Case Reports

Capecitabine may cause oromandibular dystonia

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Case

A 56-year-old Caucasian woman diagnosed with T3N2M0 rectal cancer, underwent neoadjuvant chemotherapy with capecitabine (1,500 mg BID), and presented at the emergency department after 10 days of this treatment.

The last 6 days before presentation, she developed cramps between her shoulders, a tingling feeling in both arms, and numbness of the tongue. She also had difficulty swallowing and a disability to speak. Symptoms were intermittent, but increasing and at the day of presentation leading to progressive airway obstruction. She did not have any typical features of capecitabine toxicity like hand-foot syndrome or mucositis, nor were there any other focal neurological signs or associated metabolic abnormalities. Laboratory findings were normal and no co-medication was used, in particular no dopamine antagonists.

A diagnosis of oromandibular dystonia due to capecitabine use was made, and the drug was stopped immediately. The anticholinergic drug biperiden (Akineton) 10 mg was given intravenously, after which speaking and tongue movements improved within twenty minutes.

Unfortunately, despite prescription of biperiden, it was inadvertently not given, and twelve hours after presentation the symptoms of oromandibular dystonia with difficulty of speaking and tongue numbness recurred in the same intensity as before. Again, she was successfully treated with biperiden intravenously.

After the patient was able to swallow again, she was treated with 1 mg of oral trihexyphenidyl (Artane) once daily during three days and symptoms did not reappear.

Pharmacogenetic counseling showed a normal 5-FU drug metabolism.

Discussion

We report an extremely rare, but clinically highly relevant case of capecitabine induced oromandibular dystonia leading to airway obstruction without other neurological signs.

We concluded that this was due to capecitabine use, because after discontinuing biperiden symptoms reappeared and after cessation of capecitabine she recovered completely and complaints never recurred.

Moreover, the assessment by the Naranjo causality scale, a method for estimating the probability of adverse drug reactions(1) showed that the adverse drug reaction was definitely related to the drug (see table 1).

This is the first reported case of capecitabine induced oromandibular dystonia in Caucasians. In an earlier described case a Chinese male developed oromandibular dystonia nine days after consuming capecitabine which resolved spontaneously after three days.(2) To the best of our knowledge oromandibular dystonia is never reported after the administration of other forms of 5-FU.

The mechanism by which oromandibular dystonia occurs upon capecitabine intake is unclear. One plausible explanation is that capecitabine may pass through the blood brain barrier which may lead to a disruption within the basal ganglia, the center for movement control. This is seen in other types of dystonia and other causes of oromandibular dystonia.(2) The quick improvement after anticholinergic drugs, the first choice of treatment in these other types of dystonia,(3) underlines a similar pathogenesis.
Conclusion

Capecitabine may cause oromandibular dystonia, which may be treated with anticholinergic drugs. More research is needed to clarify the pathogenesis.

Reference list


Pulmonary embolism (PE) has an incidence of 60-70 per 100,000 people per year. It is potentially life-threatening with mortality 7% at one week\(^1\).

A 46 year old woman with a history of asthma, obesity and antiphospholipid syndrome presented with acute dyspnoea. She was tachycardic, tachypnoeic and hypotensive with a clear chest on auscultation and no evidence of deep venous thrombosis (DVT). Chest radiography was unremarkable. CT pulmonary angiography (CTPA) was negative for PE. The patient was treated for a presumed lower respiratory tract infection and discharged.

The patient re-attended one month later with a similar history and clinical findings. Chest radiography was unremarkable. CTPA demonstrated saddle embolus with right heart strain. The patient underwent successful intravenous thrombolysis.

This patient had risk factors for venous thrombo-embolism and clinical findings suggestive of PE at index presentation. National Institute for Health and Care Excellence (NICE) guidelines recommend performing a two-level Wells score for suspected PE\(^2\). This patient’s two-level Wells score was 4.5, indicating ‘likely PE’. With a negative CTPA and absence of clinical evidence for DVT, NICE guidelines suggest considering alternative diagnoses.

The PIOPED II study found that combined CT-angiography and CT-venography had better sensitivity than CT-angiography alone in investigating for PE\(^3\). In contrast to NICE, PIOPED II concluded that high-risk patients warrant further investigation when clinical probability is inconsistent with imaging results. Usually this would initially involve bilateral lower limb doppler-ultrasound. This case highlights the need for clinical vigilance in investigating high-risk patients presenting with findings consistent with PE.


2. National Institute for Health and Care Excellence (NICE) CG 144 Venous thoromboembolic diseases: the management of venous thromboembolic diseases and the role of thrombophilia testing. NICE: June 2012

Case Reports

Parsonage Turner Syndrome with Bilateral Phrenic Nerve Involvement

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Aim:

To present a case of a rare cause of dyspnoea which highlights the need for detailed history and clinical examination.

Methodology:

A 40 year old man presented with a 2 week history of progressive dyspnoea on exertion and orthopnoea. On examination he was breathless on speaking with accessory muscle use. Saturations were 98\% on air, RR 18. Neurological examination showed marked atrophy of the left deltoid, triceps and supraspinatus with weakness of shoulder abduction. Further history revealed acute onset of left shoulder pain and rapid progressive left shoulder weakness 3 months prior. CXR and CT scan demonstrated bi-basal atelectasis and lung volume reduction. Spirometry demonstrated a drop in vital capacity (VC) from 2.3L (standing) to 0.45L (lying).

Outcomes and results:

EMG revealed denervation in C5/C6 innervated muscles as well as severe bilateral phrenic nerve lesions, therefore confirming a diagnosis of brachial neuritis with bilateral phrenic nerve involvement. He was treated with IV methylprednisolone followed with steroid taper, and CPAP during sleep. His symptoms gradually improved; at 4 months, his VC was 3.09L (sitting) and 1.03L (lying), with full range of left shoulder movement. He continued requiring CPAP at night, but returned to his full time job.

Conclusion:

Parsonage Turner syndrome or brachial neuritis is a rare cause of acute shoulder pain with proximal muscle weakness with atrophy, often following minor trauma. Involvement of bilateral phrenic nerve lesions is rarer still \textsuperscript{1}. This case is a good reminder that full clinical examination of patients with dyspnoea and careful history taking is always key to diagnosis.

Reference:

Case Reports

A Rare Case of Syncope: Left Atrial Compression in Oesophageal Cancer

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Barts Health NHS Trust

Introduction

Syncope is a symptom of numerous medical conditions and a common cause of acute presentation to hospital. In this case a woman with metastatic oesophageal carcinoma was found to have an unusual cause of syncope.

Case Description

A 69 year old woman with metastatic squamous cell carcinoma of the oesophagus presented with two episodes of syncope. She had been feeling generally unwell since commencing palliative radiotherapy two weeks previously. Her observations were: HR 110, BP 82/53; RR 20; SaO2 (air) 95%; Temp 37.9°C. Examination was unremarkable with the exception of facial plethora and bibasal crepitations on auscultation of the lungs. There were no other signs of superior vena caval obstruction.

Her laboratory tests were unremarkable and her chest X-ray unchanged. A presumptive diagnosis of occult sepsis was made and antibiotics commenced. Subsequent review of a CT pulmonary angiogram performed one month prior noted local progression of her tumour, with impingement of the left atrium. Bedside transthoracic echocardiogram demonstrated an echo-bright structure compressing the left atrium, reducing its area by half. Medical management consisted of careful preloading with intravenous fluids and review of medications. She symptomatically improved and returned home for end of life care.

Discussion

Literature review revealed only one other report of atrial compression in oesophageal cancer. This case presents unique learning opportunities in the recognition of a rare cause of syncope, the clinical manifestations of cardiac compression and the challenges of acute fluid balance management in the context of severe atrial compromise.
Case Reports

A case of Marantic Endocarditis

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Background

Non-bacterial thrombotic endocarditis (NBTE), formally known as marantic endocarditis, is a rare condition, most often diagnosed post-mortem. It's associated with malignancy in the majority of cases, with inflammatory conditions and also in isolation.

Clinical Presentation

A 78 year old female presented with brief transient loss of consciousness, associated with subtle dyspnoea on exertion. There was no chest pain, haemoptysis, calf swelling or stigmata of left ventricular failure.

Respiratory examination revealed a clear chest with saturations of 94%. She had normal heart sounds with a normal JVP. ECG revealed sinus tachycardia and the chest x-ray was unremarkable. Serum Troponin was elevated at 0.74 (Normal<0.04). Urgent transthoracic echocardiogram revealed echogenic masses at the tricuspid valve, with impaired RV contractility and severe pulmonary hypertension. CT Pulmonary Angiogram showed extensive bilateral pulmonary emboli with a saddle thrombus. She had initially been anticoagulated with LMWH and also given antibiotics but after the CTPA she was as NBTE, antibiotics discontinued, and commenced on lifelong anticoagulation. Pre-discharge ECHO showed full resolution.

Discussion & Conclusion

NBTE commonly affects the left side of the heart, the mitral and aortic valves, making the above case rare. The diagnosis is usually made following systemic embolisation, and the mainstay of investigation is trans-thoracic echocardiography, with laboratory investigations utilised to distinguish between infective endocarditis. The mainstay of treatment is lifelong anticoagulation, with limited data suggesting LMWH to be more effective in reducing re-embolisation. This case highlights the importance of considering NBTE as an important differential diagnosis where endocarditis may have been diagnosed.


Case Reports

Sauna use with concomitant febrile illness and undiagnosed lipid myopathy leading to an acute presentation of rhabdomyolysis

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AIM

Exercise-induced rhabdomyolysis is well described. We present a case of rhabdomyolysis induced by a combination of febrile illness, sauna use and previously undiagnosed lipid myopathy.

METHODS

A previously well 31 year old Asian male with no family history of note presented with a 4 day history of fever and lethargy. He had spent 2 hours in a sauna “to sweat it out”. His GP had prescribed Amoxicillin and Paracetamol. He then presented to our Acute Medical Unit with thigh pain, pyrexia and dark brown urine consistent with myoglobulinuria. He had no history of exercise, alcohol or illicit drug use. He was found to have metabolic acidosis, acute kidney injury, disordered liver transferases and an initial CK of 284,250. He was treated in AMU with IV bicarbonate and saline, with Piperacillin and Tazobactam for possible sepsis. Despite this his CK rose to >900000 and he was transferred to the renal unit for haemofiltration.

OUTCOME

The patient was dialysed temporarily and his renal function fully recovered on discharge. A thigh muscle biopsy showed no necrosis but a high lipid content. Viral studies were negative. There have been case reports of rhabdomyolysis following sauna use in those with sickle cell trait\(^1\), and some cases linked to metabolic diseases\(^2\). This patient’s presentation, elevated liver enzymes and high muscle lipid content on biopsy points to a possible undiagnosed carnitine deficiency\(^3\).

CONCLUSION

This is an interesting case report of a late presentation of myopathy exacerbated by sauna use, resulting in an unusual presentation of rhabdomyolysis.

References


Case Reports

An Unusual Case of Legionella pneumophila Native Valve Endocarditis

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Aim

Legionella causes atypical pneumonia with Legionella pneumophila (L.pneumophila) accounting for 90% of human infection. We present an interesting case of native valve endocarditis secondary to L. pneumophila.

Methods

A 50 year-old man with no significant past medical history presented with dyspnoea and cough after returning from Los Angeles. On presentation he was febrile with initial investigations revealing marked hyponatraemia (111mmol/L) and radiological evidence of bi-basal consolidation. Ciprofloxacin and rifampicin were initiated on day 2 for L.pneumophila identified by urinary antigen testing. Severe respiratory failure necessitated intubation and ventilation and subsequent echocardiograms revealed a mitral valve vegetation and significant aortic regurgitation. Blood, bronchial cultures, polymerase chain reaction (PCR) in conjunction with screening for HIV and other causes of endocarditis proved negative. He received 6 weeks of antibiotics and had elective valve repair 7 months later.

Results

Definitive diagnosis of Legionella requires organism isolation with specific culture media, ideally using bronchial specimens. However, urinary antigen testing provides rapid results with high sensitivity and 100% specificity for L. pneumophila. PCR is also highly specific and offers equal sensitivities to cultures [1]. Serological testing has limited clinical utility due to delayed diagnosis, but provides strong evidence for Legionella’s role in blood culture-negative endocarditis (BCNE)[2]. Of 16 reported cases 14 had previous valve replacements and two involve native valves of immunosuppressed patients [3,4].

Conclusion

Endocarditis represents a rare extrapulmonary complication of Legionella. This case is distinguished by the absence of previous cardiac intervention and immunosuppression. Therefore, a high index of suspicion is warranted particularly in BCNE.

References


A Presentation of Neurocysticercosis as a Stroke Mimic

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Aim

Presentations of sudden onset neurological signs suggestive of stroke, which are non-vascular in cause, can be termed ‘stroke mimics’. Exclusion of stroke mimics is important due to the time-dependant nature of treating ischaemic stroke with thrombolysis. This case report has been written to highlight neurocysticercosis as a rare cause of stroke mimic in the UK that is an important differential in patients from certain endemic areas.

Method

A 62 year old Nepalese gentleman presented to the Royal Derby Hospital with acute left sided weakness and sensory loss. Suspecting a Stroke a CT head scan was performed and results were felt to be atypical. Subsequent MRI scanning revealed the ‘ischaemic’ regions to be oedema surrounding cysts. Cysticercosis serology was positive and the patient met the criteria for diagnosis with neurocysticercosis1.

Outcomes

The patient has subsequently made an almost complete recovery after treatment with albendazole, praziquantel and steroids.

Conclusion

Neurocysticercosis is the most common cause of seizures worldwide2, although rarely can present with focal neurological signs. It is common in endemic areas of the world but is rare in the UK, particularly outside of London. This case highlights that seeing patients from diverse origins widens the scope for unusual diagnoses in the acute setting. In patients with a neurological presentation or suspected stroke, Neurocysticercosis should be considered in those originating from endemic areas.


Case Reports

An occult pneumothorax in a patient with an exacerbation of asthma

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NHS

AIM

We present an unusual case of an occult pneumothorax in a patient treated as an asthma exacerbation.

METHOD

An occult pneumothorax is one which is missed during initial examination and plain radiography but picked up on a CT scan (1). They are thought to complicate up to 5% of trauma registry patients (2). However, a search of the literature on EMBASE and Medline has revealed no previously published account of an occult pneumothorax in an asthmatic patient. This case is of a 57 year old asthmatic female who presented with pleuritic chest pain, difficulty in breathing and wheeze and was treated as an acute asthma exacerbation. After 24 hours she had not improved. A CT was performed to exclude a pulmonary embolism due to a history of recent surgery. The scan showed an anterior pneumothorax that was not visible on the initial chest x-ray (Figure1)

OUTCOME

The patient was treated conservatively given the small size of the pneumothorax. She received prednisolone and regular nebulisers for her asthma and her symptoms improved after 4 days.

DISCUSSION

The presence of a deep sulcus or subcutaneous emphysema on plain radiography are commonly missed signs and should raise the suspicion of an occult pneumothorax (3,4). A prospective study on the management of occult pneumothoraces has shown that 89% of miniscule and anterior pneumothoraces resolve with conservative management (5). We suggest that occult pneumothoraces be considered as a differential diagnosis in a patient presenting with an exacerbation of asthma failing to respond to conventional treatment.
Case Reports

A case of Oesophageal Rupture

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Aim: Highlighting the importance of an accurate history during the first patient encounter.

The case:

A 78 yr old presented to ED with severe sudden onset chest pain, a cxr in the department revealed a small right sided pneumothorax. This was initially managed by pleural aspirations as per the BTS guidelines. As the patients pain did not resolve and the cxr still showed a pneumothorax a chest drain was inserted. The patient was transferred to acute medicine.

On the AMU the patients drain had filled with brown fluid, on re-taking the history the patient reports he had been vomiting and retching prior to the onset of chest pain. A CT scan was performed and reported as likely haemothorax with no evidence of mediastinal air and we were advised to insert a posterior drain, however due to the high level of clinical suspicion of oesophageal rupture further discussions with the radiologists took place and a repeat CT scan with oral contrast revealed a oesophageal perforation. The patient was taken to theatre and an emergency thoractomy performed to remove food debris from his thoracic cavity, and a diversion tube inserted to occlude the site of perforation.

The case was a difficult case as generally oesophageal ruptures are left sided and the chest drain decompressed the air delaying the diagnosis, confidence in the clinical history was key.

The case demonstrates the importance of correlating radiological imaging with the history and clinical findings. Communication with the radiology department is essential and documenting why imaging is needed in the request is crucial. This will improve service delivery, performing the right test, for the right patient at the right time.
Case Reports

Weil's disease on Acute Assessment Unit

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Aim

To increase the awareness of Icteric Leptospirosis also known as Weil’s disease which is a fatal multiorgan disease that may present during on take.

Methods

A 53 year-old man presented to the Acute Assessment Unit with a 5 day history of fever, rigors and myalgia. He reported extreme pain in his lower limb and generalised lethargy. He admits of going to the park along the river to pick berries prior to this when history was retaken. On examination he was pyrexial, jaundiced and had severe calf tenderness. All other examinations were unremarkable.

Outcome and results

Patient was initiated on antibiotics however had clinical deterioration with septic shock and respiratory compromise on day 2 of admission requiring high dependency care for observation. CT Thorax showed tree-in-bud nodularity throughout both lungs. In view of drop in haemoglobin, thrombocytopenia and high oxygen demand patient underwent bronchoscopy that excluded pulmonary haemorrhage. His CK was elevated at 21040iu/L, raised bilirubin 75umol/L, ALT 309iu/L, creatinine 155umol/l and low platelet of 59x109/L. Leptospirosis was suspected in view of possible animal urine exposure. Leptospira IgM was positive with confirmatory test of Microscopic Agglutination test was positive at 1:640 consistent with acute leptospiral infection.

Conclusion

Fever and elevated CK with strong exposure to animal urine should raise the suspicion of leptospirosis. Although our patient did not have pulmonary haemorrhage it is more often seen in this reemerging zoonotic disease that leads to ARDS and fatal outcome.
Case Reports

DVT: When outpatient anticoagulation is not enough – A case of May-Thurner-Syndrome.

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AIM

This outlines an atypical management of Deep-Vein Thrombosis (DVT). We aim to increase acute physicians' awareness of:

1. propriety of admission and/or access to the vascular team in select cases of DVT
2. their crucial role as secondary preventers of recurrent DVT and post-thrombotic syndrome morbidity.

METHODS

The setting is a district general hospital in East Anglia, UK. Following the introduction of Low Molecular Weight Heparins, the management of DVTs is typically combined with warfarin loading. This is outpatient-clinic based, or same-day discharge from Accident & Emergency (A&E) if GP referred.

OUTCOME

Clinical details: 67 year old female presenting to A&E with 3-days of buttock/thigh pain and 1-day of a swelling left leg; recent long-haul flight to the USA. On examination: thigh/calf circumference left(L) and right(R); (L:52cm/42cm) and (R:45cm/37cm) respectively. No significant medical history. Investigations: Ultrasound(USS) showed popliteal-femoral-ileal thrombus. Acute medical team decision to admit but tumour markers were found normal; further USS: no pelvic mass. Vascular opinion recommended catheter-directed thrombolysis. Post-procedure venous angiogram showed iliocaval compression typical of May-Thurner-Syndrome. The left common iliac vein promptly stented.

CONCLUSION

Converse to the typical management of DVT, this patient is at lower risk of DVT recurrence associated with iliopopliteal-femoral thrombi\(^1\) and May-Thurner-Syndrome. She is less likely to suffer the morbidity of secondary vascular pathology from post thrombotic syndrome\(^2\).

We highlight this case to flag the value of admission for special cases of DVT, not necessarily increase the consideration of thrombolysis per se since this has its own pitfalls.

References


Case Reports

A rare presentation of bowel injury following suprapubic catheter change.

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Introduction

We present a 81-year-old female with an unusual complication of small bowel perforation following suprapubic catheter change.

Case report

This lady presented to our AMU with pyrexia and drowsiness. She had a background of paraplegia and loss of bladder control secondary to a spinal meningioma. This had resulting in long-term urinary catheters and recurrent UTIs. Five weeks prior to admission she had a suprapubic catheter inserted under cystoscopic guidance. Her clinical presentation and investigations were suggestive of a urinary tract infection (UTI). She was treated with Ertapenam as ESBL tagged and a decision was made to replace the catheter. The catheter following change drained only 250 mls of urine overnight. The next day in view of frank haematuria and faeculent sediment an urgent CT abdomen was done which showed the catheter was placed in the lumen of the small bowel.

An urgent laparotomy and open suprapubic catheter insertion was performed. Intraoperative findings showed no evidence of peritonitis. After a prolonged hospital stay our patient was discharged.

Discussion

Bowel injury is a recognised but rare complication of suprapubic catheterization. Patients with previous lower abdominal/pelvic surgery have been described to be at risk. The majority of these injuries are detected shortly after insertion. There are only few case reports where recognition may be delayed until the first catheter change. The likely mechanism is the injury occurred during the original insertion when the catheter passed through the bowel enroute the bladder. The ensuing inflammatory fibrosis sealed the perforation and the patient remains asymptomatic. Poor urine output and faeculent sediment post suprapubic catheter change will raise a suspicion of bowel injury. Patients should be counseled fully regarding this risk prior to undergoing a suprapubic catheter insertion or change.

References

Benjamin L Jackson, Paul C Leeder, and J. Huw Williams


Case Reports

Not Another Pyelonephritis: Diagnostic Errors in the MAU

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Not another pyelonephritis: diagnostic errors in the MAU

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Up to 15% of diagnoses we make are incorrect, resulting in serious disability in up to 50% of cases¹. We present two cases with initial diagnosis of pyelonephritis to increase awareness of diagnostic cognitive errors and explore the possible biases.

1: 80-year-old male admitted with right sided back pain, worse on movement, and pyrexia. No urinary symptoms and unremarkable examination documented, though leukocyturia and raised inflammatory markers. Senior reviewed, diagnosed with pyelonephritis and commenced on treatment. On day 3 was fully examined by another consultant and a tender palpable lump was felt on his posterior rib wall. A CT chest/abdominal revealed a mass eroding through his 9th rib, which biopsy proved malignant.

2: 60-year-old male with known multiple vascular risk factors and an aortic abdominal aneurism (AAA), presented with bilateral back pain, urinary symptoms and pyrexia. On examination flanks and suprapubic regions were very tender; acute kidney injury and leukocyturia were noted. He was senior reviewed on two occasions and the diagnosis of pyelonephritis was made. Despite optimal treatment his renal function deteriorated and the patient arrested on day 3. A bed-side USS scan showed a posteriorly ruptured AAA.

In the cases presented, a condition that is common was easily recalled ("availability bias") despite unusual features on presentation (case 1).

In case 2 the wrong diagnosis may be propagated after senior review despite poor outcome ("diagnostic momentum" bias).

Some diagnoses may be the easiest to make and we should not shy away from rethinking and reassessing at every opportunity.

References:

Case Reports

A Case of Insulin Oedema on the Emergency Assessment Unit

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Introduction / Aim

Insulin is vital to the management of patients with Type 1 diabetes.

Here we describe a case of Insulin Oedema presenting to our Emergency Assessment Unit.

We wish to highlight this complication of insulin therapy as important for physicians working in Acute Medicine to recognize.

Case

A 21 year-old female with a 10-year history of type 1 diabetes presents with a history of 7 kilograms of weight gain over 2 weeks (to 60.6 kg). She had recently endeavored to take her insulin as prescribed (total of 66 units insulin daily) having had previous poor diabetic control.

Examination identified central obesity with diffuse abdominal wall fullness and oedematous legs.

Investigations (fig 1) were essentially normal.

Specialty consultation diagnosed Insulin Oedema.

This is a complication related to commencement or intensification of insulin therapy. Hypotheses as to aetiology include insulin causing enhanced renal tubular sodium resorption, increased capillary permeability or tissue glycogen deposition with subsequent fluid accumulation.

Oedema usually spontaneously resolves within days or weeks aided by restriction of salt and fluid intake. Diuretics are rarely required.

Conclusion

Patients with poor glycemic control may well present to Acute Medical services.

They are at risk of developing Insulin Oedema on intensification of treatment.

Whilst severe cases are rare, mild cases are likely under-reported and unrecognized.

Patients with poor treatment concordance may be reluctant to maintain intensification of treatment in the event of developing Insulin Oedema. The condition must therefore be correctly anticipated and identified to enable appropriate patient counseling.
Case Reports

Unusual cause of tension pneumothorax in a patient with acute onset chest pain

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Aim

To highlight the rare cause of tension pneumothorax secondary to Boerrhaave’s syndrome in a patient presenting with acute onset chest pain

Methods

72 year old previously healthy man presented to the emergency department with acute onset left sided chest pain. The pain began after 2 episodes of emesis. He was severely dyspnoeic with respiratory rate of 44 and saturating 97% on 15L. On examination, he had no audible breath sounds on the left chest and tense but non tender abdomen. Portable chest x-ray demonstrated a large tension pneumothorax with mediastinum shift to the right with blunted left costophrenic angle (figure 1). Large bore cannula was inserted for initial decompression followed by Seldinger chest drain. Despite initial improvement, the patient deteriorated and CT was performed which showed worsening pneumothorax and effusion in the left lung base. In view of this fact, we performed an oral contrast CT that demonstrated pneumothorax secondary to spontaneous oesophageal perforation. The patient was transferred to tertiary centre for immediate oesophagectomy and repair. After initial stay in ITU, he made a good recovery and was discharged.

Conclusion

This case report describes extremely rare and serious complication of Boerhaave’s syndrome. There are only few reported cases in literature of tension pneumothorax secondary to spontaneous oesophageal rupture (1,2). It requires prompt recognition and urgent management. The diagnosis should be confirmed with oral contrast computer tomography and early surgical intervention to produce the best outcome. Here without rapid diagnosis and surgical intervention, the outcome may have differed drastically.

References:


Case Reports

Fever Does not Always Equal Sepsis: Neuroleptic Malignant Syndrome in the Acute Medicine Unit

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AIM:

Neuroleptic malignant syndrome (NMS) is a rare condition associated with the use of neuroleptics and other medications that interfere with the dopaminergic system. Its vague presentation delays diagnosis and treatment, leading to complications and a high mortality (4-30%). This case report highlights the diagnosis and management of NMS in an acute setting.

METHODS:

A 23-year-old female psychiatric inpatient presented with worsening agitation, dysautonomia (erratic blood pressure, tachycardia, diaphoresis) and pyrexia. She had been on long term aripiprazole and had been started on olanzapine to control her recent psychotic episode. The patient was agitated and uncooperative. Her muscle tone was increased. Her initial observations showed a temperature of 38.4 °C, heart rate of 125 (ECG showed sinus tachycardia) and an erratic blood pressure ranging from 120/65 to 150/100 mmHg. There was no evidence of infection, CXR and cultures were clear. Her CK on admission was 3784 u/L and there was a significant leukocytosis.

OUTCOMES/RESULTS:

All antipsychotic medications were stopped and she was treated supportively with PRN diazepam, IV fluids, antipyretics, and thromboprophylaxis. The patient improved clinically and biochemically with her CK being 951 u/L on transfer back to psychiatry five days later.

CONCLUSION:

There are no fixed criteria for diagnosing NMS, but a diagnosis is considered when the typical combination of altered mental status, muscle rigidity, hyperthermia, and autonomic instability is present. Increases in creatinine kinase support the diagnosis. Management involves withdrawal of any potentially causative medications and supportive care which prevents serious complications and decreases mortality.

REFERENCES:


Case Reports

Case Report of a Patient Admitted with Milk Alkali Syndrome due to Anti Acid Use

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Introduction:

Milk alkali syndrome is the third commonest cause of hospital admissions for hypercalcemia, but the diagnosis is often missed. It was initially described in association with excessive consumption of milk and sodium bicarbonate for the treatment of peptic ulcer disease. It is characterized by the triad of hypercalcemia, metabolic alkalosis, and renal insufficiency. The diagnosis is based on exclusion of other causes for hypercalcemia. If untreated, milk-alkali syndrome may lead to metastatic calcification and renal failure.

Case report:

An 81-year-old man with a history of smoking, alcohol abuse and a cerebrovascular accident was presented to the emergency department with a chronic paracetamol intoxication and a weak general condition. The patient was found by his neighbour laying on the floor, where he had laid for several hours. The last couple of months he suffered from severe reflux complaints and stomach ache, for which he had used large amounts of over the counter paracetamol (up to 25 gram daily) and Rennies© (calcium carbonate with magnesium carbonate) (up to 12 tablets daily). On physical examination we saw a dehydrated, cachectic, patient with a blood pressure of 120/80 mmHg, a pulse rate of 85/min and hypothermia of 34.2°C. Further physical examination showed no abnormalities. Laboratory results revealed a potassium of 2.8 mmol/L and a calcium of 3.35 mmol/L. Serum creatinine was 62 micromol/L with a BUN of 10.8 mmol/L, phosphate was 0.49 mmol/L, and magnesium was normal. Liver enzymes were normal, and there were no signs of inflammation. Bicarbonate level was unfortunately not measured. An ECG showed atrial fibrillation, a right bundle branch block and a prolonged QTc of 525 msec. The chest radiography was normal. The metabolic disturbances were considered as a result of starvation with a suspected underlying malignancy. He was admitted with surveillance of the cardiac rhythm and treatment was started with intravenous fluids and supplementation of potassium, pamidronate disodium. Furthermore acetylcystein was started for the chronic paracetamol intoxication. A vitamin D intoxication, hyperparathyroidism and multiple myeloma were excluded as a cause for the hypercalcemia. Later, a gastroscopy showed a diaphragmatic hernia of the sliding type, a hemorrhagic gastritis and oesophagitis. Biopsies revealed no Helicobacter pylori of fungal involvement.

Discussion: On hindsight our patient had a milk alkali syndrome based on the combination of hypercalcemia with modest renal insufficiency (creatinine on admission 62 micromol/L, on discharge 29 micromol/L) and no other cause of hypercalcemia. The standard treatment is withdrawal of the offending agent and correction of the volume depletion with isotonic saline. Bisphosphonates should be avoided because that may result in hypocalcemia. Indeed our patient developed a hypocalcemia of 1.83 mmol/L seven days after admission, which recovered without any complications.

We advise to measure serum calcium in patients presenting with gastrointestinal complaints and chronic use of calcium carbonate preparations. Conversely, we also advise to consider milk alkali syndrome in patients with unexplained hypercalcemia.

Conclusion: 81 year old patient admitted with hypercalcemia due to milk alkali syndrome resulting from excessive calcium/magnesium carbonate use for reflux complaints

References:

1: Kaklamanos et al, BMJ 2007 335: 397-8
2: Picolos et al, clin endocrinol 2005 Nov; 63(5):566-76
Case Reports

A New Challenge: Suicide Attempt using Nicotine Fillings for Electronic Cigarettes

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Introduction:

Electronic cigarettes are increasingly popular for smokers as an aid in the cessation of smoking. They are considered a less harmful alternative for traditional cigarettes. However, the liquid fillings of the electronic cigarettes ('e-liquid') contain high concentrations of nicotine, which can cause potentially lethal poisoning when ingested. We present a case of nicotine poisoning of a 27 year-old male who attempted suicide by ingestion of a large amount of e-liquid.

Case report:

A previously healthy 27 year-old man with a borderline personality disorder presented at our Emergency Department (ED) one hour after ingestion of five e-liquid fillings in an attempt to end his life. He had also consumed five units of wine. The e-liquid fillings contained a total of 420 mg of nicotine, and unknown concentrations of propyleneglycol and glycerine.

Before arrival at the ED, the patient had vomited three times, but upon arrival he was free of complaints.

Initial evaluation showed a sinus tachycardia of 117 bpm, a blood pressure of 144/99 mmHg and oxygen saturation of 98%. The patient was afebrile (36·1°C). Apart from excessive salivation, his physical examination was unremarkable.

Laboratory examination revealed a high anion gap (16 mmol/L (normal 8-12 mmol/L)) lactic acidosis with a pH of 7·33 and lactic acid of 3·8 mmol/L. The osmolality was 331 mOsmol/kg and ethanol 3.9 ‰. Activated charcoal was administered and repeated twice. During the observational period of 48 hours at the intensive care no adverse events were noted and the metabolic acidosis disappeared. He was discharged after psychiatric consultation.

Discussion:

Electronic cigarettes are gaining popularity among smokers as an aid in the cessation of smoking. However, there is a possibility of potentially lethal nicotine intoxication. Information about intoxications with liquid nicotine is scarce.

E-fillers are an easily accessible source of highly concentrated nicotine solutions. It is therefore highly likely that more intoxications will occur in the near future.

The symptoms of acute nicotine poisoning follow a biphasic pattern, beginning with vomiting, abdominal pain, hypertension, tachycardia, excessive salivation, and seizures due to nicotinic cholinergic stimulation. The second inhibitory phase consists of hypotension, bradycardia and dyspnea and can lead to coma and respiratory failure.

Lethality of nicotine poisoning depends on the nicotine concentration in the patient's blood. Calculation of ingested nicotine is not accurate, as actual nicotine concentrations often significantly differ from the reported concentration by the manufacturers of the e-liquid. Measuring nicotine plasma levels can be useful, however, this laboratory test is not rapidly and universally available. Furthermore, interpretation of the results can be challenging, since data of nicotine poisoning are scarce. Our patient potentially ingested up to 420mg of nicotine, which is well above the internationally accepted lethal limit of 60mg. However, a recent article by Mayer shows that this limit is questionable. He suggests that the lethal dose is 0·5g of orally ingested nicotine. In our patient the serum concentration of nicotine was 50ug/L at 2 hours after ingestion. The concentration of cotinine (the main metabolite of nicotine) was 250 ug/L. Most of the nicotine was probably eliminated by vomiting or bound by activated charcoal before absorption.
Management of nicotine intoxication consists of general supportive measures and administration of activated charcoal. Hemodialysis or other extracorporeal techniques are not useful. Proton pump inhibitors are contraindicated, as they increase nicotine absorption in the gastrointestinal tract.

In conclusion, ingestion of e-liquid can lead to potentially lethal nicotine poisoning. Given the increasing popularity of electronic cigarettes, the frequency of nicotine intoxications is likely to rise in the next years. Physicians working in the Emergency Department and general practice should become familiar with this intoxication.

References:


Case Reports

An Unusual Peripheral Blood Smear in an African man

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Introduction: Patients originating from sub-Saharan Africa may have endemic diseases even if they have not visited their country for years.

Case report: A 27 year old man from Ghana was referred to our Emergency Department (ED) with fever, persistent cough and night sweats since 3 weeks. He also reported haemoptysis and 20 kg weight loss. His medical history was unremarkable and he lived in The Netherlands since 2004. On presentation, his physical examination showed a blood pressure of 110/65 mmHg, pulse rate of 130 bpm, body temperature of 39.3°C, generalized lymphadenopathy and no hepatosplenomegaly. Laboratory results were: Hb 4.7 mmol/L, Leukocytes 1.2 x 10^9/L, Thrombocytes 54 x 10^9/L, Sodium 125 mmol/L, Ferritin 69.401 µg/L and LDH of 2967 U/L. The urinary sodium concentration was 56 mmol/l and the chest X-ray was unremarkable. We also performed a HIV test. By an emergency call, the laboratory reports an abnormal blood smear. (Figure 1 and 2)

Figure 1

The differential diagnosis of this intracytoplasmic disease with systemic symptoms is very limited and should include Histoplasmosis, Candida Glabrata, Penicillium Marneffei and specific forms of Trypanosoma Cruzi. Since histoplasmosis was most suspected due to the microscopically appearance and the origin of the patient, we started him on liposomal amphotericin B. Because of the hyponatremia adrenal involvement was suspected but ruled out by a normal synthetic ACTH stimulation test. A few days after admission histoplasmosis was confirmed by lymph node biops[y][i]. The HIV-test turned out to be positive, with an absolute CD4+ count of 0/mm^3 and a viral load of 5 million copies/milliliter. Cultures for tuberculosis were negative. We added cART to the therapy and during the following weeks the patient clinically recovered.

Discussion: It is advisable to perform a HIV test promptly in the ED in patients originating from sub-Saharan countries due to the high prevalence of HIV and the considerable consequences in diagnostic approach and treatment strategies, even when denying high risk sexual activities.

Although histoplasmosis is a rare disease in our part of the world it should be considered in patients who originate from endemic countries. Clinical manifestations consist of three types: pulmonary, disseminated and chronic cavitatory forms. Immunocompetent patients are mostly asymptomatic but 70% of the immunosuppressed patients present with disseminated disease. These patients often present with pancytopenia and hepatosplenomegaly. Adrenal involvement is common (80-90% of autopsied cases), but adrenal insufficiency is found in less than 10%. In severe forms, the diagnosis can be made microscopically, but antigen test in urine or blood are most sensitive. In HIV positive patients, treatment with liposomal amphotericin B combined with cART has shown to improve outcome.[ii]

Conclusion: Disseminated histoplasmosis is a severe potentially life threatening infection that may be associated with HIV. We demonstrate that with relatively simple means, including a good differential diagnosis, instantaneous HIV testing and awareness of the laboratory personnel, the correct diagnosis can already be established in the ED.
