

CASE REPORTS

Effect of inappropriate naltrexone use in a heroin misuser

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Naltrexone is a long acting opioid receptor antagonist used in controlled opioid withdrawal drug programmes. When taken by an opioid dependent patient an acute withdrawal reaction will be precipitated. The case is presented where a known opioid drug misuser inadvertently ingested naltrexone in conjunction with heroin resulting in severe agitation, requiring heavy sedation followed by general anaesthesia to enable investigation and management of his clinical condition.

Naltrexone is a long acting opioid receptor antagonist used in drug rehabilitation programmes to maintain opioid abstinence. However, when consumed in conjunction with an opioid substance, prolonged opioid withdrawal will be precipitated resulting in unpredictable and life threatening medical consequences. We present a case where a known drug misuser consumed naltrexone in conjunction with heroin.

CASE REPORT

A 39 year old man presented to the accident and emergency department having taken up to three, 50 mg tablets of naltrexone and having smoked an unknown quantity of heroin. He was known to be an injecting drug user and to suffer from epilepsy. No other recreational drugs, alcohol, or prescribed medications were known to have been consumed. On arrival he was extremely agitated being restrained by four police officers. He was confused, sweating, with episodes of profuse projectile diarrhoea and vomiting. Glasgow Coma Scale was 12 (spontaneous eye opening, localising to pain, and using inappropriate speech). Pupils were dilated but reactive to light. Heart rate was regular at 180 beats/minute and respiratory rate 40 breaths/minute. Blood pressure, oxygen saturation, blood glucose, and temperature were normal. There was no evidence of head injury and no history of seizure. Urea, electrolytes, full blood count, and arterial blood gas measurements were normal. Initial attempts at sedation using a combination of titrated intravenous midazolam and droperidol were unsuccessful. After receiving a total of 20 mg midazolam and 15 mg droperidol he continued to be confused, agitated, and increasingly violent. An urgent CT head scan was arranged to exclude any intracranial pathology. To expedite this he was anaesthetised and ventilated. Rapid sequence induction of anaesthesia was carried out using 200 mg propofol, and 100 mg suxamethonium. Anaesthesia was maintained with a propofol infusion and incremental paralysis with atracurium.

CT of his brain was normal. A lumbar puncture was performed while the patient was still anaesthetised. This showed no abnormality. The patient was extubated four hours after induction and transferred to the medical high dependency unit for observation. Further episodes of agitation

Box 1 Side effects of naltrexone use

- Nausea
- Vomiting
- Anxiety
- Headache
- Sleep disturbance
- Lacrimation
- Diarrhoea
- Sweating
- Muscle/joint pains

occurred overnight requiring additional sedation with intravenous midazolam. The following morning he took his own discharge. Retrospectively urine toxicology screen confirmed the presence of cannabinoids, benzodiazepines, and opioids.

DISCUSSION

Naltrexone is a comparatively new medication used in drug rehabilitation programmes to maintain abstinence from heroin and methadone and prevent relapse in former addicts. Naltrexone is a competitive opioid receptor antagonist acting at the μ and κ opioid receptors by blocking the euphoric effects of exogenous administered opioids. Naltrexone use is restricted to specialist clinics and is initially given orally in doses of 25 mg daily, increasing to 50 mg, with courses of treatment lasting many months. The total weekly dose may be divided and given on three days of the week only to improve patient compliance. Oral absorption of naltrexone is rapid with peak plasma concentrations occurring after three hours and remains metabolically active for 24–72 hours, however, the precise pharmacodynamics are not completely understood and large differences in serum concentrations of the drug are thought to reflect variable first pass mechanism.¹ Side effects of naltrexone use are outlined in box 1.

Before being given naltrexone, patients are required to be opioid free for a period of 7–10 days and undergo a supervised naloxone challenge before being accepted into a controlled detoxification programme. Although the effects of the receptor block are surmountable, addicts are cautioned that attempts would require large amounts of opioids, which may lead to a fatal overdose.² Naltrexone has also been administered to addicts, either alone or in combination with clonidine, under heavy sedation or general anaesthesia, a process known as ultra rapid opioid detoxification, in an attempt to reduce the immediate symptoms of acute opioid withdrawal and begin a maintenance oral naltrexone programme earlier.³ Recent studies have highlighted limited success using naltrexone in the treatment of longstanding alcoholism by reducing the alcohol craving in this group.^{4,5}

Accidental or intentional ingestion of naltrexone in opioid dependent people will result in an acute block of opioid receptors and precipitate a severe opioid withdrawal reaction.

Box 2 Management of naltrexone precipitated acute opioid withdrawal

- Sedation (benzodiazepines)
- Antiemetics (metoclopramide)
- Intravenous fluids
- Non-opioid analgesia (non-steroidal preparations)
- May require antispasmodic agents (hyoscine)
- May require general anaesthesia

Symptoms of withdrawal can appear after only five minutes following ingestion and may last up to 48 hours. Symptoms include confusion, agitation, hallucinations, sweating, tachycardia, abdominal pain, and episodes of profuse vomiting and/or diarrhoea, which may result in significant fluid losses. Management is supportive with sedation (benzodiazepines), antiemetics (metoclopramide), intravenous fluids, and non-opioid analgesia (non-steroidal preparations). Antispasmodic agents (hyoscine) may be required for intestinal cramps. Opioid administration has no effect and is potentially dangerous. Greater doses of opioids would be required to reverse the receptor block and the resulting respiratory depression may be deeper and more prolonged. Patients may become extremely agitated and possibly violent requiring restraint, the administration of heavy sedation, and possibly general anaesthesia (see box 2).

The problem of acute opioid withdrawal precipitated by naltrexone appears to be an increasing problem for physicians. Two case reports have been published in the literature from Italy in 1999, where an injecting heroin user⁶ and an ex-heroin addict receiving methadone treatment⁷ both consumed naltrexone. In each case, despite repeated attempts at sedation, both patients exhibited increasing agitation and delirium requiring to be anaesthetised with propofol, intubated, and ventilated. In each case the patients recovered with no adverse effects. More recently concern regarding this presentation has been voiced in Australia.⁸ In this instance drug addicts had inadvertently injected naltrexone intravenously after having been sold the preparation incorrectly as heroin by unscrupulous drug dealers. In both cases described the addicts presented in an acute state of opioid withdrawal requiring the administration of intravenous fluids, antiemetics, antispasmodics for intestinal cramps, and benzodiazepine sedation, however, neither patient required general anaesthesia. One case has been reported in Britain.⁹ In this situation at first it was unclear that naltrexone had been consumed by the drug user and a delay in initial diagnosis resulted. The patient was removed from the A&E department for disruptive behaviour by the police and then brought back in a state of acute opioid withdrawal a few hours later. General anaesthesia was not

required, however, antiemetics and oral diazepam for agitation were given. After observation for 24 hours the patient was discharged with no adverse effects.

Conclusion

The nature, severity, and duration of naltrexone induced acute opioid withdrawal varies greatly between people and the clinical course of events is unpredictable. With the trend for more addicts to be treated with naltrexone in the community, and the possibility that current addicts may see naltrexone as a misguided means to break the cycle of drug dependence, the potential exists for increasing numbers of similar presentations. Physicians involved in the emergency care of these patients must be aware of the dramatic clinical course of the ingestion of naltrexone in opioid misusers and be prepared to manage the complications.

Contributors

Stephen Boyce was involved in the research, overall coordination and writing of the paper. Peter Armstrong identified the case and contributed to the case report and literature search. James Stevenson was involved in the research and writing of the paper. Both Stephen Boyce and James Stevenson will act as guarantors for the paper.

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Life threatening haemorrhage after anterior needle aspiration of pneumothoraces. A role for lateral needle aspiration in emergency decompression of spontaneous pneumothorax

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Needle aspiration is a recognised emergency treatment of spontaneous pneumothorax and in the case of suspected tension is usually performed before chest radiography. Three cases are described of apparent life threatening haemorrhage after anterior aspiration in the second intercostal space, mid-clavicular line (2ICS MCL) requiring resuscitation, and transfer to a cardiothoracic unit. In these patients there was no evidence of haemothorax on initial presentation. Lateral needle aspiration, in the site recommended for chest drain insertion, the 5th intercostal space, anterior axillary line (5ICS ALL) is technically easy and may be a potentially safer option for decompressing pneumothoraces.

Anterior needle aspiration in the 2ICS MCL is the recognised treatment for pneumothoraces using standard ATLS¹ and British Thoracic Society Guidelines (BTS).² The BTS guidelines state that an axillary approach is an alternative to the 2ICS MCL but no precise point is identified. For suspected tension pneumothorax aspiration is usually done before chest radiography as delay could lead to a cardiorespiratory arrest. Anterior decompression is theoretically safe and can be performed rapidly as all necessary equipment is at hand. However, we report three cases of life threatening haemorrhage, which appeared to follow anterior needle aspiration. Initial examination/chest radiography, had shown no evidence of haemothorax. After aspiration chest radiographs showed haemopneumothoraces requiring drain insertion and transfer to our cardiothoracic unit.

CASE 1

A 27 year old man presented with sudden right chest pain and shortness of breath. Chest radiography showed a right tension pneumothorax with no evidence of fluid level. Needle aspiration in the right 2ICS MCL was performed (dressing and puncture wound confirmed site), after this chest radiography showed haemopneumothorax. On formal drain insertion in the 5 ICS ALL a total of two litres of blood was drained. The patient was resuscitated, transfused, and transferred to a cardiothoracic unit for further treatment. He required video assisted thoracoscopy for on going bleeding, and a persistent air leak. At video assisted thoracoscopy blood clot was adherent to the anterior lung surface with a contused area of presumed iatrogenic injury. He made an uneventful recovery.

CASE 2

A 24 year old man presented with right sided chest pain and shortness of breath. Chest radiography confirmed a pneumothorax with no fluid level. Needle aspiration in the 2ICS MCL (puncture wound confirmed site) was performed. Post-aspiration chest radiography showed an improvement of the

pneumothorax and no fluid level. He was discharged after observation, but returned the next day with increasing pain and shortness of breath. Chest radiography showed a haemopneumothorax with evidence of an apical collection. The patient had 1.2 litres blood loss after insertion of a chest drain requiring resuscitation, transfusion, and transfer to a cardiothoracic unit. The bleeding resolved and he was managed conservatively with good outcome.

CASE 3

A 27 year old man presented with left sided chest pain and shortness of breath. Chest radiography confirmed a tension pneumothorax with no fluid collection. Aspiration in 2ICS MCL followed by chest radiography, suggested a fluid collection. Chest drain insertion was followed by 1.5 litres bleeding and he was resuscitated, transfused, and transferred to our cardiothoracic unit. Emergency thoracotomy was performed for ongoing bleeding and haemodynamic instability (consultant preference). A large quantity of clot was evacuated and apical pleurectomy performed. The patient made an uneventful recovery.

DISCUSSION

Most pneumothoraces are uncomplicated by cardiorespiratory compromise and symptomatic patients can be managed by aspiration or intercostal drains depending on the size of the pneumothorax. Success rates for aspiration vary from 68%³ to 75%.⁴ Emergency decompression in the 2ICS MCL can be life saving in tension pneumothorax and although quick and reputedly safe most are performed in the accident and emergency department by comparatively junior doctors who may have limited experience. This may be exacerbated by the urgency of the clinical picture. Data on the incidence of needle aspiration in spontaneous pneumothorax and its complications are limited. In a study of 6241 major trauma patients with suspected pneumothoraces 108 (1.7%) underwent needle thoracostomy with two iatrogenic pneumothoraces reported and no other complications.⁵ Variation in chest wall thickness, insufficient cannula length, and inexperience in performing aspirations contribute to inadequate needle aspirations, and may increase the risk of complications.^{6,7} The internal mammary artery and branches medially and the sub-clavian vessels superiorly are closest to the 2ICS MCL. Intercostal vessels may also be damaged.

The cases presented occurred over a six month period and drew our attention to the possibility that the proximity of the 2ICS MCL approach to the above vascular structures could result in injury. The aspiration sites were all identifiable by the puncture wounds and all corresponded to the 2ICS MCL. These patients all required resuscitation and two required surgery. Although it is impossible to conclude that the haemorrhage was caused by the anterior aspiration it is a clear possibility because of the lack of evidence for haemothorax as

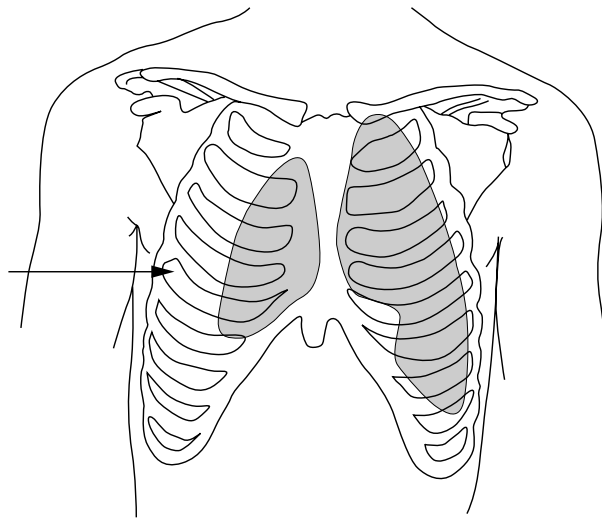


Figure 1 Arrow showing location for needle thoracocentesis in the 5th intercostal space, anterior axillary line.

part of the initial presentation. Other considerations developed the theory of potential danger with the anterior approach. After lung collapse vascular adhesions may bleed freely into the pleural space,⁸ which in the volumes involved in our three patients would be evident on percussion or on chest radiography. Initial chest radiographs all showed pneumothoraces with no fluid levels, which suggest that bleeding occurred at a time, separate from the pneumothorax. In the two cases that proceeded to surgery no bleeding vascular adhesions were seen. Unfortunately surgery could not confirm which vascular structure had been damaged; however, this is often notoriously difficult unless there is active bleeding. In all three cases the blood loss was substantial requiring active resuscitation. In the second patient whose haemothorax was confirmed the following day, we were concerned that the apical cap on the chest radiograph represented a haematoma from iatrogenic injury to the subclavian vessels.

For chest drain insertion ATLS guidelines clearly recommend an approach from the 5th intercostal space, anterior axillary line (5ICS ALL) as drainage of both air and fluid is possible, and the entry point is high enough to minimise the chance of abdominal injury. In the BTS guidelines an axillary

approach is also suggested although no precise point is identified. With most significant and especially with tension pneumothoraces, radiography shows a moderate or large lateral air space with the lung collapsed down towards the hilum (fig 1). The fact that these patients are often lying supine may suggest this procedure may be more suitable for patients with large pneumothoraces. Lateral aspiration in 5ICS ALL, is technically easy and may provide a safer option for decompressing spontaneous pneumothoraces, as it avoids major vessels and the lung that lie closer to the anterior chest wall.

Contributors

Randolph Rawlins initiated the reporting of this finding and was responsible for intellectual input. He is the corresponding author and is guarantor for the information in this paper. Cornelia S Carr was responsible for intellectual input, editing, and writing the final draft. She reviewed the literature and references. Karen M Brown was responsible for producing the line diagram showing the site for aspiration as well as reformatting, referencing, and resubmitting the article after the first reviewers comments. C R Cameron is the senior author and consultant thoracic surgeon. He was responsible for overseeing this paper, advising and making recommendations for the final paper.

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Perforation of the oesophagus and aorta after eating fish: an unusual cause of chest pain

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This report describes perforation of the oesophagus after eating fish complicated by perforation of the aorta six days later. The patient had not knowingly swallowed a fish bone. Aorto-oesophageal fistula is almost universally fatal. In the case described here, the fistula was demonstrated on contrast computed tomography before surgery, thus informing surgical management. The patient is the eighth reported survivor.

In a series of 2394 cases of retained oesophageal foreign body reported from Hong Kong, perforation occurred in 25 cases (1%) and aorto-oesophageal fistulas in two cases (0.08%).¹ A wide variety of objects was retained in the oesophagus but fish bones were the most common (60%) and chicken bones the second most common (16%). Fish and chicken bones seem to be most commonly associated with major complications—particularly in parts of the world where unfiletted fish is eaten—but other foreign bodies, for example coins, have perforated the oesophagus² and fatal oesophago-aortic perforation by a coin has been described in a child of three.³ The diagnosis is frequently missed at initial presentation, as in the case reported here.

CASE REPORT

A 57 year old woman attended the emergency department with a two hour history of dull central chest pain that radiated into her back. It had begun while eating fish (sea bass), although she had not knowingly swallowed a fish bone. There were no other symptoms and she was normally in good health. Examination and investigations (chest radiography, ECG, full blood count, and biochemistry screen) were thought to be normal. Her pain subsided apart from some discomfort on swallowing and she was discharged home. She reattended the department six days later. She complained that she had been cycling up a hill and had developed severe chest pain radiating into her jaw together with some sweating. Moreover, the discomfort of which she had previously complained had persisted. On examination she had a pulse of 92 per minute, BP 142/72 mm Hg, SaO₂ 98% on air and temperature 36.5°C. There were no cardiovascular, respiratory, or abdominal signs. There was no surgical emphysema in the supraclavicular fossae. The investigations were repeated and she now had a raised white cell count (16.3×10⁹/l with a neutrophilia), a somewhat lower haemoglobin concentration (12.7 g/dl previously 14.4 g/dl) and an increased C reactive protein concentration (46 mg/l previously <8 mg/l). The ECG was normal. Review of her first chest radiograph showed a probable pneumomediastinum. An oesophageal perforation and mediastinitis were diagnosed. Accordingly, she was given analgesia and high dose intravenous antibiotics. She was admitted but had a respiratory arrest a few hours later necessitating intubation and ventilation. There was extreme difficulty in inserting the endotracheal tube; subsequent computed tomography showed a large posterior mediastinal abscess displacing and compressing the proximal trachea. A dynamic, enhanced scan (100 ml Omnipaque 300 intra-

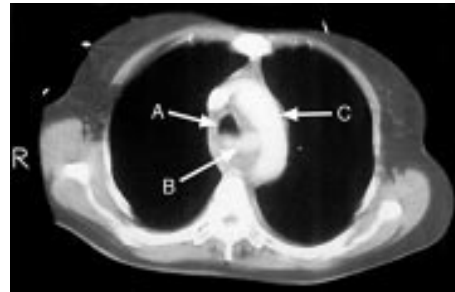


Figure 1 Computed tomography of the superior mediastinum: a large posterior mediastinal abscess displaces the trachea (A) to the right and contains free contrast (B) adjacent to the arch of the aorta (C).

venous) demonstrated pooling of contrast within the abscess cavity, closely adjacent to the aortic arch (fig 1). It was assumed that there was an aortic fistula. She was transferred to a regional centre with cardiopulmonary bypass available. She survived; the successful surgical management of her aorto-oesophageal fistula will be the subject of a separate publication (N Maynard, personal communication).

DISCUSSION

Perforation of the oesophagus and aorta by foreign bodies has been reported surprisingly often and is almost universally fatal. For example, fatal perforation of the oesophagus and aorta by a fish bone was described by Scher *et al*⁴ and these authors cited a further 86 cases of fatal aorto-oesophageal fistula. Fish bone ingestion has caused a subclavian-oesophageal fistula⁵ and has also caused perforation of the pericardium with cardiac tamponade.⁶ There is a tendency for fish bones to migrate and one has been found in the thyroid after perforation of the cervical oesophagus,⁷ and others in the liver after gastric or gastrointestinal perforation.^{8,9} Foreign bodies most commonly perforate the cervical oesophagus.¹ The second most common site for perforation is at the level of the aortic arch¹ where there is scope for fatal or life threatening vascular and respiratory catastrophe—as in the case of a 38 year old man who unknowingly swallowed part of a cocktail stick, which perforated his oesophagus and aorta and caused a catastrophic haematemesis 10 days later.¹⁰ When there is a delay between ingestion and presentation the aortic perforation may be mycotic (that is, it follows damage to the aortic wall caused by adjacent sepsis), as is probable in the case reported here. The first survivor of oesophago-aortic perforation was described in 1978 after cross clamping the aorta¹¹ and currently there are only six other survivors listed on Medline. Our own case illustrates the typically subtle initial presentation of this potentially fatal condition. Characteristically, a fatal, exsanguinating haematemesis is preceded by a minor sentinel bleed a few days earlier and mild oesophageal pain (chest pain possibly radiating to the back or root of the neck, and worse on swallowing).^{4,11} Therefore any patient with a presentation that is suggestive of a perforating oesophageal foreign body should be investigated urgently. Once a perforation has been confirmed, a dynamic contrast computed

tomogram or arch aortogram is essential to exclude vascular involvement. Any patient with an aortic fistula should be referred to a centre where cardiopulmonary bypass is available.

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Contributors

All authors had clinical responsibility for the patient and contributed to the content of the paper. The manuscript was written by Horace D'Costa, Grizelda George, and Bryan Todd.

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Chlamydia psittaci pneumonia presenting as acute generalised peritonism

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A 63 year old man presented with the signs of acute generalised peritonism in the presence of a clear chest radiograph. At laparotomy no abnormal findings were noted. Further inquiries revealed a history of recent acquisition of budgerigars, over the following days the chest radiograph developed patchy opacification. Subsequently IgG immunofluorescence confirmed the diagnosis of *Chlamydia psittaci*. The presentation of psittacosis with gastrointestinal features is well recognised. This is believed to be the first account in the literature of a human case of *Chl psittaci* pneumonia presenting with acute generalised peritonism indicating an exploratory laparotomy. It is suggested that *Chl psittaci* pneumonia should be considered in the differential diagnosis of an acute abdomen in the presence of a history of exposure to psittacine birds.

A 63 year old man was referred to the care of a surgical team with a two day history of severe abdominal pain, vomiting, and headache associated with constipation and weight loss over the preceding six months. There was no history of cough or sputum production. He had previously undergone a right sided hernia repair, left sided orchidectomy, and had been diagnosed with oesophagitis 10 years earlier after a gastroscopy. He was taking omeprazole and an alginate preparation. He denied smoking and drank little alcohol.

On admission his temperature was 38.5°C, respirations 32 settling to 18 within two hours, his chest was clear on auscultation, pulse 80 and blood pressure 120/70 mm Hg. Examination of the abdomen revealed rebound tenderness and guarding, bowel sounds were present. Initial investigations included haemoglobin concentration 15.6 g/dl, white cell count 11 100 mm³, and plasma sodium 128 mmol/l. Other electrolytes, aminotransferase activities, and amylase were within normal limits. The chest radiograph was normal, erect abdominal radiograph showed gaseous distension of the small bowel. He was initially treated with broad spectrum intravenous antibiotics but generalised abdominal guarding persisted and a laparotomy was performed 36 hours after admission. No abnormal intra-abdominal findings were noted and no samples were available for microbiological analysis.

Further inquiries revealed that he was employed as a metal-worker; there was no history of foreign travel and no risk factors for immunodeficiency. However, 10 days earlier he had purchased several budgerigars none of which had been noted to be unwell.

On day 5 of his admission he was still complaining of abdominal pain, he had not opened his bowels since the laparotomy; there was no cough or shortness of breath. Respirations were 15, crepitations were noted at the right lung base, and a further chest radiograph demonstrated patchy opacification in the right lower lobe. Ciprofloxacin was added to his treatment.

Over the subsequent three days he became increasingly short of breath and profoundly hypoxic with an arterial blood gas Pao₂

Clinical features of *Chlamydia psittaci* infection localising to the gastrointestinal system

- Diarrhoea^{2,3}
- Vomiting^{2,3}
- Anorexia³
- Abdominal pain³
- Nausea³
- Constipation³
- Hepatitis^{3,6}
- Tender hepatomegaly⁷
- Splenomegaly⁷
- Pancreatitis⁷

of 6.1 kPa on high flow oxygen via a Hudson mask. On day 8 he was transferred to the intensive care unit, and the antibiotics were changed to erythromycin and rifampicin. He was maintained on a ventilator for the following 20 days. He made a full recovery and was discharged 41 days after admission.

Infection with *Chl psittaci* was subsequently confirmed by specific IgG immunofluorescence testing, antibody values rising from <1/32 on day 7 to 1/256 on day 30.

DISCUSSION

Psittacosis is found in psittacine birds including budgerigars and can be transmitted to humans by inhalation or direct contact.¹ The presentation of psittacosis is well known to cause diagnostic difficulties relating to the late or sometimes absent onset of respiratory symptoms.² In the series published by Crosse³ the diagnosis of psittacosis was considered at the time of admission in 20% of cases, late diagnosis being related to a fulminant course and high mortality.^{4,5} The range of clinical features localising to the gastrointestinal system is summarised in the box. However, this is believed to be the first account in the literature of a human case of *Chl psittaci* pneumonia presenting with acute generalised peritonism indicating an exploratory laparotomy although there is a report of peritonism associated with chlamydia in a cat.⁸

In the case reported here the illness was characterised by gastrointestinal tract features. The patient did not complain of cough or sputum production at any time. The initial chest radiograph was normal, a finding reported to occur in 18% of patients.³ Pointers to the correct diagnosis included: hyponatraemia (49% of cases), relative bradycardia (26% of cases), and white cell count less than 11 100/mm³ (71% of cases).³ Contact with birds is reported in between 20%⁹ and 80% of cases,¹⁰ the birds are newly acquired in 15%³ of these. It has been suggested that exposure to psittacine birds is the most valuable diagnostic clue.⁴

Tetracycline is traditionally the drug of choice in treating *Chl psittaci* although erythromycin may be equally effective.¹¹ In addition, quinolones have been used successfully to treat *Chl pneumoniae*.¹²

In summary, *Chl psittaci* pneumonia infection should be considered in the differential diagnosis of peritonism in association with a history of exposure to psittacine birds even in the absence of respiratory features.

Contributors

Dr David Bourne: author and senior SHO on the medical team responsible for the case. Dr C B Summerton, consultant physician and gastroenterologist: medical consultant responsible for the case and comments on manuscript. Dr N Beck, consultant in anaesthetics and intensive medicine: intensive care consultant responsible for the case and comments on the manuscript. Guarantor: Dr David Bourne.

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An after dinner speaker?

C E Payne

Emerg Med J 2003;**20**:389–390

Mutilation of the hand and digits are commonly seen after attempts at home maintenance. Adults, adolescents, and children present to accident departments with sometimes dramatic injuries requiring immediate attention and referral to specialised units. In this case, finger entrapment in the electromagnet of a “club” sized speaker required specific management from the accident and plastic surgery departments and utilisation of the local fire brigade for quick digital release.

A 16 year old adolescent was referred from the accident and emergency department after sustaining attachment of a powerful electromagnet to his right dominant hand. He had attempted, after dinner that evening, to mend a high performance speaker. His fingers became trapped after the material cover of the speaker was removed to gain access to the voice coil while the power was still active. On examination of the right hand, the index, middle, and ring fingers were completely compressed and fixed between the two speaker magnets almost to the distal interphalangeal joints (fig 1). The magnetic gap was only 0.6 cm and the complex weighed an estimated 8.5 kg. Power could not be returned as the father had removed the magnet complex from the speaker before calling the emergency services. Marcaine digital blocks and morphine were administered while waiting for the fire brigade as more powerful equipment was required to remove the magnets. Several attempts were made with power saws to cut through the outer electromagnet with no result. The device was ultimately removed by shattering the brittle outer magnet with two pneumatic “jaws of life” so as to break it way from the inner magnet (fig 2A, B). The total ischaemic time was three hours. On radiological examination he sustained tuft fractures to the distal phalanges of the index and middle fingers and required a local anaesthetic procedure to repair the nail beds of all trapped fingers (fig 3). He suffered no soft tissue loss and was discharged the following day.

DISCUSSION

Mutilation of the hand and digits are commonly seen after attempts at home maintenance. In adults, severe injury is either by direct use or while mending an electrical item. Careless use of home power tools,¹ garden tools,² and kitchen



Figure 1 Fingers trapped in the magnetic gap (0.6 cm).

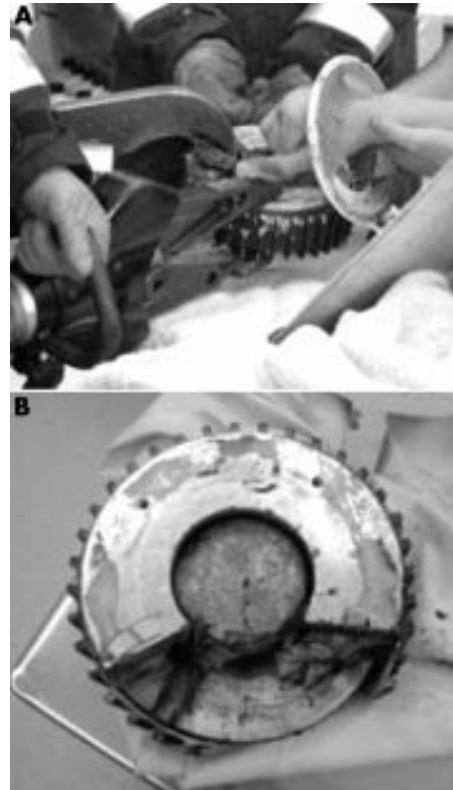


Figure 2 (A) “Jaws of life”. (B) Fractured outer magnet.



Figure 3 Fingers after release.

appliances are well known hazards. In children, it is the inquisitive fingers that are most at risk of major mutilation in the home.^{3,4} Another temptation by many unqualified home owners is to apply basic knowledge of power circuits to fix domestic electrical installations. Electrocutation because of the low voltage, alternating current of the domestic supply can cause minor injury to electrical fatalities.^{5,6} Fixing a speaker

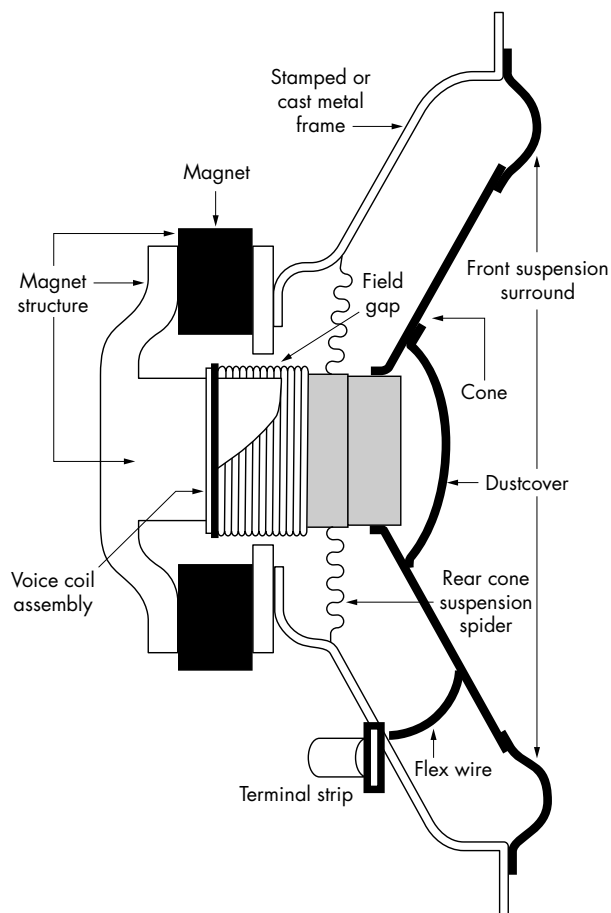


Figure 4 Schematic details of the functional units of a speaker .

highlights two possible dangers; electrocution (which may have been expected here) and the observed crushing effect of the magnetic piston mechanism.

Speakers are air pistons that move back (on the negative cycle of the signal) and forth (on the positive cycle), creating different degrees of air pressure at different frequencies. Alternate positive and negative voltages reach the voice coil rigidly mounted to the cone inside the speaker, creating an electromagnet that will either be repelled, or attracted by the fixed magnet at the bottom of the speaker. The magnets are unyielding and attracted when the power source is discontinued. The voice coil itself is mounted on a rigid cylinder, to

which it is firmly glued. At its natural resting point, the voice coil is centred within a narrow magnetic field gap (0.6 cm). The patient's fingers were pulled into this gap by the piston action of the two magnets and were solidly trapped for three hours (fig 4).

Managing this injury in the accident department required the essential primary care, referral to the hand surgeons, and the necessity of the local fire brigade. The outer magnet is composed of various materials including neodymium, strontium, and barium, among other technologically advanced metal composites, with older units using alnico-5 and other less dense alloys. These are impossible materials to cut even with powerful electric saws, but can shatter if a large enough force is applied across the magnetic ring. The use of the fire brigade equipment was essential and the only way to release the digits from the magnetic field gap.

The piston-like crushing power of these speaker magnets are enormous, they have a substantial attractive force and are impossible to separate once removed from the power source. The immediate objective, in this case, to remove the magnets was attributable to the extreme patient, and family, distress rather than the digital ischaemic time, which can be for several hours. Fortunately the damage sustained to the digits only required a local debridement and distal tip repair, with full function and sensation returning to all finger tips at 10 day follow up.

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Photographs supplied by Mr R Warner.

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Survival from accidental strangulation from a scarf resulting in laryngeal rupture and carotid artery stenosis: the “Isadora Duncan syndrome”. A case report and review of literature

P A Gowens, R J Davenport, J Kerr, R J Sanderson, A K Marsden

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In 1929 the dancer Isadora Duncan died from strangulation and carotid artery insult when her scarf caught in the wheels of a motor vehicle in which she was travelling. As part of the Edinburgh Festival scene, cycle propelled rickshaws are in popular use as short range taxis. The case is presented of a student who sustained a laryngeal rupture from strangulation with a scarf in the same way as Isadora. Despite an out of hospital cardiorespiratory arrest, severe laryngeal trauma, and carotid artery damage resulting in hemiparesis, the patient was successfully resuscitated and recovered with no neurological deficit. It is believed that this is the first recorded survival from this condition.

In the early hours of a June morning in 2001, an ambulance was dispatched to a suspected case of “choking” in a main Edinburgh street. On arrival six minutes later a 21 year old woman was found lying in the recovery position, apparently strangled from her scarf, which had become caught in the wheels of a cycle powered rickshaw. Bystanders had loosened the ligature (scarf) with difficulty but no other first aid measures had been undertaken.

Initial assessment was extremely difficult. Assessment of the airway was virtually impossible on account of gross oedema of the neck and face and massive surgical emphysema. However, the patient was apnoeic and unresponsive to painful stimuli. The neck oedema made it impossible to palpate the carotid pulses but cardiac arrest was presumed from the absence of the other major pulses. Airway control was achieved by jaw thrust; chin lift with a bystander maintaining c-spine support. The patient was ventilated by bag-valve-mask with supplemental oxygen delivered through a reservoir. Tracheal intubation was not possible, as, during laryngoscopy, the normal laryngeal landmarks could not be visualised.

Initial CPR was stopped after a few minutes because, although the pulses remained impalpable, the patient made occasional physical movement and some respiratory effort.

A collar was applied, intravenous access obtained, and the patient was made ready for rapid transport to hospital. Assisted ventilation was continued though there were some irregular spontaneous respirations with marked laryngeal noises heard.

En route to hospital the patient had a seizure followed by a right sided facial weakness.

On arrival at hospital the vital signs were RR18 with stridor, radial pulse rate 104, GCS – E1, M5, V1 and, despite the obvious partial airway obstruction, the initial SPO₂ was recorded as 99%.

There was great difficulty in securing a definitive airway because an attempt at conventional intubation by senior accident and emergency staff and anaesthetic staff failed

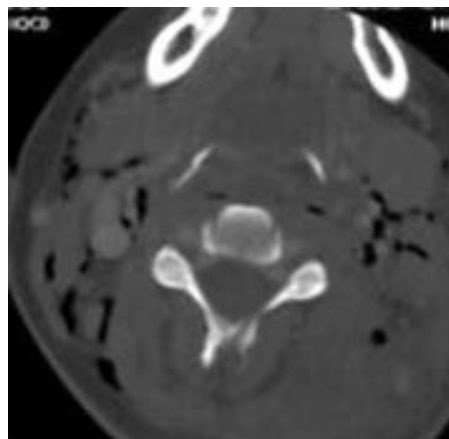


Figure 1 Fractured hyoid bone in accidental strangulation.

because of the gross dissection of the normal laryngeal architecture. These attempts had been supported by the administration of short acting agents, propofol and halothane. Eventually, nearly two hours from onset, a definitive “airway” was obtained by diathermic cricothyroidotomy carried out by a specialist ENT surgeon. The airway was secured with a cuffed tracheostomy tube.

Radiology revealed a hyoid bone fracture consistent with a strangulation injury (fig 1). The cervical spine appeared intact.

With a clinical diagnosis of traumatic carotid artery stenosis, the patient was transferred to the regional neuroscience unit for neurosurgical intensive care. Computed tomography showed no intracranial abnormality. Neither vascular investigation nor therapeutic interventions were required with the hemiparesis resolving spontaneously over the next 12 weeks. The patient was able to be discharged after four months. A full neurological recovery has taken place though the patient has required further re-constructive laryngeal surgery.

LITERATURE REVIEW

The possibility of death from strangulation by a scarf caught in the wheel spokes of a vehicle was brought to the public’s attention when the world famous dancer Isadora Duncan died on 14 September 1929. The long scarf, which she was wearing, became caught in the wire wheels of her Buggati car, stopping the vehicle. Isadora died at the scene and was later found to have sustained a fractured larynx and carotid artery injury.¹

Cycle powered rickshaws (fig 2) remain a common form of transport in some parts of India. However, the unprotected spokes of the cycle wheel can trap the long scarf (chunni) worn by Indian women and a number of cases of accidental strangulation have been described—with no recorded



Figure 2 Cycle powered rickshaw.

survivors.^{2,3} Aggarwal from the Department of Forensic Medicine at Delhi has described a number of common features including the persistence of unconsciousness from the outset with death confirmed soon after arrival at hospital.

Closed injury to the larynx most frequently follows blunt injury to the neck with some “classic” presentations including the two wheeled motorist running into an ambush wire or the unguarded tailgate of a lorry. The clinical features of dysphagia, hoarseness, and dyspnoea are related to the gross oedema and/or the distortion of the laryngeal skeleton including fracture of the hyoid and/or dislocation of the arytenoid cartilages.⁴ Carotid artery injury, also, usually follows blunt trauma and has been described following karate blows to the neck,^{5,6} diving,⁷ therapeutic manipulations,⁸ and assaults. It occasionally requires endarterectomy with the use of stents.

When the traumatic insult is severe such as in strangulation, which is described here, or in hanging, it is not uncommon for the two injuries to coexist and in some cases, the carotid artery damage is bilateral.⁹

DISCUSSION

Cycle powered rickshaws have been part of the Edinburgh scene for the past five or six years. Propelled—and patronised—by students they provide a popular “taxi” service along the pedestrianised zones in the capital centre. The occupant sits close to the ground and fairly close to the wire spokes of the rickshaw wheels. Though at first the Edinburgh accident was thought to be a rare unfortunate mishap, similar cases in India have been discovered in the literature and this raises the need for preventative measures to be introduced to avoid further occurrence. Rickshaws now in use in Edinburgh have plastic guards fitted to their wheels (fig 3).

This case raises several important points. The first is the obvious difficulty in diagnosing cardiorespiratory arrest in the presence of cervical oedema¹⁰ obscuring the carotid pulses. Initial indications at the scene (and later at hospital) were of a dismal prognosis. However, the attending ambulance crew, encouraged by occasional movements of the patient and an ECG rhythm that was potentially compatible with cardiac output, made vigorous attempts at resuscitation concentrating on basic airway care, ventilation, and oxygenation. The short response, scene and transit times (a total prehospital time of 16 minutes) ensured that hypoxia did not become established—and the initial SPO₂ of 99% on arrival at hospital was indeed very gratifying.

Secondly, of note was the impossibility of tracheal intubation in this patient even with the assistance of



Figure 3 Protected spokes on the wheel of a modern recreational rickshaw.

anaesthetic agents. This was attributable to both the anatomical distortion of laryngeal structures and the secondary complication of facial and laryngeal oedema. Although it can be speculated that there was a role for cricothyroidotomy at scene it is noteworthy that even when it came to be performed this procedure was not straightforward and required a specialist approach.

The development of traumatic carotid artery stenosis is a recognised complication of strangulation however it is of interest there are occasions where this can be managed conservatively with spontaneous recovery of the hemiparesis recovered over time.

Finally, it is remarkable that this patient survived at all. We can find no previous recorded evidence of survival from this “syndrome”. The “take home” message from this case must be that rapid intervention using good basic techniques in apparently hopeless cases can still occasionally produce remarkable results.

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Sodium bicarbonate to treat massive β blocker overdose

U R Shanker, J Webb, A Kotze

Emerg Med J 2003;**20**:393

Sodium bicarbonate is well recognised in the treatment of tricyclic overdose. But its use in the treatment of massive β blocker (propranolol) overdose has not been previously reported.

CASE REPORT

A 24 year old woman presented to the accident and emergency department with a history of overdose, taking 92 propranolol LA 80 mg, 45 paroxetine 30 mg, and 28 diazepam 5 mg tablets, two hours before admission. NPIS advised us to give activated charcoal every four hours and monitor vital signs closely and if needed intravenous glucagon. Within 30 minutes of arrival however her Glasgow Coma Score deteriorated rapidly and she developed hypotension. Arterial blood gas measurements showed metabolic and lactic acidosis. She rapidly deteriorated and went into cardiorespiratory arrest. She was intubated and standard ALS guidelines were followed. In addition NPIS was contacted and advised the following—(1) 100 μ l of 8.4% sodium bicarbonate to correct the acidosis, (2) an isoprenaline infusion to increase the heart rate, (3) an adrenaline (epinephrine) infusion to increase the blood pressure, (4) glucagon infusion to bypass the β block and act directly on cyclic GMP¹ (5) intravenous fluids to ensure adequate filling.

Despite this, the patient continued to have intermittent arrests, principally PEA (5 PEA, 1VT, 1VF). The ECG trace showed marked widening of QRS complex. Periods of arrest were getting longer and the interval between them shorter. NPIS were contacted again and advised rapid correction of arterial pH to 7.5. Thus a bolus of 300 ml of 8.4% sodium bicarbonate was given. Cardiac output was restored and the

ECG returned to a narrow complex morphology with sodium of 150 mmol. She was transferred to the intensive care unit and made a full recovery.

Propranolol is the most potent sodium channel blocker among β blockers. Sodium channels play an important part in the development of action potential in the cardiac muscle. Bradycardia caused by β blocker overdose in normal hearts is sodium channel block rather than β block.^{2,3} Treating β blocker overdose with low extracellular K⁺ and high extracellular Na⁺ increased the heart rate and restored the ability to pace thereby reversing the toxicity in isolated rat hearts.⁴ Similar experiments have been made with dogs but there are no reports of such treatment reported in humans. This is the first reported case demonstrating the importance of overloading sodium to counteract the block of the channels by β blocker overdose.

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