

CASE REPORTS

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

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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/mm³; hemoglobin 12.4 gm/dL; platelet count 280,000/mm³; 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.¹ Although there are no disease-specific signs or symptoms for acute pancreatitis,² 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.³ Romano *et al*, also reported incidental findings of pancreatitis, diverticulitis, and renal tumor in patients with suspected renal colic by using CT.⁴ 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.⁵ 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.^{4 6 7}

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

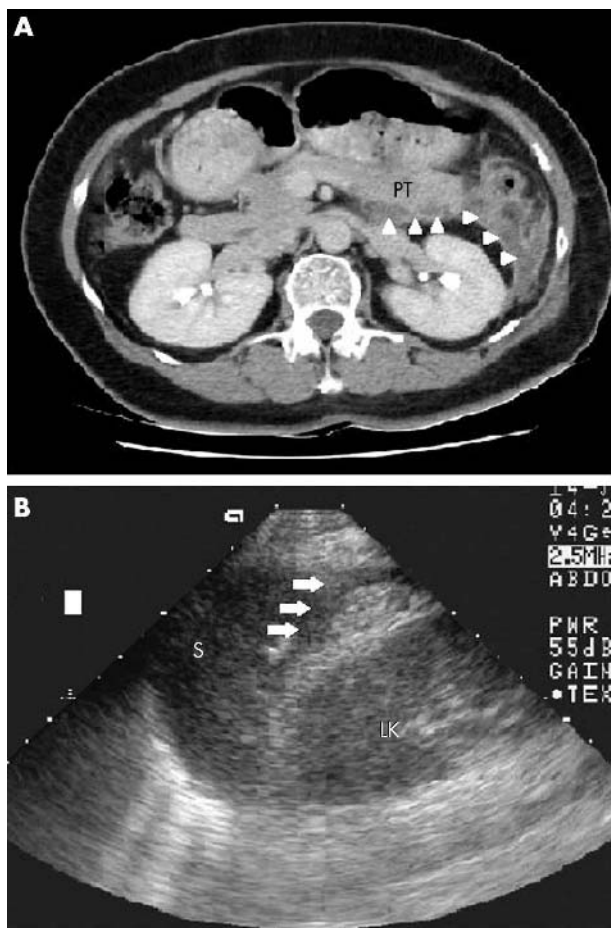


Figure 1 The computed tomography demarcated the lesion and showed a necrotic change over the pancreatic tail and abnormal fluid collection over the pancreatic tail and peri-renal space (arrowhead). The ultrasound showed fluid collection over the peri-renal spaces. A thick fluid collection (arrow) might 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.⁸

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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.¹ 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.² 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,³ urethral obstruction,⁴ obstruction of the left colon,⁵ testicular pain,⁶ peripheral neuropathy,⁷ hiccoughs,⁸ haematuria,⁹ right inguinal mass,¹⁰ and symptomatic¹¹ or even asymptomatic¹² inguinal hernia. Ijaz and Geroulakos¹³ 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.¹⁴ 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.¹

There have been three case reports in the literature of fatal cyclist and motorcyclist injuries from rear view mirrors.^{2–3} 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.^{4,5}

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.^{6,7}

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.⁸ 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 1986.⁹ 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

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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.¹ It has also been known to cause significant masseter spasm in children when used in conjunction with volatile anaesthetics such as halothane.^{2–5} 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.^{6,7} 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.⁴ 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 SAO₂ 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
 CO₂: 21–32 mmol/l
 Glucose: 65–105 mg/dl
 Lactate: 0–1.8 mmol/l

Arterial blood gases

pH: 7.35–7.45
 P_{CO₂}: 35–45 mmHg
 P_{O₂}: 80–100 mmHg
 HCO₃⁻: 22–26 mEq/l
 O₂ 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, CO₂ 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_{CO₂} 96 mm Hg; P_{O₂} 317 mm Hg; HCO₃⁻ 8.7 mEq/l and O₂ 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 CO₂ 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 CO₂ 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.⁸ Sakles and colleagues evaluated the charts of 610 intubations in the emergency department and rapid sequence intubation was used in 84%.⁹ 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.¹⁰

Masseter spasm has been implicated as an early indicator of susceptibility to malignant hyperthermia. Other markers for malignant hyperthermia include hyperpyrexia, increased end tidal CO₂, 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.³⁻¹¹ The incidence of malignant hyperthermia is unknown in adults with masseter spasm, although isolated masseter spasm is not pathognomonic for malignant hyperthermia.¹² 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 CO₂ 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.¹³ 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

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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.

Both 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.¹ 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.

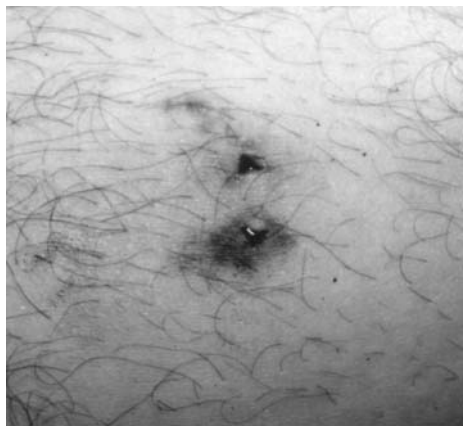


Figure 1 Leech bites in our patient resulted in a triradiate wound (consistent with reports in literature¹⁻³).

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/mm³, platelets 171 000/mm³, 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.¹⁻³ A recent clinical study has reported that leech therapy may be an effective treatment for rapid reduction of pain associated with knee osteoarthritis.² 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).^{1,3}

Leech bites are painless and results in a triradiate wound which remains open for a long time and heals slowly^{1,3} (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.^{1,3,4} Prolonged haemorrhage may result in anaemia, and deaths from excessive exsanguination have been reported.³ 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).⁵

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.⁶ 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.³

Contamination with pathogenic microorganisms may result in erysipelas and submucosal abscesses.^{1,3} 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.⁷ As a medicinal leech bite heals, ecchymosis and scarring are not uncommon sequelae.¹

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.¹ 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.¹

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 Avşaroğulları, 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 × 10⁹/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 through a diaphragmatic stab wound is a well recognised, although infrequent cause of cardiothoracic embarrassment,¹ while tension gastrothorax or colothorax appears in the literature occasionally as a case report.² Intrapleural effusion caused by herniation of the abdominal contents has likewise been noted on occasion.^{3,4} Radiological diagnosis of diaphragmatic rupture is difficult, and many

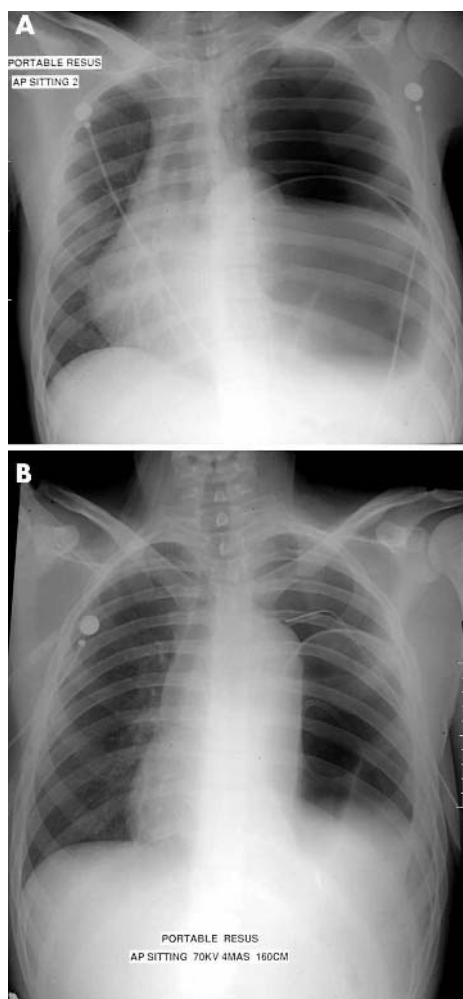


Figure 1 Chest radiographs taken before (A) and after (B) insertion of drain.

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

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.⁶ 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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