granulomatous inflammation, negative for acid fast bacilli. A serum angiotensin converting enzyme (ACE) level was elevated at 142iu/L (normal range 8-65), supporting a diagnosis of sarcoidosis.

Outcome
Oral corticosteroid therapy (prednisolone 30mg once daily) was initiated for the treatment of hypercalcaemia secondary to systemic sarcoidosis. The patient displayed an excellent symptomatic and calcium lowering response (Figure 1).

Discussion
Hypercalcaemia is a common clinical presentation. 90% of hypercalcaemia is secondary to malignancy and hyperparathyroidism, however granulomatous disease should be considered in cases refractory to saline and bisphosphonate therapy. Steroids are the first-line treatment for sarcoidosis-induced hypercalcaemia, which is subsequent to macrophage overproduction of calcitriol in an immunoregulatory response to granuloma formation. An elevated level of serum ACE is of value in cases of diagnostic uncertainty.

References
AIM
There is a paucity of evidence on how to manage superficial thrombophlebitis (ST). We present a case of ST complicated by pulmonary emboli (PE) and review the evidence.

METHODS
An 84-year-old female presented with a two week history of a swollen right leg and increased shortness of breath. She had no risk factors for venous thromboembolism (VTE) but bloods revealed a raised D-dimer. On examination her chest was clear, but with oxygen saturations of 95%. An US doppler showed ST (see figure 1) and a CTPA revealed multiple PEs (see figure 3).

OUTCOMES/RESULTS
Literature review quoted an unexpectedly high incidence of VTE from ST, between 11 - 57%\(^1\). The risk is increased if the ST extends close to the saphenofemoral junction (SFJ), if there are no varicose veins\(^1\), or if risk factors are shared with those for VTE. It is suggested that treatment with low molecular weight heparin (LMWH), can reduce the risk of VTE by 85%\(^2\), with prophylactic dosing as effective as treatment dose\(^3\). Studies suggest a short treatment period, between ten to forty-five days. A disadvantage is the increased bleeding risk and there is also the question of the cost-effectiveness of this approach\(^4\).

CONCLUSION
ST should not be dismissed as a benign and self-limiting disease and patients should have a comprehensive assessment with a detailed history and examination aimed at highlighting risk factors shared with developing VTE, as well as symptoms and signs including those associated with PE. In those presenting clinically, an US doppler should always be performed. In those patients with a ST extending close to the SFJ, with no varicose veins, there is evidence to suggest that treatment with prophylactic LMWH, for up to forty-five days, can both alleviate symptoms and reduce the risk of developing a VTE.

We present a case of spontaneous tension pneumothorax in pregnancy. A 37 year old woman developed acute dyspnoea and pleuritic chest pain, presenting to hospital 4.5 hours after symptom onset. She was 19+4/40 pregnant with known tuberous sclerosis (TS) and stable renal angiomyolipomas. Initial examination was unremarkable with O$_2$ saturations of 92% on air. Pulmonary embolism (PE) was suspected initially; due to concerns over unnecessary radiation exposure a chest X-ray (CXR) was not performed. The patient subsequently deteriorated rapidly and portable CXR with abdominal shielding confirmed a left-sided tension pneumothorax. She underwent needle decompression and Seldinger chest drain insertion with rapid improvement. After 3 weeks in hospital, the remainder of her pregnancy progressed normally with delivery of healthy twins by elective Caesarean section at 37 weeks.

**Discussion**

The pregnant patient with breathlessness or chest pain is a common presentation on the acute medical take. Dyspnoea and tachycardia may be physiological but the differential diagnosis includes life-threatening conditions such as pneumonia, PE and pneumothorax. Pregnancy increases the risk of the latter two conditions with increased likelihood of adverse outcomes. Only 56 cases of pneumothorax in pregnancy are reported in the literature with risk factors including asthma, cocaine use, hyperemesis and previous pneumothorax. TS is known to increase the risk of pneumothorax although a case during pregnancy has not previously been described. Diagnosis of pneumothorax in pregnancy may be challenging. However, concerns over radiation exposure appear unfounded - a single CXR equates to 3 days of UK average background dose or one third that of a transatlantic flight. Abdominal shielding further reduces foetal exposure. Prompt CXR may have facilitated diagnosis and treatment in this case. CXR in pregnancy is generally considered safe even in the first trimester where risk is greatest and therefore merits consideration when investigating the pregnant patient.

**References**

The one in a million “clotting screen”

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Aim
Routine “clotting screens” are discouraged in the evaluation of iron-deficiency anaemia. We describe a case where review of clotting screens performed outwith the guideline recommendations would have expedited a very rare diagnosis.

Case
A 68-year old lady on aspirin was seen acutely with symptomatic iron deficiency anaemia (haemoglobin concentration (Hb) of 47 g/L, mean cell volume 58.9 fl, ferritin 2 µg/L), transfused with packed red cells and discharged awaiting outpatient endoscopy. She was readmitted with anaemia 5 weeks later with an Hb of 67 g/L, and this time gave a recent history of dark stools. Inpatient upper gastrointestinal endoscopy revealed mild duodenitis, so aspirin was withheld; she was discharged on oral iron supplements, and subsequently received an outpatient iron infusion. Six weeks later, she was readmitted with extensive spontaneous right leg bruising and a markedly prolonged activated partial thromboplastin time (aPTT). It was then noted that “routine” clotting screens performed on previous admissions had shown a very prolonged aPTT and normal prothrombin time, suggestive of an intrinsic pathway abnormality. Further investigation revealed an Acquired Haemophilia A due to Factor VIII inhibitor (anti-Factor VIII antibodies), which has a reported UK incidence of 1.34 to 1.48 per million. She remains well on immunosuppressive therapy, and subsequent investigations for underlying systemic disease have been negative.

Conclusion
Very rare causes of common medical presentations are often not covered by national guidelines. Reviewing results of non-indicated investigations can lead to serendipitous discovery of a rare diagnosis.

References:
The use of L-carnitine in the management of sodium valproate overdose
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Abstract
Assessment and management of the poisoned patient, along with knowledge of poison-specific antidotes and relevant toxicology form an important part of the AIM curriculum.(1)

Aim
To describe the approach to management in the patient with valproate overdose.

Design
Case report and a review of the literature.

Case report
A 25 year old female presented to the department after having intentionally ingested 14g of sodium valproate. Following admission her GCS was noted to be 13 (E3 V4 M6). After being monitored for 12 hours she continued to be drowsy and was noted to have elevated serum valproate and ammonia levels. She was treated with intravenous L-carnitine, which demonstrated a rapid clinical improvement and reversal of serum ammonia back to baseline. She was discharged the following day. The patient has had no adverse effects of treatment to date.

Discussion
Sodium valproate is a commonly used antiepileptic drug used in the management of tonic-clonic seizures, and as maintanence therapy in primary generalised epilepsy.(2) Peak plasma concentrations can occur at 1-2 hours, though this may be extended in large overdoses. Hepatotoxicity is also a common feature. It is important to monitor bloods including U&Es, glucose, LFTs, blood gases and serum ammonia levels.(3) L-carnitine is recommended in patients with evidence of hepatotoxicity, hyperammonaemia or in serum valproate levels over 450mg/L.

Conclusion
L-carnitine has been found to be a safe drug with no adverse effects reported in a recent study,(4) and should be considered in valproate overdoses with any of the features outlined above.

References
Things that go pop in the night...
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Pneumatoceles are acquired air cysts that develop after lung infection, pulmonary trauma (laceration), or hydrocarbon ingestion. They represent an area of localized pulmonary over inflation caused by transient bronchial or bronchiolar obstruction of the check-valve type.\(^1\) Organisms that may lead to this condition include *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Haemophilus influenzae*, *E. coli*, *S. pyogenes*, *Serratia marcescens*, *K. pneumoniae*, adenovirus, *M. tuberculosis*, and *Pneumocystis jiroveci*.\(^2\)

A 42 year old lady presented to AMU with shortness of breath, fever and a dry cough. She was diagnosed as having severe community acquired pneumonia and was treated with intravenous antibiotics. Atypical serology and a HIV test were taken. On Day 2 of her admission she developed type 1 respiratory failure and despite a trial of CPAP, deteriorated and required transfer to ITU for intubation and ventilation. Her HIV test was positive with a viral load of >2500000 copies/ml so Co-Trimoxazole, steroids and anti-retrovirals were commenced. Bronchoalveolar lavage was positive for *Pneumocystis jiroveci* nucleic acid. A chest X-ray taken on day 5 showed lucency in the left upper zone, as well as widespread interstitial opacification. A CT thorax was undertaken to evaluate this area of lucency, it was reported as being a large pneumatocele with no evidence of a pneumothorax. However on day 7, the patient became difficult to ventilate and CXR taken at the time shows a tension pneumothorax. An intercostal drain was inserted and ventilation improved once the lung had re-expanded.

Learning points
- An uncommon manifestation of *Pneumocystis jiroveci* pneumonia is upper lobe cystic disease – pneumatocele
- Rapid growth of these pneumatoceles may mimic a massive pneumothorax
- Rupture of these pneumatoceles may lead to spontaneous pneumothorax
- The risk of rupture is increased by positive pressure ventilation\(^3\)
- The risk of rupture is also higher in patients with *Pneumocystis jiroveci* pneumonia\(^4\)

References
Unnecessary Treatment Can Cause Harm - A Case of Trimethoprim-Induced Stevens Johnson Syndrome

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Aim
To present a case of a rare but potentially fatal reaction to a commonly prescribed antibiotic.

Methods
A 74 yr old Jamaican lady with epilepsy was admitted following a seizure. She had complained of a sore throat and cough but denied urinary symptoms. Routine observations found a temperature of 38 C. Despite no symptoms, urine dipstick testing was undertaken and was positive for nitrites and leucocytes. She was commenced on trimethoprim to treat presumed urinary tract infection (UTI). Within 24 hours she developed two blisters on her right buttock and the antibiotic was stopped. Over 8 hours the patient’s skin developed severe blistering affecting 25-30% of her total body area with mucosal involvement. She had a documented penicillin reaction in her notes and closer questioning suggested this reaction had been similar.

Outcome/Results
The patient was managed with intravenous fluid support, analgesia and blister dressings as advised by the local burns unit. After a protracted course, complicated by thrombocytopenia, she made a slow recovery and was discharged 3 weeks later.

Conclusion
Stevens Johnson Syndrome is a rare but potentially fatal known complication of medications including Trimethoprim[1,2,3]. It is often first line treatment for UTIs but in this case, was prescribed to an asymptomatic patient. Urine dipstick testing in asymptomatic patients and treatment of positive tests in these patients are not recommended in the European urinalysis guidelines [4]. To minimise risk to our patients we must ensure that all prescribing is appropriate, necessary and with a full allergy history.

243 words

References
Clinical scenario
A 27 year old nurse presented with facial swelling and rash 4 days post injection of “Prostap SR”, a gonadorelin (LHRH) analogue an injection being used to treat her endometriosis. She was otherwise well, on no medications, but had been hospitalised as a child with a penicillin allergy. On examination she was tachycardic, blood pressure 117/94, had an urticarial rash on her face and torso and swelling of the eyelids. A diagnosis of allergic reaction was made and treatment initiated with intravenous hydrocortisone and chlorpheniramine. She was discharged with oral chlorpheniramine and prednisolone.
She represented 24 hours later with the same symptoms and a new wheeze. A diagnosis of anaphylaxis was made and adrenaline administrated intramuscularly.
Over the next 6 weeks she suffered approximately 30 further allergic reactions, both out of hospital and during her 7 spells in hospital. The trigger was presumed to be Prostap, a depot medication that remains at a steady state from day 5 following injection to day 117.
It was unclear whether the allergic response was mast cell or bradykinin mediated. C3, C4 and serum tryptase levels were normal. Immunologist advice was to use a range of medications in the hope of reducing admissions, (see discharge medication table). Adrenaline is the only medication that will treat shock. Patient and partner were trained in use of epipens, using these successfully on multiple occasions. She had no further admissions after six weeks.

Anaphylaxis
Anaphylaxis is a serious allergic or hypersensitivity reaction that is rapid in onset and may cause death. It is under-recognised and under-treated. False reassurance of a normal blood pressure is a common pitfall. Treatment involves removal of the trigger - not possible in this case. Early intramuscular administration of 0.5mg of adrenaline aims to prevent the development of shock and reduce fatalities¹.

Case Presentation
A 37yr man presented with a week of tiredness, dyspnoea and chest pain. He was known with asthma, OSA and a past medical history of pulmonary embolism. He complained of severe inspiratory chest pain and was tachypnoeic. He felt extremely uncomfortable and distressed. There was widespread bilateral wheezing. His blood results showed mildly raised WBC, CRP <1. CXR showed a possible right basal consolidation. He had been started in A&E on back to back nebulisations; oral prednisolone, IV co-amoxiclav, 10mg IV morphine. ABG in A&E on 15L O2: pH 7.46; pCO2 3.6; pO2 31.5; Lac 2.8. He was transferred to AMU overnight with hourly nebulisations. At the morning PTWR excruciating muscle pain was the main complaint and lactate was 4.2. Review of his notes revealed a similar worsening lactic acidosis during previous admission. We prescribed analgesics and diazepam as a muscle relaxant. We decreased the frequency of nebulisations resulting in a lactate level of 2.3. Only Ketamine administered in ITU could ultimately reduce the lactate induced extreme myalgia.

Discussion and Conclusion
The mechanisms by which β-agonist therapy can induce a type B lactic acidosis are not completely understood. Multiple case reports and two small prospective studies have addressed this issue. High lactate needs to be contextualised with the clinical picture. β-agonist induced lactic acidosis could enter the differential. Transitioning the patient to less frequent nebulised therapy and/or change to a non β-agonist regimen needs to be considered to avoid deterioration.

References
39 year old lady who was previously fit and well, presented acutely with a two week history of progressive breathlessness. She was pyrexial on admission with raised inflammatory markers. Chest xray was abnormal with evidence of consolidation in both lower zones. She was treated for community acquired pneumonia but failed to improve despite escalation of antimicrobial therapy. Blood cultures remained negative. Bronchoscopy showed no endobronchial abnormalities and no organisms were cultured from bronchial washings.

CT thorax showed bilateral dense airless consolidation in both lower lobes with volume loss. She proceeded to have a video assisted thoracoscopic wedge biopsy from the right lung. Histology revealed “patchy but prominent organising fibrosis with fibroblastic plugs distending bronchioles and peri-bronchiolar alveolar spaces with an inflammatory infiltrate constituting lymphocytes, macrophages and eosinophils”.

An organising pneumonia was the main differential diagnosis following the lung biopsy. She received a three day course of high-dose Methylprednisolone, resulting in dramatic clinical improvement within 48 hours. She was discharged on a reducing course of oral prednisolone. Follow-up CT scan 3 months later showed almost complete resolution of the previous abnormalities consistent with a diagnosis of cryptogenic organising pneumonia.

Organising pneumonia can be cryptogenic (COP - formerly known as bronchiolitis obliterans organising pneumonia, BOOP) or secondary to infection, drugs, or malignancy. COP is a disease of unknown aetiology characterised by ‘plugging’ of alveolar spaces with granulation tissue. It typically presents as a slow or non-resolving chest infection, often after several antibiotic courses and should be considered in such patients. Diagnosis is made on clinical features, high resolution CT and biopsy. It is important to search for secondary causes, which was thought most likely to be infection in this case. Mainstay of treatment is steroids; immunosuppressants can be used if response to steroids is poor (1).

References
“If I be waspish, best beware my sting…” Type I Kounis syndrome caused by vespid envenomation

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Aim
We describe a case of type I Kounis syndrome in a patient with anaphylactic shock from vespid envenomation.

Case
A 45-year-old gentleman developed anaphylactic shock after being stung by a wasp, requiring adrenaline for haemodynamic support. Serum mast cell tryptase was raised at 147 µg/L (normal 0 – 14 µg/L). 12-lead electrocardiography (ECG) revealed transient widespread ST-segment depression (Figure 1A), which resolved spontaneously (Figure 1B). Serum troponin T was raised at 119 ng/L (normal < 15 ng/L). He was subsequently discharged with adrenaline auto-injectors and immunology follow up. Transthoracic echocardiography showed no regional wall motion abnormality, and an exercise stress test was normal. IgE assay performed on outpatient review revealed raised venom-specific IgE, and he subsequently successfully received wasp venom desensitization immunotherapy.

Conclusion
Other causes of acute ECG ST-segment deviation with cardiac biomarker release apart from acute coronary syndromes (ACS) include takotsubo cardiomyopathy, vasospastic (Prinzmetal’s) angina, myopericarditis, and Kounis syndrome. Kounis syndrome is a recently described entity which often goes unrecognised. Diagnosis is based on simultaneous manifestations of acute type 1 hypersensitivity and apparent ACS. We review the clinical presentation and proposed pathophysiology of Kounis syndrome, and discuss its classification into type I (no coronary artery disease), type II (concomitant culprit coronary artery disease), and type III (inflammatory coronary thrombus) subgroups.

References