

## CASE REPORTS

# Left flank pain as the sole manifestation of acute pancreatitis: a report of a case with an initial misdiagnosis

J-H Chen, C-H Chern, J-D Chen, C-K How, L-M Wang, C-H Lee

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Acute pancreatitis is not an uncommon disease in an emergency department (ED). It manifests as upper abdominal pain, sometimes with radiation of pain to the back and flank region. Isolated left flank pain being the sole manifestation of acute pancreatitis is very rare and not previously identified in the literature. In this report, we present a case of acute pancreatitis presenting solely with left flank pain. Having negative findings on an ultrasound initially, she was misdiagnosed as having possible "acute pyelonephritis or other renal diseases". A second radiographic evaluation with computed tomography showed pancreatitis in the tail with abnormal fluid collected extending to the left peri-renal space. We performed a literature review and discussed this rare occurrence of acute pancreatitis. We also discussed the clinical pitfalls in this case.

In physicians' clinical experiences, pancreatitis can manifest solely as left flank pain, but very rarely. However, in a review of the literature, we were unable to identify a report specifically mentioning "left flank pain" as an isolated finding. We present a case of pancreatitis presenting solely with left flank pain. Due to a negative ultrasound report and the misinterpretation of clinical presentations, the on-duty physician missed the diagnosis initially.

### CASE REPORT

A 63 year old female patient visited our ED with a complaint of back pain on her left side for 5 days. The patient had no fever, abdominal pain, chest pain, dyspnea, or symptoms related to the urinary system. No recent trauma was noted. A review of her medical history revealed that she had a 5 year history of hypertension and type 2 diabetes mellitus with regular treatment, but no history of cardiac disease, stroke, or renal disease (including urolithiasis). She did not smoke or consume alcohol. A physical examination revealed prominent left flank pain with percussion, but was otherwise unremarkable.

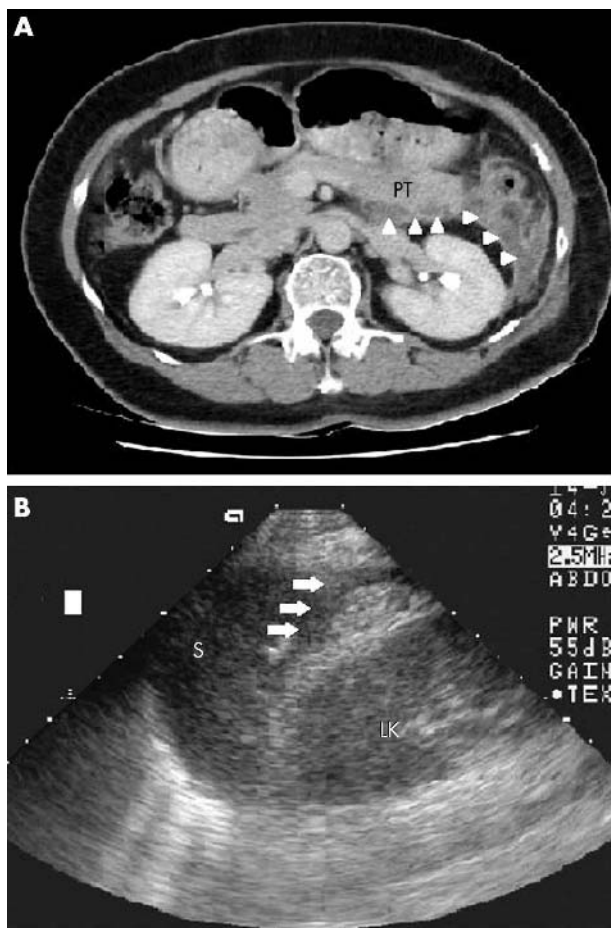
Laboratory data were as follows: white blood cells 7,970/mm<sup>3</sup>; hemoglobin 12.4 gm/dL; platelet count 280,000/mm<sup>3</sup>; blood urea nitrogen 9 mg/dL; serum creatine 0.6 mg/dL; serum glucose 280 mg/dL; and C-reactive protein (CRP) 10.3 mg/dL. A urinary analysis was normal and abdominal plain films did not reveal a radiopaque lesion or other significant abnormal findings. Due to the elevated CRP level and marked flank pain, an ultrasound was performed to evaluate the left kidney or surrounding organs. The ultrasound report by radiology suggested there were no abnormal findings in the areas of the kidneys, spleen, pancreas, or hepatobiliary system. Given this report, the on-duty senior resident decided to treat the patient in the ED-attached observation room.

On further review of the patient's case 2 hours after the ultrasound examination, a decision was made to obtain a computed tomography (CT) scan due to concern over the limitation of ultrasound studies in some clinical conditions. The CT showed abnormal fluid collection over the peri-renal space and pancreatic tail as well as necrotic changes and swelling of the pancreatic tail (fig 1). Serum pancreatic enzymes revealed a normal amylase (90 u/L) and a slightly elevated lipase level (336 u/L). The patient was diagnosed to have acute pancreatitis and admitted for supportive treatment and monitoring. During her admission she was also noted to have hyperlipidemia (triglyceride 980 mg/dL and cholesterol 319 mg/dL). The left flank pain was resolved after a 7 day treatment and she was discharged with the recommendation that she needed to follow up as an outpatient for long-term the lipid management.

### DISCUSSION

The clinical manifestations of acute pancreatitis can include upper abdominal pain, nausea, vomiting, and elevated levels of amylase and lipase.<sup>1</sup> Although there are no disease-specific signs or symptoms for acute pancreatitis,<sup>2</sup> making the diagnosis is usually not difficult, using a combination of clinical, laboratory, and imaging findings. Combinations of both upper abdominal and left flank pain are common in the presentation of pancreatitis. However, presenting solely with left flank pain is rare in the clinical experience. After reviewing the literature, we were unable to identify a report specifically mentioning the incidence of left flank pain as sole manifestation of acute pancreatitis. A few reports have described this rare clinical manifestation indirectly. Dalla Palma *et al*, reported using CT to diagnose urolithiasis in patients with flank pain and suggested its usefulness in detecting extraordinary lesions that can mimic renal colic.<sup>3</sup> Romano *et al*, also reported incidental findings of pancreatitis, diverticulitis, and renal tumor in patients with suspected renal colic by using CT.<sup>4</sup> As early as 1975, Hodges *et al*, suggested that pain typical of reno-ureteral diseases could emanate from any adjacent organs or any organs with the same innervations. Pancreatitis is listed in the differential diagnoses.<sup>5</sup> According to the literature, from 0.47% to 3.1% of patients with a flank pain were determined to have pancreatitis during their evaluation for the possible urolithiasis.<sup>4 6 7</sup>

This case report presents several points of interest in recognizing an unusual presentation of a common clinical problem. First, the causes of flank pain should not include only renal-ureteral diseases, but a wide range of clinical conditions. Pancreatitis should be included in the differential diagnosis, especially when renal-ureteral causes fail to adequately explain the clinical picture. Second, ultrasound may have limitations in identifying pancreatitis or other lesions around the pancreatic tail. Additionally, thick fluid collection in peri-renal space and the pancreatic tail (fig 1 in



**Figure 1** The computed tomography demarcated the lesion and showed a necrotic change over the pancreatic tail and abnormal fluid collection over the pancreatic tail and peri-renal space (arrowhead). The ultrasound showed fluid collection over the peri-renal spaces. A thick fluid collection (arrow) might have been misinterpreted as bowels by an inexperienced hand. (PT: pancreatic tail, S: spleen, LK: left kidney)

the same patients after retrospective review, arrowhead) may be confused with bowel on an ultrasound examination. Understanding this limitation and making use of CT may be necessary. Another interesting finding in this report is the level of the CRP. Although it is a nonspecific finding, an elevated CRP should raise the physician's suspicion to look for serious disease in the light of initially negative findings (for example, negative ultrasound). The CRP level has been shown to be well correlated with the severity of acute pancreatitis.<sup>8</sup>

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## Ruptured abdominal aortic aneurysm presenting as buttock pain

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This is the first case report of a ruptured aortic aneurysm presenting with acute right buttock pain. The patient was an 80 year old man. A literature search revealed one report of ruptured internal iliac artery aneurysm presenting with acute hip pain and another of an unruptured aortic aneurysm presenting with chronic hip pain. Thus the present case is another unusual presentation of ruptured abdominal aortic aneurysm and highlights the importance of careful history taking and clinical examination. A high index of clinical suspicion of aneurysm rupture should be maintained in elderly patients presenting with a history of collapse.

An 80 year old man presented to our accident and emergency (A&E) department with a history of severe pain in the right buttock for 15 minutes followed by collapse. He was unconscious for five minutes, and, on regaining consciousness, he was still having pain. On arrival at the hospital, his Glasgow Coma Scale score was 15/15, respiratory rate 20/min, pulse rate 88/min, and blood pressure (BP) 104/60 mm Hg. There were no acute changes on electrocardiography. The only significant past medical history was hypertension and myocardial infarction five years ago.

While in A&E, he started sweating profusely and lost consciousness momentarily. His BP at that time was 60/00 mm Hg and pulse rate 136/min. He recovered spontaneously in the

next few minutes without any resuscitation. He still complained of right buttock pain but no abdominal or chest pain. Examination of the hip joint was unremarkable. Both lower limbs were adequately perfused with palpable pulses and there was no sensory or motor deficit. Abdominal examination revealed a huge soft pulsating mass in the umbilical region, with an audible bruit.

In the absence of abdominal pain, we did not consider a bedside ultrasound examination appropriate to confirm a retroperitoneal leak.<sup>1</sup> Since his pulse was 84/min and BP 110/60 mm Hg, an abdominal computed tomography (CT) scan was arranged to confirm the diagnosis of a leaking abdominal aneurysm. The CT scan showed an extensive haematoma in the right perinephric and paranephric regions extending into the right iliac and inguinal regions, associated with a clearly leaking 10.2 cm infrarenal aortic aneurysm (fig 1). While still in the scanner he collapsed again and was transferred directly to theatre.

Intraoperatively we found a 10 cm infrarenal abdominal aortic aneurysm with a massive retroperitoneal haematoma extending from the upper abdomen to the whole of the pelvis, more on the right side. Following aortic grafting, the patient made an uneventful recovery and he was discharged on day 9 with no buttock or hip pain.

## DISCUSSION

The rupture of an aneurysm is a potentially life threatening complication of a diseased aorta, which may be preceded by leaking of variable duration. If the aneurysm ruptures into an open cavity such as the peritoneum the patient collapses and may even die before reaching hospital. But if the rupture occurs within a contained space such as the retroperitoneum the patient may improve without resuscitation, as in the present case. The classic triad of hypotension, back pain, and pulsatile abdominal mass may be present in only 50% of patients.<sup>2</sup> The presentation of this disease can often deviate from the classic clinical picture, resulting in erroneous diagnosis that may have lethal consequences.

The reported unusual presentations of leaking or ruptured abdominal aortic aneurysms include renal colic,<sup>3</sup> urethral obstruction,<sup>4</sup> obstruction of the left colon,<sup>5</sup> testicular pain,<sup>6</sup> peripheral neuropathy,<sup>7</sup> hiccoughs,<sup>8</sup> haematuria,<sup>9</sup> right inguinal mass,<sup>10</sup> and symptomatic<sup>11</sup> or even asymptomatic<sup>12</sup> inguinal hernia. Ijaz and Geroulakos<sup>13</sup> reported a case of a patient presenting with acute hip pain due to a ruptured internal iliac artery aneurysm. Chronic hip pain has been associated with an unruptured aortic aneurysm cured by elective repair.<sup>14</sup> A ruptured abdominal aortic aneurysm presenting with acute buttock pain has not previously been reported.

The commonest diagnosis considered in an elderly person with hip pain after a fall is a fractured neck of femur. Our patient had pain in the hip before he fell. Common causes of sudden hip pain in elderly people include septic arthritis, acute flare-up of osteoarthritis, and Leriche's syndrome. It is not unusual for the patients to present with a hip fracture after an episode of collapse irrespective of the cause. Therefore, in our case the presentation was misleading and a failure to examine the non-painful abdomen could have led to a delay in diagnosis with fatal consequences.

The pain in the hip experienced by our patient could be accounted for by the irritation of the posterior cutaneous nerve of the thigh or sciatic nerve at its origin. It is also possible that the retroperitoneal bleeding, depending on its volume and extent, may irritate the ilioinguinal nerve (L1) or the femoral branch of genitofemoral nerve (L1) or the



**Figure 1** Computed tomography scan (lateral view) showing abdominal aortic aneurysm.

femoral nerve with its branches (L2, 3, 4) or the lateral cutaneous nerve of thigh (L2, 3), which present with groin pain, testicular pain, anterior thigh pain, or lateral thigh pain, respectively.

The present case emphasises the importance of considering the diagnosis of a leaking or ruptured abdominal aortic aneurysm in patients presenting with collapse, regardless of the presenting symptoms.

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# Serious paediatric head trauma caused by vehicle rear view mirrors

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We report five cases of serious isolated head injury inflicted on children by rear view mirrors mounted on vehicles (table 1). All the injuries occurred between 1996 and 2001 and were admitted to our unit. So far there has been scant reporting of this particular type of injury. The risk of injury from rear view mirrors to pedestrians can potentially be prevented by modification of vehicle design and use of new technology.

In our series of five patients, all had serious head injuries requiring admission to the intensive care unit and three needed neurosurgical intervention. At follow up, two of the patients had persisting neurological problems. The cause of these head injuries was a very high pressure resulting from a force applied to a small area, in this instance a rear view mirror.

The relatively small surface area of a rear view mirror can transmit a large force carried by the vehicle. Even when a relatively small impact is applied over a small area, it is converted to a large force with the potential to cause substantial tissue damage. Ahmed, in discussing “stiletto heel” penetrating fractures of the skull, gave a good example of this by estimating that the force exerted per square centimetre by the heel of a woman’s stiletto shoe is greater than that of an elephant’s foot on the ground on which it treads.<sup>1</sup>

There have been three case reports in the literature of fatal cyclist and motorcyclist injuries from rear view mirrors.<sup>2–3</sup> The deaths were all caused as a result of head injury. Additionally there have been published case reports of perforating eye

injuries from extended rear vision mirrors because of shattering of the mirror in motor vehicle accidents.<sup>4,5</sup>

Head injury prevention must be the primary goal in management for all care providers. Clearly children of any age should be supervised while crossing the road, but thousands of young lives are lost every year as a result of accidents, and trauma remains the number one cause of paediatric death. There is a pattern and regularity to children’s injury: the pedestrian child has usually been the victim of a road traffic accident and in 75% of these cases has suffered head injury.<sup>6,7</sup>

Over the years there have been significant steps taken to make roads safer. Roadway design improvement, such as removal of fixed objects from roadsides, widening roadside recovery zones, and installing dividers between opposing lanes of traffic, has been effective in reducing crashes and injuries. Speed restrictions in urban areas and the use of traffic cameras have probably caused a reduction in the number of lethal crashes.

There have been significant improvements in the last 30 years in all aspects of vehicle design to make them safer for occupants in the event of a collision with research conducted to help minimise forces exerted to the occupant’s head.<sup>8</sup> These improvements include lap and shoulder belts for car occupants, the use of automatic air bags for front and side impacts, head restraints, and even the use of automatic roll bars for vehicles that overturn in a collision.

**Table 1** Five cases of serious isolated head injury inflicted on children by rear view mirrors mounted on vehicles

Age, sex	GCS on admission	Injury, including CT findings	Treatment, including operative findings	Follow up at 6 months
7, M	14	Fronto-parietal skull fracture and scalp laceration, brain contusion with small subdural haemorrhage and some midline shift	Laceration sutured, non-operative treatment of injury	Reported to have slurred speech and poor attention span
4, M	3	Generalised seizure, depressed fronto-parietal skull fracture, ipsilateral brain swelling with ventricular effacement	Depressed fracture elevated, small extradural haemorrhage removed, underlying brain contused	No further seizures but MRI brain showed persisting abnormality in subcortical frontal and parietal white matter bilaterally
13, F	3	Scalp laceration, fronto-parietal skull fracture, temporal lobe contusion, zygomatic fracture	Compound depressed skull fracture elevated	Well with no problems
11, M	15	Occipital skull fracture, frontal lobe contusion, subarachnoid haemorrhage	Non-operative treatment	No problems reported
8, M	11	Orbital fracture, basal skull fracture, extradural, subdural, and intracerebral haemorrhage	Staged surgical treatment, initially evacuation of haematoma then parietal bone grafting	Evidence of left sided hemiparesis

CT, computed tomography; F, female; GCS, Glasgow coma score; M, male; MRI, magnetic resonance imaging



Less attention has been directed at making vehicles safer for pedestrians in the event of a collision. The Department for Transport sets out the requirements for mirrors on motor vehicles in the Road Vehicles Regulations act 1986.<sup>9</sup> Where the bottom edge of an exterior rear view mirror is less than 2 m above the ground, the mirror should not project more than 0.2 m beyond the overall width of the vehicle.

New technologies have been introduced to vehicles in order to minimise pedestrian injuries. These include modifying vehicle exterior structures, such as wing mirrors, including size reduction and fold down designs. The authors agree that there is a clear benefit in the use of rear view mirrors in accident prevention; however, a significant number of manufacturers still build large or extended mirrors on all types of vehicles, including trucks, vans, and buses. We believe this area needs to be given greater focus and that wing mirror injuries are potentially preventable if technology allows.

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## Succinylcholine induced masseter spasm during rapid sequence intubation may require a surgical airway: case report

**S J Bauer, K Orio, B D Adams**

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Succinylcholine has long been the neuromuscular blockade agent of choice for the emergency physician for rapid sequence intubation because of its rapid onset and relatively brief duration of action. However, it has many known life-threatening side effects and contraindications including allergy, histamine release, dysrhythmias, hyperkalaemia, and malignant hyperthermia.<sup>1</sup> It has also been known to cause significant masseter spasm in children when used in conjunction with volatile anaesthetics such as halothane.<sup>2–5</sup> In adults, succinylcholine can also produce transient masseter spasm that resolves when fasciculation stops. This potentially deadly side effect has been noted in other specialties but the incidence in adults is unknown.<sup>6,7</sup> The generally agreed upon treatment is to stop the anaesthetic and reschedule the procedure at a later date with different agents and evaluation for malignant hyperthermia.<sup>4</sup> However, in the emergency department that management option is not available to the emergency physician. Knowledge of the potential side effects of this commonly used medication is paramount to successful airway management.

We present a case of succinylcholine induced masseter spasm in the emergency department requiring surgical

cricothyroidotomy for airway control. We believe that this is the only reported case of masseter spasm resulting in a failed airway requiring surgical cricothyroidotomy during rapid sequence intubation.

#### CASE REPORT

The patient was a 56 year old man brought in by ambulance for altered mental status and hypotension. His vital signs in the field were: blood pressure 97/58 mm Hg; heart rate 135; respiratory rate 19; and SAO<sub>2</sub> 98% on 100% oxygen. Finger stick whole blood glucose was normal. The ambulance team reported that the patient's apartment was covered with bloody vomitus and melaena, and the patient was noted in the field to be covered in what appeared to be old blood and stool. He presented complaining only of irritation at the site of his recently inserted percutaneous endoscopic gastrostomy (PEG) tube and vomiting. He was awake and alert but slow to respond.

His past medical history was significant only for squamous cell carcinoma of the head and neck of unknown staging. The PEG tube had been inserted for feeding just two weeks before he was brought to our emergency department. He stated that

### Normal values, Brooke Army Medical Center Laboratory

#### Serum chemistry

Blood urea nitrogen: 4.8–20 mg/dl  
 Creatinine: 0.7–1.3 mg/dl  
 Sodium: 138–146 mmol/l  
 Potassium: 3.5–5.1 mmol/l  
 Chloride: 99–109 mmol/l  
 CO<sub>2</sub>: 21–32 mmol/l  
 Glucose: 65–105 mg/dl  
 Lactate: 0–1.8 mmol/l

#### Arterial blood gases

pH: 7.35–7.45  
 P<sub>CO<sub>2</sub></sub>: 35–45 mmHg  
 P<sub>O<sub>2</sub></sub>: 80–100 mmHg  
 HCO<sub>3</sub><sup>-</sup>: 22–26 mEq/l  
 O<sub>2</sub> saturation: 95–98%

his only medications were “nausea medications” and he had no known drug allergies. He was an ex-smoker and regularly consumed large quantities of alcohol. He had had the last drink three days prior to arrival.

His physical exam was significant for hypotension (98/59 mm Hg) and tachycardia at 135 beats per minute, and he had a rectal temperature of 39.7 °C (103.4 °F). He appeared much older than his stated age of 56 years. His exam was remarkable for dried blood in the posterior pharynx, he had a Mallampati class II airway with three fingers width mandibular opening, and his trachea was in the midline. His lungs demonstrated scattered rhonchi; his PEG tube was excoriated and leaked haeme positive brown fluid but otherwise showed no signs of infection. His abdomen was not tender and there were no peritoneal signs. Rectal exam demonstrated obvious melaena with mixed blood. His extremities were cool, and his skin was covered in old blood and stool but there were no rashes or petechiae. His neurological exam was non-focal.

We placed the patient on oxygen by non-rebreathing mask, inserted two large bore intravenous catheters, and administered 3 l of normal saline by 1 l boluses. Urinary output was monitored by inserting a Foley catheter. The patient was placed on a cardiac monitor, which showed sinus tachycardia. He was typed and crossed for four units of packed red blood cells. He was given ceftriaxone 2 g intravenously. His electrocardiogram (ECG) showed only sinus tachycardia and a portable chest x ray was negative for infiltrates. Laboratory studies revealed sodium 125 mmol/l, potassium 2.6 mmol/l, chloride 78 mmol/l, CO<sub>2</sub> 25 mmol/l, creatinine 1.3 mg/dl, and glucose 124 mg/dl (see box for normal values). His complete blood count showed leucocytosis at 20.4 million white cells with left shift and haematocrit 37%. Urinalysis showed only trace ketones. Lactate was >12 mmol/l. Cardiac enzymes and a head computed tomography (CT) scan were negative. A lumbar puncture was performed and was negative.

The patient had required occasional doses of midazolam 2 mg intravenously for facilitation of studies and procedures. Although he would occasionally become agitated, his mental status never deteriorated and his vital signs normalised after resuscitation and one unit of packed red blood cells.

Approximately four and a half hours into his resuscitation the patient began having difficulty maintaining his airway with sonorous respirations and became unrousable. An arterial blood gas (ABG) at that time was: pH 7.228; P<sub>CO<sub>2</sub></sub> 96 mm Hg; P<sub>O<sub>2</sub></sub> 317 mm Hg; HCO<sub>3</sub><sup>-</sup> 8.7 mEq/l and O<sub>2</sub> saturation 99%. The patient was prepped for intubation by placing into the sniffing position, suction was made ready, and bag

and mask with end tidal CO<sub>2</sub> detector was ready for use. As the patient had normal external anatomy with a Mallampati class II airway with three fingers width mandibular opening he was deemed to be a safe candidate for rapid sequence intubation. Etomidate 20 mg and succinylcholine 100 mg were administered through the intravenous catheter. Cricoid pressure was maintained throughout the procedure. Fasciculations were noted, and flaccid paralysis was noticed approximately 30 seconds after administration of succinylcholine. The mouth could not be opened and the masseter muscles appeared to be in spasm. The teeth were clenched together and movement of the mandible was not possible. An additional 100 mg of succinylcholine without atropine was administered without effect on the spasm. The on-call anaesthesia service was notified, and the patient was ventilated by bag and mask without difficulty. The attending anaesthesiologist and senior resident arrived within minutes and attempted to insert a nasotracheal airway with fibreoptic visualisation but were unable to see the patient’s vocal cords. Anticipating a potential failed airway, the patient’s anterior neck was prepped with Betadine for a cricothyroidotomy. The patient eventually became difficult to ventilate, and his oxygen saturation dropped to 82% and could not be brought back up. Flaccid paralysis of the extremities was noted throughout the procedure. A failed airway was declared and a surgical airway was deemed necessary. No additional anaesthetic medications were administered. However, the entire time course was less than 10 minutes and the etomidate originally administered should have been sufficient for anaesthesia and amnesia.

A 2 cm vertical incision was made over the cricothyroid membrane with a number 10 blade scalpel. The membrane was visualised and horizontally incised with the scalpel. The membrane space was dilated with haemostats and an attempt at passing a 5.0 mm endotracheal tube was made but the tube would not pass. A second attempt with a 5.0 mm endotracheal tube was made, and it successfully passed into the trachea. A colorimetric end tidal CO<sub>2</sub> detector showed yellow colour change with six breaths from the bag with a subsequent rise in his pulse oximetry to the mid-90s. Bilateral breath sounds were auscultated in four lung fields. At this time midazolam 5 mg was administered intravenously. Five minutes after the completion of the cricothyroidotomy, the attending anaesthesiologist was able to place a 7.0 mm nasotracheal airway. The 5.0 mm endotracheal tube was removed from the cricothyroidotomy site as the 7.0 mm tube allowed easier ventilation and the site was closed with sutures.

The patient was transferred to the medical intensive care unit and was extubated four days later. He eventually required reintubation with awake nasotracheal intubation for respiratory distress. He died 14 days while in intensive care. Autopsy demonstrated a large 3×4 cm gastric ulcer that had eroded into a submucosal artery.

## DISCUSSION

Rapid sequence intubation has been shown to be successful and safe when used in the emergency department for airway control. Dufour examined the charts of 219 patients who underwent rapid sequence intubation and only one patient had a fatal outcome unrelated to the intubation and 32 patients had mild complications.<sup>8</sup> Sakles and colleagues evaluated the charts of 610 intubations in the emergency department and rapid sequence intubation was used in 84%.<sup>9</sup> Of those intubated with rapid sequence intubation, 99% were successfully intubated with one adverse outcome of hyperkalaemic cardiac arrest. The use of neuromuscular blocking agents has been shown to increase both the safety and success rate of rapid sequence intubation.<sup>10</sup>

Masseter spasm has been implicated as an early indicator of susceptibility to malignant hyperthermia. Other markers for malignant hyperthermia include hyperpyrexia, increased end tidal CO<sub>2</sub>, generalised rigidity, autonomic instability, and rhabdomyolysis. The exact incidence of patients who develop masseter spasm and actually go on to develop malignant hyperthermia is unknown. An incidence as high as 50% has been reported in children, but some authors believe that this may be due to overreporting.<sup>3-11</sup> The incidence of malignant hyperthermia is unknown in adults with masseter spasm, although isolated masseter spasm is not pathognomonic for malignant hyperthermia.<sup>12</sup> Although not given to our patient, the treatment for malignant hyperthermia is dantrolene 1 mg/kg administered intravenously. This may be repeated until symptoms resolve or a maximum dose of 10 mg/kg is reached.

Did malignant hyperthermia cause our patient's masseter spasm? We do not think so. The patient did not develop generalised rigidity, nor did he develop increased end tidal CO<sub>2</sub> based on repeat blood gases. His creatinine phosphokinase never rose above 100 IU/l and he did not develop myoglobinuria. A repeat rectal temperature approximately 30 minutes after masseter spasm was 38 °C (100.4 °F). It has been recommended that patients who develop masseter spasm undergo muscle biopsy for evaluation of potential susceptibility to malignant hyperthermia. We did not take a muscle biopsy from our patient, however, his family was extensively questioned and there was no family history of anaesthetic reactions. His grown children were advised to have biopsies done to determine if they were susceptible to malignant hyperthermia.

Many lessons can be learned from this case report. First and foremost, succinylcholine has many adverse effects that must be anticipated. Secondly, a patient's external anatomy should not be entirely relied upon as an indicator of the ease of intubation as many other factors such as medication side effects can contribute to a failed airway. Finally, dantrolene administration should be considered if masseter spasm is encountered after succinylcholine as this may signal the development of malignant hyperthermia.

## SUMMARY

Succinylcholine is the neuromuscular blocking agent of choice in rapid sequence intubation due to its rapid onset

of action and relatively rapid return of muscle tone.<sup>13</sup> Emergency physicians need to be aware of the significant adverse side effects of succinylcholine and must be prepared to deal with them, including the potential for masseter spasm and malignant hyperthermia.

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# Bleeding due to a medicinal leech bite

İ İkizceli, L Avşaroğulları, E Sözüer, Y Yürümez, O Akdur

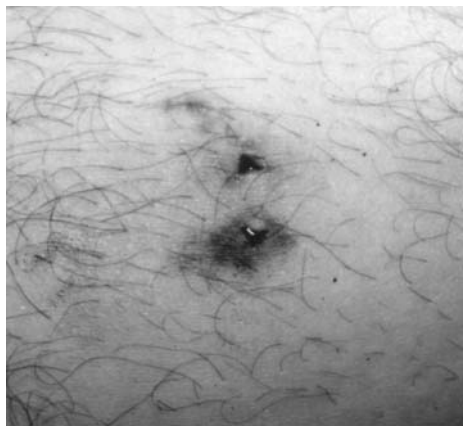
*Emerg Med J* 2005;**22**:458-460. doi: 10.1136/emj.2003.013847

This paper reports a case of prolonged bleeding following application of leeches to treat chronic pain. The paper discusses the characteristics of the wounds and possible complication of prolonged bleeding following medicinal leech application. The principles of treatment are also described.

**B**oth doctors and lay health practitioners use leeches of the phylum Annelida for therapeutic purposes in many countries worldwide. The medicinal leech, *Hirudo*

*medicinalis*, is used for bloodletting and pain relief in traditional folk medicine, as well as by doctors to maintain local circulation after reimplantation, flap repairs, and breast and thoracic wall reconstructions. Complications related to leech bites are not commonly seen in emergency departments (ED). To date, only one patient with bleeding secondary to a leech bite, who received advice from an ED by phone, has been reported.<sup>1</sup> Here we describe a patient who presented to the ED with prolonged bleeding from a leech bite, the first such report in the emergency medicine literature.





**Figure 1** Leech bites in our patient resulted in a triradiate wound (consistent with reports in literature<sup>1-3</sup>).

## CASE REPORT

A 19 year old man presented to our ED complaining of bleeding from both legs. He had applied leeches to both legs at midnight to treat chronic leg pains of over a year's duration. After the leeches spontaneously detached, he dressed his wounds and went to bed. When he woke up three hours later, he saw bloody bandages. The exact time of the onset of bleeding was unknown. As the bleeding did not stop in spite of compression and wrapping with tight bandages, he came to the ED at 8 45 am. He had no remarkable medical history.

On physical examination, he appeared generally healthy, was alert and oriented, and in no acute distress. Vital signs were as follows: blood pressure 110/80 mm Hg, heart rate 92 beats per minute, respiration rate 16 per minute, temperature 36 °C. Blood was oozing from two lacerations on his right leg, one 3 cm below and the other 4 cm medial to the tibial tuberosity. The distance between them was 7 cm. In addition, there were two lacerations on the lateral head of the gastrocnemius muscle on the left leg that had stopped bleeding. The distance between them was 5 cm. No ecchymosis, swelling, or erythema was present. His physical examination was otherwise normal. Laboratory findings were as follows: haemoglobin 18.1 g/dl, haematocrit 56%, mean corpuscular volume 98.1 femto l, white blood cells 6400/mm<sup>3</sup>, platelets 171 000/mm<sup>3</sup>, prothrombin time 13.48 seconds, activated partial thromboplastin time 35.3 seconds, and international normalised ratio 1.19. The peripheral blood smear was normal.

The wounds were rinsed with antiseptic solutions and bandaged with sterile gauze, following which he was observed in the ED for three hours. He was then discharged as no further bleeding had occurred. The wounds were unremarkable three days later, but the patient stated that the wounds had started oozing again one hour after discharge from the ED. When he had changed the dressings later that day, he had noticed clots on the wounds with bruising in the surrounding tissues, but there was no redness, warmth, or pain in the areas surrounding the wounds.

## DISCUSSION

The medicinal leech has been used for medical purposes since at least 200 BC and is still used in Asia and Africa.<sup>1-3</sup> A recent clinical study has reported that leech therapy may be an effective treatment for rapid reduction of pain associated with knee osteoarthritis.<sup>2</sup> Our patient used leeches for alleviating pain and placed them at painful periarticular sites of the knee.

*H. medicinalis* has an approximately 10 cm long, cylindrical body with two suckers: one present anteriorly on the head, and the other on the posterior end. The mouth lies in the

anterior sucker and has three jaws with teeth well designed for biting. The leech can ingest blood almost ten times its own weight (5–15 ml).<sup>1-3</sup>

Leech bites are painless and results in a triradiate wound which remains open for a long time and heals slowly<sup>1-3</sup> (fig 1). The commonest complication of leech application is oozing, as was the case in our patient. The amount and duration of bleeding vary according to the area bitten, with bleeding from the vagina, rectum, urinary bladder, and pharynx having been reported.<sup>1-3-4</sup> Prolonged haemorrhage may result in anaemia, and deaths from excessive exsanguination have been reported.<sup>3</sup> In our patient, the bleeding continued for three hours and persisted intermittently for the next 18 hours, although he did not have any haematological problems. The mean duration of bleeding from leech bite wounds in one report was 10 hours (range 6.5–23).<sup>5</sup>

The saliva of the leech contains hirudin, which inhibits thrombin in the clotting process, and histamine-like substances which may cause continuous bleeding by preventing closure of capillaries.<sup>6</sup> Munro *et al* reported that hirudin has only a transient antithrombin effect, lasting only about 15 minutes in humans. The prolonged duration of bleeding can be attributed to collagen–platelet interaction, along with possible modifications of the vascular walls by proteases or other enzymes secreted by the leech during feeding.<sup>3</sup>

Contamination with pathogenic microorganisms may result in erysipelas and submucosal abscesses.<sup>1-3</sup> Leech application can also cause infection with *Mycobacterium marinum*, a parasitic bacteria usually hosted by salt water fish, or with *Aeromonas hydrophilia*, which leeches carry in their gut.<sup>7</sup> As a medicinal leech bite heals, ecchymosis and scarring are not uncommon sequelae.<sup>1</sup>

As regards treatment, if the leech is still in place, it should be removed with the help of table salt, a saline solution, or vinegar. It should not be forcibly removed because its jaws may remain in the wound, causing infection.<sup>1</sup> After removing the leech, pressure should be applied to the wound. If the bleeding persists, sterile gauze soaked in thrombin solution may be applied. After control of bleeding, the wound should be rechecked for signs of infection.<sup>1</sup>

In our patient, the leech had detached before arrival to the ED, and we had no thrombin solution to apply with the bandages. There was intermittent bleeding for an additional 18 hours. Leeches application in the evening or night should be avoided because bleeding cannot be noticed during sleep. Patients with bleeding disorders should not apply leeches to avoid prolonged bleeding. Emergency physicians should exercise caution when removing leeches, and they should not be surprised if patients present with persistent bleeding after removal.

## CONTRIBUTORS

I İkizceli: data collection and drafting of the report. L Avsarogullari, E Sözüer: critical revision and drafting of the report. Y Yürümez, O Akdur: data collection and treatment and follow up of the patient during his admission and stay in the emergency department.

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# Stabbing chest pain: a case of intermittent diaphragmatic herniation

P Ransom, P Cornelius

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A case of traumatic diaphragmatic herniation is described in which gross mediastinal shift was caused by a combination of the herniated abdominal organs, purulent exudate, and air. This complex presentation might best be described as a case of gastrocolopneumothorax, diagnosis of which was further complicated by the intermittent nature of the herniation.

A case of traumatic diaphragmatic herniation is described in which gross mediastinal shift was caused by a combination of the herniated abdominal organs, purulent exudate, and air.

## CASE REPORT

A 21 year old man presented to our emergency department complaining of a 2 day worsening of anterior and posterior chest pain with shortness of breath, and a 2 month history of intermittent vomiting, dysphagia, and weight loss. Three months previously, he had sustained a stab wound to the left chest and presented to hospital. A chest radiograph taken on that admission was reported as normal, and an abdominal ultrasound had shown only a small left pleural effusion. Shortly after, he had self discharged against medical advice. One month later he began the first of six visits to two separate emergency departments complaining of episodic abdominal pain and vomiting, leading to admission on two occasions. Blood results for these visits showed white cell counts of between 16 and  $23 \times 10^9/l$ , and varying degrees of dehydration, on one occasion leading to acute pre-renal failure with urea of 33 mmol/l. During the later visits, confusion had been noted. A chest radiograph taken 3 weeks prior to his presentation to us had been reported as normal by the radiologist and treating doctors, and oesophagogastroduodenoscopy (OGD) performed during that admission found a normal stomach, pylorus, and duodenum, with a degree of oesophageal candidiasis. This, together with his emaciated appearance, had led to a suspicion of immunocompromise. HIV testing was subsequently performed, which had proved to be negative. In the light of the normal chest radiograph and unremarkable gastroduodenal examination, a diagnosis of self induced vomiting had been given on his last discharge.

On examination at our hospital, he was emaciated and distressed, tachypnoeic at 30 breaths/min, with pulse oximetry on air of 97%. He was normotensive at 110/70, with a pulse of 110 beats/min. He was vomiting an almost clear fluid. Examination of the chest showed a trachea deviated to the right, absent breath sounds on the left side, with percussion note being resonant over the left upper chest and dull from the midline down. A small, healed scar was seen below the anterior axillary line of the 10th rib.

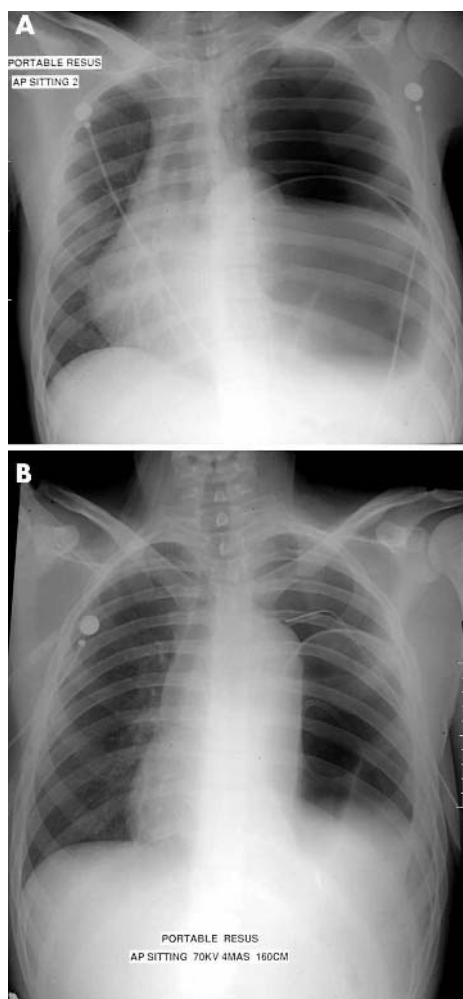
The initial portable chest radiograph showed massive diaphragmatic herniation of stomach and bowel, with a left sided pneumothorax and what appeared to be a collection of fluid reaching to the mid-thorax, all of which had caused marked rightward mediastinal shift (fig 1A).

As the patient's oxygen saturation was normal and the radiographic appearances were not those of a classical tension pneumothorax, we elected to place a small (size 12) chest drain anteriorly through the second intercostal space rather than perform a needle thoracocentesis. A hiss of air was noted upon drain insertion, and during the patient's initial breaths, continuous bubbling of the drain bottle was seen throughout the respiratory cycle. Within minutes, his chest pain had decreased. Over the next 15 minutes, 1.4 litres of thick, purulent fluid was discharged. A second radiograph taken 40 minutes after the first showed the stomach partially decompressed, some remaining fluid, and the mediastinum shifted back towards the midline (fig 1B).

Thoracotomy revealed that the greater part of the stomach and a loop of colon had herniated through a 6 cm defect of the left diaphragm. There was still an appreciable amount of purulent exudate, but no perforation of the abdominal organs and no damage to the lung structures. A sample of the fluid showed white cell debris, and culture revealed scanty growth of upper respiratory tract flora. He made an uneventful recovery, and once again self discharged against medical advice.

## DISCUSSION

Herniation though a diaphragmatic stab wound is a well recognised, although infrequent cause of cardiothoracic embarrassment,<sup>1</sup> while tension gastrothorax or colothorax appears in the literature occasionally as a case report.<sup>2</sup> Intrapleural effusion caused by herniation of the abdominal contents has likewise been noted on occasion.<sup>3–4</sup> Radiological diagnosis of diaphragmatic rupture is difficult, and many



**Figure 1** Chest radiographs taken before (A) and after (B) insertion of drain.

case series have demonstrated the unreliability of radiographs or computed tomography scans to pick up this injury.<sup>5</sup>

This case is unusual, however, in that the abdominal herniation, exudate, and the air under tension had all combined to cause respiratory compromise in what would best be called a tension gastropneumothorax. To our knowledge, no previous cases have been described in which these three features have co-existed simultaneously.

The contribution of the air to the tension is of interest. Classically, a tension pneumothorax is caused by a one way

valve in a breached lung, allowing air into, but not out of, the pleural cavity. However, the lung in this case had not been breached, but had collapsed. Here, presumably, the combined volume of the bowel, the inflated stomach, and the exudate had together exerted an increasing pressure in the left chest that was only partially compensated by the rightward shift of the mediastinum. The hissing of air as the drain was inserted, and the initial bubbling of the chest drain independently of the respiratory cycle provide evidence for the pleural cavity being under a degree of air tension.

Perhaps the most diagnostically challenging feature of this case concerns the unremarkable chest radiograph and OGD performed 3 weeks before presentation to our hospital. The normal results of these investigations argue strongly that the herniation was intermittent in nature, as it is extremely unlikely that another pathology existed for his symptoms. Presumably the defect in the diaphragm had on previous occasions allowed the herniated abdominal contents to reduce spontaneously, until he presented to our hospital and reduction was this time no longer possible.

Stabbings are becoming a more frequent presentation to the emergency department.<sup>6</sup> Patients with thoracoabdominal stab wounds or blunt abdominal trauma should perhaps be warned before discharge about the possibility of diaphragmatic herniation in the future, as the symptoms may be delayed by many years and the originating trauma forgotten or disregarded as the cause of subsequent, potentially life threatening, symptoms.

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## CASE REPORTS

# Hereditary motor and sensor neuropathy: a cause of acute stridor

T Jaiganesh, A Bentley

*Emerg Med J* 2005;22:666–667. doi: 10.1136/emj.2003.009886

We present an acute stridor secondary to bilateral vocal cord paresis in a patient with demyelinating form (type I) of hereditary motor and sensory neuropathy (HMSN). Management problems are discussed and HMSN reviewed.

A 48 year old female attended the emergency department with complaints of cough, breathing difficulty, and flu-like symptoms for one day. She suffers from hereditary motor and sensory neuropathy (HMSN) Type Ia, which had been detected by isolating DNA from a blood sample for the presence of duplication on Chromosome 17. She had been treated for asthma for two years and had a history of nocturnal choking episodes. Examination revealed inspiratory stridor with indrawing of neck muscles. The ear, nose, and throat (ENT) surgeons, consultant anaesthetist, and intensivist were contacted. ENT examination through a fibre optic flexible laryngoscope revealed smooth white paramedian position of the vocal cords, which were not swollen. Neurological examination revealed peripheral lower motor neurone (LMN) wasting of the arm and legs distally with areflexia. Family history was suggestive of an autosomal dominant pattern of inheritance. There was no evidence of diaphragmatic or respiratory muscle weakness.

She was transferred to theatre recovery suite and had a gaseous induction for endotracheal intubation. Further inspection of the vocal cords confirmed they were partially adducted. The patient was intubated and subsequently transferred to the intensive care unit (ICU). Forty eight hours later she went to theatre for an examination of vocal cords using a flexible laryngoscope prior to consideration of extubation. The vocal cords remained in paramedian position and were now oedematous. She therefore underwent a formal tracheostomy and returned to ICU where she was weaned and was discharged home with tracheostomy in situ with an early follow up to the regional neuromuscular clinic.

### DISCUSSION

HMSN, previously known as charcot marie tooth (CMT) disease or peroneal muscular atrophy (PMA), was initially described in 1868 by two French neurologists (Charcot and Marie)<sup>1</sup> and one English neurologist (Tooth)<sup>2</sup> as a familial condition causing distal muscular atrophy. HMSN is a slowly progressive genetic disorder, which causes deterioration of the peripheral nerves resulting in weakness and sensory loss of the distal limbs. There are a few identified cases that involve the vocal cords and/or the respiratory muscles, particularly the diaphragm (table 1).

Cranial nerves can be involved in the pathological process of HMSN. In a cohesive overview only 11 cases of HMSN with

**Table 1** Clinical presentation of HMSN related to the genetic defects

Type	Chromosome	Age of onset (in years)	Early symptoms	Tendon reflexes
HMSN 1: dominant; demyelinating				
1 A	17	5–10	Distal weakness	Absent
1 B	1	5–10	Distal weakness	Absent
1 C	Unknown	10–15	Distal weakness	Reduced
1D	10	10–15	Distal weakness	Absent
X	X	10–15	Distal weakness	Absent
HMSN 2: dominant; axonal				
2 A	1	10	Distal weakness	Absent
2 B	3	10–20	Distal weakness	Absent
2 C	Unknown	1 <sup>st</sup> decade	Sensory loss & distal weakness	Absent
2 D	7	16–30	Distal weakness	Reduced
HMSN 3 (Dejerine-Sottas)				
HMSN 4: recessive; demyelinating/axonal Involves ethnic groups				

bilateral vocal cord paresis have been observed and of that only two have occurred in type I.<sup>3</sup> Vocal cord and diaphragm involvement is usually a feature of type Iic.<sup>4</sup> Most of the patients with HMSN tolerate vocal cord paresis remarkably well but can result in quick rapid decompensation, as shown in our case, possibly related to inter-current infection. The patient's mother also suffered from a similar condition and her son is currently suffering from a the same condition, supporting autosomal dominant inheritance, but none of them have developed stridor. This is possibly because there may be some re-innervation and recovery of function.

Vocal cord paresis in HMSN is most often bilateral and is not restricted to type Iic as was previously thought. Vocal cord paresis may be overlooked or misdiagnosed as asthma, as in our patient. The small number of cases with

**Abbreviations:** CMT, charcot marie tooth; ENT, ear, nose, and throat; HMSN, hereditary motor and sensory neuropathy; ICU, intensive care unit; PMA, peroneal muscular atrophy



tracheostomy in children suggests that in adults there may be a partial recovery. In our case this will be reviewed in the regional neuromuscular clinic.

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## Recurrent elbow dislocation—an uncommon presentation

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A 58 year old female attended our A&E department following a fall in the garden with swelling and bruising of the right arm and the elbow. Anteroposterior and lateral radiographs were interpreted as showing a normal elbow joint. A diagnosis of soft tissue injury to the elbow was made and the patient was discharged with advice. She returned 2 days later, did not have an x ray, and again given advice. Three weeks later she was referred back to A&E by the general practitioner with persistent swelling of the elbow. Further radiographs showed a posterolateral dislocation of the elbow. The elbow was reduced under sedation but was subsequently dislocated at follow up, and was treated by external fixator and transolecranon pin. The fixator was removed at 4 weeks and the elbow was then stable. This case highlights that recurrent elbow dislocations due to significant ligament injuries can present in joint and subsequently dislocate. A high index of suspicion is necessary and appropriate referral to the specialist must be made to avoid the morbidity associated with recurrent dislocation. It also emphasises the need to always assess the patient on his or her own merits despite previously normal investigations.



**Figure 1** Anteroposterior radiograph of elbow at initial presentation. Reproduced with permission.

The elbow is one of the more highly constrained and stable joints in the body, yet dislocation is not uncommon.<sup>1 2</sup> Because of its intrinsic stability, redislocation is rare in the elbow in contrast to the shoulder. Most acute elbow dislocations are posterior. There are no previous reports in the literature of a recurrent elbow dislocation presenting as a normal elbow and subsequently dislocating. We describe the management of this patient, highlight the lessons learnt, and describe best practice for similar cases.

#### CASE REPORT

A 58 year old female presented to the accident and emergency (A&E) department with extensive bruising and swelling of the right arm and the elbow following a fall in the garden. The elbow movements were restricted with no distal neurovascular deficit. Anteroposterior and lateral radiograph

of the elbow was interpreted as normal and the patient was discharged with analgesics and limb elevation advice (figs 1 and 2). The patient returned to the A&E 2 days later with increasing pain and deformity. She was advised that the management plan was correct as the initial radiograph showed no dislocation.

Three weeks later the general practitioner referred the patient to the on call orthopaedic doctor because of continued pain and swelling. On examination the elbow was still swollen with a restricted range of movement. Radiographs of the elbow were repeated, which showed a posterolateral dislocation of the elbow (figs 3 and 4). The elbow was reduced in the A&E department and referred to the fracture clinic. At two weeks review in the clinic, the elbow had dislocated again. Subsequent treatment involved examination under anaesthesia (EUA) in the theatre where the elbow was found to be grossly unstable; after application of external fixator and transolecranon pin (fig 5), which was removed at 4 weeks, the elbow was found to be stable. There had been no further dislocation and the patient was referred to the physiotherapy department for mobilisation of the elbow. At four months review the elbow was stable with an extension loss of about 30°.

**Abbreviations:** A&E, accident and emergency; EUA, examination under anaesthesia



**Figure 2** Lateral radiograph of elbow at initial presentation. Reproduced with permission.

## DISCUSSION

Recurrent instability of the elbow joint is uncommon but it is a debilitating disorder when present. The elbow is the second most commonly dislocated large joint after the shoulder in adults and the most common in children.<sup>2</sup> Recurrent dislocation is an uncommon sequelae. It usually occurs in association with intra-articular fracture or generalised ligament laxity, but can occur following simple dislocation.<sup>2-3</sup> We can find no mention in the literature of an elbow dislocating two days after the index injury, when at initial presentation the elbow was found to be in joint.

The elbow is a highly constrained joint and the stability depends on the bony architecture and the integrity of the ligaments, capsule, and muscles around the joint. The articular surface of the elbow is congruent and the bony surfaces minimise the risk of dislocation. The medial and lateral collateral ligaments are strong and play an important role in the stability of the joint. Theories on the most important factors in the stability of the joint have changed over 20 years.<sup>4</sup> The medial collateral ligament was believed to be the important stabiliser and posterolateral dislocation was not possible if the medial collateral ligament remained intact.<sup>5</sup> Recently attention has focussed on the lateral ulnar collateral ligament as the primary constraint to posterolateral instability<sup>6</sup> and the secondary constraints of the extensor muscle origin, fascial bands, and lateral intermuscular septum.<sup>7</sup>

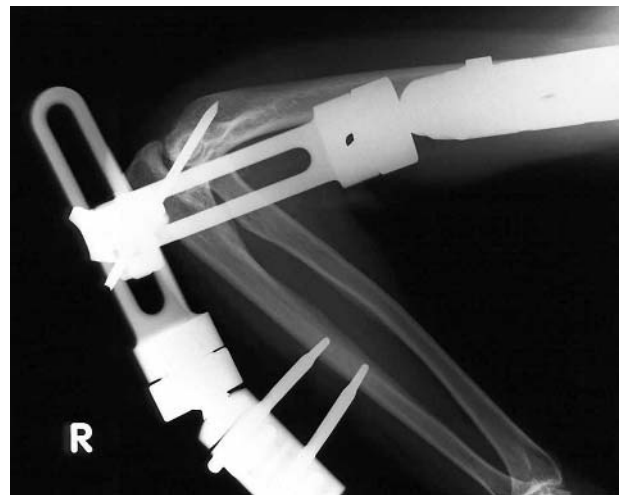
Elbow instability may be classified by time (acute, chronic, or recurrent), by direction (medial, lateral, posterolateral, or anterior), by associated injuries (fracture of the coronoid, radial head, capitellum, medial and lateral epicondyle, disrupted proximal radio-ulnar joint), or by mechanism of injury (hyperextension, axial compression, valgus and supination, or valgus stress).



**Figure 3** Anteroposterior radiograph of the dislocated elbow. Reproduced with permission.



**Figure 4** Lateral radiograph of the dislocated elbow. Reproduced with permission.



**Figure 5** Radiograph of the elbow treated with external fixator and transolecranon pin. Reproduced with permission.

Clinical evaluation includes careful history of the injury and the events causing instability and examination, including the valgus stress test and the posterolateral instability test.<sup>6</sup> Routine anteroposterior and lateral radiographs may reveal associated bony injuries, degenerative changes, and heterotrophic bone around the elbow. In posterolateral rotatory instability, the lateral view with the forearm supinated may show widening of the humero-ulnar joint with inferior subluxation of the radial head.

Magnetic resonance imaging and computed tomography arthrogram may be used to identify ligamentous injuries.<sup>8</sup> EUA may be indicated when the history is convincing or the clinical examination unhelpful.

## LESSONS LEARNT AND BEST PRACTICE

Our patient had clearly suffered a significant ligamentous injury, which allowed the elbow to dislocate, reduce spontaneously, and then subsequently redislocate. This case highlights that recurrent elbow dislocations can present in joint. Although the initial radiographs showed that the elbow was not dislocated, careful scrutiny of the radiographs retrospectively showed tiny bony avulsions from the medial and lateral epicondyles. This suggested that the elbow had

sustained significant ligament injury, which may predispose to recurrent instability. This needs to be specifically noted on the initial *x* rays. The subsequent attendance two days later was documented as showing deformity of the elbow. The mistake was in assuming that the patient's condition remained the same and that there was no need to re-evaluate the situation and take more *x* rays. The elbow was almost certainly dislocated at this stage. It emphasises the need to assess the patient on his or her own merits despite previously normal investigations.

Once the elbow was found to be dislocated at three weeks, a reduction in theatre would have been more appropriate as an EUA at this stage would have revealed the gross instability and a stabilising procedure could have been performed forthwith. Finally, the patient was referred to the fracture clinic with a two week appointment. All dislocated joints should be seen at the next available clinic, no more than a few days from the injury, to ensure that the relocated joint remains in place.

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## Right upper lobe consolidation: an unusual complication of an uneventful endotracheal intubation

**D Sunderamoorthy, S Ahuja, A Grant, T Mian**

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A 28 year old fit and healthy Caucasian man had a Bankart's repair of the left shoulder under general anaesthetic for a recurrent dislocation of the shoulder. The operative procedure was uneventful. Following extubation he was tachycardic and saturation dropped in the recovery room. The chest radiograph revealed shadowing in the right lung and he was diagnosed to have right middle lobe collapse. Subsequently the radiograph was reported as right upper lobe consolidation by the radiologist. We wish to report this unusual complication and the difficulty in diagnosis of such a complication occurring following an uneventful anaesthetic.

Cuffed endotracheal tubes are used for airway management in the operating room, emergency room, and intensive care unit. Anaesthetist and intensivists are aware that right main stem intubation may precipitate left-sided pulmonary atelectasis.<sup>1</sup> Inadvertent right main bronchus intubation can also cause paradoxical collapse of a portion of the right upper lobe.<sup>2</sup> There had been reports of right upper lobe collapse occurring after an uneventful endotracheal intubation.<sup>3</sup> There had also been reports of right middle lobe collapse occurring, in isolation or in combination with a right upper lobe collapse, after an uneventful endotracheal intubation. Right upper lobe consolidation as

a complication of endotracheal intubation has not been reported. We wish to report such a complication occurring in a young man who had an uneventful endotracheal intubation where he was thought to have had a right middle lobe collapse and subsequently reported to have right upper lobe consolidation. The radiological features of right middle lobe collapse and right upper lobe consolidation and ways to diagnose them are discussed.

#### CASE REPORT

A 28 year old Caucasian underwent a Bankart's repair of the left shoulder under general anaesthetic for a recurrent dislocation of the shoulder. He was fit and healthy non-smoker with no premorbid medical conditions (American Society of Anaesthesiologists (ASA) Grade 1). He had an uncomplicated endotracheal intubation and his electrocardiogram (ECG), blood pressure, SaO<sub>2</sub>, ETCO<sub>2</sub>, and oxygen inspiration were satisfactory throughout the operative period. The operative procedure was uneventful. Following extubation he was tachycardic and SaO<sub>2</sub> was 82% in the recovery room. Clinically there was reduced air entry over the apex and the middle of the right chest with good air entry in the lung base. He was resuscitated immediately and his SaO<sub>2</sub> improved

**Abbreviations:** ASA, American Society of Anaesthesiologists; ECG, electrocardiogram





**Figure 1** Anteroposterior radiograph showing right upper lobe consolidation. Reproduced with permission.

to 95% (60% oxygen inhalation). ECG was normal and an x ray of the chest (fig 1) revealed shadowing in the right lung, which was thought to be right middle lobe collapse. Postoperatively he was treated with intravenous antibiotics, humidified oxygen, and chest physiotherapy. He was discharged home after 5 days of treatment. At 2 weeks follow up he had no chest problems and a chest radiograph showed complete expansion of the lung (fig 2). The radiologist reported his chest radiographs as right upper lobe consolidation (fig 1).

## DISCUSSION

Right bronchial airway obstruction is an infrequent complication of endotracheal intubation. Following emergency resuscitation procedures, it is more common than generally acknowledged.<sup>4</sup> The upper lobe bronchus on the right side arises as an offshoot from the right main bronchus, whereas the left upper lobe bronchus arises further away from the carina as a bifurcation of the main trunk. In adults, the right main bronchus is only 2 cms and shorter. The right upper lobe bronchus can also arise from the lower end of the trachea. Therefore, occlusion of the upper lobe opening occurs when the right lobe bronchus is inadvertently intubated.<sup>5</sup>

Complete obstruction of the left main stem bronchus produces rapid atelectasis of the lung.<sup>5</sup> Collapse of the right upper lobe is a rarely reported form of lobar atelectasis in the intubated patient. This usually follows inadvertent intubation of the right main bronchus. Seto *et al* observed that main stem intubation does not cause immediate collapse of the left lung, right lung preferential ventilation does not preclude right upper lobe collapse, and right upper lobe collapse can occur very rapidly after inadvertent intubation of the right main stem bronchus.<sup>2</sup>

Numerous mechanisms have been hypothesised: anatomical considerations of the right upper lobe bronchus, Bernoulli-type mechanism, compression of the upper lobe from hyperinflation of the lower lobes, and resorption atelectasis are the various factors leading to the right upper lobe collapse following intubation.<sup>2-3</sup> Halpern *et al* had reported the aetiology as multifactorial. This complication can occur at any time following an eventful or uneventful intubation.<sup>3</sup>

In our patient, the chest radiographs taken in the recovery room showed homogenous opacity in the right middle zone and the right heart border could not be seen. A diagnosis of right middle lobe collapse was made. Seto *et al* had reported right middle lobe collapse, either alone or in combination with right upper lobe collapse, occurring as a complication of inadvertent right bronchus intubation.<sup>2</sup>

It was a surprise when the radiologist reported the radiographs as right upper lobe consolidation. There has been no report of patients developing acute right upper lobe



**Figure 2** Radiograph showing the cleared consolidation. Reproduced with permission.

consolidation following an endotracheal intubation. In right middle lobe collapse the horizontal fissure and lower half of the oblique fissure move towards one another. In right upper lobe consolidation, the consolidation is confined by the horizontal fissure inferiorly and the upper half of the oblique fissure posteriorly. This can be best seen in the lateral projection. The signs of right middle lobe collapse are subtle in frontal projection and hence the difficulty in differentiating between right upper lobe consolidation and right middle lobe collapse.

In our patient the ventilation was uneventful throughout the operative procedure. One probable explanation was that there might have been a mucous plug obstructing the right upper lobe bronchus causing the consolidation and then clearing subsequently with treatment. Another possible reason was that the endotracheal tube might have displaced when transferring the patient from the operating table to the bed. This could lead to the endotracheal tube migrating into the right main bronchus thereby causing a collapse of the right upper lobe and then the patient subsequently developing an acute consolidation. If there is doubt in the differentiating between a collapse and a consolidation on an anteroposterior view of a chest radiograph, obtaining a lateral view would help in diagnosis for the reasons described earlier.

Anaesthetists, intensivists, and the emergency physicians need to be aware of right upper lobe consolidation occurring as complication of endotracheal intubation and the difficulty in the radiographic diagnosis of such a complication.

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# Traumatic atlantoaxial rotatory subluxation

T B Crook, C A Eynon

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Atlantoaxial rotatory subluxation should be considered in the presentation of traumatic torticollis. This case report discusses the characteristic radiographic findings and appropriate management.

**T**orticollis is a relatively frequent presenting sign to an emergency department. It describes lateral flexion of the neck and contralateral rotation, with a variable degree of flexion. The causes may be divided into traumatic and non-traumatic. The differential diagnosis of non-traumatic torticollis should include, particularly in children, congenital cervical spine anomalies, head and neck infection (for example, otitis media, pharyngitis, or retropharyngeal abscess), and tumours in the posterior fossa or upper cervical spine.<sup>1</sup> Other causes include drug-induced torticollis—for example, with phenothiazines—and the movement disorder spasmodic torticollis. Traumatic causes of torticollis include atlantoaxial rotatory subluxation, atlantoaxial dislocation, cervical vertebral fractures, and injury to the cervical musculature.

This article highlights a case of atlantoaxial rotatory subluxation with the aim of improving awareness of this condition to enable early recognition. Appropriate management options and outcomes are discussed.

## CASE HISTORY

A 15 year old girl tackled an opponent while playing rugby. During the tackle her head was forcibly extended and rotated to the right. She attended her local emergency department complaining of neck pain with her neck in fixed rotation to the right. She had no neurological sequelae. Plain x rays showed abnormality at the atlantoaxial joint (fig 1). A computed tomography (CT) scan confirmed a 45° rotatory subluxation of C1 on C2, with forward subluxation of the lateral mass of the atlas off the axis on the left and posterior subluxation on the right (fig. 2). The atlantodental interval was not increased and no fracture was identified. The patient was transferred to the regional neurosurgical centre. Management consisted of analgesia, sedation, and application of halo skull traction. This allowed clinical reduction of the subluxation within one hour of application. Reduction was confirmed by CT scan. The patient remained in traction for six days before being mobilised in a rigid neck collar. She was discharged without further problems after 11 days. The rigid collar was removed after eight weeks. Outpatient review at three months revealed a full range of painless neck movement.

## DISCUSSION

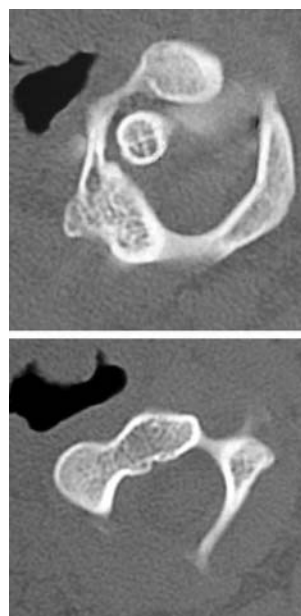
The atlantoaxial joint primarily facilitates rotation and is stabilised in the anteroposterior plane by the transverse ligament and joint capsule. The alar ligaments, which pass from the lateral occipital processes to the posterolateral margin of the odontoid apex, prevent anterior shift of the atlas on the axis but mainly function in preventing excessive rotation at the atlantoaxial joint. There is evidence from magnetic resonance imaging to suggest that alar ligament disruption is the mechanism by which rotatory subluxation occurs.<sup>2,3</sup> The lateral mass of the atlas rotating posteriorly



**Figure 1** Plain anteroposterior x ray of cervical spine demonstrating torticollis with chin directed to the right.

locks behind the ipsilateral lateral mass. Conditions with associated ligamentous laxity or congenital atlantoaxial abnormalities therefore carry an increased incidence of rotatory subluxation. These include Down's syndrome, Morquio's syndrome, Marfan's syndrome, and rheumatoid arthritis. Grisel's syndrome describes non-traumatic subluxation of the atlantoaxial joint from inflammatory ligamentous laxity following an infectious process.<sup>4</sup>

Traumatic atlantoaxial rotatory subluxation is predominantly a paediatric phenomenon, with rare occurrence in adults.<sup>5,6</sup> It should be included in the differential diagnosis of patients presenting with torticollis following even minor trauma. Two classification systems for rotatory subluxation



**Figure 2** Computed tomography scan of cervical spine showing approximately 45° rotation of the skull and C1 to the right in relation to C2. Approximately three quarters of the C2 facet is exposed.

have been described: the Fielding and Hawkins system,<sup>7</sup> and that of White and Panjabi.<sup>8</sup> These are based on radiological findings, describing the direction of atlantal displacement and the pivotal axis.

Radiographic features of rotatory subluxation are of persistent asymmetry of the odontoid peg in relation to the lateral masses of the atlas, uncorrected by head rotation. Plain x rays of the cervical spine will detect approximately 93% of rotatory subluxations compared with a detection rate of 54% with CT. When combined, a sensitivity of 99% has been described.<sup>9</sup> Complex motion tomographic studies or dynamic CT have also been recommended for the diagnosis of rotatory subluxation.<sup>6,9</sup>

Acute traumatic atlantoaxial rotatory subluxation is often reduced easily.<sup>2,6,10</sup> The length of time between injury and reduction appears to correlate with rates of recurrence and failure of reduction by non-surgical techniques.<sup>10</sup> Evidence supports the use of early cranial traction in conjunction with benzodiazepines.<sup>6,10</sup> This is followed by external immobilisation (Philadelphia collar or Guilford orthosis) for six weeks. The patients achieve good long term stability.<sup>6,10</sup> In cases of recurrence, further cranial traction followed by external immobilisation (once reduced) for three months is advocated.<sup>10</sup> For recurrent or irreducible subluxation, open reduction and posterior atlantoaxial fusion may be required.<sup>6,7,10</sup>

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## Primary intraventricular haemorrhage in an 11 year old child

B S Rayen, F M Russell

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An 11 year old boy walked into our accident and emergency department with his mother having woken up with headache four hours ago. He was known to have mild learning difficulties for speech and language, but was otherwise previously fit and healthy. He was not on any medications, and he did not have any history of trauma. He had vomited twice at home. On initial examination, he did not have a high temperature, rash, or meningeal symptoms, and his Glasgow Coma Scale score was E4 M6 V5. He had no neurological signs or papilloedema and the pupils were normal. He had no neck stiffness or meningeal signs. His pulse rate was 60 per minute and a subsequent electrocardiogram showed sinus bradycardia. His blood pressure was normal.

During further examination his conscious level deteriorated. He became vague and disinterested, and then he began babbling. He had a fluctuating conscious level, at best occasional spontaneous eye opening, localising to pain with inappropriate words. His respiratory rate was normal and he was maintaining his airway. At this point, the most likely clinical diagnosis was acute encephalitis and he was treated with intravenous cefotaxime and aciclovir. Investigations including full blood count, urea and electrolytes, C-reactive protein, glucose, liver function tests, and clotting profiles

were normal. An urgent computed tomography (CT) scan of brain revealed recent haemorrhage in predominantly the right lateral, third, and fourth ventricles (fig 1). There was no evidence of acute hydrocephalus. Following our initial management he was transferred to the neurosurgical unit for continuing care. He then developed mild weakness of his left limbs. He underwent further CT scanning of brain and was treated conservatively. The CT scan and a cerebral angiogram performed at a later date did not reveal any underlying cause, such as an arteriovenous malformation or aneurysm.

Four months later the mild left sided weakness persists and it was planned to carry out magnetic resonance imaging (MRI) after six months of the haemorrhage to exclude an angiographically occult lesion such as cavernoma. The MRI confirmed the findings of the CT scan performed previously.

Spontaneous intracranial haemorrhage is rare in adolescence. Primary intraventricular haemorrhage is an uncommon subgroup. Little is known about the clinical and imaging features of primary intraventricular haemorrhage. Livingston *et al* reported that the clinical features in this condition were not specific, but that altered consciousness, headache, vomiting, and focal signs were common.<sup>1</sup> Vascular malformations may result in intraventricular haemorrhage in children. Such presentation accounts for 3% of adult





**Figure 1** Computed tomography scan of an 11 year old boy presenting with spontaneous intracranial haemorrhage.

intracranial bleeds but relevant data about this association have not been reported in children.<sup>2</sup> Certain prognostic factors like initial level of consciousness,<sup>3</sup> early hydrocephalus,<sup>4</sup> and delay in diagnosing intracranial aneurysms<sup>5</sup> may adversely affect the outcome with severe neurological deficits

and high mortality. In our case an early cranial CT scan ensured urgent neurosurgical referral. The absence of the above-mentioned prognostic factors might explain the reasonable outcome in our patient with little in the way of neurological deficit. Therefore to diagnose this serious condition, a high degree of clinical suspicion and a low threshold for early use of CT is essential, particularly in children who have difficulties with expression and speech.

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## An unusual presentation of baclofen overdose

C-F Chong, T-L Wang

*Emerg Med J* 2005;**22**:673–674. doi: 10.1136/emj.2003.011908

Baclofen has become increasingly popular in the treatment of spasticity disorders. Its availability for misuse has also increased. We report a case of baclofen overdose in a 20-year-old man, who manifested atypical symptoms of baclofen overdose—that is, delirium and rhabdomyolysis. He was treated successfully with full supportive management, and was discharged from the hospital on the 12th day following admission. If a past medication history is not immediately available, baclofen overdose should be included in the differential diagnosis of an acutely confused patient complicated with rhabdomyolysis, as routine toxicology screening does not include baclofen.

**B**aclofen is commonly used to treat spasticity. Reported adverse effects of its overdose include somnolence, coma, seizures, respiratory depression, and cardiac conduction abnormalities. We describe a patient with baclofen overdose presenting to the emergency department (ED) with atypical symptoms of acute delirium and rhabdomyolysis.

#### CASE REPORT

A 20-year-old man was found lying unconscious on the floor in his apartment and was brought to the emergency department by ambulance. Initially, no past medical history could be obtained. Physical examination at admission showed a young man with obvious disorientation (Glasgow coma scale score 13). Vital signs were: temperature 37.4°C, blood pressure 120/70 mmHg, pulse 92 beats/min, and respiratory rate 24 breaths/min. Pupils were 2.5 mm diameter bilaterally, with prompt reaction to light. External ocular movements were full and free. Nuchal rigidity or carotid bruits were not detected. Findings on examination of the heart, lungs, vascular system, and abdomen were unremarkable. Neurological examination revealed full strength of both upper and lower limbs equally. Knee jerks were normal, but ankle jerks were decreased bilaterally. Plantar responses were bilaterally downward (Babinski's sign). Light touch and pinprick sensation, cranial nerve examination, and cerebellar examination could not be performed because of the patient's restlessness. Laboratory tests revealed normal haemoglobin and platelet counts but the

**Abbreviation:** CPK, creatinine phosphokinase

white blood cell count was elevated at  $15.6 \times 10^3$  cells/mm<sup>3</sup>. Glucose, blood urea nitrogen, creatinine, sodium, potassium, and ionised calcium were within normal limits. Initial creatinine phosphokinase (CPK) level was greater than  $10 \times 10^3$  IU/L (normal range 24–204), and test for urine myoglobin was positive. Urine drug screen was negative for narcotics, cocaine, and amphetamines. ECG showed sinus tachycardia. Computed tomography of the head and cerebrospinal fluid studies were negative.

The patient was admitted to the intensive care unit with an initial diagnosis of meningoencephalitis and rhabdomyolysis. In addition to supportive management, he also received vigorous hydration, alkaline diuresis, and empirical antibiotics for coverage of probable meningitis. Renal function remained normal and CPK rapidly fell to within normal limits 2 days after admission. He was transferred to the medical ward on day 3 and was able to recall that he had ingested 30 pills of baclofen (Befon; 5 mg/tablet) in a suicide attempt about 36 hours before his arrival to the ED. He admitted that he irregularly took baclofen (5–10 mg a day) for treatment of spasticities due to amyotrophic lateral sclerosis. Co-ingestion of other drugs or alcohol was denied. The patient also had pains, swelling, and some pressure sores in his left buttock and thigh. An electroencephalogram performed 7 days after admission revealed diffuse cortical dysfunction compatible with encephalopathy. Other studies performed included somatosensory evoked potentials and magnetic resonance imaging of the brain, which were normal. He was discharged in excellent neurological function on day 12.

## DISCUSSION

Baclofen, a lipophilic analogue of the naturally occurring neurotransmitter gamma-aminobutyric acid (GABA), is the drug of choice for treatment of spasticity from spinal cord lesions and multiple sclerosis. It appears to act as an agonist at bicuculline insensitive GABA receptors in the spinal cord to decrease neurotransmitter release from presynaptic terminals.<sup>1</sup>

After a single therapeutic dose, baclofen is rapidly absorbed from the gastrointestinal tract. Blood levels peak within 2 hours. The serum half-life is 2–6 hours, which can be significantly prolonged after an overdose. The majority of this drug is excreted unchanged in the urine.<sup>2</sup> Signs of toxicity have been reported after ingestion of as little as 100 mg of

baclofen.<sup>3</sup> Adverse effects of baclofen overdose are well defined in the literature, and include somnolence, coma, seizures, encephalopathy, respiratory depression, flaccidity, hyporeflexia, and cardiac conduction abnormalities.<sup>4</sup> None of the patients described previously in the literature presented with rhabdomyolysis and acute delirium as in our case. Although uncommon, agitated confusion can be a pattern of baclofen encephalopathy. The mechanism for rhabdomyolysis in this case is uncertain. Drugs impairing the central nervous system, including baclofen, can cause rhabdomyolysis by pressure induced ischaemia due to prolonged immobilisation and muscle compression. This is supported in our case by the presence of pressure sores on the patient's left gluteus and thigh. Unwitnessed seizures may also have contributed to muscle breakdown in this case.

In conclusion, in the absence of detailed past medication history, an acutely confused patient complicated with rhabdomyolysis should have baclofen overdose included in the differential diagnosis, as routine toxicology screening does not include baclofen. Such atypical signs should be recognised early, as drug discontinuation and full supportive treatment result in good outcome, provided no hypoxic or ischaemic insult has occurred before medical attention.

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# An unusual swelling in the neck

**A A Abbasi, N S Harrop**

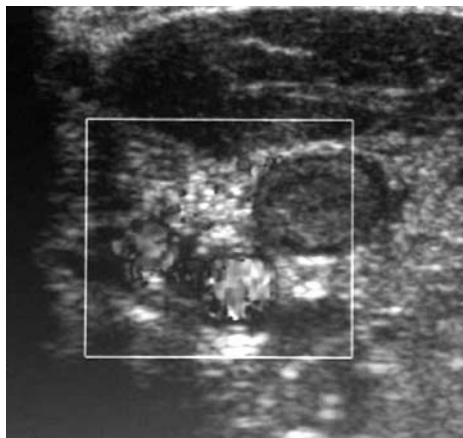
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Venous thrombosis is a fundamental pathological entity. Our patient provides an opportunity to consider etiology in terms of Virchow's classic triad. We also draw attention to the effort syndrome, in which recurrent, vigorous exertion of an upper extremity is thought to produce venous thrombosis by virtue of local endothelial trauma.

## CASE HISTORY

A 44 year old man was referred by his General Practitioner to the A&E department with a 4 to 5 day history of left-sided neck swelling, symptoms of pleuritic chest pain, and weight loss of one stone.

He had just returned from holiday in Africa and had suffered from altitude sickness whilst climbing Mount



**Figure 1** Showing an extensive thrombus within the left juglar vein, which extends to brachiocephalic and left axillary veins.

Kilimanjaro. He had then suffered a prolonged bout of diarrhoea.

At a first visit, the GP had advised fluids because of dehydration. After a few days, the patient developed swelling in the left supraclavicular region with intermittent chest pain. He was, therefore, referred to A&E.

On examination he looked well. There was a non-pulsatile, diffuse swelling, in the left supraclavicular region. It extended to the base of the neck. Its margins were not palpable. The swelling was red, hot, and tender. There was no lymphadenopathy.

The chest was clear and the heart sounds were pure. Abdominal examination was unremarkable.

He was thought to have an abscess. However, chest x rays showed broadening of the left superior mediastinum with a small left basal effusion. Ultrasound of the neck demonstrated extensive thrombus in the left jugular, subclavian, and axillary veins (figure 1). A thoracic CT scan demonstrated diffuse tissue swelling in the left side of the neck and superior mediastinum. CT did not disclose any lymphadenopathy. There were bilateral pleural effusions, more so on the left (figure 2).

The patient was admitted to a medical ward and he was treated for venous thrombosis with low molecular weight heparin initially, then Warfarin. The swelling improved. His CRP, initially significantly elevated (200), returned to normal. His full blood count, renal functions and liver functions were normal.

The patient was discharged after 5 days of hospital admission with advice to continue Warfarin for 6 months. He will have another CT scan after 6 weeks.

## COMMENT

Spontaneous thrombosis of the axillary or subclavian veins was first postulated as a cause of upper extremity pain and swelling by Sir James Padget in 1875.<sup>1</sup> Von Schroetter demonstrated thrombotic occlusion of upper limb veins, hence the Padget-Schroetter syndrome. More recently, the association between strenuous repetitive movement of the upper extremity and axillosubclavian thrombosis has been recognised and termed effort syndrome.<sup>2</sup> Apart from iatrogenic



**Figure 2** Showing an extensive thrombus within the left juglar vein, which extends to brachiocephalic and left axillary veins.

factors such as catheter placement, venous thrombosis of the axillary, or subclavian vein may be caused by external compression by an anatomical structure such as a congenital cervical rib or by the pathological development of a local tumour.<sup>3</sup>

Virchow's triad explains thrombosis in terms of venous stasis, vessel wall abnormality or disorders of the blood itself.<sup>4</sup>

In this patient, it is suspected that venous stasis may have been added to local tissue trauma. The patient had worn a rucksack, possibly obstructing venous flow as well as causing trauma to the vessel wall. He had suffered altitude sickness and it is of interest that hypoxia may also produce endothelial injury.<sup>5</sup> Finally, he had suffered a period of significant dehydration likely to have been attended by hemoconcentration and increased blood viscosity. In other patients, it is important to bear in mind that the occurrence of thrombosis at an unusual anatomic location may provide the first evidence of a hypercoagulable state, especially if there is a previous personal or a positive family history of thrombosis.<sup>6</sup> Suitable investigations would then include a haemophilia screen.

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# Butane encephalopathy

D Harris, Z Mirza

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Volatile solvent abuse (VSA) is defined as the “intentional inhalation of a volatile substance for the purpose of achieving a euphoric state”. The lifetime prevalence of VSA in the UK remains steady at around 15%, the fourth highest rate in Europe, and VSA is the most common form of drug abuse in the 11–15 year age group in England and Wales. A 13 year old girl presented to the accident and emergency unit following inhalation of butane based deodorant, which resulted in a prolonged semiconscious state with encephalopathic symptoms.

Volatile solvent abuse (VSA) is defined as the “intentional inhalation of a volatile substance for the purpose of achieving a euphoric state”.<sup>1</sup> The lifetime prevalence of VSA in the UK remains steady at around 15%, the fourth highest rate in Europe,<sup>2</sup> a figure aided by the ready availability of these products. We present the case of a 13 year old girl who presented to the accident and emergency (A&E) unit following inhalation of a large quantity of butane based deodorant, which resulted in a prolonged semiconscious state with encephalopathic symptoms.

## CASE REPORT

The patient, who was markedly obese (body mass index >30) was brought by ambulance to A&E, having been found unconscious on her bedroom floor by her mother. On arrival, the patient was agitated and combative with a patent airway, tachycardia (131 beats/min), and tachypnoea (28 breaths/min). She was normotensive with a low grade pyrexia (37.6 °C tympanic). Her Glasgow Coma Score (GCS) level was 10/15 (E2/4, M5/6, V3/5).

Arterial blood gas analysis, performed on a sample taken from the patient’s right radial artery, showed respiratory acidosis (pH 7.35, pO<sub>2</sub> 10.4 kPa, pCO<sub>2</sub> 7.2 kPa on 5 litres oxygen/minute via face mask). Venous blood tests (full blood count, clotting screen, urea and electrolytes, liver function tests, bone profile, paracetamol and salicylate levels, random glucose, C reactive protein, and amylase) were within normal limits. Further examination found four superficial “deliberate self harm” scars on her forearms. Her mother reported that she had recently been the victim of bullying at school and that the third anniversary of her father’s death was approaching. The paramedic crew had found two new, but empty cans of a commercial deodorant at her bedside, the principle inactive components of which are butane and hydrofluorocarbon 152A, which act as propellants. An electrocardiogram showed sinus tachycardia, and chest x ray showed no abnormalities.

After consultation with the paediatric consultant on call, prophylactic broad spectrum intravenous antibiotics and antivirals were commenced, assuming a possible infective cause for her symptoms. Prophylactic corticosteroids were not given.

The patient remained stable 18 hours post-admission, but with a GCS of 10/15 (E2/4, M5/6, V3/5). Lumbar puncture was performed and cerebrospinal fluid (CSF) analysis

showed no organisms, normal glucose, and slightly elevated protein. Blood, urine, and sputum samples were sent for microscopy, culture, and sensitivity, and a urine sample was sent for “drugs of abuse” screening (amphetamines, opiates, barbiturates, benzodiazepines, cannabinoids, and cocaine), all of which were found to be negative.

By 24 hours post-admission, the patient was alert but experiencing episodes of drowsiness and disorientation. She also had a marked nystagmus, and on day 3, when she was first able to stand, she was ataxic with slurred speech. Magnetic resonance imaging of her brain performed 4 days after admission was unremarkable. Electroencephalogram performed the same day found abnormal delta and theta waveforms, consistent with a drug induced encephalopathy.

While in hospital, the patient was fully assessed by the paediatric psychiatry team, during which she admitted regular solvent misuse. It appeared that the various recent traumatic events in her life had provoked a significant increase in the amount of solvent used. She denied using any other recreational drugs or alcohol.

She was discharged 14 days after first presenting, the length of admission partly explained by her complicated social and psychological problems. To facilitate the administration of her antibiotics for a further week, a peripheral long line was placed in her distal right saphenous vein. All CSF, blood, sputum, and urine samples sent for microbiology had shown no abnormalities or growth.

In view of her investigation results and her admission to butane use, a diagnosis of butane induced encephalopathy was made, one similar case having been reported previously in the medical literature.<sup>3</sup> Despite the elimination half life of butane being around 10 minutes, the prolonged duration of her symptoms could well be explained by her obesity, the large quantity of butane inhaled, and the extreme lipophilic nature of the misused solvent.<sup>4</sup> The decision to continue antibiotic treatment was made on the basis that as little was known by the clinical teams about solvent use and its effects, thus as an additional safety measure the normal infection protocols should be adhered to despite all investigations showing no infective process.

## DISCUSSION

Butane is a low molecular weight aliphatic hydrocarbon and the principle propellant used in spray on deodorant since the elimination of chlorofluorocarbons. It is also the most commonly misused volatile solvent in the UK, and was the cause of 52% of the 64 known solvent related deaths in 2000.<sup>5</sup> Of great concern is that 43% of these deaths occurred in young healthy subjects who were trying VSA for the first time. The majority of people who misuse solvents are aged between 12 and 17 years, with a peak around 15 years, although there are recorded cases of use among children as young as 7 years.

While there are multiple case reports of cardiac and trauma related deaths from VSA in Europe and the USA,<sup>7–9</sup> neurological complications are less common, with only one

**Abbreviations:** A&E, accident and emergency; CSF, cerebrospinal fluid; GCS, Glasgow Coma Score; VSA, volatile solvent abuse

**Table 1** Clinical effects of butane inhalation

CNS depression / acute confusional state
Cardiac arrhythmias
Nausea and vomiting
Dizziness
Headache
Abdominal pain
Ataxia
Muscle weakness
Photophobia
Hallucinations
Diplopia
Insomnia
Depression
Fatigue

Collated from reference 3, 4, 7–11, and 13.

previous report of butane associated acute encephalopathy,<sup>3</sup> one of chronic cerebral atrophy (neurodegeneration),<sup>10</sup> and one of prolonged hemiparesis.<sup>15</sup>

Cardiac arrhythmias and vagal stimulation leading to cardiac arrest, along with anoxia, oedema, and respiratory depression<sup>11</sup> are the principal causes of death. By spraying butane directly into the throat, the jet of fluid can cool rapidly to  $-20^{\circ}\text{C}$  by expansion, causing prolonged laryngospasm.<sup>13</sup> "Sudden sniffing death syndrome", first described by Bass in 1970,<sup>12</sup> is the commonest single cause of solvent related death, resulting in 55% of known fatal cases.<sup>13</sup> The myocardium is hypersensitized to epinephrine by the inhaled hydrocarbon, so that any sudden stimulation or excitation of the user can result in arrhythmias brought on by intrinsic epinephrine release. This also explains the poor outcome of attempted resuscitations during which more epinephrine is routinely administered. Edwards and Wenstone<sup>14</sup> suggest the use of amiodarone over epinephrine in cardiac arrest where VSA is suspected.

The protean presentation of VSA in A&E (table 1) and the lack of awareness among clinical staff mean the true underlying cause of presentation is often not realised. Very few people who present will have any obvious aroma of solvent about them or a perioral "huffer's rash". Greater awareness of VSA may aid in the management of young people who present to A&E with unusual symptom patterns. Knowledge of the contraindication of epinephrine for cardiac arrest in VSA, earlier definitive airway control and the need to nurse patients in softly lit and quiet areas, where excitation and stimulation are less likely, will improve the often poor outcome in resuscitation following VSA.

The enormous number of household products open to misuse and the exclusion of VSA in most drug awareness

programmes mean that eradication of solvent misuse is unlikely. Legislation such as The Intoxicating Substance Supply Act of 1985 and The Cigarette Lighter Refill Regulation Act of 1999 are difficult to enforce, with only 53 convictions since their introduction.

The high prevalence and varied clinical presentation of volatile solvent misuse will continue to present a challenge to emergency physicians until the technical problems of developing a non-toxic solvent can be overcome.

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# Hyperthermia associated with central anticholinergic syndrome caused by a transdermal hyoscine patch in a child with cerebral palsy

A Frampton, J Spinks

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Patients frequently present to the Emergency Department with elevated body temperature; the distinction between “fever” and “hyperthermia” is vital. Many commonly prescribed drugs can cause hyperthermia.<sup>1</sup> The goal of treatment is to reduce core temperature and oxygen demand before organ damage occurs.<sup>2</sup> We present the case of a 14 year old boy who presented with hyperthermia due to a transdermal hyoscine patch used to control his symptoms associated with cerebral palsy.

## CASE

A 14 year old boy with spastic quadriplegia presented to the Emergency Department (ED) having been found unresponsive at home in the early hours of the morning. On arrival in the ED he was drowsy and agitated with dilated but reactive pupils and a fever of 42°C. His heart rate was 120/min, blood pressure 100/70 and capillary refill time instant. No rash or neck stiffness was present but it was noted that despite the high fever he was not sweating and he had dry mucous membranes. The presence or absence of urinary retention was not commented on at initial examination. Examination was otherwise unremarkable except for the presence of a gastrostomy feeding tube and a hyoscine patch sited on the skin behind his ear to control oropharyngeal secretions, which was removed. His parents stated that he had been well the previous evening and had no other recent symptoms. The hyoscine patch was not a recent addition to his medication and no previous problems with its use were reported by the parents.

His temperature was rapidly reduced using paracetamol and fanning and following a 20 ml/kg bolus of fluid he became more responsive and his tachycardia settled. Two sets of blood cultures, urine, and sputum were sent for culture and broad spectrum antibiotics (a cephalosporin and metronidazole) were commenced. Lumbar puncture was not felt to be indicated immediately in the ED because of the initial rapid improvement (subsequent to the blood results being made available, LP was not undertaken because of the significant coagulopathy) and chest x ray was unremarkable. He was transferred to the Paediatric High Dependency Unit for observation.

Over the course of the day he remained apyrexial. However, his urine output and mental state deteriorated and he became hypotensive. It became clear that he was developing multi-organ failure. He was transferred to the Paediatric Intensive Care Unit (PICU).

On PICU, he required invasive ventilation and cardiovascular support with moderate doses of adrenaline and noradrenaline. He developed rhabdomyolysis with a peak serum creatine kinase of 70,000U/l and myoglobinuria. He also developed anuric renal failure, for which he required continuous veno-venous haemofiltration for 7 days, and acute hepatic failure with a coagulopathy.

No source of sepsis was ever identified despite multiple sets of blood and sputum culture (including sputum for viral organisms) and his temperature remained below 38°C. Antibiotics were stopped 72 hours after his admission to

PICU and his condition improved with supportive therapy alone. Urine was not sent for toxicology, however, he had no access to any drugs other than those prescribed to him and administered by his parents. In the absence of any proof of sepsis and in view of the pattern of deterioration in his condition we diagnosed multi-organ failure secondary to hyperthermia in association with central anti-cholinergic syndrome caused by the hyoscine patch. He made a full recovery.

## DISCUSSION

Fever is an elevation of body temperature that exceeds the normal daily variation and occurs in conjunction with an increase in the hypothalamic set point. Hyperthermia, in contrast is characterised by an unchanged (normothermic) setting of the thermoregulatory centre in conjunction with an uncontrolled increase in body temperature that exceeds the body's ability to lose heat. Heat exposure, increased endogenous heat production (vigorous exercise, malignant hyperthermia) and decreased heat loss (over wrapping in blankets, anticholinergic medications) may precipitate hyperthermia.<sup>1</sup>

The primary injury in hyperthermia is due to cellular toxicity of temperatures above 42°C. Above this temperature cell function deteriorates due causing widespread damage to all the major organ systems.<sup>3</sup> The primary goal of treatment is rapid cooling either by evaporative or direct thermal methods.<sup>1</sup>

Central anticholinergic syndrome (CAS) is a potentially life-threatening syndrome caused by toxicity from a variety of drugs with anticholinergic effects, most notably atropine and hyoscine.<sup>4</sup> It is produced by the inhibition of cholinergic neurotransmission at muscarinic receptor sites centrally or peripherally. Central nervous system manifestations range from an agitated or excitatory state with hyperthermia to a state of extreme depression with decreased respiratory drive or coma.<sup>5</sup> Peripheral manifestations include sinus tachycardia, anhidrosis, functional ileus, urinary retention, hypertension, tremulousness, and myoclonic jerking.<sup>6</sup> Hyperthermia, which is a feature in over 25% of cases,<sup>7</sup> can be caused by the central action of the drug in combination with absence of sweating which raises the temperature further. Common signs and symptoms may be remembered by the mnemonic – “red as a beet, dry as a bone, blind as a bat, mad as a hatter and hot as a hare”. CAS has been reported following intentional overdose, inadvertent ingestion, geriatric polypharmacy and an idiosyncratic reaction to drugs with anticholinergic properties.<sup>8</sup> Systemic effects have been reported with topical eye drops.<sup>9</sup>

This case has been reported to the committee for safety of medicines using the yellow card reporting system and to the authors' knowledge this is the first report in the literature of CAS occurring as a result of a transdermal hyoscine patch in a child with cerebral palsy. The authors are aware of two other cases occurring in different centres; unfortunately the information is anecdotal only and no details of these cases are available to us. The only previous case report of CAS in a child we have been able to find is that of a 9 year old boy who

developed CAS secondary to a transdermal hyoscine patch after an anaesthetic.<sup>10</sup>

No specific diagnostic studies exist for anticholinergic syndrome and serum drug concentrations are not helpful and rarely available to aid in the initial management. The antidote for anticholinergic toxicity is physostigmine salicylate. However, this drug has potentially serious cardiac side effects and is no longer recommended for use.

Prolonged hyperthermia may lead to devastating multi-organ failure and rapid cooling is essential to reduce the damage caused by excessively high temperatures. CAS has been previously well recognised in the elderly, but has rarely reported in children. Hyoscine patches are commonly used to control symptoms in children with cerebral palsy, often to good effect. The possibility of this syndrome, however, should be considered in any child on anticholinergic medications with a fever or the other symptoms described, particularly in the absence of sweating.

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# Cardiovascular complications induced by cannabis smoking: a case report and review of the literature

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Cannabis is generally considered a drug of low toxicity. Although attention has focused on its neuropsychiatric effects, little has been given to cardiovascular side effects. Here we report a case of atrial tachyarrhythmias following cannabis use, and review the literature on its cardiovascular effects and complications.

Cannabis is generally considered a drug of low toxicity. Despite this a variety of cardiovascular complications have been documented. Here we report a case of atrial tachyarrhythmias and review the literature.

A 35 year old Afro-Caribbean female presented with a 1 month history of headaches and was found to be hypertensive with a sitting blood pressure of 179/119 mmHg on average. Her past medical history included polycystic ovary disease. She smoked 20 cigarettes a day and had a history of infrequent cannabis use although none in the year preceding presentation. She denied using any other recreational drugs and was on no regular medication. There was a positive family history of essential hypertension. Clinical examination was unremarkable. She was admitted to hospital for further investigations particularly to rule out secondary hypertension.

Laboratory investigations showed normal urine analysis, biochemistry, thyroid function tests, plasma aldosterone/renin ratio, and 24 hour urinary catecholamines. Abdominal CT scan was normal. A 12-lead electrocardiogram (ECG) suggested left ventricular hypertrophy using the Sokolow and Lyon voltage criteria (R in V5 + S in V1  $\geq$ 35 mm), however, the patient was slim built. An echocardiogram showed

normal ventricular wall thickness and cavity size, good biventricular function, and normal transmitral inflow Doppler profile and atrial dimensions.

The patient was started on amlodipine tablets 10 mg once daily and her blood pressure improved to 159/107 mmHg. Whilst in hospital, she smoked cannabis, and approximately 20–30 minutes later she developed palpitations, chest pain, and shortness of breath. Her blood pressure was found to be elevated at 233/120 mmHg with a pulse rate of 150 beats per minute. An ECG showed a narrow complex tachycardia, which was confirmed as typical atrial flutter with 2:1 atrioventricular block, following the administration of intravenous adenosine. The cardiac rhythm shortly degenerated into atrial fibrillation at a rate of 146 beats/minute. She was treated with intravenous flecanide to relieve significant discomfort and sinus rhythm was promptly restored. Cardiac troponin at 12 hours was normal. Urine toxicology was positive for cannabis but no other recreational drugs were detected. Two weeks post discharge her blood pressure was 117/85 mmHg on amlodipine 10 mg once daily and atenolol 25 mg once daily. A 24 hour ECG Holter monitoring demonstrated normal sinus rhythm. She remains well with excellent blood pressure control and has not smoked cannabis since.

#### DISCUSSION

The arrhythmogenic potential of cannabis smoking has rarely been reported. It is likely that the real incidence of arrhythmias is substantially underreported given the prohibition of cannabis use. Epidemiological data indicate that cannabis users are significantly more likely to experience palpitations with the majority being dose related sinus tachycardia.<sup>1,2</sup> Other reported arrhythmias include sinus



bradycardia, second-degree atrioventricular block and atrial fibrillation.<sup>3-5</sup> The onset of arrhythmias can begin within a few minutes of smoking cannabis, reaching a peak within 30 minutes, but can persist for longer than 90 minutes.<sup>1</sup> The cardiovascular effects of cannabis are largely related to its biphasic effect on the autonomic nervous system.<sup>6</sup> At low or moderate doses, the drug leads to an increase in sympathetic activity and a reduction in parasympathetic activity, producing a tachycardia and increase in cardiac output. Blood pressure therefore increases. At high doses, sympathetic activity is inhibited and parasympathetic activity increased, leading to bradycardia and hypotension. Animal data suggests that the bioactive constituent of cannabis may cause sympathetic inhibition via CB1 receptors on the presynaptic nerve terminals of postganglionic sympathetic fibres.<sup>7</sup> The influence of the autonomic nervous system in relation to the mechanism of atrial fibrillation has been previously examined.<sup>8</sup> Both sympathetic and parasympathetic mechanisms have been implicated.

Vagal stimulation reduces action potential duration, shortens the atrial refractory period and produces cellular hyperpolarisation.<sup>8</sup> The net result is a reduction in the wavelength of atrial activation, predisposing to the re-entrant mechanism of atrial fibrillation.<sup>8</sup> Vagal induced atrial fibrillation is usually seen in hearts with no obvious structural disease. Adrenergic stimulation reduces action potential duration and alters the electrophysiological characteristics of the atria, favoring automaticity, triggered activity and micro-re-entry. Adrenergic mediated atrial fibrillation is usually seen in patients with structural heart disease often with a triggering event such as drugs, sepsis, or post operatively. Our patient, who used cannabis infrequently, may have had adrenergic induced atrial flutter. The heightened sympathetic response following cannabis use may have destabilized the arrhythmogenic substrate, in this case a hypertensive atrium, initiating atrial flutter. Adenosine, which shortens atrial refractory period, may have been the trigger that induced atrial fibrillation.

Evidence-based guidance for the management of cannabis induced atrial tachyarrhythmias is limited to case reports,<sup>3,4</sup> but some general principles can be used to guide treatment. Haemodynamically unstable patients require prompt DC cardioversion. In haemodynamically stable patients a period of observation is recommended as the majority of these arrhythmias usually spontaneously revert to sinus rhythm. If the arrhythmia persists then the usual management protocols, as recommended by the American College of Cardiology/American Heart Association and the European Society of Cardiology,<sup>9</sup> should be followed. Class 1a antiarrhythmic agents (flecainide and propanolone) can be used to terminate these arrhythmias in structurally normal hearts.

Cannabis use has also been associated with premature ventricular contractions,<sup>1</sup> and other reversible ECG changes affecting the P and T waves, and the ST segments.<sup>6</sup> Although it is not clear if these changes are related to drug ingestion independent of effects on heart rate.

In patients with ischaemic heart disease, cannabis increases the frequency of anginal symptoms at low levels of exercise,<sup>10</sup> owing to an increase in heart rate and myocardial contractility, and a reduction in the oxygen carrying capacity of blood due to the formation of carboxyhaemoglobin.<sup>11</sup> These adverse haemodynamic changes may trigger plaque rupture in vulnerable individuals culminating in myocardial infarction,<sup>11</sup> and sudden cardiovascular death.<sup>12</sup> Myocardial infarction has also been reported in the presence of normal coronary arteries, suggesting coronary vasospasm.<sup>12</sup> Other reported cardiovascular effects associated with cannabis consumption include transient ischaemic attacks and strokes.<sup>12</sup>

These recent reports confirm that cannabis use alone can result in cardiovascular complications. Furthermore, cannabis may often be used in combination with other recreational drugs such as cocaine and amphetamines, which may have synergistic cardiovascular effects.<sup>6</sup> A careful recreational drug history should always be elicited, particularly when associated with unusual cardiovascular presentations and altered mental states.

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## CORRECTION

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In the case report titled, Left flank pain as the sole manifestation of acute pancreatitis: a report of a case with an initial misdiagnosis (*Emerg Med J* 2005;**22**:452-3) the affiliation for Dr Jiann-hwa Chen was incorrect. The correct affiliation is Department of Emergency Medicine, General Cathay Hospital-Taipei, Taiwan, ROC.