Aim:

To improve the management of all patients with infective endocarditis referring to a case report that provides some important, global learning points.

Methods:

Infective endocarditis is missed because it is a rare disease with non-specific symptoms [1]. Prompt diagnosis and treatment is vital due to high mortality rates [2,3,4].

This case describes a 44 year-old lady who presented twice to the Acute Medical Unit (AMU). On the first admission she was treated as an exacerbation of asthma and discharged. One month later she presented with similar symptoms but developed acute pulmonary oedema requiring invasive ventilation following a blood transfusion for anaemia. Further investigation revealed infective endocarditis that required surgical treatment. Review of the case highlights areas of her management including examination findings, microbiological and haematological abnormalities that could have led to earlier diagnosis.

Outcomes/results:

New protocols have been implemented to improve diagnosis. Microbiologists can now refer directly to cardiologists when a ‘typical organism’ for endocarditis is isolated on blood culture, leading to earlier review and specialist investigation. The case report has raised awareness of the diagnosis on the AMU and highlights the need for Duke’s criteria to be used in the diagnostic process.

Conclusion:

This is an interesting case with a relatively rare diagnosis but one that will occur in all acute medical units on occasion. It highlights important areas for improvement of patient management that may lead to reduced morbidity and mortality. The lessons learned from this case can be applied to all acute medical units.
References:


Aims
To highlight the following:

1. Pulmonary embolism (PE) is a leading cause of unexpected death in hospitalised patients.\textsuperscript{1}
2. Superficial thrombophlebitis (STP) is not just a benign self limiting condition.\textsuperscript{2,3}
3. Although rare, STP is associated with PE and should be considered as a risk factor.\textsuperscript{4,2}

Methods
A 71yr old was admitted with a one week history of dyspnoea and reduced exercise tolerance. Examination was unremarkable other than saturations of 96% on 24% oxygen. No risk factors for deep vein thrombosis were elicited in the initial history. Past medical history included severe angina and a coronary stent. This prompted an initial diagnosis of congestive heart failure. The patient rapidly deteriorated overnight and became peri-arrest on the ward, requiring thrombolysis for suspected PE. This was later confirmed on imaging. The patient later revealed she had been treated by her general practitioner six weeks previously for STP of the thigh.

Outcome
The potential risk of STP and associated PE are discussed:
- In contrast to deep vein thrombosis, STP is usually regarded as self limiting and benign.\textsuperscript{4}
- Verlato et al found unexpectedly high rates of PE in those with STP.\textsuperscript{2,3}
- Sobreira et al also discovered that rates of PE could be as high as 28.3% in patients with STP.\textsuperscript{5}

Conclusion
The association between PE and STP is controversial.\textsuperscript{4} There are several reports which suggest that STP may not always run a benign course.\textsuperscript{2,3,5} Although rare, we should be alert to the hazard of associated PE.\textsuperscript{2,3}

References:
Surgery 1999; 30(6):1113-1115


Aim

To increase awareness about the possibility of opiate retrieval from over the counter medications.

Methods

We describe the case of a young girl who experimented to obtain Codeine from an over the counter medication consisting of a combination of Aspirin and Codeine in order to get ‘high’.

Outcomes and Results

An 18 yr old student was brought to A&E by her friend after an episode of loss of consciousness and confusion following recovery. This was preceded by tinnitus and dizziness. History was suggestive of staggered overdose with Aspirin 400mg + Codeine 8mg tablets. Clinical assessment revealed tachycardia and a GCS of 14 with no other signs of opiate toxicity. Routine blood tests and blood gases were normal except for mild neutrophilia. ECG showed sinus tachycardia. Toxicology screen showed high salicylate level of 317mg/L. Thereafter, she was managed on the AMU as per TOXBASE recommendations.

Following recovery, she admitted to abuse cannabis and marijuana. She informed that she learnt about the process of obtaining codeine from combination tablets, through an internet article which recommended crushing the tablets to powder, suspending it in water, allowing it to stand for sometime and then filtering it to obtain codeine in the filtrate. She initially consumed filtrate obtained by this process with 32 tablets of above strength and repeated it twice over next three days resulting in above admission.

Conclusion

This case highlights the dangers of such readily available preparations containing low dose opiates to a susceptible population and warrants further review of drug regulations.
Aim

This case study illustrates the potential dangers of the antiplatelet and anticoagulant drugs used in the acute coronary syndrome (ACS) protocol. This topic is of great relevance to acute physicians given the frequency of chest pain as a presentation.

Methods

A 54-year-old man with a background of epilepsy, alcohol dependency, type 2 diabetes and hypertension presented to the Emergency Department via ambulance with chest pain. He had also suffered a tonic-clonic seizure en route which terminated spontaneously. On arrival, a fourteen-hour history of non-radiating central chest pain was elicited. Serial electrocardiograms revealed sinus rhythm with fixed T wave inversion in the lateral leads. He was loaded with aspirin, clopidogrel and dalteparin and referred to the medical team.

Outcomes/Results

Shortly afterwards, the patient began to complain of difficulty swallowing. On examination a large haematoma with accompanying oedema had originated at the site of a lateral tongue bite sustained during the earlier seizure. Intravenous dexamethasone and tranexamic acid were administered and an urgent anaesthetic opinion sought. The patient did not require advanced airway support although he was unable to eat, drink or take oral medication for 24 hours. Blood results subsequently revealed a negative troponin, a macrocytic anaemia and thrombocytopenia.

Conclusion

The ACS protocol should be applied with caution, particularly in patients with bleeding risk factors such as alcohol misuse. Occasionally it may be prudent to await the troponin result, particularly if the patient presents when a 12-hour level may be taken immediately. This may be further facilitated by point-of-care testing.
Aim

Subclavian vein thrombosis (SCDVT), accounting for 1-2% of DVT, is a rare but potentially morbid disease with progression to venous gangrene and pulmonary embolism. Two main causes are Paget-von Schrotter and Subclavian vein catheterisation. We report a patient presenting with spontaneous SCDVT.

Method

51 year old female presented with 2 weeks history of pleuritic chest pain and cough despite prior treatment as viral illness and developed left sided neck swelling and dysphagia. Past history revealed epilepsy and hypertension. Clinically, 4 cm ill defined mass was felt in left supraclavicular region. Ddimer was 2.33 and differential diagnoses of pharyngeal pouch, abscess and axillary vein thrombosis were considered and promptly anti coagulated. CT Thorax revealed left SCDVT extending to the confluence of left jugular vein with possible thymic mass compressing brachiocephalic vein. Biopsy revealed a mature thymus gland without neoplasia and whole body CT ruled out associated neoplasms.

Outcome

Patient was thrombolysed with Tenecteplase using a catheter into the thrombus and discharged with heparin and 3 monthly follow up CT Thorax.

Conclusion

Acute clinicians need awareness of SCDVT in patients presenting with acute unilateral neck swelling. If idiopathic, there is a need for long term follow up. There is ongoing research into possible link between SCDVT and incident lung cancer. Early recognition of SCDVT is important as fatality rates for untreated related pulmonary embolism is 10%. There is some evidence for localised thrombolysis in these patients.

Reference:

Recurrent hyperammonemic encephalopathy post ureterosigmoidostomy

Idrisu Sanusi, Steven Twigg, María-Belén Espina

A 52 year old man was admitted to hospital after being found unresponsive at home. On arrival, Glasgow coma score was six with upper motor neurone signs. External examination was unremarkable and a midline scar was noted on his abdomen. Arterial blood gas showed a hyperchloraemic normal anion gap compensated metabolic acidosis (Table 1). Inflammatory markers, clotting, liver function tests, renal function and electrolytes were normal. Brain computed tomography was also normal. He was admitted to intensive care unit as he remained deeply unconscious with a persistent metabolic acidosis. Serum ammonia was raised, 239 µmol/L (16-60 µmol/L). A review of his medical notes revealed he had a history of congenital hypospadias, urethral strictures and ureterosigmoidostomy at childhood and confirmed two previous admissions with hyperammonemic encephalopathy. Metabolic acidosis was corrected with 1.26% intravenous sodium bicarbonate. He received lactulose and empirical antibiotics for presumed urinary tract infection. Complete clinical recovery occurred after two days, bicarbonate level normalised and serum ammonia was dramatically decreased (Table 1). Hyperammonemic encephalopathy is a recognised but rare complication of ureterosigmoidostomy due to increased absorption of ammonia by intestinal tissue whilst acidosis is due to increase sodium bicarbonate secretion 1,2. Liver disease is the most common cause of hyperammonemic encephalopathy, however, non-hepatic causes should be suspected in the absence of overt liver disease. Prognosis is good with supportive treatment and alternative urinary diversion surgery needs to be considered. Determination of serum ammonia may be useful in aiding the diagnosis of unconscious patients without a clear diagnosis.

References


Aim

To increase awareness about a rare but potentially lethal complications of opiate misuse to a wider acute medicine community.

Methods

We describe the case of a 45 year old male who was abusing heroin and cocaine. He had a background of alcohol abuse and bipolar affective disorder. He was taking clozapine, quetiapine, thiamine and vitamin B tablets.

Outcomes and Results

Patient attend A&E due to agitation and was referred to acute medical team for control of opioid withdrawal. He was hypertensive and tachycardic. Temperature was 36.8. His only symptom was of severe back spasms.

Clinically his entire musculature was rigid and the possibility of tetanus considered in view of IVDA.

Initial bloods were normal. ECG showed a sinus tachycardia (QTc 445ms).

Patient admitted to smoking £40.00 of crack cocaine and injecting a similar value of heroin into his left ante-cubital fossa. The acute medical team suspected the possibility of heroin or contaminate injection leading to progressive rhabdomyolysis and muscle rigidity. Bloods performed on the AMU confirmed CK was 24,000 U/L. Aggressive fluid resuscitation was commenced. He was catheterised and was passed good volumes of urine. We monitored for signs of acute compartment syndrome.

National Drugs Poisons Team were consulted and alkalinisation of urine performed. Anti-psychotics were withheld. He received regular diazepam for his agitation and muscle cramps. CK peaked to 114,210 U/L day 2. By day 4 it fell to 33,867 U/L. He continued to pass good volumes of urine and renal function remained stable.

He was discharged at day 6.

Conclusion
Muscle rigidity is an unusual clinical finding but despite obvious clinical appearance initial medical team where blinded to this. This case highlights the importance of clinical examination in ascertaining a diagnosis and awareness of rarer complications of drugs of misuse.
AIM

We describe an important case of a rectus sheath haematoma (RSH) developing subsequent to anti-platelet and low molecular weight heparin treatment.

Clinical features/Methods

A 79 year old female with chronic atrial fibrillation, on warfarin, and ischaemic heart disease developed unstable angina following admission to hospital with digoxin overdose. Whilst awaiting an inpatient coronary angiogram she developed stabbing lower abdominal pain and distension.

Outcome/Results

On examination, the main clinical findings were of a firm tender lower abdominal mass in the right iliac fossa and pelvis (image 1). Investigations revealed a haemoglobin level of 7.4g/dl, INR of 1.9 with platelets of 226000 /µL. Biochemical tests were normal. An abdominal ultrasound scan revealed a large RSH and this was consequently confirmed with CT (computed tomography) scanning (image 2).

Our patient was managed conservatively with bed rest, volume resuscitation with intravenous fluids and blood products and reversal of anticoagulation with vitamin K and fresh frozen plasma. She made an uncomplicated recovery and was discharged three weeks later.

CONCLUSION

RSH may present as an acute abdomen and is a great mimicker of other intra-abdominal pathology. CT scanning is a reliable imaging modality and should not be delayed. Our case emphasises that the diagnosis must be considered in any patient who develops abdominal pain and is on anti-platelet and anticoagulant therapy. This is especially relevant given the high frequency use of these therapeutics in modern medicine.

REFERENCES:

Case Report

Aim: The case highlights a rare but life threatening effect of severe nutritional deficiency, in this case due to anorexia.

Case:

A 49 yr old patient with a 20 year history of severe anorexia, weighing 34kg, was admitted to the emergency department with low GCS, hypoglycaemia and hypothermia.

The patient was intubated in the Emergency Department and admitted to the Intensive care unit. She remained on the unit for a week, she was warmed, glucose corrected, vitamins replaced and started on a slow feed. The patient never received any sedative drugs and never regained consciousness. A CT scan was carried out which revealed appearances suggestive of demylination of the corpus callosum. The patient was reviewed by neurology who also agreed that this was the likely cause of her persistent low GCS and most likely due to her severe malnutrition. This rare condition is known as Marchiafava-Bignami disease.

Marchiafava-Bignami disease is a rare neurological disorder seen on imaging as demylination of the corpus callosum. Most cases are seen in chronic alcoholics but a few cases have been reported in anorexics. The condition can present in milder forms but the more severe form has a very poor outcome.

This case highlights a rare cause of low GCS in nutritional deficiency. It is important we are aware of the condition and always remember to correct vitamin deficiencies and assess nutritional states in patients regardless of the cause.
Aim: To describe an unusual case of Acute Kidney Injury.

Case Report: A 31-year-old HIV-positive man was admitted for investigations after deterioration in his renal function. He had recently been treated with antibiotics for a respiratory tract infection and high creatinine was noted; in the following weeks a further rise in creatinine occurred, from 101 to 391 umol/L (59-104). He was clinically nephrotic with worsening limb and abdominal oedema, high urine PCR (642) and low albumin (25g/L [40-52]).

He had travelled in Spain a few months before and had suffered from episodes of vomiting prior to admission. He denied any fevers or night sweats and stated that he had put on weight secondary to his increasing oedema. He required a number of blood transfusions and suffered from a few episodes of epistaxis. CT scan of his abdomen demonstrated hepatosplenomegaly (spleen 20cm) and multiple enlarged lymph nodes extending into inguinal regions, considered suspicious of lymphoma.

Renal biopsy was regarded as too high-risk. Histology from inguinal lymph node biopsy showed multiple macrophages containing amastigotes consistent with leishmaniasis. Specialist advice was obtained from the Hospital of Tropical Diseases and the patient commenced treatment with Amphotericin B.

Outcome: Follow-up in outpatient clinic 4 weeks after discharge showed a resolution of oedema and improving renal function.

Conclusion: Renal impairment in leishmaniasis is well described, often due to immune-complex-mediated interstitial and glomerular nephritis. Nephrotic syndrome is a documented consequence of leishmaniasis (especially with HIV co-infection). This interesting patient highlights the importance of considering wide differential diagnoses for familiar symptomatology in the setting of immunocompromise.
AIM

Toxic alcohol poisoning is a rare medical problem with potentially serious short-term and long-term clinical consequences. A low index of suspicion is required for diagnosis & appropriate investigations need to be arranged urgently as delay in diagnosis and treatment could result in severe organ system dysfunction. This case report highlights a rare presentation of toxic alcohol poisoning.

METHOD

Retrospective analysis of an acute admission on medical take

CLINICAL CASE

A 60 year old gentleman presented to accident and emergency with dysphasia and loss of co-ordination after a fall. Significant past medical history included COPD, ischemic heart disease, previous CVA, alcohol excess, depression & deliberate self-harm. After excluding an acute intra-cranial bleed on CT head, patient was referred to the medical team as '? CVA'. Patient suddenly dropped GCS level & ABG was done which showed severe metabolic acidosis (pH 7.05, HCO3 2.8, pCO2 1.8, BE -25, Lactate 0.8) and hyperkalemia (K 6.6). Urgent venous bloods confirmed hyperkalemia and also showed acute kidney injury & normal serum calcium. He did not have any clinical or biochemical evidence of severe sepsis. BM was 6.4 with normal serum & urinary ketones. Further investigation showed a high anion gap (30), high serum osmolality (336), high measured alcohol levels, high osmolar gap (32), normal Paracetamol & Salicylate levels and a drop in serum calcium. He was transferred to ITU because of refractory hyperkalemia & metabolic acidosis despite adequate measures. After consultation with clinical biochemist & toxicologist, he was commenced on IV Ethanol for a presumed diagnosis of 'Toxic alcohol poisoning' and serum sample for Ethylene Glycol & Methanol levels were sent.

OUTCOME

Ethylene Glycol level was significantly elevated confirming Ethylene Glycol poisoning. He was commenced on IV Fomepizole (antidote for Ethylene Glycol poisoning). He required haemofiltration for hyperkalemia & acute kidney injury. He made a slow yet complete recovery & was successfully extubated and discharged from ITU.

CONCLUSION

This case highlights a rare presentation of a rare medical problem. This case also emphasise on the importance of thinking colaterally in face of changing clinical picture with common medical presentations.
like dysphasia & confusion.