A common but under recognised cause of Encephalitis

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Introduction:

Anti-N-methyl-d-aspartate (NMDA) receptor encephalitis results from an immune response against NR1 subunit of the NMDA receptor.

Case Report:

A 35 years old female presented to us in AMU from psychiatry ward where had been admitted for two weeks with headache, acute confusion, agitation, hallucinations, catatonia and echolalia. She had no past medical history. She was given sertraline which she did not tolerate and then started on olanzapine but continued to deteriorate. She had no focal neurology and normal examination. Her CT brain was normal. She was started on IV acyclovir after Lumbar Puncture which showed CSF protein 0.43, glucose 3.6, WCC 19 with 90% lymphocytes. Her viral PCR, HIV and autoantibodies were negative. MRI brain was normal. We had a strong suspicion of Anti-NMDA receptor encephalitis so urgent immunology was sent. She was empirically started on IV methylprednisolone and then given IV immunoglobulins for 5 days but remained unwell. Once results confirmed she was started on IV cyclophosphamide with neurology and rheumatology input. She made a good recovery and discharged home with further cyclophosphamide sessions planned. She had extensive imaging to look for malignancy though nothing found. She would be followed up by neurologists and continued to have psychiatric support.

Discussion:

Anti-NMDA receptor encephalitis is common yet under recognised cause of encephalitis. Most cases occur in young patients with strong female predominance. The disorder is characterised by acute behavioural change, catatonia, seizures and autonomic dysfunction. Diagnosis is confirmed by elevated anti-NMDA receptor antibody titres. The disorder can be associated with underlying malignancy, most common being ovarian teratoma. As it’s becoming more recognised more cases being identified without underlying malignancy. Treatment includes immunotherapy with removal of the tumour if found. High index of suspicion with cases of encephalitis with early immunotherapy may lead to improved morbidity and mortality.

References


A hobby with an unusual consequence - yet another cause of stroke

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Introduction:

Spontaneous Cervicocerebral Arterial Dissections (CAD) account for only about 2% of all ischaemic strokes, but they are an important cause of ischaemic stroke in young patients and account for up to 25% of cases.¹

Case:

A 45 year old gentleman with no significant past medical history presented with a sudden occipital headache in November 2013. A subarachnoid haemorrhage (SAH) was suspected. He underwent a CT head which was normal. He declined a lumbar puncture and was discharged.

A week later, he presented again with a sudden occipital headache. On arrival, he was dysarthric with right facial asymmetry and mild right hemiparesis. Again, a CT head was normal. Given the history of suspected SAH, thrombolysis was not considered. An urgent MRI brain revealed an acute right midbrain infarction.

He was treated with antiplatelet therapy. During admission, his neurodeficit completely resolved. He recalled that the first episode had occurred whilst rifle-shooting, which was his hobby. The onset was hyperacute following a sharp kick-back from the gun. The second episode happened after a week in exactly the same mode whilst rifle-shooting with friends.

A subsequent Magnetic Resonance Angiogram (MRA) of extracranial vessels revealed a right vertebral artery dissection. A follow up MRA after 6 months showed complete recanalisation.

Discussion:

Extracranial vertebral dissections account for about 15% of all CAD.² Aetiopathogenesis of CAD is incompletely understood, though trauma, respiratory infections and underlying arteriopathy are important associations. MRI/MRA, and CT angiography are useful non-invasive diagnostic tests. The treatment of extracranial CAD is mainly medical using anticoagulants or antiplatelets agents. Prognosis is more favourable in extracranial CAD. Recurrences are rare in CAD.²

This case again highlights the importance of taking a good history at the onset of neurological symptoms and how such information can aid in organising the most appropriate investigations to best tailor management.

References


Case Report:

Anisocoria is defined as unilateral pupillary dilation which has a number of benign and sinister causes. In the setting of acute stroke, this worrying clinical sign requires urgent investigation to exclude an impending neurological emergency. We present a rare and unusual benign cause for asymmetrical pupils in a patient following a stroke resulting from contamination of the eye with nebulised ipratropium.

A 79 year old female presented with dyspnoea and a productive cough. There was no other significant past medical history and she was an ex-smoker with a 40 pack-year-history. Initial examination revealed a tachycardia of 140 beats-per-minute and pyrexia of 38.5°C with coarse right-sided crepitations on chest auscultation. Initial bloods revealed an elevated WCC of 17x10^9/L and CRP 131mg/L. All other blood tests were unremarkable. A chest x-ray confirmed right basal consolidation and electrocardiography revealed fast atrial fibrillation, therefore intravenous antibiotics and digoxin were commenced. The following day she developed a new right-sided facial droop and dysphasia and urgent CT head demonstrated an acute left occipitoparietal infarct. Despite treatment, she continued to deteriorate and developed a widespread expiratory wheeze. Arterial blood gas sampling revealed chronic type II respiratory failure and nebulised salbutamol and ipratropium were started to treat a presumed infective exacerbation of undiagnosed COPD.

Shortly after commencing nebulisers, a new right-sided mydriatic pupil was noted and an urgent repeat CT head was arranged to exclude extension or haemorrhagic transformation and revealed no new acute changes. Following cessation of nebulisers, the anisocoria resolved spontaneously.

Anisocoria secondary to direct contamination of the eye with nebulised ipratropium has been reported in only a handful of cases in the literature and is thought to arise from direct antimuscarinic effects on parasympathetic neurones. This rare phenomenon should be considered in any patient presenting with anisocoria following nebulisers after imaging has excluded a neurological aetiology.
A rare differential diagnosis of unilateral calf swelling in the Ambulatory Care Unit (ACU).

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Introduction

We present a rare differential diagnosis of unilateral calf swelling in the Ambulatory Care Unit (ACU).

Case Report

A 30 year old lady, of Indian descent, was referred to the ACU to rule out Deep Vein Thrombosis (DVT) with history of worsening unilateral leg swelling and pain. On examination her left calf was swollen and tender with some erythema. There were no systemic infective symptoms and her blood work including C-reactive protein was normal. Her father had history of treated Pulmonary Tuberculosis (TB) and she was investigated by the respiratory physician 6 months ago and discharged following normal results.

Ultrasound Doppler was negative for DVT but showed a large echogenic area to the left lateral calf and an enlarged left inguinal node. Left leg X-ray (picture 1) and MR (picture 2) of the left lower calf showed evidence of osteomyelitis affecting the distal third of fibula and fluid collection over the posterior calf.

She was referred to the TB clinic; 5ml of pus was aspirated from the calf swelling which was smear and culture negative. Clinical and radiological diagnosis to TB osteomyelitis and abscess was made and she was treated with a standard 6 months course to quadruple anti-TB treatment. She made a good clinical and radiological recovery.

Discussion

Bone and joint TB accounts for some 10–15% of non-respiratory disease, with approximately 50% in the spine, and 50% in other bones and joints (1). Unlike respiratory TB where cultures are gold standard for diagnosis, due to low bacterial load in non-respiratory TB, microbiology results are not always positive. Anti-tuberculous drugs are the mainstay of treatment (2). Diagnosis & treatment can be difficult due to atypical and late presentation, multidisciplinary approach is crucial along with a thorough history and examination.

References

(1) Tuberculosis Clinical diagnosis and management of tuberculosis, and measures for its prevention and control. NICE clinical guideline 117 March 2011

Acute Dysphagia as the Presenting Feature of Syringobulbia in a Sixteen-Year-Old Patient

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Case Report

A sixteen-year-old female with no past medical history presented with a one-week history of painless dysphagia to solids and liquids and a two-week history of bowels not opening. Neurological examination on admission revealed left-beating nystagmus but no other cranial nerve or peripheral neurological deficits. CT Head showed a longitudinal Cerebrospinal Fluid-density lesion in the Medulla and Cervical Cord. Within eight-hours of her initial neurological examination, the patient developed bilateral Glossopharyngeal, Vagus, Accessory and Hypoglossal Nerve palsies, bilateral nystagmus, and glove-and-stocking paraesthesiae. MRI spine showed an extensive, complex hydro-syringomyelic cavity extending from the junction of the Fourth Ventricle and central Spinal Canal to the T10 level, with associated cord expansion. The patient underwent neurosurgical excision of the tumour and decompression of the syrinx within three days of admission. She subsequently made a full neurological recovery. Histology revealed a Grade 1 haemangioblastoma. The patient is being investigated for other features of Von Hippel Lindau Syndrome.

This case illustrates the importance of regular neurological examination to detect evolving signs which may indicate life-threatening rises in intra-cranial or intra-spinal pressure. It also demonstrates that an extensive spinal cord space-occupying lesion in a young patient may be part of an underlying condition such as Von Hippel Lindau Syndrome, and therefore prompt imaging of the Central Nervous System should be considered in any patient with a focal neurological deficit. Finally, it serves as a reminder that timely referral to other specialties can be life-saving for patients admitted on the Acute Medical Take.
Allopurinol related deranged liver function and eosinophilia – Think DRESS!

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Aim
To increase awareness of DRESS (Drug reaction with eosinophilia & systemic symptom) and allopurinol hypersensitivity

Methods
47 years old Asian male presented with 1 week history of malaise with fever, night sweats and rash over body and arms. He is generally fit and well with no previous medical history. He was not taking any regular medications apart from allopurinol 300mg od which was commenced by his GP for asymptomatic hyperuricaemia 2 months ago. He is non-smoker and does not drink alcohol. He has no recent travel history and no contact history of tuberculosis.

Initial examination revealed SIRS with temperature of 39’ C and heart rate of 115 bpm but other vital parameters were within normal limit. There was diffuse erythematous rash involving his face, trunk and upper limbs. There was no cervical lymphadenopathy or organomegaly.

Laboratory examination reveals abnormal liver and renal function test with high white cell count mainly eosinophils and neutrophils.

Outcomes / Results
Initial diagnosis was viral illness and he was treated with IV fluids, IV antibiotics. Blood tests for liver screen including quantiferon test for TB all came back as normal. USS of liver and biliary tree is normal. However, his liver and renal functions did not improve, so liver biopsy was performed which showed drug induced reaction. Definitive diagnosis is DRESS secondary to allopurinol. Steroids therapy (prednisolone 40mg od) was commenced and his condition improved subsequently.

Conclusion
DRESS is a potentially life threatening, drug induced hypersensitivity reaction presenting with rash, fever, eosinophilia, lymphadenopathy and single or multiple organ involvement (liver, kidney, lung).

Commonly used medications such as allopurinol & antiepileptics are the most frequently reported causes.

Prompt withdrawal of causative drug is essential in the management of DRESS.

The latency between drug exposure and onset of symptoms is considerably longer (2-6 weeks) in DRESS than in most drug eruption.

References
An unusual case of breathlessness

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Case Report

Breathlessness is a common presenting complaint on the acute medical post take ward round, and there is a broad range of differential diagnoses. This case highlights a rare but important cause of breathlessness.

A 62 year old man was admitted with a two week history of progressive breathlessness, affecting him when lying flat. He reported a recent diarrhoeal illness, but no other cardiovascular, respiratory or neurological symptoms. He was a lifelong non-smoker, and had no occupational or environmental risk factors for lung disease.

Clinical examination was normal apart from the onset of acute dyspnoea upon attempting to lie flat.

Routine blood tests, ECG and D-dimer were normal, however his chest X-ray (CXR) showed bilateral elevated hemi-diaphragms. Pulmonary function tests showed an extra-pulmonary restrictive defect and sniff inspiratory muscle pressures were markedly reduced, indicating a diaphragmatic weakness.

He was diagnosed with bilateral phrenic nerve palsies and referred to the Neurology team. He went on to have a lumbar puncture which showed an elevated protein count, and nerve conduction studies showed absent phrenic response bilaterally. He was diagnosed with Guillain-Barre Syndrome and was treated with intra-venous immunoglobulin and respiratory support with nocturnal non-invasive ventilation.

Phrenic nerve palsies should be considered in patients who present with exertional dyspnoea and orthopnoea, with apparent normal cardiac function and CXR appearances. Symptoms are more pronounced in bilateral palsies, though these are much rarer. Common causes include metastatic infiltration of the spinal cord, trauma and inflammatory neuropathies. Suspicion of phrenic nerve palsy can be confirmed with spirometry demonstrating an extra-thoracic restrictive pattern and a significant reduction in sniff inspiratory mouth pressures. Patients with underlying lung disease or bilateral palsies have a worse prognosis and they are more likely to require long term ventilator support.
An unusual case of leg swelling

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Case Report

A swollen painful leg is a common presentation to acute medical take, often attributed to deep vein thrombosis. Therapeutic dose of low molecular weight heparin while awaiting Doppler ultrasound to confirm diagnosis is widely used approach in such presentation. However, differential diagnosis is broad and includes some rare conditions likely to be missed if not considered.

We report the case of a 69 year old patient who presented with a two weeks history of leg pain, swelling and reduced mobility. Neurological examination revealed left leg weakness (MRC grade 4/5) associated with hyporeflexia and bilateral plantar flexor. There was altered sensation throughout the limb without sensory level. Rest of systemic examination was normal with no evidence of cellulitis or inguinal lymphadenopathy.

Past medical history includes right hemicolectomy for colonic adenocarcinoma. Blood tests were normal apart from the mildly elevated C-reactive protein and D-Dimer. A Doppler ultrasound excluded DVT. MRI spine performed to look for diagnosis which showed a huge (16 x 14cms) rather unusual abdominal aortic aneurysm eroding into the L3 and L4 vertebral bodies and plexus indicating significant chronicity, with no evidence of rupture or leak, accounting for his neurological signs. Patient underwent uneventful open repair and grafting in a tertiary centre with good outcome.

Compression effect of large AAA can rarely leads to direct erosion of vertebrae and plexus which can present with unilateral leg pain, swelling and weakness. Our case highlights the importance of thorough history, detailed examination, considering sinister and unexpected pathologies and arranging appropriate investigation. In our patient the diagnosis was made on MRI spine done to exclude spinal cord pathology.

Unilateral leg weakness could be an unusual presentation of AAA, vigilance should be applied when initial diagnosis is not certain. Spinal cord imaging should be considered in cases with painful swollen leg with neurological signs.

References

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An Unusual Cause of Breathlessness

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Case Report

A 77-year-old Japanese lady presented to the Acute Medical Take at 3am with a seven-day history of worsening breathlessness and flu like symptoms. She was normally an independent lady with no smoking history.

On arrival she was tachypnoiec, tachycardic with a BP of 120/70. Her ECG revealed a sinus tachycardia. Initial blood tests revealed an AKI with eGFR of 34, CRP -71, Na -110, WCC-14.9 with a neutrophilia of 12 and a troponin - 66 (normal<14). An arterial blood gas revealed type 1 respiratory failure and a metabolic acidocis. A chest radiograph (Figure 1) showed a bibasal effusion and cardiomegaly.

A provisional diagnosis of severe sepsis and pneumonia was made for which antibiotics were initiated. As she was struggling NIV was in initiated. An hour into her admission and 30 minutes after initiating NIV she deteriorated and became hypotensive (BP 80/50). At this stage a focused cardiac ultrasound revealed a cardiac tamponade with right ventricular collapse and a large right-sided pleural effusion (Figure 2).

She subsequently had a PEA cardiac arrest. An emergency pericardiocentesis removed 400 ml of blood stained fluid. A right sided chest drain was inserted which drained 1500ml of blood stained fluid. She regained cardiac output and was transferred to Intensive Care. A CT CAP reveled bilateral consolidation with right-sided pleural effusion. Pericardial fluid cytology later revealed an adenocarcinoma consistent with a lung origin. She made a full recovery and was discharged from hospital after ten days with a view to have a pericardial window and chemotherapy.

This case highlights the importance of swift diagnostic decision making in the acutely unwell patient. It also provides evidence to support the need for the acute medical registrar to be trained in focussed ultrasound. This will aid correct diagnosis and manage the patient appropriately and most importantly save lives.

Word Count 300. Patient consented.

(Video clips of cardiac tamponade available if selected for oral presentation)
An Unusual Presentation of Post-Partum Hypo-pituitarism

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Aim

We describe an unusual presentation of a young lady 2 weeks post-partum with acute panhypopituitarism.

Case

A 28 year old primipara presented with a 2 day history of wide based gait, diplopia, nausea and vomiting 2 weeks postpartum.

Following an uneventful pregnancy her delivery was complicated by a third degree tear and significant blood loss resulting in an episode of hypotension. She had been unable to breast feed since the birth.

A day prior to presentation she developed dysarthria and had a tonic/clonic seizure on arrival in ED, requiring high dependency unit care.

Blood tests showed acute hyponatraemia, very low Random Cortisol, hypopituitary hypothyroidism and hypopituitary hypogonadism.

A CT head and MRI pituitary done thereafter showed an enlarged postpartum pituitary.

She was treated with steroid replacement resulting in complete resolution of her hyponatraemia. Following normalisation of her electrolytes she was commenced on Levothyroxine and made a rapid clinical recovery.

Conclusion

Differential diagnoses of Pituitary apoplexy and cavernous sinus thrombosis were considered due to visual and neurological symptoms. Although Sheehan’s syndrome (acute-type 1) was our primary differential classic symptoms of hypotension and tachycardia were absent at presentation.

Her symptoms and seizure were secondary to acute hyponatraemia which is an uncommon manifestation of Sheehan’s syndrome\(^1\). This illustrates that cases of acute pan hypopituitarism can present atypically to the acute medical take causing potentially life threatening illness.

We also demonstrate that a diagnosis of Sheehan’s syndrome cannot be made on acute imaging of the pituitary and sequential imaging might be necessary\(^2\).

References

Atrioventricular block associated with benzodiazepine use.

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Case Report

I report a case of transient second degree atrioventricular block associated with administration of benzodiazepine for control of agitation in a young male.

A 15 year old boy was brought to the emergency department (ED) after being found at home drowsy and difficult to rouse. A few tablets of diazepam were found on him. Initial vital signs were within normal parameters. He had GCS 14; E4V4M6. Pupils were dilated 6mm bilaterally, equally reactive. Physical examination was unremarkable. His admission ECG revealed normal sinus rhythm with PR interval of 192ms (Figure 1). He became agitated and aggressive in the ED requiring physical restraint. He received Haloperidol 5mg IM, Diazepam 10mg IV, and titrated doses of Midazolam up to total 60mg IV over 50 minutes. He was transferred to the medical admissions unit for further management.

Over the course of the next fourteen hours he remained drowsy and developed second degree heart block (Mobitz Type 1), this was documented on serial ECGs performed at six hours (Figure 2), eight hours (Figure 3), twelve hours (Figure 4) and fourteen hours (Figure 5) post benzodiazepine administration. He was bradycardic with heart rate between 50 and 60 beats/min, requiring intermittent fluid boluses for hypotension. His serum calcium level was 2.14mmol/L, treated with 10mls 10% calcium gluconate IV. The atrioventricular block resolved spontaneously sixteen hours following the benzodiazepine administration, ECG performed at the time revealed normal sinus rhythm (Figure 6, 7). He admitted to ingesting diazepam but denied any other substance abuse. He was discharged from hospital the following day.

Discussion

Benzodiazepines (BZD) bind to specific receptors in the gamma aminobutyric acid (GABA) receptor complex, which enhances the binding of this inhibitory neurotransmitter. A second class of benzodiazepine binding sites, called the peripheral type benzodiazepine receptor (PBR), is found in peripheral tissues including the myocardium (1). BZD receptor agonists can reduce the current through transient and long-acting calcium channels, hence BZD may behave like weak calcium channel blockers. There are also reports of the direct action of BZD on the suppression of atrioventricular node conductivity (2). This would account for the BZD associated atrioventricular block seen in our patient and explain the use of flumazenil in the reversal of benzodiazepine induced atrioventricular block (3). Flumazenil is a competitive antagonist of the BZD receptor.

Given the frequent use of benzodiazepines in management of agitation in patients, this case would serve to remind clinicians of the potential complication associated with exposure to this agent.

References

Beyond the Scope – A Rare Cause of Recurrent Upper Gastrointestinal Haemorrhage due to Gastric Varices Secondary to an Isolated Splenic Vein Thrombosis

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Case Report

Acute upper gastrointestinal haemorrhage (GIH) due to bleeding oesophageal or gastric varices is often associated with high risk of morbidity and mortality. In the absence or clinical suspicion of underlying chronic liver disease and right-sided portal hypertension, very often such varix can be overlooked and missed during oesophagogastroduodenoscopy (OGD). We present an exceptionally rare case of a patient presenting with recurrent GIH due to massive gastric varices secondary to an isolated splenic vein thrombosis with no evidence of pancreatic or liver disease.

A 59 year-old-male with a background of colonic carcinoma, diabetes and renal transplant following chronic pyelonephritis presented with melaena in March 2012. OGD revealed a small 3cm segment of Barrett’s oesophagus thought to account for his presentation. He was subsequently commenced on high-dose omeprazole and discharged. There was no significant smoking or alcohol history. Over several months he presented with recurrent episodes of GIH and repeated OGDs were all unremarkable. In December, he presented with haematemesis and haemoglobin was noted to drop from 139 to 93g/L. Urea was elevated at 28mmol/L. Repeat OGD revealed prominent gastric mucosal folds suggestive of a varix. Ultrasonography of the liver was normal with no evidence of portal hypertension. CT angiography revealed moderate gastrosplenic varices with an isolated splenic vein thrombosis. Appearances of the liver and pancreas were unremarkable. Due to ongoing haematemesis an emergency splenectomy was performed and the patient was discharged home three days later.

Varices due to left-sided portal hypertension is rare and most often occurs in association with splenic vein thrombosis secondary to pancreatic pathology. Isolated thrombosis within the splenic vein is exceptionally rare and most probably occurs due to an underlying myeloproliferative disorder. In the absence of chronic liver disease and portal hypertension, a bleeding varix is often easily missed, therefore this rare entity should be considered in any patient presenting with recurrent GIH and normal serial OGDs.
Bilateral Internal Jugular Thrombosis complicating Ovarian Hyperstimulation Syndrome

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Case Report

A 37 year old lady presented to the emergency department complaining of tightness in her chest and pain in her left neck. She described the pain as an intermittent "squeezing pain", non-pleuritic in nature. She was feeling generally lethargic and had nasal congestion for a week prior to presentation. Following a successful cycle of IVF, she was 10 weeks pregnant. The confirmatory scan further diagnosed mild ovarian hyperstimulation syndrome (OHSS). She had no significant past medical history.

Physical examination was normal and vital signs normal. An electrocardiogram tracing was normal. Her blood tests showed a marginally raised white cell count with increased neutrophils. She was re-assured, at the time, that this was most likely a mild viral infection, and discharged.

She re-presented to the emergency department one week later, complaining of ongoing pain and tightness in her chest, and now complained of pain on both sides of her neck. Physical examination of her neck revealed tender, well defined supraclavicular fullness in her neck. There was no palpable lymphadenopathy and her carotid arteries were palpable bilaterally. A bedside ultrasound was performed which confirmed a diagnosis of bilateral subclavian vein thrombosis.

As the patient was within her first trimester, she was managed with treatment dose of low molecular weight heparin for the duration of her pregnancy. A repeat ultrasound of her neck showed decreased thrombotic burden. She delivered her baby at term with no further complication from the OHSS.

This case illustrated a number of valuable clinical lessons for acute medical physicians:

- Thrombotic disease is an important complication of Ovarian hyperstimulation syndrome.
- Due to the rarity of upper extremity DVT, a secondary cause should always be considered
- Thrombosis
- Bed side ultrasound can be used to diagnose jugular vein thrombosis.
- Low molecular weight heparin is the treatment of choice for DVT in pregnancy
**Brugada syndrome unmasked by fever – commoner than we think?**

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**Aim**

We describe two patients with Type 1 Brugada morphology noted on 12-lead electrocardiography (ECG), both unmasked by concurrent febrile illness.

**Cases**

A 54-year-old lady was admitted with pyelonephritis. An ECG done when febrile at 39°C revealed Type 1 Brugada morphology, which reverted to normal when afebrile. Subsequent ajmaline provocation testing confirmed Brugada syndrome (Figure 1).

A 51-year-old gentleman with type 1 diabetes mellitus presented with a febrile illness, which was eventually determined to be due to a urinary tract infection. An ECG taken when febrile at 38.2°C showed Type 1 Brugada morphology, which reverted to incomplete right bundle branch block with defervescence (Figure 2).

**Discussion and conclusion**

Brugada syndrome is a hereditary cardiac sodium channelopathy, and the Brugada ECG pattern is estimated to exist in 0.05% of the population.\(^1\) Recognised potentiators of sodium channel blockade include certain drugs such as class I antiarrhythmic agents, and metabolic conditions such as hyperkalaemia. Fever has also been reported to unmask Brugada ECG morphology, and recent studies suggest that the Brugada ECG pattern is 20 times more common in febrile patients.\(^2\) We discuss the different Brugada ECG patterns, as well as the pathophysiology and prognostic implications of an inducible versus fixed ECG pattern and subsequent risk stratification. We also propose that febrile patients should have opportunistic ECG recording to identify this potentially lethal condition.

**References**

Case Report

A 33 year old male presented with a five day history of vomiting associated with severe cramping abdominal pain. On examination: clinically dehydrated, generalized abdominal tenderness, voluntary guarding but no peritonism and bowel sounds present. He had an erythematous rash to his upper thighs and buttocks which was blanching. He had deranged renal function with a stage 3 AKI. Venous blood gas showed no metabolic derangement. Urgent US KUB showed no evidence of obstruction. With aggressive fluid resuscitation his renal function improved rapidly back to baseline within 48 hours.

He had found bathing in a hot bath improved his symptoms and the rash was felt to be from scalding. He also admitted to daily cannabis use over many years.

On review of his past medical notes he’d had multiple admissions stretching back at least five years with similar presentations. He had been extensively investigated with US abdomen, gastroscopy and full renal screen including immunology on prior occasions. With the only abnormality detected being mild gastritis.

He had features consistent with cannabis hyperemesis syndrome. He met diagnostic criteria as proposed by the Mayo Clinic\(^1\). These include: long-term cannabis use, severe cyclical vomiting, relief of symptoms with hot showers or baths, abdominal pain, weekly use of marijuana, normal bowel habit and negative laboratory, radiological and endoscopic test results.

Given the widespread use of cannabis, 6.6% of adults aged between 16 and 59 in England and Wales in 2014\(^2\), cannabis hyperemesis syndrome needs to be considered in all cases of unexplained vomiting.

References


Discitis: A pain in the neck

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Introduction

Neck pain is a common reason for presentation to health services. Most patients have simple mechanical neck pain but a small proportion have serious pathology. We present a case of infective cervical discitis with associated abscess due to haematogenous spread from a pseudomonas aeruginosa catheter related urinary tract infection.

Case

A 71 year old gentleman with benign prostatic hypertrophy and recent Pseudomonas catheter related urinary tract infection presents to hospital with a 6 week history of progressive neck pain radiating to both shoulders and hands despite treatment with paracetamol, anti-inflammatories and opiates by his General Practitioner. The main clinical findings were cervical tenderness and bilateral upper limb weakness 4/5 with no sensory deficits. Temperature was normal.

Investigations showed a leucocytosis of 16.7, neutrophillia of 14 and raised C-reactive protein of 73. Blood cultures were negative. Urinalysis was negative for leucocytes and nitrites. An MRI C spine showed marrow oedema affecting C5-6 vertebral bodies with an associated extradural abscess causing cord compression. The patient was transferred urgently to a neurosurgical centre for surgery. Disc cultures confirmed pseudomonas aeruginosa. He was treated with intravenous ceftazidime for 8 weeks followed by oral ciprofloxacin for 4 weeks.

Discussion

Discitis in adults has a slow insidious onset which may cause delay in diagnosis leading to morbidity. Neck pain with localised tenderness exacerbated by movement is the initial presenting complaint. Clinical red flags such as fever, abnormal neurology and urinary retention are well recognised but may not be present as in this case where progressive neck pain and arm weakness was the main complaint. This case serves to highlight to physicians the insidious nature of discitis and the diagnostic challenges that spinal pain poses. Discitis should be actively considered in patients with atraumatic acute or sub-acute back pain particularly with raised inflammatory markers.

References

A 37 year old gentleman, known to have inflammatory arthritis, was found to have left VI nerve palsy and was seen in ophthalmology clinic. Whilst awaiting further investigations he was admitted, acutely unwell, with quadriaparesis. During his admission he was also found to have a right VII nerve palsy and had a partial complex seizure. He proceeded to have a lumbar puncture and was treated for presumed encephalitis and atypical Guillain-Barre syndrome (GBS) with intravenous acyclovir and intravenous immunoglobulins (IVIg). His cerebro spinal fluid (CSF) analysis showed a grossly raised protein level and the presence of oligoclonal bands. An MRI scan this admission showed inflammation of the left orbit. He improved and was subsequently sent for nerve conduction studies, which showed the presence of a primary acute demyelinating polyradiculopathy. After discussion with the neurologists, it was felt that all his symptoms were in keeping with GBS. He underwent rehabilitation and was discharged home. However, two weeks later he re-presented to us for management of Lyme's disease. His CSF was found to be positive for Borrelia infection. His GP had discussed this with the microbiology team, after he developed an allergic reaction to oral penicillin, and he was therefore admitted for intravenous ceftriaxone treatment. During the second admission he happened to be under the care of the rheumatologists who investigated him further and found that he actually has IgG4 disease. On reviewing the notes it was felt that his raised oligoclonal bands and CSF protein were due to a systemic immune response secondary to IgG4 disease, and that the initial administration of IVIg for presumed GBS had resulted in a false positive Borrelia screen. Therefore, we present a unique case of IgG4 disease, and possibly GBS with asymmetrical VI and VII nerve involvement, which mimics a case of Lyme's disease.
Euglycemic ketoacidosis: not just for patients with diabetes

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Background

Euglycemic ketoacidosis is a rare endocrine emergency and a rare presentation of diabetic ketoacidosis. If left undiagnosed without treatment with insulin and dextrose, it can have dire consequences. This is a case of a non-diabetic presenting with acute confusion and profound euglycemic ketoacidosis which improved with insulin and fluids.

Case

A 56-year-old lady presented to A&E with confusion, GCS12, and hypoglycemia. Her husband reported reduced oral intake for 12 hours and vomiting. Past medical history included subdural hematoma, depression and hypothyroidism. Medications included quetiapine, levothyroxine, and sertraline.

Blood tests revealed AKI, CRP579, WCC28. CT head and abdominal ultrasound showed no acute pathology, CXR showed bilateral consolidation. Initial blood gas pH7, BE-23, glucose 1.7 and lactate 0.5. Toxicology screen, hepatitis and HIV were negative.

She was initially treated with IV sodium bicarbonate, IV fluids and antibiotics for sepsis. 8 hours later she was more coherent, and blood gas improved to pH7.25. 14 hours post-admission ketones were measured at 4.9, and treatment for euglycemic ketoacidosis was commenced with insulin/dextrose for 42 hours. She improved clinically and blood gas was normal.

Outcome and follow-up

She was discharged home 8 days later and is being investigated for carnitine deficiency.

Discussion

Although euglycemic ketoacidosis has been diagnosed in the context diabetes(1), it is not well known in patients without insulin deficiency. Prater et al discuss a case where it presented in the context of alcohol-related pancreatic injury and starvation for over 1 week leading to inhibition of gluconeogenesis(2). Joseph et al report the condition in a type 1 diabetic whose ketoacidosis was triggered by starvation secondary to severe depression(3).

However, this case presents a lady with normal HbA1c, a period of starvation not exceeding 12 hours, acutely presenting with profound euglycemic ketoacidosis. Other causes of anion gap metabolic acidosis were excluded, and her acidosis improved with sodium bicarbonate initially, then with insulin and dextrose infusion.

Learning points

- The importance of ketone testing in anion gap metabolic acidosis, even in the context of euglycemia
- The early role of ITU and critical care outreach in patients presenting with acidosis

References

Excessive belching as a presenting feature in aortic dissection: A case report

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Case Report

A 74 year old male presented to A&E with acute severe central lower chest/upper abdominal pain. PMH: COPD, gastro-esophageal reflux, and duodenal-ulcer. SHx: ex-smoker (70-PY), drinks 24u alcohol/week. Initial examination: BP:182/91mmHg, HR:48 bpm, SPO2:97%OA, mild epigastric tenderness.

ECG: sinus bradycardia. U&E, amylase and LFT normal. CXR: Cardiac magnification (AP projection). He was treated for acute coronary syndrome (ACS) and also received pantoprazole. 12-hour troponin I: negative. Bradycardia resolved.

In a few hours pain diminished to 2/10 and remained mainly epigastric. His main complaint now: new onset severe belching. History revisited: the original pain started in his shoulder blade, then chest and abdomen, radiating to back. The epigastric nature, worse on eating and lying flat, was similar to his indigestion though no response to ranitidine.

Biliary ultrasound: normal. Systolic BP in both arms revealed 34 mmHg discrepancy. Repeat ECGs: no dynamic changes. CT aortogram reported as acute aortic dissection(AD) from above the renal arteries to the aortic arch.

He was transferred to a cardiothoracic unit and managed conservatively for reportedly two penetrating aortic ulcers(PAU) and extensive intramural haematoma(IMH) with labetolol infusion.

Discussion: Acute aortic syndromes(AAS) encompass PAU, IMH and AD. Prompt recognition is essential as they carry high rates of morbidity and mortality. A high index of suspicion is important as AAS is an uncommon cause of chest/abdominal pain. Good history taking, as always, is paramount. Our patient with predominantly gastrointestinal complaints after the first few hours could have been mismanaged with a gastrointestinal diagnosis. Our literature search did not reveal previous documentation of an association between belching and AAS, whilst belching is a known complaint in ACS. Our proposed mechanisms are: vagal-stimulation, air-swallowing (due to pain), or, given the close anatomical relationship between oesophagus and aorta, a direct pressure from a swollen aorta affecting oesophageal peristalsis and lower oesophageal sphincter.

References


Iatrogenic disseminated Bacillus Calmette-Guérin infection from immunotherapy for bladder cancer

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Shiva Sreenivasan
Andrew White

Aim

We describe a patient with fever caused by disseminated Bacillus Calmette-Guérin (BCG) infection secondary to local immunotherapy for bladder cancer.

Case

A 67-year-old gentleman with a history of recurrent bladder cancer treated with intravesical BCG immunotherapy presented with a 2-week history of weight loss and rigors. Radiographic and computed tomographic (CT) imaging revealed a classic miliary pattern of disseminated mycobacterial disease (Figure 1). Bronchoscopic washings and interferon-gamma release assay testing were negative for Mycobacterium tuberculosis. A clinical diagnosis of disseminated BCG infection was made, and the patient responded well to a 9-month course of anti-tuberculous treatment.

Discussion

Intravesical BCG is commonly used to treat superficial bladder cancer. Disseminated BCG infection is a rare and often under-recognised complication of BCG immunotherapy, with recent studies reporting an incidence of 4.3%.

Clinicians should have a low threshold for suspecting and treating this in susceptible patients.

References

May-Thurner syndrome-related iliofemoral deep vein thrombosis as the initial clinical presentation of perforated sigmoid diverticulitis

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Shiva Sreenivasan

Aim

We describe a case of a patient with iliac vein compression (May-Thurner) syndrome-related deep vein thrombosis (DVT) as the initial presenting feature of perforated sigmoid diverticulitis.

Case

A 67-year-old gentleman presented with a 3-day history of painful left leg swelling. Examination was normal apart from a swollen left leg. Ultrasonography demonstrated extensive left iliofemoral deep vein thrombosis (DVT) and anticoagulation was commenced. Subsequent computed tomographic (CT) imaging revealed pulmonary emboli, sigmoid diverticulosis, and a left iliofemoral DVT caused by left common iliac vein compression by the right common iliac artery (May-Thurner syndrome). There was also the surprising finding of free peritoneal gas of unclear aetiology (Figure 1).

Repeat history and examination revealed neither gastrointestinal symptoms nor abdominal signs, but over the next few days the patient developed progressive abdominal pain with localised peritonism. There was a rise in his serum inflammatory markers, and repeat CT revealed peritoneal collections of gas and fluid with extensive mesenteric fat stranding, typical of perforated sigmoid diverticulitis (Figure 2). He was treated conservatively with parenteral antibiotics, and subsequently discharged on long-term anticoagulation.

Discussion and conclusion

Whilst a relationship between diverticular disease and subsequent venous thromboembolic disease has been established\textsuperscript{1}, to our knowledge this is the first reported case of May-Thurner syndrome-related iliofemoral DVT as the presenting clinical feature in a patient with perforated diverticulitis.

References

Ocular myasthenia gravis mimicking brainstem stroke

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Case Report

This 82 year old lady was referred urgently to the high risk TIA clinic by her Ophthalmologist after she presented to him with a two week history of acute deterioration in vision in her left eye, diplopia and left sided ptosis. She had previously seen him for left eye Age-related Macular Degeneration and her vision impairment was attributed to this. Her other past medical history included hypertension, peripheral vascular disease, abdominal aortic aneurysm and hypothyroidism.

On examination in the TIA clinic this patient had diplopia on upward, left and downward gaze with normalisation of the image on covering her left eye. She also had marked ptosis and decreased visual acuity in the left eye. Examination of the right eye was normal. Both pupils were equal and reactive to light. The remainder of the cranial nerve examination was normal. She also had normal power and sensation in all limbs.

Routine blood tests were unremarkable. MRI brain showed no restricted diffusion to indicate recent infarct. There was only mild generalised involutional and periventricular white matter change. A CT of the orbits showed no abnormality. Anti-acetylcholine receptor antibodies came back significantly raised.

This patient has ocular myasthenia gravis but remains at risk of the generalised form. Myasthenia gravis is an under-recognised condition, especially in the elderly. In the context of sudden onset unilateral signs and the absence of a clear history of fatigability it can easily be misdiagnosed as a stroke, especially in the acute setting. Considering this important differential diagnosis in your patients with suspected brainstem stroke could save them from significant morbidity.
Orthostatic headache in a 21-year old free runner

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Paresh Mistry

Introduction Headache caused by intracranial hypotension is often reported as being orthostatic in nature.¹ Low cerebrospinal fluid (CSF) volumes and pressure can results from dural trauma or can be spontaneous.²

Case report We present the case of a 21-year old free runner who was admitted to our Acute Medical Unit in a London District General Hospital with a worsening headache. The patient reported an orthostatic headache since sustaining a head injury 10 days previously. He experienced no loss of consciousness but complained of dizziness, photophobia and vomiting on more than 2 occasions. The patient’s GCS was 15/15 and a full neurological examination demonstrated diplopia on right lateral gaze but was otherwise normal.

A CT head scan, and subsequently an MRI head scan was performed, that showed no skull fractures or intracranial haemorrhages but low-lying cerebellar tonsils – initially thought to be an incidental finding of Chiari malformation.

After a review by a neurology consultant, the patient was diagnosed with a headache secondary to intracranial hypotension. Inferior displacement of cerebellar structures is one of the known potential MRI findings in intracranial hypotension.³

The patient was managed conservatively with bed rest and IV hydration, and made an uneventful recovery.

Discussion CSF leak from a dural tear, in this case caused by head trauma, is a recognised cause of intracranial hypotension.² Headache can also occur from spontaneous intracranial hypotension, which is rare with a reported annual incidence of 5 per 100,000.⁴ These patients are often misdiagnosed or it may be months to years before a diagnosis is made.⁴

We want to highlight intracranial hypotension as a rare, but possible, diagnosis in patients presenting with a headache to the Acute Medical Unit, and emphasise the importance to establish the orthostatic nature of headaches.

References

Snap! Crackle! Pop! The perils of complementary therapy

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Aim

Complementary therapy is often considered as either benign or equivalent to placebo. We discuss two patients admitted with life-threatening complications of complementary therapy for chronic back pain.

Cases

A 76-year-old lady was admitted with pleuritic chest pain and dyspnoea six hours after having acupuncture. Chest radiography confirmed a left pneumothorax (Figure 1), which required intercostal drainage.

A 65-year-old lady was admitted with severe back pain and dyspnoea following chiropractic therapy the previous day. Computed tomographic imaging confirmed a leaking thoracic aneurysm with features of a type B aortic dissection (Figure 2), which necessitated urgent transfer to a national cardiothoracic centre. We postulate that her chronic back pain was secondary to an expanding thoracic aneurysm.

Discussion and conclusion

Acupuncture-induced pneumothorax is rare, occurring only twice in nearly a quarter of a million treatments.\textsuperscript{1} We also discuss other reported complications, which include spinal cord injury, hepatitis B, and cardiac tamponade.\textsuperscript{1} Serious complications of chiropractic therapy include vertebrobasilar strokes, disc herniation, and cauda equina syndrome.\textsuperscript{2} Few prevalence studies exist of serious adverse effects after chiropractic therapy.\textsuperscript{3} Patients should be fully informed of potential risks of complementary therapy.

References

Stress Cardiomyopathy (Takotsubo) Associated with Duloxetine Overdose

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Case Report

Objective: To describe a case of Takotsubo Cardiomyopathy in a patient after an overdose of the serotonin norepinephrine reuptake inhibitor Duloxetine.

Method: Presentation of a case report including both clinical and laboratory data and a review of the current relevant literature on Takotsubo Cardiomyopathy and the use of serotonin norepinephrine reuptake inhibitors.²

Results: A 74 year-old woman presented to the emergency department after an overdose of duloxetine prescribed for major depression. No chest pain was reported. Her electrocardiogram showed anterolateral T-wave inversion. Her troponin-I and creatine kinase levels were elevated. Echocardiogram showed mid-apical hypokinesia. Coronary angiography excluded coronary artery disease. Cardiac MRI showed wall motion abnormalities and acute oedema involving the mid to apical left ventricle circumferentially in a non-coronary distribution but no late gadolinium enhancement, characteristic of Takotsubo Cardiomyopathy.

Conclusions: To our knowledge this is the first reported case of an overdose of Duloxetine associated with Takotsubo Cardiomyopathy. With recent trends in prescribing towards selective norepinephrine reuptake inhibitors for selective serotonin reuptake inhibitor-resistant major depressive disorder, duloxetine overdoses are increasingly likely to present to the emergency department in the future.³ As yet, there are few reported cases of duloxetine overdose,⁵ and clinicians working in acute medicine should be aware of Takotsubo Cardiomyopathy as a potentially fatal complication.

References


⁵ Darracq M. et al. A retrospective review of isolated duloxetine-exposure cases. Clinical Toxicology. 2013. 51:2 106-110
The Double-edged Sword of Dsypnoea - Three Rare Mistaken Cases of Pulmonary Hypertension

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Case Report

Primary pulmonary hypertension is a rare and often underdiagnosed condition. Without correct treatment most cases progress to right heart failure and death however, appropriate treatment can significantly slow progression of the disease and improve symptoms. If patients are deteriorating or not responding to treatment one should question the primary diagnosis and remain inquisitive to other potential causes.

We report three unusual cases encountered over two years in our medical assessment unit:

Case 1 - A 23 year-old female was seen in MAU in Spring 2013 with increasing dyspnoea on exertion on a background of poorly controlled asthma for a year. Admission SpO2 was 77% on air and her friends noted marked cyanosis after walking half a mile.

Case 2 - A 54 year-old female visited her GP with dyspnoea which was extensively investigated and attributed to hyperventilation and anxiety for over a year. Exercise tolerance markedly reduced and her admission SpO2 was 89%.

Case 3 - A 53 year-old female presented with a history of persistent palpitations and feeling generally unwell for 6 months on a background of asthma, hypertension and retroviral disease on treatment. On admission she was found to be dyspnoeic with peripheral oedema and SpO2 of 92% on air.

Due to failure to respond to current treatment for the initial presentation, all of the above patients were re-reviewed for alternative diagnoses. Pulmonary hypertension (primary or secondary) was suspected in each due to the presence of right-sided heart strain patterns on electrocardiography which was subsequently confirmed via echocardiography. In each case CT pulmonary angiography was negative for thromboembolic disease. Right ventricular systolic pressure was elevated at 78mmHg in the first case, 59-64mmHg and 55-60mmHg in the second and third cases respectively.

All patients were referred to the regional pulmonary hypertension unit at Papworth Hospital for further assessment and were commenced upon diuretics, anticoagulation and pulmonary artery vasodilator agents. All patients subsequently responded to treatment and their quality of life improved.

These three cases all illustrated a pivotal lesson in the importance of re-reviewing and assessing an initial working diagnosis particularly when patients fail to respond to treatment and continue to deteriorate highlighting the need to broaden the differential diagnoses to include rare and life limiting conditions such as pulmonary hypertension.

References


The Grey Man

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The Grey Man

A 45-year-old gentleman presented to the Acute Medical Take with an episode of syncope. He was working as a chef and had no significant medical history. From the records he had one episode of drug overdose in the past.

On arrival he was severely cyanosed, tachypnoiec, tachycardic and hypotensive with a BP of 70/40. His ECG revealed a sinus tachycardia. He was confused and had a GCS of 13/15. His whole torso was grey in colour. He had admitted to have taken vodka on the same day.

Initial blood tests revealed normal CRP, WCC and a raised ALT at 89. An arterial blood gas revealed a metabolic acidosis (pH 7.32, pCO2 3.3, pO2 49 lac11, BE -11, HCO3 12.9, SaO2 69% and a methaemoglobin of 69%. A chest radiograph was normal. A focused echo scan revealed normal biventricular function.

Drug overdose or poisoning was suspected. The case was discussed with the local poisons unit and they advised to treat with methylene blue. As he was profoundly hypotensive intensive care was involved to provide inotropic support. 2mg/kg IV Methylene blue was administered. After one hour of administration the patient’s complexion turned from grey to pink in complexion. Blood pressure improved, acidosis improved and the methaemoglobin levels reduced in level slowly on intensive care and he was discharged a few days later. The case was reported to public health in order to investigate the composition of the vodka.

This case was important as it dealt with a rare and unfamiliar presentation where it is known that severely raised methaemoglobin above 70% can lead to seizures and death itself. Prompt recognition by the acute physician and treatment with methylene blue was vital in managing this condition.
Think before you treat a Troponin!

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Case Report

A 50 year old Non-UK resident presented to the Emergency Department complaining of epigastric pain. His English was limited but he explained that the pain had been there for 10 days, occasionally radiating to the back with some relief with Ibuprofen. He worked as a truck driver, smoked 10-15 cigarettes a day and occasionally drank alcohol.

On examination he appeared mildly dehydrated with audible wheeze. Heart rate was 100/minute, blood pressure 108/76mmHg. Nurses had noticed he was dizzy on standing and was walking as if he was drunk.

Bloods showed a Troponin of 214ng/L (normal <40), CRP 22mg/L, Hb 108g/L, MCV 80fl and Urea of 19.6mmol/L. ECG showed sinus tachycardia along with Left Ventricular Hypertrophy. Chest X-ray, Amylase and Creatinine were normal.

He was treated with Aspirin and IV fluids for a diagnosis of NSTEMI and transferred to the Tertiary Cardiac Centre.

He received further Clopidogrel and low molecular weight Heparin. Later he was found collapsed in the toilet, appeared pale with a heart rate of 140/min and a systolic of 70mmHg. He complained of dizziness and developed frank hematemesis. Hb was 3.5g/L. After resuscitation a Gastroscopy revealed a 3cm ulcer in the incisura of the stomach with 2 visible vessels covered by a fresh clot. The ulcer was treated with 14mls of adrenaline, 2 clips and heater probe. Patient was subsequently discharged to have repeat gastroscopy in 8 weeks.

Troponin is known to be elevated in Gastrointestinal Bleeding\(^1\). This patient was thought to have a NSTEMI in view of an elevated troponin; however a raised urea, low haemoglobin, resting tachycardia and orthostasis on a background of NSAIDS use would point towards a Peptic Ulcer. This case underlines the importance of careful clinical assessment while forming a diagnosis and the implications it has when we treat patients.

References

Think! It Could Be HIV

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Case Report

Despite advances in management and diagnosis, HIV infection continues to cause considerable morbidity and mortality in the UK\(^{(1)}\). The rate of undiagnosed cases remains high; an estimated 26,100 patients in the UK are undiagnosed \(^{(2)}\).

We present a patient who was diagnosed with HIV after admission to acute medicine with diarrhoea. A simple mnemonic is suggested to identify groups at risk who should undergo HIV testing.

A 43 year old female, visiting on business from Kenya presented with a one month history of diarrhoea worsening over the previous week. The patient opened her bowels six times daily with blood mixed in her stool and also some mild rectal bleeding. She also reported a one week history of nausea and intermittent cramping abdominal pain, as well as fatigue, decreased appetite, and weight loss. Her abdominal pain became constant on the day of admission, eased by defecation and exacerbated by eating. Examination was unremarkable aside from left iliac fossa tenderness. Tests revealed a mild microcytosis and lymphopenia. Stool cultures were positive for Shigella flexneri. The patient received a five day course of azithromycin and made a prompt recovery. No risk factors for HIV infection were identified in the patient history apart from her origin. She consented to an HIV test and was found positive with a high viral load (HIV-1 RNA 207000 copy/ml, CD4 count 0.00). The patient will be followed up locally in the sexual health clinic.

This case illustrates demographic risk factors and symptoms which the British HIV Association recognises as indications for HIV testing (see table 1, fig 1) \(^{(3,4)}\). A simple mnemonic is suggested in figure 1. The AMU can play a key role in diagnosing HIV. A joint effort between Acute Medicine and public health can effectively decrease the incidence, morbidity and mortality caused by HIV.

References


Type 1 spontaneous diaphragmatic rupture – an unusual cause of chest pain and dyspnoea

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Aim

Spontaneous rupture of the diaphragm (SRD) is a rare condition that often presents with chest pain. We describe a patient with SRD who presented with chest pain, dyspnoea, and radiographic appearances of a left pleural effusion.

Case

A 39-year-old construction manager presented with sudden-onset left sided chest pain that had woken him up from sleep five days previously. He described it as severe, worse on lying flat, and associated with new-onset dyspnoea. Physical examination and subsequent chest radiography was suggestive of a moderate left pleural effusion (Figure 1), which could not be confirmed with ultrasonography. Thoraco-abdominal computed tomography (CT) performed after the administration of oral and intravenous contrast revealed a large left diaphragmatic deficit with thoracic herniation of retroperitoneal fat, omentum, and the tip of the pancreatic tail (Figure 2). Following cardiothoracic surgery review, elective repair is planned in view of persistent symptoms.

Conclusion

Traumatic diaphragmatic rupture is well described following blunt abdominopelvic injury, but spontaneous rupture is exceedingly rare, with a quoted incidence of 1% of all diaphragmatic ruptures. Usual presenting features of SRD include chest or abdominal pain, vomiting, and dyspnoea. A suggested SRD classification is into type 1, which does not involve herniation through the chest wall, and type 2, in which abdominal organs herniate through the diaphragm and chest wall. Our case strengthens the importance of pleural ultrasound in confirmation of radiologic appearances of pleural effusion, as well as the need to think laterally when patients present with chest pain and dyspnoea.

References

Case Report

Giant cell arteritis (GCA) is a large vessel vasculitis that predominantly affects medium sized vessels such as arteries found in the head and neck. The prevalence is highest in females aged 70 years and it typically presents with symptoms of temporal arteritis. We present a case of atypical large vessel vasculitis with resulted diagnostic difficulties.

A 70 year old Asian male who was admitted onto the rapid assessment unit with a 2 week history of lethargy, productive cough and fever, weight loss and poor appetite. Examination was unremarkable. A septic screen was inconclusive; however with a high C reactive protein (CRP) of 212, patient was treated for an infection of unknown source with intravenous (IV) antibiotics. The patient however, remained symptomatic in addition to the raised CRP, and erythrocyte sedimentation rate (ESR) of 120. Further investigations including an immunology screen, cANCA, C3/C4 level, echocardiogram, bone marrow examination, colonoscopy, and computed tomography (CT) were performed which were negative of any pathology. A possibility of tuberculosis (TB) was raised in view of his ethnicity and symptoms. A decision was being made to start him on anti-TB medication. PET CT was done to rule out occult malignancy which showed extensive underlying vasculitis involving ascending aorta, descending aorta, iliac, femoral, subclavian and carotid arteries. Patient was managed with a 3 day course of IV methylprednisolone followed by 60mg prednisolone to which he responded well with improvement of symptoms.

Conclusion: This case demonstrates the atypical features of GCA with involvement of almost all large vessels and how these create challenges in prompt diagnosis of the disease. Furthermore, it signifies the role PET CT imaging has in diagnosing GCA where temporal arteritis isn't the initial presenting problem. Acute physicians should be aware of the use of PET CT in diagnosing occult vasculitis.
Vertebral Artery Dissection - not as uncommon as we think?

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Case Report

We present two cases of vertebral artery dissection (VAD) – along with neck vessel and cerebral imaging. Both cases presented in an atypical fashion.

Vertebral artery dissection is thought to be a rare cause of ischaemic stroke. Observational studies have demonstrated that the classical features of minor cervical trauma, neck pain and headache were not found in many patients with proven VAD\(^1\). The true incidence of VAD may be higher due to the paucity of reliably associated clinical signs.

A high index of suspicion is essential, particularly in young patients presenting with atypical neurological symptoms.

CASE 1 - A 33 year old man presented to ED with a short history of frontal headache, unsteadiness, clumsiness, and falls. Neurological examination was normal with no cerebellar signs. An initial plain CT brain scan was reported as normal. The patient was discharged with a diagnosis of atypical migraine. However, a subsequent neuroradiology review revealed a small right superior cerebellar infarct. The patient was recalled to ED the next morning and CT angiogram confirmed a right vertebral artery dissection (VAD).

CASE 2 - A 47 year old lady presented with loss of consciousness lasting 30 seconds during a pre-match hockey warm up. Afterwards she suffered from confusion and amnesia which lasted 12 hours. Initial CT brain scan and lumbar puncture was normal, but subsequent MRI revealed multiple embolic infarcts in the occipital lobes. CT angiogram confirmed a left VAD.

References

Weight-lifting: more of a headache than it's worth?

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Case Report

Intracranial hypotension most commonly occurs following lumbar puncture, with spontaneous cases often misdiagnosed, leading to treatment delays.¹ The classic orthostatic headache may be accompanied by various neurological symptoms and signs.

We describe the case of a 46 year old fireman who presented with such a headache two weeks following minor cervical discomfort while weight lifting. MRI confirmed intracranial hypotension with slumping of the mid brain, bilateral extra-axial collections, foramen magnum crowding and smooth dural Gadolinium enhancement. He improved with bed rest and hydration, and was discharged with lifestyle advice. Four weeks later he re-presented with worsening headache, facial dyskinesia, restless limbs and unusually gregarious behaviour. Repeat MRI confirmed progression of intracranial hypotension, with bilateral subdural haematomas compressing his cerebral hemispheres, distortion of midbrain and pons, and bilateral uncal herniation (figures 1&2). Interestingly, he had none of the clinical features typically associated with herniation due to raised intracranial pressure. A thirty degree head down tilt caused marked symptomatic improvement over two days, provided he remained recumbent. MR myelogram could not identify the source of his CSF leak and he was treated with two non-targeted lumbar epidural blood patches, eight days apart. The second resulted in sustained symptomatic resolution, with repeat MRI showing an epidural haematoma but relatively little change intracranially.

Intracranial hypotension due to spontaneous CSF leak should be considered in patients presenting with orthostatic headache. Associated movement disorders are uncommon, and facial dyskinesia has not been previously reported. Diagnosis relies on clinical suspicion and cranial MRI, given CSF pressures and CT may be normal.² Symptoms can resolve with conservative management: bed rest, hydration, simple analgesia and caffeine.³ If unsuccessful, epidural blood patching is indicated, though with a success rate of 33-57% multiple patches are often needed.⁴,⁵ Failing this, epidural fibrin glue, continuous epidural infusion or surgical repair may be considered.³

References

You take my breath away - another cause of hyperlactaemia

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Background
A 22-year-old lady was admitted to the Emergency Department complaining predominantly of dyspnoea. An arterial blood gas demonstrated a high lactate. Initially she was treated for sepsis. We discuss another cause of hyperlactatemia and the importance of ruling out other causes.

Case Presentation
She presented with dyspnoea and pleuritic chest pain. An arterial blood gas demonstrated a hyperlactaemia (5mmol/L) and respiratory alkalosis. A CXR and CTPA were unremarkable. Her ECG showed sinus tachycardia. She was referred to the Acute Medical Unit (AMU) with "sepsis - ?source". On arrival to AMU she hyperventilated and had a panic attack. Her observations demonstrated a pulse of 125 bpm and a respiratory rate of 38. She described a similar episode at home which subsequently led to her admission. Her phosphate was 0.3mmol/L. Her inflammatory markers and other electrolytes were normal. The hypophosphataemia caused hyperventilation leading to a panic attack. She was given IV and oral phosphate replacement.

Discussion
Insufficient dietary intake or absorption; raised renal losses or redistribution of body phosphate stores can cause hypophosphataemia (<0.8mmol/L). Hyperventilation results in a fall in the partial pressure of CO2 in arterial blood, causing an alkalosis. As CO2 diffuses freely across cell membranes the alkalosis becomes intracellular and extracellular. Intracellular alkalosis causes a stimulation of phosphofructokinase, the rate limiting enzyme in glycolysis. (Figure 1) As a result phosphate is redistributed in the intracellular space thus reducing extracellular phosphate concentration causing hypophosphataemia. Glycolysis also produces lactate causing hyperlactatemia.

Clinical manifestations of hypophosphatemias include skeletal muscle weakness, rhabdomyolysis, haemolysis, altered mental state and panic.

Learning Points
- Life threatening pathology must always be excluded first
- High lactate does not always mean sepsis
- If a patient has a high lactate but the clinical picture does not fit, explore other causes
- It is important to check electrolytes and correct any abnormalities

References
Young and Confused (A case of acute cerebellitis and fulminant hepatitis in a young confused adult presenting to the Acute Medical Unit)

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Diego Maseda

Introduction

Confusion is an unusual, challenging presentation within younger cohorts. We present a rare case of acute cerebellitis and fulminant hepatitis presenting as confusion.

Case Presentation

28-year-old gentleman presented to A&E with a dull headache and lethargy for 2 days, he displayed unusual behaviour and was discharged, thought to be under the influence of illicit drugs. The following day he was to be found ‘not himself’. He became increasingly confused, photophobic and re-presented. He returned 2 months ago following 5 years of travel (South-east Asia) PMH: dengue fever (2011) He was confused (GCS 14/15) unable to give a history, pyrexial, with multiple mosquito bites and severely elevated transaminases, CK, LDH and deranged hepatic synthetic function. CT head showed bilateral cerebellar hypodensity within the white matter extending to the cortical grey matter laterally. MRI brain diagnosed acute cerebellitis. Extensive serological testing including HIV, CSF analysis (WBC: 1 per mm² 100% lymphocytes) and CT abdomen/pelvis were normal, except west nile virus IgG. 3 days later he developed a vesicular rash on his scalp which was positive for varicella zoster. He was treated as viral encephalitis and acute hepatitis. His confusion improved slowly over 12 days. He later admitted to using heroin from the internet with the use of a needle exchange. Diagnosis was varicella zoster and/or toxin induced acute cerebellitis and fulminant hepatitis. The hepatitis completely resolved over a 2 month period and a repeat MRI head showed almost complete resolution.

Discussion

Acute cerebellitis is an inflammatory condition of the cerebellum commonly seen in children, but rarely in adults. It’s usually viral or post-immunisation related (1) and defined as fever and signs of meningeal irritation with additional nausea, headache, altered mental status (including loss of consciousness/convulsions) acute onset of cerebellar symptoms and characteristic neuroimaging (2). Particularly MRI can diagnose acute cerebellitis but has no prognostic value. Diffuse hemispheric abnormalities represent common imaging presentations (3) It has been linked to viruses including varicella-zoster (4) and less so to external toxins (5).

References