Aim
To describe a case of focal myositis presenting to Acute Medicine.

Methodology
A 24 year old Bangladeshi male office worker presented to Acute Medicine with a six hour history of swelling and heaviness in his left upper arm. He had no history of trauma, no systemic symptoms, no symptoms of autoimmune, joint or connective tissue disease and no recent travel history. His only past medical history was hypertension and he had no risk factors for developing thromboses. He denied intravenous drug use. He was afebrile with no signs of systemic disease. He had obvious, firm, non-fluctuant swelling extending from his left shoulder to the middle of his forearm. The skin was not hot, there was no erythema or tenderness and his arms were neurovascularly intact. All joints were normal.
Routine blood tests were normal. His creatine kinase level was markedly elevated at 4014. A duplex ultrasound was normal. MRI of the left arm demonstrated fibrillar high signal on the T2-weighted sequences and enhancement from the long head of the triceps muscle, consistent with a myositis.

Outcomes and Results
With regular non-steroidal anti-inflammatory drugs the swelling improved, matched by a drop in creatine kinase levels from 4014 to 1084. Muscle biopsy was consistent with an inflammatory myopathy.

Conclusion
Focal myositis is a benign inflammatory condition causing profound swelling of a solitary muscle. It does not affect neurovascular function. Clues towards the diagnosis are increased muscle enzyme levels and high intensity on T2 weighted MRI. Histology of interstitial connective tissue infiltration confirms the diagnosis.

References
Note: Not referenced in the above, but relevant to a discussion of the case.
Title: A lethal DVT mimic: Early recognition can avoid catastrophe!

Aim
A 55-year-old HIV-positive on antiretroviral therapy (HAART) presented to A&E with non-traumatic right leg swelling. A Deep Vein Thrombosis (DVT) was provisionally diagnosed and therapeutic fondaparinux commenced. No blood tests were done prior to anticoagulation. He was discharged and early outpatient Doppler sonogram scheduled.

Methodology
Three days later, he presented to Acute Medical Unit with new confusion and worsening right leg oedema. Examination revealed gross right leg oedema with erythema and warmth. Treatment was commenced for cellulitis. However, rapid deterioration ensued within hours, with pyrexia, severe hypotension and GCS of 3. CT Head excluded an intracranial bleed. Blood tests revealed Haemoglobin of 6.7g/dl, normal Platelet count, Prothrombin Time of 25 seconds and grossly abnormal partial-thromboplastin time (APTT) of 125 seconds. CD4 count was 485/cmm.

Outcomes and Results
Provisional diagnosis of acute compartment syndrome due to right calf haematoma and acquired hemophilia was made with aggressive resuscitation and transfer to ICU. Intracompartimental pressures were raised confirming diagnosis. Surgical fasciotomy was performed to evacuate large calf haematoma with simultaneous aggressive correction of the abnormal clotting using Factor Eight Inhibitor Bypass Activity (FEIBA) along with Factor VII. Further investigations confirmed diagnosis of acquired haemophilia (Factor VIII deficiency). After prolonged ICU stay complicated by right-sided weakness due to cerebral infarct, patient recovered with full consciousness and is undergoing neuro-rehabilitation.

Conclusion
In suspected DVT, assessment of coagulation profile prior to anticoagulation can avoid catastrophes akin to our case. Furthermore, since clinical diagnosis of DVT is inaccurate in 50% of cases(1), fastidious ultrasound scanning (within 24-hours) is crucial for patient safety.

References
Title: A rare cause of meningitis with cranial nerve involvement: a case report

Aim
Varicella zoster infection is a common condition but only infrequently causes neurological disease in immunocompetent adults as is described in this patient.

Outcomes and Results
A 39 year old environmental consultant presented with a 12 hour history of visual disturbance and facial asymmetry, and a three day history of headache and nausea. Past medical history included asthma and childhood chicken pox, and he took no medication. His partner had recurrent shingles and had had a recent episode. He recently returned from field work in Spain.

He was well, afebrile, with no signs of meningism, a rash or bites. The only abnormalities on neurological examination were a right sided seventh nerve palsy sparing the forehead and subjective diplopia on right lateral gaze, although objective assessment of eye movements was normal.

Blood tests, chest radiography and magnetic resonance imaging of his head were normal. Cerebrospinal fluid analysis showed an opening pressure of 27mmH2O, the cell count was 98 WBCs/mm3 (96 lymphocytes) and 0 RBCs/mm3. The protein was elevated at 1286mg/L and VZV DNA was detected by polymerase chain reaction. Serological testing was negative for Borrelia and human immunodeficiency virus. The patient was discharged home on valacyclovir and prednisolone.

Conclusion
CSF analysis in 40-50% of individuals with herpes zoster infection shows a lymphocytic pleocytosis but only a subgroup develop meningitic symptoms. The increasing use of viral PCR is showing recurrent VZV infection to be a commoner cause of neurological abnormalities than previously thought and often a coexistent rash may be absent.

References

Aim
This case underlines the importance of pursuing for the root cause of the disorder like in this young adult who was finally diagnosed to have a very unusual genetic cause of stroke.

Methodology
Retrospective analysis of journey of diagnosis

Outcomes and Results
47 years old right-handed cleaner presented to stroke services with a history of right leg pain & weakness and increasing problems with his gait. He was a non-smoker & drank alcohol in moderation. Examination revealed brisk tendon reflexes on right hand side with flexor planters, and was otherwise normal. His family history suggested a possible genetic disorder causing cardiovascular disease at young age involving male members of family. His brother died of stroke in his 50s & father died of multi-infarct dementia early 60s. On closer questioning he admitted to chronic muscular pain in both thighs exacerbated by exertion. Neuro-imaging revealed multiple white matter lesions which were suggestive radiologically of ischaemia or inflammation. CSF studies showed mildly raised CSF protein, otherwise within normal limits. Carotid ultrasound was normal. Visual evoked responses and nerve conduction studies were normal. He was found to have low serum alfa-galactosidase & he was finally confirmed to have Fabry disease with a mutational alfa-galactosidase gene. End organ evaluation revealed proteinuria, LVH & mild corneal deposits (corneal-verticillata). Referred to Department of Lysosomal disorders at Manchester for enzyme replacement therapy.

Conclusion
This case shows the importance of pursuing for the root cause of the Disorder and also of importance of good history making to be successful in diagnosing a very rare condition like this.

References
Aim
We describe a patient who was seen five weeks following insertion of a permanent pacemaker and presented a diagnostic challenge.
A 72 year old lady was admitted with breathlessness and chest discomfort following insertion of a dual chamber permanent pacemaker. Reduced air entry at the left lung base and raised white cell count. Chest x-ray (Figure 1) showed cardiomegaly and likely left lower lobe infection. The marked lateral position of the pacemaker lead was unfortunately over-looked, and lower respiratory tract infection diagnosed. She received antibiotics and allowed home.

Methodology
She presented again a week later with symptoms of left ventricular failure and non resolving pneumonia. She was given intravenous furosemide and a different antibiotic. A second chest radiograph showed bilateral pleural effusions. The echocardiogram indicated a large anterior and posterior pericardial effusion with signs of impending tamponade. Pericardiocentesis was performed. During this procedure, a pacemaker lead was visualised in the pericardial space, close to the right ventricular apex, confirming right ventricular perforation.

Outcomes and Results
Urgent transfer was made to a cardio-thoracic centre where median sternotomy was performed, the pacemaker lead removed, and right ventricular perforation repaired (Figure 2). Unfortunately she died, despite the insertion of temporary pacing electrode.

Conclusion
In our case, diagnosis was delayed because lead position was overlooked on chest radiographs, and alternative diagnosis was considered. Although a rare complication, we suggest that right ventricular perforation by pacemaker lead is always considered in symptomatic patients following implantation of a permanent pacing system, that chest x-ray is carefully scrutinized for lead position.

References

Aim
Hypercalcaemia is a common metabolic abnormality amongst hospital inpatients, with malignancy and primary hyperparathyroidism accounting for the majority of these cases. We present a case of adult T-cell leukaemia/lymphoma secondary to Human T-cell Lymphotropic Virus type 1 (HTLV-1), as a rare cause of severe hypercalcaemia.

Outcomes and Results
A 67 year old Jamaican woman was admitted with a two week history of diarrhoea, anorexia and weight loss. She had no other symptoms suggestive of hypercalcaemia. She was on medication for hypertension and type 2 diabetes mellitus. On examination, she was mildly dehydrated and had no lymphadenopathy, hepatosplenomegaly or rash. Laboratory investigations showed severe hypercalcaemia (4.18mmol/L) and a normal parathyroid hormone level (1.8pmol/L). Full blood count showed a microcytic anaemia with electrophoresis consistent with alpha thalassaemia trait. A lymphocytosis (13 x10^9/L) was also seen with abnormal clover-shaped CD4 and CD25 positive lymphoid cells on the blood film, suggesting a diagnosis of adult T-cell leukaemia/lymphoma. HTLV-1 serology was positive. CT imaging of her chest and abdomen were normal. Her calcium level normalised after intravenous fluids and a pamidronate infusion. She was then transferred to the Haematology service for ongoing treatment.

Conclusion
The initial detection of a normal parathyroid hormone concentration in the presence of severe hypercalcaemia suggested primary hyperparathyroidism but this case demonstrates the importance of interpreting these results in the context of other abnormalities in the history and on laboratory testing. It also emphasises the importance of the patient’s country of origin, despite her limited travel in the last fifty years.

References
Aim
Madelung’s disease is a condition characterised by deposition of multiple unencapsulated lipomas in different areas of the body, sparing distal arms and legs and usually in a symmetrical fashion. A case that presented to our acute medical unit is described and the literature reviewed.

Methodology
A 52 year old Caucasian gentleman admitted to our ward was noted to be obese, but with an abnormal distribution of the body fat in keeping with a diagnosis of Madelung’s disease. The case is described with a review of the literature.

Outcomes and Results
The patient had large amounts of fat deposition on the trunk and proximal aspects of all the four limbs, giving him a pseudo-athletic appearance (image). This is typical of Madelung’s disease. This disease has been classified into 2 clinical types (2). Type I: the fatty deposits are well circumscribed, distinct protruding from the body surface. They can also invade the mediastinum and cause compression of the aerodigestive tract. Type II: lipomatous tissue is present all over the body giving the patient an external appearance of obesity. The majority of patients have polynuearopathy and autonomic neuropathy can be a cause of sudden death (3). Surgical excision of lesions or liposuction is effective, but lipomas can recur. (4) Weight loss and abstinence from alcohol are advised though have not proven to be effective in stopping progression.(1)

Conclusion
This patient fitted the criteria for Madelung’s disease type II, based on history and clinical examination. This is a rare but important condition to recognise because of the important associations including neuropathy.

References
2. Enzi G. Multiple symmetric lipomatosis: an updated clinical report. Medicine 1984; 63:56-64
Aim

Neurological complications following percutaneous coronary intervention (PCI) are more frequently caused by cerebral embolism or haemorrhage. Cerebral extravasation of contrast is rare but a recognised complication of using high doses of contrast media1,2

Methodology

A 76 year old gentleman was admitted with NSTEMI. He was given on 1mg/kg enoxaparin, aspirin 300mg, clopidogrel 300mg and initiated on tirofiban. Coronary angiography confirmed significant circumflex disease necessitating the deployment of two intracoronary stents. Due to significant vessel tortuosity the procedure was prolonged and 500ml of Iopamidol contrast agent was used. Thirty minutes following the procedure, the patient became acutely confused and clinical examination confirmed a receptive dysphasia and a left hemiplegia. CT scan of the brain revealed widespread high density changes within subarachnoid space of the right hemisphere and left frontal lobe area (Figure 1 ) consistent with subarachnoid haemorrhage.

Tirofiban was immediately stopped and the patient received two units of platelets and 30mg of protamine sulphate. The patient was observed closely and his symptoms resolved within 36 hours. A repeat CT of brain after 41 hours revealed complete resolution of the high density areas within the subarachnoid space (Figure 2). The patient made a full neurological recovery and was discharged to continue aspirin and clopidogrel.

Conclusion

In the context of neurological symptoms following coronary PCI, the importance of considering the diagnosis of contrast agent neurotoxicity, is not only to avoid unnecessary treatment and investigation but to also allow subsequent continuation of dual antiplatelet therapy to minimise the risk of stent thrombosis.

References


Title: The first case report of H1N1 associated Supraventricular tachycardia

Aim
We describe the first reported case of multifocal atrial tachycardia in a previously well middle aged patient suffering from confirmed H1N1 infection with no significant past medical history or cardiac risk factors.

Methodology
A 38 year old lady presented to A&E with headaches, vomiting and a pyrexia of 38°C. Examination was unremarkable. Initial ECG showed a supraventricular tachycardia of a rate of 210 which subsided with 6mg of adenosine. Investigations for electrolytes, inflammatory markers and troponins were normal.

Subsequently the patient began developing further runs of AVNRT requiring intravenous metoprolol to convert to a regular sinus tachycardia. On day two she was confirmed as being H1N1 positive.

On day three she deteriorated acutely; desaturating to 88% on room air. Outreach were informed to consider NIV for respiratory support. Eventually the patient began complaining of a productive cough and the diagnosis of Swine Flu with secondary pneumonitis was made.

Outcomes and Results
Gradually over eight days she improved clinically as the infection resolved. She required 50mg of metoprolol tds on discharge to control her rate however subsequent follow up demonstrated no structural cardiac abnormality and she stopped taking her medication experiencing no further problems.

Conclusion
Our case highlights another complication of the H1N1 spectrum, which all acute physicians should be aware of. Secondary bacterial infections are well documented; however the association of Swine flu causing AVNRT demonstrates further complications that could manifest in a previously well individual. Here, we also show that although she became significantly unwell, the SVT resolved once the acute condition subsided.

References
World Health Organisation Pandemic (H1N1) 2009
Aim
To increase awareness about the McKittrick-Wheelock Syndrome, which can cause life-threatening electrolyte disturbances. For many years, unexplained deaths occurred among patients with colonic villous adenomas. However, McKittrick and Wheelock were the first to describe in 1954 major electrolyte disturbance that can be associated with these tumours [1].

Methodology
A case report.

Outcomes and Results
An 81 year old man with a history of ischaemic heart disease was admitted with lethargy. He also complained of passing mucoid, “jelly”-like stools, nausea and occasional vomiting over a period of few weeks. On examination, he had dry mucous membranes and cold peripheries. His pulse was 83 bpm, BP 130/104 mmHg and temperature 34.5 C. His abdomen was soft with no palpable masses.

Investigations: Laboratory blood tests showed major electrolyte abnormalities with acute renal failure, hypokalaemia and hyponatraemia (Table 1). Stool samples were negative for C-difficile toxin and enteric pathogens. He subsequently underwent a flexible sigmoidoscopy which showed a large 14 cm villous adenoma extending from the rectum to the sigmoid colon (Figure 1). Histology confirmed villous adenoma and staging CT and MRI scans showed no evidence of invasive cancer.

The patient successfully underwent laparoscopic low anterior resection with loop ileostomy formation. His electrolytes and renal function normalised within few days after the operation, and stayed essentially within normal limits at follow up over a year later (Table 1).

Conclusion
The McKittrick-Wheelock Syndrome is a rare but potentially fatal presentation of villous adenomas. It should be suspected in a patient with chronic diarrhoea, renal failure and electrolyte disturbance[2].

References