Introduction

Rhabdomyolysis is a clinical condition characterised by damage to striated muscle membranes with resulting biochemical and clinical sequelae. Relatively uncommon and underdiagnosed, it is potentially severe and debilitating with life threatening complications.

Aims

1) To determine the aetiology of severe de-novo rhabdomyolysis necessitating acute hospitalisation
2) To highlight deficiencies in the regulation and governance of the fitness industry
3) Suggest recommendations to the Public Health Agency

Methods

Six cases of severe debilitating de-novo rhabdomyolysis were admitted to the acute medicine unit over a 4 week period. Each patient presented with crippling myalgia, dark urine and a significant CK elevation. Previously healthy but exercise-naïve, presentation was within 72 hours of structured exercise under ‘qualified’ supervision (spin class).

A comprehensive ‘exercise history’ was elicited to determine the nature and duration of the activity and establish the degree of supervision and advice given to clients.

Furthermore, biochemical interrogation excluded inborn errors of metabolism.

Outcome/Results

Poor physical conditioning and intensive prolonged exercise coupled with inadequate hydration precipitated rhabdomyolysis.

Previously fit and healthy with no preceding rhabdomyolysis, the patients were all clients at the same Health & Fitness facility, received similar training advice with an identical training schedule.

Conclusion

The author suggests more stringent regulation of the Health & Fitness Industry with nationally recognised professional qualifications to minimise potential harm caused by overzealous training regimes.

Outcome

The Public Health Agency were informed and liaised directly with the Health Club. Subsequently, there have been no similar acute medicine admissions.
References


Aim

To describe a case of colchicine associated hepatotoxicity.

Methods

A 60 year old caucasian male presented to acute medicine with subacute onset of painless jaundice. He had felt nauseous, lethargic and faint for a few days. He had completed a six day course of colchicine (6mg in total) for presumed acute mono-articular gout two weeks prior to presentation. He denied any fever, pruritis, change in stools, but did say his urine was darker. He had no other past medical history except hypertension for which he used lisinopril. He denied any other drug use. He drank excessively in the past, but denied any alcohol intake for the last five years. He had no significant surgical, occupational, sexual or travel history. Apart from jaundice, he had no signs of systemic upset. He had no organomegaly, ascites or signs of chronic liver disease. Blood tests were normal except for elevated bilirubin 138 umol/L, ALT 2108 IU/L, and ALP 227 IU/L. Liver tests a year ago had been normal. Serum paracetamol levels were undetectable. Abdominal ultrasonography was essentially normal except for suggestion of mild fatty change in liver.

Outcomes

Subsequent negative screening tests for viral, autoimmune and other aetiologies supported the diagnosis by exclusion. Liver function was improving gradually till last follow-up. His symptoms had completely resolved.

Conclusion

Colchicine has generally not been associated with acute liver injury or abnormal liver function tests except with significant overdosage. This case suggests that hepatotoxicity is possible even with therapeutic doses.
A previously fit 47-year-old man was admitted with severe muscle pain and spasms affecting his trunk and limbs, exacerbated by movement. Two days earlier he had surgery under general anaesthesia for hand trauma. He described the pain as having ‘played the first football match of the season.’ He flinched and called out in pain when spasms occurred. His blood tests revealed a raised creatinine kinase (CK) of 655 IU/L. The anaesthetic chart showed he received suxamethonium, a known cause of myalgia.

He was managed conservatively for 24-hours with IV fluids and diazepam for symptomatic relief. His renal function remained stable and after 24 hours his CK fell to 271IU/L. Muscle pain lasted for four days.

Background

Post-suxamethonium myalgia was first described in 1954 [1] and is well documented[2]. The incidence may be as high as 89%[3]. Usually it begins on the first post-operative day, lasts for several days and is self-limiting[4]. Predisposing factors include short operation times, lower baseline muscular fitness, middle-age and female gender[4]. In this case, emergency intubation with suxemethonium was required during the operation due to failed airway protection from a supraglottic device.

Post-suxamethonium myalgia is not usually associated with low plasma cholinesterase levels and in this patient the enzyme level was normal.

Theories as to why this complication occurs include fasciculation, muscle fibre rupture and potassium release[4]. Multiple therapies have been tried to reduce the incidence of post-suxamethonium myalgia. This patient had symptomatic improvement with diazepam, although robust evidence for benzodiazepines is lacking.

References


The management of the acute blue finger is controversial with many regarding this as a benign condition. [1, 2] However, we would argue that it could be an emergency that requires prompt management. We present a challenging case of a 43-year-old woman who presented acutely to a district general hospital medical admissions unit with 1 week history of sudden onset blue discolouration of left 5th digit, and a 6-week history of episodic joint problems. Examination showed bilateral normal radial and ulnar pulses. Following blood investigations showing positive rheumatoid factor, high C-reactive protein and ESR, an initial working diagnosis of early rheumatoid arthritis with associated Raynaud’s phenomenon was made. Also, a differential diagnosis of infective endocarditis was considered due to temporary misleading physical signs. Later, CT angiography of left upper limb arteries showed a significant proximal left subclavian stenosis. Subsequently, a diagnosis of left subclavian arteritis associated with digit ischaemia from embolic debris was made and the patient underwent left subclavian angioplasty. However, the delay in management resulted in a necrotic digit, which was left to auto-amputate. In conclusion, acute blue finger is a diagnostic challenge. A vascular surgical opinion should be sought early in the presentation to prevent potentially catastrophic complications.

References


Aim

To highlight the non-specific and varying clinical presentation of acute basilar artery thrombosis along with need for prompt vascular imaging to facilitate accurate diagnosis and urgent reperfusion therapy.

Methods

The case histories of two patients were reviewed. Patient 1: A 75 year old man presented with sudden onset dizziness, diplopia and occipital headache along with fluctuating GCS between 8 to 15. Initial CT brain was unremarkable. CT angiogram demonstrated subtotal basilar artery occlusion. Patient 2: An 83 year old man presented with collapse and GCS 3. CT angiogram demonstrated complete basilar artery occlusion and no distal blood flow.

Outcomes/ results

Both patients were transferred to a tertiary centre with access to interventional radiology. They underwent endovascular thrombectomy and received intra-arterial thrombolysis. Patient 1 had clot removed from the basilar artery which had been intermittently occluding causing brainstem hypoperfusion and fluctuating GCS. He made a full recovery with follow up CT brain demonstrating only minor occipital ischaemia. Patient 2 had a large segment of clot removed from the basilar artery and distal flow restored. Peri-procedurally he was managed in intensive care made a full neurological recovery.

Conclusion

A high index of suspicion is required to diagnose and investigate basilar artery thrombosis. Knowledge of brainstem physiology and varying clinical presentation is important. These patients should have urgent vascular imaging and reperfusion therapy as appropriate.
Title: An Unusual Cause of Ischaemic Stroke
Category: Case Reports
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Introduction:
During thrombolysis for pulmonary embolism (PE), stroke usually results from intra-cerebral haemorrhage. Ischaemic stroke due to paradoxical embolism is unexpected by clinicians.

Case report:
A 60 year old lady presented after collapsing. She had noted breathlessness for several weeks. Positive examination findings included reduced oxygen saturations, hypotension, tachycardia and left leg swelling.

Arterial blood gases demonstrated hypoxia. The ECG showed sinus tachycardia, right axis deviation and strain. D-dimer was raised.

Enoxaparin was commenced for presumed PE. Unfortunately, she deteriorated and became profoundly hypotensive. She was thrombolysed with intravenous alteplase. After ten minutes, dense right sided weakness and aphasia were observed. The alteplase was stopped.

Urgent CT brain revealed left middle cerebral artery (MCA) thrombus. She was transferred to a stroke centre. Thrombolysis was completed. Unfortunately, a malignant MCA syndrome developed. Hemicraniectomy was undertaken.

Left-leg duplex scanning revealed a proximal deep vein thrombosis. “Bubble echo” demonstrated a patent foramen ovale (PFO).

She is currently receiving neuro-rehabilitation.

Discussion
Massive PE carries a high mortality (1). Thrombolysis is potentially life-saving (2). It is not without risk; intracranial haemorrhage being the most feared complication (3).

Cases of paradoxical embolism after thrombolysis for major pulmonary embolism have been reported only rarely (4). However, patients with a PFO, in one series, were seen to have twice the death rate when compared to those without. This may be due to thrombolysis generating fragments of clot that paradoxically embolise (5).

Mechanical clot retrieval is an alternative option where a PFO has been previously detected by echocardiography (6).
References


(4) Bracey T, Langrish C et al. ‘Cerebral infarction following thrombolysis for massive pulmonary embolism’ Resuscitation. 2006: pp 135-137


BACKGROUND: African Tick Bite Fever (ATBF) is a newly described disease which can prove diagnostically challenging, even to experienced physicians.

CASE PRESENTATION: The first author recently travelled to Swaziland. Upon return, a small purpuric lesion in the left groin was noted. This subsequently developed into a small pustular lesion. A self-diagnosis of Staphlococcus infection was made and treatment with topical antibiotics and dressings commenced. A second furuncular lesion was noted several days later, upon which expert infectious disease opinion was sought. A provision diagnosis of Tumbu Fly infection was made and topical Vaseline applied. Further symptoms of high fevers, widespread lymphadenopathy, headache and rash developed 2 days later. The diagnosis was revised to African Tick Bite Fever. Doxycycline was prescribed, with resolution of symptoms. Typical eschars did not develop until topical treatments were ceased.

INVESTIGATIONS: Lymphopaenia, raised transaminases, elevated CRP and low albumin supported the diagnosis of ATBF.

DISCUSSION: Previous series of ATBF have described patients presenting at the point of becoming systemically unwell, at which eschars, if present, have fully developed, making the diagnosis relatively straight-forward. The use of topical treatments hindered typical eschar formation. The case underlines the importance of being willing to revise diagnoses when new symptoms emerge and the necessity of repeated review of unwell returning travellers.
Bilateral Ptosis in Thalamic Infarction – an unusual presentation of Acute Stroke

Introduction

Bilateral ptosis caused by unilateral thalamic lesion is an extremely rare condition. The exact anatomical cortical and sub-cortical basis of the lesion is still undefined.

Case History

74 year right handed caucasian male with history of Type 2 diabetes mellitus, hypertension and hypercholesteremia was admitted with sudden onset of dizziness, confusion and drowsiness along with bilateral drooping of eyelids. On examination he had fluctuating symptoms every 5-10 minutes intervals with varying degree of GCS (4-13), dysarthria, asymmetrical bilateral ptosis and opthalmoplegia. The NIHSS score varied between 4-28. Urgent CT brain (Figure 1) and CT Angiogram did not reveal any evidence of vertebral or basilar artery clot although there was some evidence of frail posterior circulation suggesting unstable atheroma. Subsequent MRI brain scan showed hyperintensity in medial aspect of right thalamic region on DWI, T2 and Flair sequences (Figure 2). Intramuscular injection of Neostigmine methylsulphate did not result in any improvement of symptoms. EMG, EEG, Carotid Duplex and Trans-thoracic Echocardiogram were normal. He was started on dual anti platelet therapy. The patient was discharged after 5 days on secondary stroke prevention therapy. The ptosis improved gradually over next 4 weeks with no other additional neurological deficit during this period.

Conclusion

Bilateral ptosis with transient change in the state of alertness can be the only symptoms following unilateral thalamic infarct. Although uncommon, this presentation needs to be recognised early as it is a treatable condition having both short and long term impact on the functional outcome of stroke patients.

Reference

Aim

Distinguishing intracardiac masses between thrombus, tumour or vegetation may be challenging, but has large implications for acute patient management. We identify diagnostic features and consider management dilemmas.

Methods/outcomes

Case 1: 68 year old man with hepatocellular carcinoma had two months of progressive dyspnoea leading to severe type 1 respiratory failure. CT pulmonary angiography reported small non-occlusive pulmonary embolism and a large right atrial filling defect considered to be thrombus. He was considered for surgical embolectomy and thrombolysis until a metastatic tumour was subsequently diagnosed on a cardiac magnetic resonance (CMR) study.

Case 2: 19 year old woman with acute lymphoblastic leukaemia and central line for chemotherapy had a sinus tachycardia. Echocardiography demonstrated a large mass in the right atrium. CMR showed no internal enhancement suggestive of thrombus and anticoagulation was commenced, however interval scanning after one week showed atypical gadolinium penetration and CT imaging was consistent with soft tissue tumour.

Conclusion

Systemic symptoms, signs of obstruction or embolisation, and haematological or biochemical markers, do not consistently differentiate between cardiac tumours, infection or thrombus, yet distinction between these is critical to ensure appropriate therapy.

Discussion points: (1) overlap of clinical features, (2) requirement for multiple imaging modalities to provide sufficient information to improve diagnostic confidence, (3) establishing blood flow patterns as peripheral or central in the lesion as a means to identify thrombus and tumour respectively, (4) treatment may need to include full anticoagulation and antibiotic cover until diagnosis is clarified, (5) serial imaging where diagnostic uncertainty remains.
AIM

We present a rare case of nausea, vomiting and dizziness caused by concurrent vertebral artery dissection and contralateral occlusion, causing bilateral cerebellar infarctions.

METHODS

n/a

OUTCOMES

Our 34 year old patient presented with nausea, vomiting and dizziness. He reported 7 days of a dull, intermittent occipital headache. His initial examination was unremarkable except for a fluctuating hypertension. CT head was normal. He was diagnosed with severe labyrinthitis, and admitted for ongoing symptom management. On reassessment 12 hours later, he developed horizontal nystagmus, and continued to have nausea and dizziness. Twenty-five hours post admission, he was found unresponsive, GCS E2, M6, V2. He displayed a fixed right upwards gaze with horizontal nystagmus, and constricted unreactive pupils. Repeat CT head demonstrated bilateral cerebellar infarcts and early hydrocephalus. He underwent an emergency decompressive occipital craniectomy and external ventricular drain insertion. Post-operative imaging showed a vertebral artery dissection with contralateral vertebral artery occlusion. The patient had a good response to Neurosurgery, and is now undergoing neuro-rehabilitation.

CONCLUSION

This case illustrates the potential sinister aetiology of common symptoms such as nausea, dizziness and vomiting. Early CT imaging was not helpful with this rare bilateral vertebral artery pathology. Regular reassessment of signs in young patients with vestibular symptoms, including blood pressure and cranial nerve examinations, is vital to differentiate between labyrinthitis and vertebral artery pathology. Early identification of potential vertebral artery pathology will enable Acute Physicians to request appropriate imaging and optimise treatment, saving lives and brains.
INTRODUCTION
Headache is a common presentation to Acute Medical Units (AMUs) accounting for 3% of all referrals (1). These patients are usually assessed in the ambulatory area and most of them can be managed safely as outpatient. However, we do encounter atypical cases where we need to think about the rare causes. We present two patients who presented to our AMU with headache.

CASE PRESENTATION
The first patient presented with a sudden severe headache. Computerised Tomography (CT) showed an enlarged pituitary. Subsequent investigations revealed a low cortisol and TSH. Further review of imaging by neuroradiologist suggested pituitary apoplexy. The second patient had a more prolonged history of headache and a sepsis-like syndrome which failed to resolve with antibiotics. Review of admission blood tests showed a suppressed TSH with low thyroxine and triiodothyronine. Further investigation showed steroid deficiency. Neuroimaging confirmed diagnosis of pituitary apoplexy.

DISCUSSION
Pituitary Apoplexy is an uncommon common condition. It is a clinical syndrome consisting of headache, vomiting, visual impairment and decreased conscious level. The infrequency with which it is encountered makes it a considerable diagnostic challenge.

Whilst providing an accurate diagnosis of headache is desired, ruling out a serious cause of primary severe headache is paramount. Subarachnoid haemorrhage, meningitis and space occupying lesion account for less than 10% of all headaches and an argument can be made for ambulatory assessment of primary severe headache(1). However, rarer causes should be considered before discharge or treatment is considered.

References
1. Acute Med. 2012;11
Background:

Hyperkalaemia is a life threatening medical emergency. However occasionally high potassium could be a spurious result due to delay in analysis and haemolysis. Pseudohyperkalaemia is a term given to hyperkalemia in the absence of any haemolysis. There is elevated serum potassium concentration with a concurrent normal plasma potassium level.

Aim:

Pseudohyperkalaemia may be identified early during a patient’s admission if considered as part of differential diagnosis. Unnecessary treatment may potentially lead to a reduction in serum potassium with a variety of consequential physiological effects. We report a case of incidental hyperkalaemia.

Case report:

66 year old lady referred by GP to AMU with routine blood tests showing potassium of 6.6, previously between 5.6 to 6.5 over past 6 months. Concurrent history of Polycythaemia rubra vera on Hydroxyurea and periodic venesection. On admission the patient was asymptomatic, with no clinical signs and a normal ECG. Renal functions and bicarbonate were normal. Repeat samples showed a persistent hyperkalaemia. Concurrent plasma and serum samples were sent, to detect pseudohyperkalaemia. The plasma sample reported a potassium of 5.3 mmol/L, whilst the heparinised sample showed levels of 4.1 mmol/L. This allowed a diagnosis of pseudohyperkalaemia, and the patient was discharged.

Conclusion:

Pseudohyperkalaemia has been recognised in a setting of high blood cell counts such as polycythaemia and thrombocytosis. In these situations, intracellular potassium from blood cells is released into plasma giving falsely high serum potassium. High index of suspicion is needed to identify these patients with an appropriate clinical background.

References:

A 20-year-old female with insulin dependent diabetes presented with a 6-day history of central chest pain and breathlessness. On arrival she was tachycardic, tachypnoeic, hypotensive and pyrexial. Examination revealed a clear chest with normal heart sounds. Electrocardiogram indicated a sinus tachycardia and chest x-ray was normal. Pulmonary embolus was initially suspected. An urgent Computed Tomography Pulmonary angiogram revealed no embolus, but a large pericardial effusion (fig 1). A repeat ECG showed ST segment elevation (fig 2) and an echocardiography confirmed evidence of early cardiac tamponade. An emergency pericardiocentesis drained pus, confirming a diagnosis of purulent pericarditis.

Following transfer to the cardiothoracic centre further imaging demonstrated a contained rupture of a mycotic aneurysm at the aortic root. The patient underwent an aortic root replacement; Staphlococcus Aureus was cultured from the explanted aortic root.

Mycotic aortic aneurysm of the ascending aorta without previous cardiac surgery is rare, being reported in only 2.9% of cases.\(^1\) Due to its rapid expansion and tendency to rupture it is life-threatening\(^2\), and if left untreated it is almost universally fatal. The most common pathogens isolated from mycotic aneurysms are Salmonella and Staphylococcus species.\(^3\) Surgical intervention is always required, however even after a successful repair and insertion of a graft, prognosis is poor, with an in-hospital mortality of 11.8%.\(^3\)

Pericarditis should be considered in young patients with chest pain and physicians should be mindful of atypical presentations. Although often self-limiting this case demonstrates the serious complications that can occur. Despite the life-threatening features this patient made a full recovery.

References:


AIM

5-aminosalicylates are a group of medications commonly used in Crohn's disease. Sulphasalazine is a combination of 5-ASA and sulphapyridine which acts as a carrier. In mesalazine (5-ASA), the specific sulphapyridine-related side effects, especially pulmonary reactions, are avoided. However, we present a case of lung fibrosis which was associated with mesalazine in a Crohn's patient.

METHODS

An 82 year old male whose Crohn's disease was diagnosed in 1964, was in remission on prednisolone after recent flare-ups. He was commenced on Pentasa (mesalazine) while awaiting thiopurine methyltransferase (TPMT) in view of starting azathioprine.

Nine days later he was admitted to HDU with dyspnoea and hypoxia, a CXR showed consolidation and pleural effusion and he was treated as for pneumonia.

The patient continued to deteriorate, a high resolution CT thorax showed ground glass changes consistent with lung fibrosis with no obvious causes from the history or investigations undertaken.

RESULTS

Medical literature was reviewed, a few cases of mesalazine lung injuries were found, therefore Pentasa was discontinued and the patient was placed on iv methylprednisolone followed by oral steroids.

As a result we witnessed a significant clinical improvement, the patient went off non-invasive ventilation, was discharged to the ward and C-reactive protein (CRP) had fallen dramatically.

Clinical findings were supported by a repeat HRCT thorax a few weeks after discharge.

CONCLUSION

Mesalazine should be considered seriously as a potential cause for lung disease in a Crohn’s patient.

Currently, mesalazine is considered clinically no more effective than placebo for active Crohn’s disease (AGA & ECCO 2009)
Case

A 40-year old man was referred with ‘cellulitis’ of the right foot that had not responded to co-amoxiclav, metronidazole or flucloxacillin. Three weeks earlier he was walking in the sea in Crete and developed a sudden, excruciating pain in his right foot. There were two puncture marks on the sole of the foot. A local doctor attended the patient at the beach and gave adrenaline. Initially the foot improved but despite antibiotics it started to swell again.

The sole of the foot had a small necrotic area. The right foot was swollen and red but not oedematous.

Having been a victim of such an injury on August Bank Holiday 1990, a co-author suspected that this was a weever fish injury.

Management

An orthopaedic opinion was sought and the patient was managed conservatively with bed rest and elevation of the right foot. Intravenous flucloxacillin was commenced and doxycycline was added to cover Vibrio vulnificus, a rare cause of cellulitis and found in warm, salt water. Subsequent care was ambulatory. The foot improved and was almost healed after a further ten days.

Discussion

Weever fish are present in UK waters in warm summers. They produce a heat labile toxin. The immediate management is to submerge the affected foot in hot water to destroy the toxin; the temperature being as hot as can be tolerated without skin scolding. In patients with marine injuries, ‘atypical’ causes of infection should be considered. A UK death from a weever fish injury was reported in 1927.

NOTE: Patient has given permission for use of images and presenting the case at SAM.
This is a clinical case report of a 59 year old woman whom presented to the AMU with a flaccid quadriparesis, anaemia, metabolic acidosis, and weight loss following a prolonged episode of depression. She had been taking Nurofen Plus (3-4 grams/daily) for a period of 6 months for lower back pain. She reported a loss of weight (2 stones approximately) in that time, increased feelings of lethargy and fatigue, and subsequent weakness until her presentation.

On examination the patient had a thin appearance, was pale, and demonstrated a 1-2/5 power in all four extremities. Her reflexes were globally diminished with no loss of sensation. Her examination was otherwise unremarkable.

Biochemistry tests demonstrated a loss of serum bicarbonate, sodium, potassium, and chloride. A normocytic anaemia was also present. Urine free electrolytes demonstrated increased potassium loss. CT staging and MRI/EEG were also unremarkable. Slow intravenous replacement of lost electrolytes resulted in improved muscle function and tone. Other potassium mediated channelopathy investigations were largely normal.

DIAGNOSIS:

1. Hypokalaemic Quadriparesis 2. Renal Tubular Acidosis (type 2) following Nurofen excess 3. B12 deficient anaemia

DISCUSSION:

Nurofen Plus (Ibuprofen + Codeine Phosphate) is a non-steroidal anti-inflammatory agent used in painful inflammatory conditions. The drug has been found to cause renal tubular acidosis in some patients, and rarely excess potassium loss. Case reports have previously highlighted muscle weakness with consistent misuse.

CONCLUSION:

1. Practitioners should be aware of the role of potassium channels in assessing patients with motor weakness
2. A thorough medical history is fundamental to contextualizing patient symptoms, including medication reconciliation
3. Renal Tubular Acidosis from NSAIDS is a common finding; and it can lead to electrolyte loss over an extended period of time
Aim

To increase awareness about a rare but fatal lung condition that may present to the acute medicine setting as recurrent pneumothorax.

Methods

The case of a 29 year old lady who presented to the acute medicine unit with bilateral spontaneous pneumothoracies will be described. She had no significant past medical history and was fit and well.

Outcomes and results

The patient had attended the ED 3 days earlier for aspiration of a right sided pneumothorax. She presented to the acute medicine unit with dyspnoea and chest pain. Chest radiographs revealed a resolving right pneumothorax and a new left sided pneumothorax in otherwise normal looking lungs.

On further history taking it was noted that the patient had been treated for a pneumothorax one year previously.

A computerised tomography scan was requested to look for underlying abnormality. This revealed multiple well-defined small cysts throughout both lungs, consistent with diagnosis of lymphangioleiomyomatosis.

This is a slowly progressive cystic lung disease affecting mainly women of child bearing age. Cyst rupture will often lead to pneumothorax, leading to respiratory failure over one to two decades.

Conclusion

The majority of spontaneous pneumothoracies occur in males and are benign; however it is important to consider lymphangioleiomyomatosis as a diagnosis in recurrent pneumothorax in young females.

Further investigation with computerised tomography and respiratory referral is essential for diagnosis. This is not reflected in the BTS guidelines for pneumothorax, which do not provide guidance for investigation of recurrent pneumothorax.


AIM

To report an unprecedented case of female monozygotic twins, both suffering from Dercum’s disease, presenting simultaneously with multiple pulmonary emboli.

METHODS

A 67-year-old woman presented with a four-month history of shortness of breath on exertion. The D-Dimer was elevated and Ventilation/Perfusion (VQ) scanning revealed multiple pulmonary emboli. Two weeks later, the patient’s monozygotic twin was admitted with a six-month history of worsening shortness of breath on exertion. The recent admission of her twin with symptoms which mirrored her own had prompted the patient to seek medical attention. VQ scanning detected multiple pulmonary emboli. Further investigations, including blood tests for thrombophilia and imaging to exclude mechanical venous compression, revealed no underlying cause in either twin.

OUTCOMES/RESULTS

Although several genetic determinants have been associated with increased risk of venous thromboembolism (VTE), the overall influence of genetic factors is unknown. A Danish study in 2003 demonstrated an increase in the relative risk of VTE in male monozygotic twins but as yet there is no published evidence that supports an intra-twin pair similarity for women (Larson et al 2003). Dercum’s disease (Adiposis Dolorosa) is a rare condition characterised by multiple, painful lipomas. The underlying pathology of the disease is poorly understood, but as yet there has been no recorded association with an increased risk of VTE.

CONCLUSION

This case is of clinical interest due to the improbability of a pair of twins presenting at the same time with the same condition, for which no intra-twin increased relative risk has ever been proven, and with no identified underlying cause.

REFERENCES

AIM

To highlight an unusual cause for bilateral leg swelling which can present in young healthy patients on the acute medical unit

METHODS

A previously fit and well 32 year old man with no family history of note presented with left flank pain and bilateral leg swelling. On examination he had oedema to the groins bilaterally (Figure 1) and visibly dilated abdominal wall veins. Bloods revealed a D-dimer of 4.6mg/L (normal range <0.5) and ultrasound doppler of lower limb veins revealed occlusive DVT in the common femoral, femoral and popliteal veins bilaterally. Subsequent CT scan of abdomen showed an abnormal mid inferior vena cava thought to represent a congenital aplasia of the IVC (Figure 2).

OUTCOMES

The patient was treated with low molecular weight heparin, warfarin and compression stockings. He was referred to the regional vascular unit who recommended continuing conservative management and seeking haematology advice regarding the duration of anticoagulation required. His leg swelling improved considerably with this management.

CONCLUSION

Congenital IVC malformation is a rare vascular defect found in approximately 5% of unprovoked DVTS in patients under 30 years old\(^1\). It is more common in men and DVT can be provoked by intense physical activity. DVT can be unilateral but is more commonly bilateral. It can be diagnosed with CT scanning and is managed with anticoagulation and compression stockings. The necessary duration of anticoagulation has yet to be established as patients with this anomaly can be at increased risk of recurrent DVT\(^2\).
AIM:

To highlight the significance of sarcoidosis-lymphoma syndrome.

METHODS:

Published studies and case reports on sarcoidosis-lymphoma syndrome were studied. These were compared with findings in our patient.

OUTCOME:

A 44-year-old Caucasian lady presented to the GP in October 2010 with a two-month history of right-sided neck swelling. CT scan showed no other lymphadenopathy. Biopsy was consistent with granulomatous lymphadenopathy. Serum ACE level was elevated and sarcoidosis was diagnosed.

Two years later, the patient was admitted to the Acute Medical Unit with complaints of left sub-mammary discomfort, painful lymphadenopathy, lethargy, headaches, joint pain, neck pain, sweats and rigors. Examination revealed bilateral cervical and axillary lymphadenopathy and hepatosplenomegaly.

Laboratory findings revealed thrombocytopenia of 6 and elevated CRP of 190 mg/l. CT scan of the thorax, abdomen and pelvis showed progressive splenomegaly and generalized lymphadenopathy. Bone marrow aspirate and biopsy was non-diagnostic. Lymph node biopsy confirmed classical Hodgkin’s disease with mixed cellularity; CD 30 positive/ CD 15 positive/ EBV positive. Diagnosis of Stage IV Hodgkin’s disease was confirmed and chemotherapy was initiated.

CONCLUSION:

When faced with possible sarcoidosis-lymphoma syndrome, consider three characteristic features proposed by Brinker:

1. The lymphoid malignancy occurs after a preceding history of sarcoid (median interval of 24 months).[i]
2. It tends to occur in older patients (median age 41).[i]
3. Hodgkin’s disease is more commonly associated than other types of lymphoma.[i]

Our patient fulfilled these criteria. Understanding this association will lead to early screening for lymphoproliferative disease and subsequently better patient outcome.
Introduction

Loss of Consciousness is an uncommon presentation of stroke and it is more often seen in haemorrhagic strokes\(^1\). With the emergence of new acute therapies for stroke, in particular thrombolysis it has become essential to be able to recognize ischaemic strokes early and evaluate patients for possible treatment\(^2\). We present a case where recognising a Stroke in an unconscious patient can be challenging and understanding variants in cerebrovascular anatomy can be invaluable.

Case

80-year-old gentlemen admitted unconscious to the emergency department, GCS - 3. He had fixed dilated pupils but his CT Head showed no significant abnormality. CT Intracranial angiogram showed no basilar thrombus. The patient was intubated and transferred to ITU. The following morning he was successfully extubated, GCS - 15. Neurological examination demonstrated difficulty with focus and conjugate eye movements. MRI showed acute bithalamic infarcts; a diagnosis of the artery of percheron occlusion was made.

Discussion

The artery of Percheron was first described in 1973 by the French medical scientist Gerard Percheron\(^3,4\). It is an anatomic variation in the brain vascularisation, a single arterial trunk arises from the posterior cerebral artery (PCA) to supply both sides of the thalamus and midbrain\(^4\). It may be present in up to a third of people\(^4\). The most widely reported clinical signs of bithalamic infarcts are vertical gaze palsy, memory impairment, confusion and coma\(^5\). Even though it is uncommon, it is necessary for physicians to be aware of this anatomic vascular variant and its clinical consequences\(^4\).
Title: Use of Clonidine in Acute Medicine to reduce sedation induced Morbidity & Mortality.

Category: Case Reports

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AIM

Clonidine is an ideal agent in the AMU for example in patients on CPAP or BIPAP to provide sedation and analgesia. It does not obtund protective airway reflexes or cause respiratory depression unlike the commonly used benzodiazepines and opiates. It is commonly used in Anaesthesia & Intensive Care to provide anxiolysis and analgesia. It provides potent analgesia. It is not commonly used in Acute or Emergency Medicine and there are limited publications in this setting. We have often used it in Anaesthesia & Intensive Care and describe its use in Acute and Emergency Medicine.

METHODS:

Two case reports.

OUTCOMES:

Patient A was a 50 year old man who was intoxicated and had dislocated his shoulder. He was morbidly obese (BMI 40) was not fasting and had a difficult airway. He was given conscious sedation morphine 10mg, midazolam 10mg, and fentanyl 100mcg to facilitate shoulder reduction but his painscore remained very high and shoulder reduction wasn't possible. He was then given clonidine 150mcg total. This maintained his airway reflexes, avoided CNS depression and provided excellent analgesia facilitating easy shoulder reduction. This avoided the risks of a General Anaesthetic in this high risk patient. Patient B was an obese patient with COPD Gold stage 4 who presented with an infective exacerbation of her COPD. She was obese with OSA. She was unable to tolerate BIPAP. She did not wish to be admitted to ICU but wished to be comfortable. Titration of clonidine 100mcg provided her with adequate analgesia and anxiolysis necessary for her to tolerate BIPAP and avoided the risks of opiates and benzodiazepines in a patient who did not want tracheal intubation and who was high risk. She was discharged home a few weeks later.

CONCLUSION:

Clonidine may be used to improve patient safety and satisfaction in Acute Medicine. I propose to carry out a RCT comparing it with benzodiazepines and opiates used for sedation and analgesia. I anticipate that use of clonidine will greatly improve the risk:benefit ratio and that it will become a very popular analgesic in Acute Medicine.