CASE REPORTS

Cocaine/heroin induced rhabdomyolysis and ventricular fibrillation

B McCann, R Hunter, J McCann

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A case of cardiorespiratory arrest in a 28 year old man after cocaine and heroin ingestion is described. The arrest is attributed primarily to hyperkalaemia/rhabdomyolysis—a recognised consequence of each of these drugs. The administration of naloxone may have been contributory. He developed acute renal failure, disseminated intravascular coagulopathy with consequent lower limb compartment syndrome requiring fasciotomy. Ventricular fibrillation was identified at thoracotomy.

A 28 year old man was brought to the emergency department after intravenous ingestion of cocaine and heroin. He was noted to be pale, cyanosed, bradypnoeic (6/min), and hypotensive (70/40).

Initial treatment consisted of oxygen via a facemask, and 800 µg of naloxone intravenously. Within two minutes he developed cardiorespiratory arrest with pulseless electrical activity (PEA). QRS complexes looked normal. PEA protocols were started according to Advanced Life Support (ALS) guidelines.[1] Ventilation was begun using bag and mask, proceeding to endotracheal intubation. Drugs administered included adrenaline (epinephrine) 1 mg, naloxone 1200 mg, sodium bicarbonate 8.4% 50 mmol with a fluid bolus of 500 ml of normal saline.

His cardiac rhythm converted to asystole within five minutes. Atropine 3 mg, and second dose of adrenalin were given. Ten minutes after cardiac arrest an irregular baseline was noted on his cardiac monitor. A series of shocks at 200, 300, and 360 joules was delivered. This had no impact on the underlying rhythm, which was deemed to be asystolic.

At 15 minutes after arrest left thoracotomy was performed. The heart was noted to be coarsely fibrillating. Chest leads were checked and the gain on the cardiac monitor increased. Monitor rhythm retained its asystolic appearance. Internal cardiac massage was started. A single internal counterdefibrillatory shock of 30 joules converted the heart (and the monitor) to sinus rhythm. Heart rate was 80/minute, sustaining a blood of 128/96. His 12-lead electrocardiograph (after arrest) showed no features compatible with hyperkalaemia or ischaemia.

Shortly thereafter the patient began to gag on the endotracheal tube, opened his eyes, and tried to lift his head off the bed. He was sedated and paralysed. Chest drain was inserted, chest wound was closed and he was transferred to the intensive therapy unit.

It was established (subsequent to time of arrest) that the time of cocaine and heroin ingestion was at least 12 hours before presentation.

INVESTIGATIONS

The following investigations were recorded: sodium 139 mmol/l; potassium 7.5 mmol/l; urea 8.9 mmol/l; glucose 6.7, Hb 15.2; WCC 18.6; platelets 167. Arterial blood gas (after restoration of spontaneous circulation): pH: 7.03; $Paco_2$: 6.29 kPa; Pao_2: 24.3 kPa; base deficit: 18.0 mmol/l; lactate: 8.4 mmol/l. Creatine phosphokinase: 90 500 IU/l (normal range 33–194). Urine: myoglobin: positive; cocaine metabolites: positive; opioids: positive.

Consequent upon his rhabdomyolysis he developed acute renal failure requiring haemodialysis, disseminated intravascular coagulopathy and right lower limb compartment syndrome requiring fasciotomy. Echocardiogram showed a left ventricular ejection fraction of 53%. Respiratory function remained stable. At day 10 (after tracheostomy) he had spontaneous eye opening, flexed to pain and had no response to verbal commands. Brain stem reflexes were intact. Computed tomographic scan of his brain was normal.

The patient died two months later. Cause of death was bronchopneumonia complicating multiorgan failure.

DISCUSSION

Rhabdomyolysis is a well documented complication of cocaine and heroin ingestion.[2] In the past the diagnosis was often made on the basis of a massively raised creatine phosphokinase, which was being measured as a marker for damage to the myocardium. The presence of urinary myoglobin confirms the diagnosis. There is a danger that with the advent of "cardiac specific" markers and the resultant demise of phosphokinase, that the diagnosis will not be detected as readily as in the past. It is important to be mindful of the potential for rhabdomyolysis in relation to cocaine and heroin overdose, and to check the urine for myoglobin. It follows that hyperkalaemia should be considered as a probable concomitant and treated appropriately.

Naloxone is an effective opioid antidote that is not without harmful side effects.[3] This patient had injected heroin and cocaine at least 12 hours before presentation. The timing of the ingestion was unknown at presentation. In retrospect his clinical status at that time did not reflect acute overdose but a complication of same—that is, rhabdomyolysis. Against this background naloxone may have been harmful for two reasons: (1) administration of naloxone in the patient with combined opioid and sympathomimetic intoxication may provoke life threatening manifestations of sympathomimetic toxicity by removing the protective opioid mediated CNS depressant effects[4]; (2) the arrhythmogenesis of naloxone is well documented and may have been increased in this case on a background of hyperkalaemia.[3–7] Establishing the timing of ingestion of narcotics in relation to the time of presenting complaint can be crucial.

The "hunt for VF" (ventricular fibrillation) is listed as the second of the 10 commandments for ACLS (Advanced Cardiac Life Support) of the American Heart Association.[8] In this case PEA was followed by apparent asystole, which was confirmed in standard fashion by changing lead and increasing gain. The

Abbreviations: PEA; pulseless electrical activity; ALS; Advanced Life Support

discovery of ventricular fibrillation macroscopically at thoracotomy, confounded this diagnosis. The identification of ventricular fibrillation in this case was serendipitous and not the result of a hunt. Our equipment—a Lifepak Physiocontrol 9—subsequently passed electronic medical engineering assessment. The decision to proceed to thoracotomy was based on patient age, the probability of underlying toxic but reversible insult, and failure to re-establish a cardiac output following standard ALS protocols. Our intention was to improve cardiac output by internal massage[9 10] pending reversal of a toxic insult. We could find no report in the literature that described thoracotomy to identify ventricular fibrillation. This case reinforces the advice contained in resuscitation literature,[11–13] which suggests that we defibrillate asystole if in any doubt about the cardiac rhythm.

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Accidental colchicine overdose. A case report and literature review

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Colchicine overdose is uncommon but potentially life threatening. It is a safe drug when used according to established therapeutic guidelines but causes serious systemic effects if ingested in doses that exceed the recommendations. Overdose must therefore be recognised early and treated appropriately to optimise the outcome. A fatal case of colchicine overdose caused by inappropriate self medication is reported and to the best of the authors' knowledge, there has been no report of fatal accidental overdose in the United Kingdom. The pharmacology of colchicine, the clinical features associated with overdose, and the options for treatment are discussed.

A 41 year old white man was brought to the accident and emergency (A&E) department after ingesting 53×500 µg colchicine tablets over the previous 24–48 hours in an attempt to alleviate the pain of acute gout. The number of tablets was calculated from those remaining in a recently prescribed bottle.

On arrival he reported abdominal pain, diarrhoea, and vomiting for the preceding 24 hours. He was alert and orientated but was peripherally vasoconstricted and tachycardic with a blood pressure of 108/70. Breathing was laboured and shallow with a respiratory rate of 40 breaths/minute but lungs were clear. There was mild abdominal distension but bowel sounds were normal. Electrocardiogram was unremarkable.

While in A&E the patient had a witnessed electromechanical dissociation (EMD) cardiac arrest without warning. He was intubated, cardiopulmonary resuscitation started, and 1 mg adrenaline (epinephrine) given intravenously. Full recovery occurred after two minutes and he was extubated. After arrest ECG showed multifocal ventricular ectopics. He then had two further witnessed EMD cardiac arrests of identical pattern, although he was not intubated, and again completely recovered after each. The patient was then transferred to the intensive care unit for further observation and treatment.

Laboratory investigations showed a white cell count 31.3×10^9/l, platelets 341×10^9/l, urea 10.4 mmol/l, creatinine 391 mmol/l, ALP 1320 U/l, ALT 82 U/l, bilirubin 43 µmol/l, INR 3.2, magnesium 0.58 mmol/l, and corrected calcium 2.03 mmol/l. Arterial blood gas results showed a profound metabolic acidosis (pH 7.00, pCO_2 6.30, pO_2 7.72, HCO_3^- 11.5).

His clinical condition and acidosis worsened (pH 6.90, HCO_3^- 9.7) and he was intubated and given a bicarbonate infusion with some improvement (pH 7.05, HCO_3^- 15.5). Chest radiography showed bilateral patchy shadowing throughout both lung fields. He remained cardiovascularly unstable and required dopamine, adrenaline and noradrenaline infusions to maintain blood pressure.

Oliguria and then anuria ensued and were not responsive to supportive measures. He continued to deteriorate and death occurred approximately 11 hours after admission.

DISCUSSION

Colchicine is a naturally occurring alkaloid with weak anti-inflammatory activity derived from the autumn crocus *Colchicum automnale* and the glory lily *Gloriosa superba*. It has been used extensively in the treatment of gout for many centuries and also been recommended in preventing attacks of familial Mediterranean fever[1] and in the treatment of primary biliary cirrhosis,[2] amyloidosis,[3] and condyloma acuminata[4]

Colchicine has potent anti-mitotic activity, which is caused by its binding, both reversibly and selectively, to tubulin, the microtubular protein that disrupts the function of the mitotic spindles in those cells capable of dividing and migrating. Although colchicine is taken up equally by all cells it is thought that those which have the highest cell turnover (that is, the greatest mitotic activity) are most affected.[2 5–7]

Colchicine is rapidly absorbed from the gastrointestinal tract after ingestion. It undergoes significant first pass hepatic metabolism, which primarily involves deacetylation. Subsequent to this, the metabolites undergo widespread enterohepatic recirculation before being excreted in bile and faeces. It is thought that the extended time period during which the gastrointestinal mucosal cells are exposed to colchicine may explain the prominence of the gastrointestinal symptoms of toxicity. Renal clearance also accounts for 10%–20% of colchicine removal and if normal renal function exists larger fractions can be excreted via this route if a toxic amount has been ingested. Increased urinary excretion also occurs in the presence of hepatic disease, as there is a reduction in the capacity for deacetylation. However, if renal and hepatic diseases coexist the possibility of toxicity greatly increases.[5–8]

Overdose with colchicine is uncommon and we are not aware of similar report of fatal accidental overdose in the United Kingdom. It exhibits a low therapeutic index although there is great variation in the dose required for significant morbidity. Patients have survived ingestion of more than 60 mg[9] but conversely others have died after ingesting only 7 mg over a prolonged period.[10] There does not seem to be any clear cut separation between non-toxic, toxic or lethal doses of colchicine. Indeed, symptoms of gastrointestinal toxicity such as nausea, vomiting, diarrhoea and abdominal pain are seen in 80% of patients on full therapeutic doses and are used as the clinical endpoint in dose titration.[5]

Overdose with colchicine constitutes a toxicological emergency and rapid intervention is required. The symptoms of toxicity are well described in the literature and can be separated into three characteristic phases (table 1).

This patient had three EMD cardiac arrests, from which full recovery was made each time, and an episode of self limiting ventricular tachycardia. Cardiotoxicity is much reported upon in the literature. Commonly, this manifests as arrythmias, namely sinus bradycardia, sinus tachycardia, ventricular fibrillation, and complete atrioventricular block. ECG changes of ST elevation in leads I, II and V3-V6 have also been reported.[11 12] However, the pattern of repeated cardiac arrests and a self limiting arrhythmia that we describe in this case have not previously been reported.

There are various suggestions to explain the effect that colchicine has on the heart. It is thought that there may be a direct toxic effect on the myocardial cells with impairment of impulse generation and cardiac conduction.[13 14] This has not been proved, although a similar mechanism of direct toxicity is seen on the cells of skeletal muscle.[15] It is also possible that the profound acid-base disturbances and electrolyte derangements associated with overdose will play a significant part.[8]

Gastrointestinal decontamination with gastric lavage and activated charcoal is often performed, and may help despite colchicine being rapidly absorbed because there is extensive enterohepatic recirculation.[8] Consequently, it is important that efforts are made to remove any remaining colchicine because the retrieval of even small amounts can greatly benefit prognosis.[11]

Table 1 Phases of colchicine toxicity

Phase	Symptoms
I 0–24 hours	Nausea, vomiting, diarrhoea, abdominal pain, and anorexia Electrolyte imbalance and hypovolaemia Peripheral leucocytosis
II 2–7 days	Bone marrow hypoplasia, profound leucopenia, and thrombocytopenia Cardiac arrythmias and cardiovascular collapse Respiratory distress, hypoxia, pulmonary oedema, and ARDS Oliguric renal failure Rhabdomyolysis Electrolyte derangements Metabolic acidosis Mental state changes Seizures Peripheral neuropathy and ascending paralysis
III 7th day onwards	Rebound leucocytosis Transient alopecia

The large volume of distribution of colchicine and the fact that 50% of its plasma concentration is linked to proteins means that methods of extracorporeal removal are ineffective. Therefore, haemodialysis, although of benefit in the treatment of any associated renal failure, is not used to increase elimination.[2 5]

Currently in the United Kingdom there is no specific treatment commercially available for the treatment of colchicine toxicity. However, the successful outcome after the use of colchicine specific Fab fragments has been reported.[5 16] Colchicine specific Fab fragments consist of the light chain and variable region of the heavy chain and are derived from goats.[2] Their mechanism of action is similar to that of digoxin specific Fab fragments, namely binding to the target drug allows redistribution into the intravascular compartment and thus the removal of substantial amounts from peripheral sites.[12] There is a high affinity between the Fab fragment and colchicine and this acts to prevent the drug returning to these peripheral binding sites.[9]

CONCLUSION

Overdose with colchicine is associated with a high mortality rate with death occurring secondary to rapidly progressive multiorgan failure. It is important therefore that the potential dangers of this drug are recognised by clinicians on its prescription, and that patients are given an understandable explanation of its effects including the point at which to cease ingestion. A careful watch must also be made of the number of tablets prescribed to avoid unintentional overdose of this potentially lethal drug.

Contributors

MJM initiated the idea, reviewed the literature and wrote the paper. PM initiated the idea, helped with the literature search and writing the paper. PP reviewed the manuscript and helped with writing the paper. PP acts as the guarantor of the paper.

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Toxicity of brake oil

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Brake oil is an automobile transmission fluid composed of a mixture of toxic glycols and glycol ethers. Three cases of poisoning with toxic glycol based brake fluid are reported who presented with mild metabolic acidosis and acute renal failure. As all the cases had presented late, treatment with ethanol was not started. All of them were treated successfully with haemodialysis.

CASE 1

A 30 year old man presented to the emergency room 72 hours after having consumed 100 ml of brake fluid after intake of 60 ml of whisky (40% proof). He had three vomits immediately after the consumption and a progressive decrease in his urine output the next day. At admission, the pulse was 92 per minute and blood pressure 164/100 mm Hg. The systemic examination was normal. Laboratory investigations revealed normal complete blood counts. The serum biochemistry values are given in table 1. The urine examination did not reveal any crystals of calcium oxalate; however, numerous red blood cells could be seen.

The presence of renal failure necessitated immediate haemodialysis. Hypertension worsened requiring antihypertensive therapy with nifedipine and atenolol. The presence of prolonged oliguria raised suspicion of renal cortical necrosis but the renal biopsy showed only acute tubular necrosis. After receiving 15 haemodialyses over a period of five weeks he went into the diuretic phase of acute renal failure and was discharged soon thereafter in a satisfactory condition.

CASES 2 AND 3

These were two brothers aged 40 years and 35 years. They presented to the emergency services 24 hours after having consumed approximately 40–60 ml of brake oil with 80 ml of rum. Both had many vomits over the next few hours followed by declining urinary outputs. At admission, the pulses and blood pressures of both were normal and systemic examination of both patients did not reveal any abnormality. Laboratory indices obtained showed normal complete blood counts. Serum biochemical values of both are presented in table 1. The urine examination of both patients showed a 2+ proteinuria, plenty of red blood cells but no crystals of calcium oxalate.

Both patients were taken up for immediate haemodialysis. The urine outputs of both remained in the oliguric range. After receiving six haemodialyses in one and eight in the other, both patients went into the diuretic phase of acute renal failure and were discharged a week later in a satisfactory condition.

DISCUSSION

Both ethylene glycol and diethylene glycol are colourless, odourless, sweet tasting compounds with widespread commercial use. Whereas ethylene glycol is used as antifreeze, coolant, and preservative, diethylene glycol has been used as a replacement for glycerine. The earliest reported toxicity of diethylene glycol dates back to 1937 when it was used as a vehicle for preparing sulfanilamide elixir.[1] According to a document by Dow Chemical Company, a leading producer of automobile liquids, brake fluid is a transmission fluid composed of a mixture of several glycols like ethylene glycol, diethylene glycol, polyethylene glycol, polypropylene glycol

Table 1 Laboratory parameters of patients presenting with brake oil intoxication

	Sodium/ potassium (mmol/l)	Urea in mmol/l and creatinine in μmol/l	Calcium/ phosphorous (mmol/l)	Arterial pH	Arterial Po_2 kPa	Arterial Pco_2 kPa	Serum bicarb (mmol/l)
Case 1	136/4.6	64.3/972.4	1.5/2.4	7.25	12.6	3.3	15
Case 2	137/4.9	53.6/371.2	1.8/1.7	7.30	13.0	4.6	20
Case 3	134/5.2	71.4/848.6	2.4/2.0	7.27	12.5	3.9	10

and glycol ethers. To the best of our knowledge, so far there has been only one case report of poisoning resulting from the consumption of brake fluid.[2]

After ingestion of ethylene glycol, inebriation occurs but the typical smell of alcohol is lacking. Ethylene glycol is metabolised by the enzyme alcohol dehydrogenase to glycoaldehyde, glycolic acid and glyoxylic acid, which are responsible for most of the clinical effects of ethylene glycol poisoning.[2] Cardiovascular toxicity of ethylene glycol usually appears after a period of 12–24 hours and is characterised by tachycardia, hypertension, and pulmonary oedema.[2] In all the three cases, apart from nausea and vomiting, the other clinical features were not present. Acute renal failure usually occurs as a delayed manifestation after 24 hours of ingestion in 73%–84% of cases of ethylene glycol poisoning [2] and was present in all of our cases. The presence of proteinuria in all cases was consistent with acute tubular necrosis and was confirmed on biopsy of the kidney in case number 1. All of our cases also had microscopic haematuria.

For patients presenting early with ethylene glycol poisoning treatment with ethanol is preferred. Ethanol acts by competing with ethylene glycol for the enzyme alcohol dehydrogenase thus limiting the formation of toxic metabolites. All three patients had presented late (>24–72 hours) with established renal failure. As the elimination half life of ethylene glycol is three hours [2] and more than five times the elimination half life had elapsed, there would have been little ethylene glycol left in the body. Hence, treatment with ethanol would not have served any therapeutic purpose. The modality of treatment chosen was haemodialysis to facilitate removal of toxic metabolites of ethylene glycol and to combat uraemia.

Besides ethylene glycol, the other components of brake fluid may also have played some part in the manifestations of this unusual poisoning. Diethylene glycol has been incriminated earlier in a case report of five cases of acute renal failure complicating the use of diethylene glycol based silver sulfadiazene ointment.[3] Recently, diethylene glycol has been implicated as the causative factor for renal failure in the paediatric population of Bangladesh, Haiti, and India.[4–6] The mode of poisoning was from contamination of the available liquid peadiatric medications with diethylene glycol. In another case report, propylene glycol has also been suspected of having led to hyperosmolarity and cardiorespiratory arrest in an infant.[7] All toxic glycols are metabolised by alcohol dehydrogenase resulting in profound metabolic acidosis attributable to the accumulation of organic acids. In all our cases, it is possible that the consumption of ethanol probably led to partial saturation of the enzyme alcohol dehydrogenase thereby limiting the formation of organic acids. The resultant metabolic acidosis was mild in all cases and significant toxicity on systems other than the kidneys was avoided.

Contributors

Navneet Sharma was the treating physician who diagnosed, managed and treated the patients in the medical emergency. Sanjay Jain, a senior consultant in the emergency department, actively participated in the discussion of treatment modalities and review of this paper. Navneet Sharma is the guarantor of the paper.

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Metal fume fever: a case report and review of the literature

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Metal fume fever is an acutely noxious inhalation syndrome secondary to metal oxide fumes. Despite preventative strategies sporadic cases are likely to continue to present to emergency departments.

A 55 year old man presented to the emergency department at 9 pm. He complained of feeling generally unwell for the previous five hours. He described malaise, fatigue, cough, fever, nausea, and dyspnoea at rest. He had no previous medical history of note and was usually fit and well. He was a non-smoker. He worked as a plumber and on the day of admission had been using an oxyacetylene torch to remove a steel tank.

On examination, he was unable to talk in full sentences. His respiratory rate was 24/min with an oxygen saturation of 94% in room air. Chest examination was normal. His pulse rate was 100/min and he was feverish at 39°C. There were no other findings of note. Blood gas analysis demonstrated acute type I respiratory failure with an arterial oxygen partial pressure of 8.8 kPa. There was a neutrophil leucocytosis but no other abnormality of baseline pathology. A chest radiograph revealed patchy opacification in the right perihilar area.

His 18 year old son, who had been working with him all day, presented to the emergency department simultaneously. He complained of malaise, nausea, vomiting, and cough. He had no previous medical history. Examination and investigation were unremarkable.

A further coworker presented to a local minor injury unit the same night with similar symptoms to the 18 year old patient. He required no medical intervention.

A diagnosis of metal fume fever was made and the 55 year old man admitted for observation and oxygen therapy. His son was discharged. By the following morning both had made a full recovery.

DISCUSSION

Metal fume fever (MFF)—"brass founders ague", "zinc shakes", "Monday morning fever"—is a self limiting inhalation fever attributed to a number of metal oxide fumes. The history is characterised by fever, headache, myalgia, fatigue, and dyspnoea. Other features include cough, thirst, a metallic taste, salivation, and a neutrophil leucocytosis. Radiography may demonstrate bilateral diffuse infiltrative pulmonary lesions. Pulmonary function tests demonstrate a significant reduction in vital capacity, transfer factor and arterial oxygen partial pressure. Urine and plasma metal levels may be increased.

Onset is typically rapid, occurring between three and 10 hours after exposure. Spontaneous recovery occurs within 24 hours. No long term complications are known.[1]

MFF is classically associated with zinc oxide fume exposure from welding galvanised steel or brass. It is also seen in association with high temperature zinc coating processes and metal pouring in brass foundries. Magnesium and copper oxide fumes are more rarely the causative agents. Approximately 2000 cases are reported annually in the United States.[2 3]

The pathophysiology is unclear but seems to reflect a direct toxic effect. The lack of a latent period and the fact that large proportions of a single workforce can be affected are against an immunological basis for the disease. [4] There is evidence of an exposure dependent neutrophil alveolitis in association with tumour necrosis factor α, interleukin 6, and interleukin 8 cytokine release from pulmonary cells.[4] Interestingly there is evidence of rapid adaptation after repeated exposure though the transient nature of this tolerance is reflected in the synonym "Monday morning fever". It has been postulated that tolerance occurs because of induction of metallothionein protein synthesis. These proteins bind to heavy metals preventing toxic metal accumulation. [5]

Diagnosis is based on clinical suspicion, clinical findings, and rapid resolution. Evidence of possible exposure is critical. Treatment is symptomatic. The syndrome needs to be differentiated from serious MFF seen after military smoke exposure, which typically has a biphasic response with severe relapse 24 to 48 hours after initial remission.[6] It should also be differentiated from true chemical pneumonitis after metal fume exposure. This is particularly associated with cadmium fumes but also occurs with manganese, mercury, and nickel. In the early stages, it may be indistinguishable from MFF but the pneumonitis is progressive and usually complicated by non-cardiogenic pulmonary oedema. Cadmium also injures the renal tubules resulting in acute renal dysfunction.[1]

Preventative strategies for MFF are aimed at reducing fume exposure concentrations.[7] "Toxic" levels have not been established. However, Fine *et al* have demonstrated that inhalation of traditionally safe levels of zinc oxide can produce MFF symptoms and a rise in plasma interleukin 6.[8] In the UK, the Reporting of Injuries, Diseases and Dangerous Occurrence Regulations (1995) place responsibility on employers to report MFF to the Health and Safety Executive once it has been diagnosed in writing by a doctor.[7]

CONCLUSION

MFF is a common, acute, severe occupational syndrome. Despite preventative strategies, sporadic cases continue to present to emergency departments. Recognition of the possibility of an inhalation syndrome requires an understanding of the aetiology and an adequate occupational history. Early recognition can prompt a more directed management approach and permit the exclusion of more serious inhalational syndromes.

Contributors

PK and HY were responsible for the diagnosis and management of the case, reviewed the literature and wrote the paper. IOS reviewed and advised on the paper and is the guarantor for the paper.

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Methylene blue: a treatment for severe methaemoglobinaemia secondary to misuse of amyl nitrite

B Modarai, Y K Kapadia, M Kerins, J Terris

Emerg Med J 2002;**19**:270–271

Poisoning with inhalational nitrites is a recognised cause of methaemoglobinaemia presenting to the emergency department. Methaemoglobin (MetHb) is the oxidised form of haemoglobin and incapable of carrying oxygen. The concentration of MetHb does not exceed 1%–2% in the normal physiological state. Previously reported cases [1 2] include patients with severe poisoning who were comatosed on presentation or required repeat treatment with methylene blue (MB). Two cases of severe methaemoglobinaemia secondary to misuse of amyl nitrite are presented. A MetHb level of greater than 50% was measured in each case, however, both patients were conscious and talking on presentation and showed clinical and biochemical response to treatment with one dose of MB.

CASE REPORT 1

A 32 year old white woman was brought by ambulance to the emergency department. She had been found "collapsed" on the street but was alert and appropriately responsive. She admitted sniffing half the contents of a small bottle of amyl nitrite, drinking one unit of alcohol, and smoking cocaine. On examination a deep blue-grey discoloration was noted of her skin especially over the lips, nose, and ears despite 15 l/min high flow oxygen therapy. A pulse oximetry reading of 82%, together with a pulse rate of 130 beat/min, blood pressure 100/50 mm Hg, and a respiratory rate of 22 breaths/min were noted. Neurological examination was unremarkable with a Glasgow coma score of 15, no focal neurology, and normally reactive pupils.

An arterial blood sample was chocolate brown coloured and had a MetHb level of 59.9%, po_2 34.7 kPa, pco_2 3.1 kPa, pH 7.30, HCO_3 11.4 mmol/l, and base excess −13.3 mmol/ l.

She was treated with 1.5 mg/kg of intravenous MB given over five minutes, one litre intravenous isotonic saline over six hours, and high flow oxygen therapy.

Forty minutes after administration of MB the patient's colour was beginning to improve and a repeat arterial blood gas measurement showed MetHb level of 4.8%, po_2 52.7 kPa, pco_2 4.28 kPa, pH 7.32, HCO_3 16.1 mmol/l, base excess −8.5 mmol/l.

Clinically the patient remained well and was admitted overnight for observation. She self discharged the following morning.

CASE REPORT 2

A 28 year old man of Mediterranean origin was brought to the emergency department by ambulance at 03 05 am from a local night club. He had complained of difficulty in breathing and chest pain. On arrival he was agitated and uncooperative. He admitted to drinking six units of alcohol but denied other substance misuse.

On examination he had a navy blue discoloration of his skin, particularly around his face. His Glasgow coma score was 14 (confusion) and his pupils were mid-size and normally reactive. He had a tachycardia of 140 beat/min, a blood pressure of 80/40 mm Hg, and a respiratory rate of 30 breaths/min. His chest was clear and pulse oximetry registered 74% on 15 l/min high flow O_2 therapy.

An arterial blood sample was chocolate brown coloured and had a MetHb level of 63.3%, po_2 9.78 kPa, pco_2 2.29 kPa, pH 7.202, HCO_3 6.6 mmol/l, and base excess of −19.4 mmol/l.

Further treatment was started with 2 mg/kg of intravenous MB over a period of five minutes and one litre isotonic saline immediately. High flow oxygen therapy was continued. Within 10 minutes the patient had improved systematically and a further five minutes later the patient was no longer cyanosed. A repeat arterial blood gas sample showed MetHb level of 1.4%, po_2 30.61 kPa, pco_2 3.68 kPa, pH 7.369, HCO_3 15.6 mmol/l, base excess −8.5 mmol/l.

The patient remained well overnight and before discharge admitted he had inhaled a bottle of amyl nitrite while in the night club.

Table 1 Clinical effects of methaemoglobinaemia

% MetHb	Clinical effects
0–15	Clinical effects unlikely
15–30	Mild: Cyanosis (tongue, lips, earlobe), fatigue, dizziness, headache
30–50	Moderate: Weakness tachycardia, tachypnoea, mild dyspnoea
50–70	Severe: Stupor, coma, convulsions, respiratory depression, cardiac dysrhythmias, acidosis
>70	Potentially fatal

DISCUSSION

Haemoglobin is oxidised from the ferrous (Fe^{2+}) to ferric (Fe^{3+}) form. The ferric form is known as MetHb and is incapable of transporting oxygen. In a healthy adult the concentration of MetHb is 1%–2%. Increased levels of MetHb lead to tissue hypoxia and can be fatal. Two enzyme systems within the erythrocyte are responsible for reducing MetHb back to Hb. These are the cytochrome-b_5-MetHb reductase system and reduced nicotinamide adenine dinucleotide phosphate (NADPH)-MetHb reductase.[3 4]

Exposure to nitrites and nitrate compounds (for example, amyl nitrite, glyceryl trinitrite) is the commonest cause of acquired methaemoglobinaemia.[5] Fatal methaemoglobinaemia secondary to ingestion of nitrite contaminated well water was first reported in an infant.[6] Other causes of methaemoglobinaemia include local anaesthetics (for example, benzocaine, prilocaine, lignocaine (lidocaine)),[7] aniline dyes,[8] sulphonamides, dapsone and quinones.[9]

Although the response to varying levels of MetHb differs from one person to another, the typical effects from increased concentrations of MetHb are shown in table 1.[10]

Immediate treatment entails attention to the airway, prevention of further substance absorption, and reduction of

Abbreviations: MB, methylene blue; MetHb, methaemoglobin

the ferric form of haemoglobin with intravenous MB.[11] Treatment with MB is advised when the MetHb level is >30%–40% but each case must be treated individually on clinical grounds and symptoms. The recommended dose is 1–2 mg/kg given intravenously over five minutes. The different doses (both within the recommended range) used in our two patients were at the discretion of the attending emergency physician.

MB acts as a substrate for the enzyme NADPH-MetHb reductase. The reduced MB produced by the action of this enzyme in turn reduces MetHb back to haemoglobin. NADPH is a necessary cofactor for the enzyme and is produced using G6PD (from the hexose monophosphate shunt). In people with G6PD or NADPH-MetHb reductase deficiency MB is ineffective and alternative treatments such as exchange transfusion, hyperbaric oxygen, or packed cell transfusion must be used.[4 12]

Pulse oximetry in the presence of methaemoglobinaemia is inaccurate. This device uses light absorbance at two wavelengths (660 nm and 940 nm) to calculate the relative concentration of oxy-haemoglobin and deoxy-haemoglobin. MetHb absorbs more light at both wavelengths than do the other two forms of haemoglobin but has a disproportionately greater absorbance at 660 nm. When MetHb concentration reaches 65% or more of the total haemoblobin concentration, the 660 nm to 940 nm light absorbance ratio approaches 1.27. This generates a (falsely high) SaO_2 reading of 80%, even though the maximum possible value is 35%.[13 14]

Co-oximetery avoids this problem by using spetrophotometric techniques to estimate the oxy-haemoglobin percentage of total haemoglobin concentration in the blood sample. It measures light absorbance at four different wavelengths to calculate relative concentrations of oxy-haemoglobin, deoxy-haemoglobin, carboxy-haemoglobin, and MetHb.[15]

Arterial blood gas analysis can also be misleading. Values obtained are a measure of the dissolved oxygen in the sample and not of the oxygen bound to haemoglobin. Calculations of oxygen saturation are based on the assumption that all haemoglobin present has the capacity to carry oxygen. Thus in the presence of a high MetHb concentration, calculated po_2 levels will be an overestimation and may mask severe tissue hypoxia.[16]

The use of volatile nitrites as drugs of misuse make them a possible cause of methaemoglobinaemia presenting to the emergency department. These are known on the street as "poppers". The composition of the liquid varies including amyl, butyl and isobutyl nitrite. A high level of awareness and index of suspicion is required to diagnose the condition and successfully reverse potentially fatal sequelae associated with its misuse.

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Avulsion of the triceps tendon

C Rajasekhar, T K Kakarlapudi, M S Bhamra

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Avulsion of the triceps tendon is the least common of all tendon injuries.[1] In a review of 1014 tendon ruptures over a nine year period by Anzel *et al*,[2] 2% constituted the triceps tendon.The rupture could be partial or complete with or without associated fractures. The usual mechanism of injury is fall onto an outstretched hand but can occur after direct contact injuries. Although ruptures at the musculotendinous junction have been reported, the commonest location is the osseo-tendinous insertion. We report a case of triceps avulsion in a 42 year old heavy manual worker treated by open surgical repair.

CASE REPORT

A 42 year old man presented to the accident and emergency department with pain in his left elbow when he landed awkwardly on it while carrying a barrow of soil up a plank, three feet high and slipped. Clinical examination revealed diffuse swelling and tenderness in the region of the left elbow. A definite gap was palpable just above the olecranon and weakness of arm extension was clearly evident. Lateral radiograph of the elbow showed a "flake" fracture of the olecranon. A diagnosis of complete rupture of the triceps was made. Through a posterior midline incision, the area of rupture was

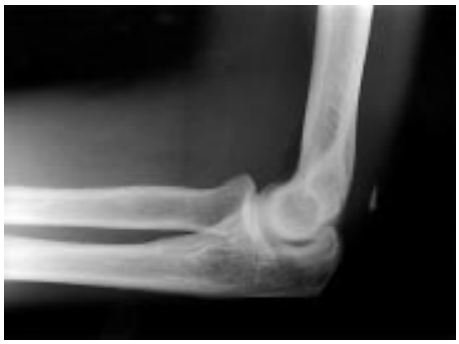

Figure 1 Lateral radiograph of the injured elbow showing the "flake" fracture avulsed from the olecranon.

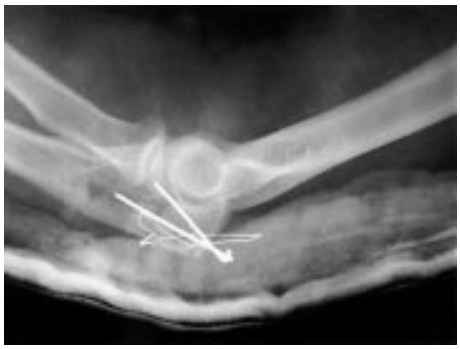

Figure 2 Lateral radiograph of the elbow showing the reattachment of the avulsed tendon using k-wires and circlage wire.

exposed and the flake of bone with the triceps tendon was reattached using two k-wires reinforced with a circlage wire. Postoperatively the arm was immobilised in a back slab at 80 degrees for four weeks after which active flexion was commenced. Extension was permitted after a period of eight weeks. The k-wires had to be removed at three months after the operation. One year after the operation he has full range of movement of the elbow with complete recovery of the triceps power.

DISCUSSION

Being comparatively uncommon, triceps injuries are frequently missed in a normal accident and emergency setting. Triceps avulsion should be suspected in patients presenting with pain and swelling about the elbow after trauma. It usually follows indirect trauma but can be seen after a direct blow or fall on the elbow. Injury to the triceps can also be sustained in a variety of sports including weight lifters and body builders. It has also been described in patients with hyperparathyroidism and in haemodialysed patients with renal failure.[3]

Clinical examination will reveal swelling and a palpable gap proximal to the olecranon. Significant loss of range of motion of extension and strength usually suggests a complete rupture. This may be difficult to elicit because of the pain, swelling, and muscle spasm.

Roentgenographic examination usually reveals a "flake" fracture, which is an avulsion fracture of the olecranon (fig 1). Careful inspection of the radiographs and if necessary oblique views of the elbow should be requested to rule out other fractures. Levy *et al*,[4 5] described radial head fractures associated with triceps ruptures in two reviews. Ultrasound examination or magnetic resonance imaging may be needed if the diagnosis is uncertain.

Complete avulsion rupture of the triceps needs surgical exploration and repair. Reattachment of the triceps tendon to the olecranon via drill holes within the olecranon is usually successful. If the avulsed flake of bone is of reasonable size fixation may be attempted as in our case (fig 2). Neglected ruptures and ruptures at the musculotendinous junction will require more extensive procedures including V-Y advancement and tendon gafting.[6]

Avulsion of the triceps tendon is a rare injury. It can occur after direct or indirect trauma and is usually at the osseo-tendinous junction. A high index of suspicion, physical examination for a palpable gap and "flake" fracture on lateral radiograph will aid in diagnosis. Surgical repair will usually yield excellent results.

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Spontaneous haemopneumothorax: are guidelines overdue?

S R Hart, C Willis, A Thorn, L Barfoot

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Spontaneous life threatening haemopneumothorax is an unusual but treatable cause of unexpected circulatory collapse in young patients. Two case histories are presented to illustrate the management of this condition. Diagnosis and initial management depends on early recognition of the clinical pattern by accident and emergency (A&E) staff and/or hospital physicians. Problems may arise for two reasons. Firstly, as the incidence of life threatening spontaneous haemopneumothorax is low, admitting medical staff may not have experienced this condition in the absence of trauma. Secondly, unlike surgeons, staff in these specialties are unlikely to have received training of either traumatic or spontaneous haemopneumothorax. The cases illustrate potential problems. Not only early recognition of the clinical pattern but also proactive intervention in the A&E department are necessary before referral to a cardiothoracic surgeon. Furthermore, we suggest treatment would be improved by the introduction of management guidelines.

Historically, a haemothorax complicates 2% to 5% of spontaneous pneumothoraces[1 2] and may be life threatening. Spontaneous haemopneumothorax was first described in 1876[3] and fatal cases were reported at the beginning of the last century.[4 5] Non-traumatic haemopneumothorax is 30 times more common in men than women; a gender difference much larger than for spontaneous pneumothorax.[6] Bleeding most commonly results from a torn adhesion between parietal and visceral pleura or rupture of a vascularised bulla.[7] When blood loss has been substantial, early placement of an intrapleural chest drain is necessary and thoracotomy may be required to achieve haemostasis.[8 9]

Prompt diagnosis is essential but may not be readily apparent on first presentation. Problems associated with delayed diagnosis and medical treatment are exemplified by two patients who presented to the A&E department with life threatening features.

CASE REPORTS

Case 1

A 28 year old previously well man presented to the A&E department with a 15 hour history of upper abdominal pain exacerbated by movement. Examination was unremarkable except for a tender epigastrium. Haemoglobin was 12.3 g/dl, white cell count 15.4, electrolytes, liver function tests and amylase were normal. Gastritis was diagnosed but Gaviscon was ineffective. Four hours later the pain had begun radiating to the right anterior chest. During observation in the A&E department, he was sweating, looked very unwell and had a tachycardia of 120 beats/minute with a blood pressure of 130/70 mm Hg. Four units of blood were cross matched. Epigastric tenderness with rebound were described. Blood glucose concentration was 26.6 mmol/l. A sliding scale of insulin was started with an infusion of one litre of normal saline over eight hours. An arterial blood gas sample showed pH 7.28, p_{O_2} 31.3 KPa, p_{CO_2} 6.1 KPa, HCO_3 21.6 mmol/l while inhaling oxygen at 5 l/min. A surgical opinion was then sought and having inspected a chest radiograph, a right lower lobe pneumonia was diagnosed and antibiotics were started.

Seven hours after presentation the A&E staff requested a medical opinion, which revealed breathlessness, chest pain, and indigestion. On examination, he looked very unwell and was sweating. The blood pressure was 140/50 mm Hg but suddenly fell becoming unrecordable. Tachycardia had increased to 140 beats/minute and respiratory rate was 36 per minute, although there was no other sign consistent with pulmonary embolism. The trachea was deviated to the left with a dull percussion note at the right base. The chest radiograph was reviewed and now judged to show a right haemopneumothorax with mediastinal deviation to the left.

A large bore cannula was inserted into the right pleural space for aspiration of 1500 ml of blood after which the blood pressure recovered transiently to 180/90 mm Hg. Central venous access showed the CVP was −8 cm. So far, the patient had received normal saline at a rate of one litre per eight hours. A blood transfusion was started and although three units of blood were given rapidly, the blood pressure fell again to 97/50 mm Hg. While still in the A&E department, a cardiothoracic surgeon was called and inserted an intercostal drain, which revealed a further 1700 ml of blood. The patient was transferred to the intensive care unit with a blood pressure of 120/80 mm Hg and a second haemoglobin was 8.3 g/dl. A further eight units of cross matched blood were titrated to maintain a CVP of +2 cm and a systolic blood pressure above 120 mm Hg. Five hours after admission to the intensive care unit (ICU), an additional three litres of blood had been collected via the chest drain. The blood pressure then fell suddenly and the chest drain was temporarily clamped but was released before surgical intervention. The patient had received 12 units of blood preoperatively. Nine hours after admission to ICU, a right thoracotomy revealed two litres of clotted blood and a small spurting artery in an apical parietal pleural adhesion.

Case 2

A 36 year old man presented to the A&E department with a two day history of sudden right sided chest pain and breathlessness. One month before, he had a minor car accident with head and chest bruising. These injuries had not required hospital treatment.

On examination, he appeared very unwell, sweating, with a heart rate of 130 beats/minute and a blood pressure of 80/50 mm Hg. His respiratory rate was 12 per minute and there was reduced respiratory excursion on the right side of the chest, which was dull to percussion. A chest radiograph was judged to indicate a right haemopneumothorax (see fig 1).

He was referred to the medical team and given two litres of Hartmann's solution intravenously and one litre of air was aspirated from the second intercostal space through a venflon. Six units of blood were cross matched. Arterial blood gas analysis while breathing air showed a p_{O_2} 9.7 KPa, p_{CO_2} 6.4 KPa, pH 7.32, HCO_3 24.7 mmol/l.

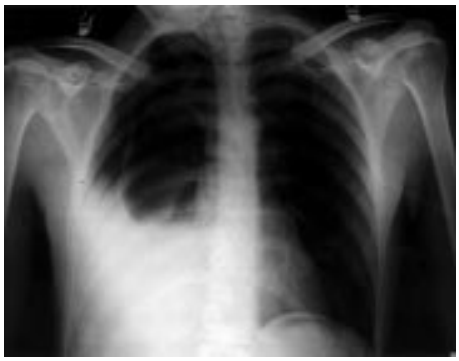

Figure 1 Chest radiograph of a right spontaneous haemopneumothorax.

A drain was inserted into the intrapleural cavity, which rapidly drained air and two litres of blood. This drain was then clamped. Haemoglobin was 12.7 g/dl, white cell count 15.7, platelets 193. Three hours later, the blood pressure was 111/50 mm Hg, heart rate 60 beats/minute and the drain was swinging having been unclamped.

The following day the blood pressure was 100/50 mm Hg and the drain was clamped for two hours but discharged 500 ml of blood when re-opened. A cardiothoracic surgeon advised against further clamping of the drain before performing an urgent thoracotomy to remove a large clot from the right pleural cavity. No active bleeding was observed but apical bullae were stapled.

Both the patients were discharged one week after surgery and had made a full recovery six weeks later.

DISCUSSION

There are no guidelines for management of spontaneous haemopneumothorax. When traumatic haemopneumothorax becomes life threatening because of substantial bleeding, early insertion of two large bore chest drains to evacuate the accumulated blood and air is recommended.[9] This often permits re-expansion of the lung resulting in haemostasis by tamponade of bleeding vessels in apposition to the parietal pleura.

In our first case, diagnostic delay resulted in near fatal circulatory collapse, which could have been avoided by an early educated appraisal of the chest radiograph. In the absence of trauma, this condition may not be immediately recognised by emergency staff or by surgeons, neither of whom may be familiar with spontaneous haemopneumothorax. Physicians are likely to be involved in management of these patients but may not have knowledge of the guidelines for traumatic haemopneumothorax or have had experience or training for spontaneous haemopneumothorax.

In case 1, evacuation of blood to relieve intrathoracic pressure permitted the rapid restoration of blood pressure before the circulating volume had been restored by transfusion. This suggested that tension was the life threatening factor although circulatory failure was exacerbated by hypovolaemia. Both patients were acidotic; in case 2 this was respiratory and in case 1 both metabolic and respiratory.

Subsequent clamping of chest drains to cause tamponade of a vessel by the haemothorax is controversial. Currently it is not widely recommended (personal communication). When bleeding continues, survival will depend on early surgical haemostasis during open or laparoscopic thoracotomy. Indications for urgent thoracotomy have been clearly defined for a traumatic haemothorax and include: aspiration of more than 1.5 litres of blood on insertion of a chest drain and continued loss of more than 200 ml/h according to Advanced Trauma Life Support guidelines.[10] Consensus is less clear for spontaneous haemopneumothorax. We suggest these indications for urgent thoracotomy should form the basis of guidelines for spontaneous haemopneumothorax.

In conclusion, diagnosis of a spontaneous haemopneumothorax depends on recognising the clinical pattern of sudden chest pain, dyspnoea, shock, and clinical chest signs. Successful treatment of a large spontaneous haemopneumothorax depends on early recognition, proactive intervention, and early consideration by a cardiothoracic surgeon. We also suggest treatment would be improved by the establishment of guidelines similar to those for traumatic haemopneumothorax.

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A rare case of a pedunculated lipoma in the pharynx

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Emerg Med J 2002;**19**:275

While lipomas on the trunk and limbs are common, they are rare in the upper aerodigestive tract. A case is reported of an 18 cm long pedunculated lipoma arising from the hypopharynx in a 73 year old man. The tumour was asymptomatic until it appeared in the mouth of the patient after a coughing episode.

A fit and healthy 73 year old man presented to the accident and emergency department with a pendulous mass protruding from his mouth (fig 1). The mass appeared after a coughing episode during a meal. Until this time the patient was asymptomatic with no history of airway obstruction, throat discomfort or dysphagia. The patient was referred to the on call ENT team.

Examination of the oral cavity and throat revealed a long pendulous mobile mass arising from the posterior hypopharyngeal wall. It was removed transorally under general anaesthesia.

Macroscopically, the elongated piece of tissue was 18 cm long and 1 cm wide at it greatest diameter at the base. It was covered with mildly inflamed hyperplastic squamous mucosa with the centre consisting of mature adipose cells and a vascular mesh work. The overall appearance of the mass was consistent with a pedunculated lipoma.

DISCUSSION

Lipomas are benign slow growing neoplasms composed of mature white fat cells.[1] The discovery of a lipoma in the subcutaneous tissue of the body does not usually evoke much interest, except when it is large enough to cause a cosmetic problem or interfere with function as a result of its anatomical position. While 13% of lipomas occur in the head and neck region, only rarely do they occur in the pharynx.[2]

In our case it is most intriguing that a space occupying lesion arising from the hypopharynx can grow to a length of 18 cm without causing any specific symptoms. In the presence of such a long pharyngeal mass one would expect at least some degree of dysphagia, throat discomfort or occasional respiratory embarrassment associated with coughing episodes if the tumour became lodged in the upper airway. The first reported case of an elongated epiglottic lipoma resulted in multiple choking episodes before the death of the patient.[3] In 1952 Penfold documented a case of a fatal laryngeal obstruction secondary to a lipoma attached to the posterior cricoid area.[4] More recently a lipoma arising from the left aryepiglottic fold and flopping into the laryngeal inlet was thought to be responsible for a sudden fatal respiratory arrest.[5] It is fortunate that our patient did not experience any upper airway obstruction during his coughing episode.

Figure 1 The lipoma protruding from the mouth (patient lying supine on operating table with a gag in the mouth).

From the size of the tumour and well known slow growth rate of a lipoma, it is our conjecture that it was present for a considerable period of time before diagnosis. We did not immediately consider lipoma in the differential diagnosis of the mass because lipomas tend to be soft, spherical, and cystic and this lesion was firm and pendulous. Furthermore, lipomas occur most frequently where fat cells are abundant and the pharynx is a muscular tube with very little fatty tissue. So although lipomas in the pharynx are rare, this case highlights the need to consider it in the differential diagnosis of throat masses. This case also illustrates that a lipoma of the pharynx can reach a considerable size without causing any symptoms until it appears in the mouth. The first port of call for such patient is usually the accident and emergency department.

This case illustrates a rare addition to our differential of oral cavity masses. We recommend immediate referral to the ENT team and complete surgical excision as soon as possible to prevent serious complications such as airway obstruction and death.

Contributors

Ricardo Persaud reviewed the literature and wrote the paper. Rohit Kotnis contributed to the discussion of core ideas and to the revision of the penultimate version. Chun Ong admitted and operated on the patient. David Bowdler initiated the idea to write up the paper and was the consultant responsible for the patient and overall supervision. Ricardo Persaud is the guarantor.

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