deaths from stingrays are reviewed in an article by Cooper and include fatalities from thoracic and abdominal penetration.\textsuperscript{5} Debris, including the spines and portions of the integumentary sheath, may contaminate the wound. Secondary infection is common and because of the unique milieu created by sea water, pathogenic marine microbes tend to be halophilic (a bacterium which can grow in salt culture medium), heterotrophic (a micro-organism which requires complex substances for its nutrition, as it possesses limited powers of synthesis), motile, and Gram negative rod forms. The genus Vibrio is specially common and poses a serious risk to the immunocompromised.\textsuperscript{2}

**TREATMENT**

Treatment should attempt to reverse the local and systemic effects of the venom, alleviate pain, and prevent infection.\textsuperscript{1,2,6} The wound should be immediately irrigated with whatever diluent is available, which in the field is usually sea water. Any visible pieces of the integumentary sheath or spines should be removed. As soon as is practically possible, the wound should be soaked in hot water (about 40-5°C) for 30 to 90 minutes, with appropriate care to avoid thermal damage. The heat will inactivate any thermostable venom. If the pain persists, it may be relieved by further hot soaks or alternatively a regional nerve block or local infiltration can be performed. Vesicular fluid should be removed promptly and aseptically.\textsuperscript{2} The wound should then be re-irrigated, explored, and debrided of necrotic tissue and foreign material. Radiographs may help identify retained radio-opaque fragments of the spines.\textsuperscript{2,3} The wound may need to be left open for delayed primary closure or healing by secondary intention. Prophylactic antibiotics are controversial, but should be given in the immunocompromised or with high risk wounds.\textsuperscript{2,6} In all cases, antitetanus prophylaxis should be remembered. Patients who are treated should be observed for up to four hours from the time of envenomation to detect a systemic reaction.\textsuperscript{5} Occasionally, an envenomation will be accompanied by an allergic reaction and treatment for anaphylaxis may be necessary.\textsuperscript{6}

Fortunately many stingrays lose or tear their integumentary sheaths (45% in one study of 4000\textsuperscript{7}), so that stings often result in trauma without envenomation.

We thank Mr A D Meredith, Consultant Orthopaedic Surgeon, for allowing permission to publish the case report.


**Oesophageal rupture in the course of conservative treatment of bleeding oesophageal varices**

Agnieszka Crerar-Gilbert

**Abstract**

Fatal oesophageal rupture is described as a complication of the management of bleeding oesophageal varices with repeated sclerotherapy and tamponade using the Sengstaken-Blakemore tube. The importance of chest radiographs is stressed in the early detection and prevention of malposition of the Sengstaken-Blakemore tube, as inflation of the gastric balloon in the oesophagus can result in oesophageal rupture.\textsuperscript{(J Acud Emerg Med 1996;13:225–227)}

Key terms: gastrointestinal haemorrhage; oesophageal rupture; Sengstaken-Blakemore tube

Oesophageal perforation is a rare complication following treatment of bleeding oesophageal varices with the Sengstaken-Blakemore tube\textsuperscript{1,3} but carries a high mortality in patients already compromised by gastrointestinal haemorrhage. In the unusual case presented here a massive oesophageal tear occurred in the thoracic part of the oesophagus and resulted in a most striking radiograph showing the gastric balloon of the Sengstaken-Blakemore tube inflated in the right hemithorax (figure).

Chest radiographs should be taken following the Sengstaken-Blakemore tube insertion to ascertain the correct position before inflation of the gastric balloon. Further chest radiographs taken after the gastric balloon is inflated are essential to detect dislocation into the oesophagus.

The probable mechanism and the underlying pathology of the oesophageal perforation are discussed.
Case report

A 35 year old Asian male, an electronics engineer, was admitted with a short history of haematemesis and melaena. There was no significant past medical history. He smoked 10 cigarettes a day and was known to be a heavy drinker.

On examination yellow sclera were noted. There were no spider naevi or dilated veins over the abdominal or chest wall.

His pulse rate was 90/min, blood pressure 120/80 mm Hg, with no postural hypotension and normal capillary refill. The abdomen was soft and not distended, with no signs of ascites. The liver was tender and palpable 4 cm below the costal margin.

The initial base line haematological and biochemical investigations, as well as a chest radiograph, were normal.

The clinical diagnosis of upper gastrointestinal bleeding from oesophageal varices or peptic ulcer was made. The patient was treated with intravenous fluids and intravenous ranitidine, and his vital signs were monitored. A few hours after admission he had a further haematemesis of 250 ml and melaena, leading to a blood pressure drop to 90/60 mm Hg and pulse rising to 120/min.

Six hours after admission, he underwent emergency oesophago-gastroduodenoscopy, which showed a normal stomach and duodenum and a single large bleeding oesophageal varix. The varix was injected twice with 1.5 ml of ethanolamine olate, and a Sengstaken-Blakemore tube was inserted, with inflation of the gastric balloon to 500 ml. On chest x ray the gastric balloon was found to be positioned low and traction was applied, using the weight of a one-litre normal saline bag. In spite of these procedures the patient had a further haematemesis.

Twelve hours after admission he was transferred to the intensive therapy unit as he was haemodynamically unstable. He also became restless and his breathing deteriorated. At that stage he was intubated and ventilated. He was transfused with fresh frozen plasma, platelet concentrate, and packed cells. Twenty four hours after admission he underwent the second endoscopy with further injection of the bleeding varix. The Sengstaken-Blakemore tube was reinserted and the gastric balloon inflated as before. However, he continued to have haematemesis and his condition rapidly deteriorated.

A chest x ray taken a few hours after the second tamponade with the Sengstaken-Blakemore tube showed the gastric balloon occupying most of the right hemithorax (figure), leading to the diagnosis of oesophageal rupture.

Once the diagnosis was made, even though the patient was unstable, it was decided to perform an emergency laparotomy. Intraoperatively, blood pressure and oxygen saturation were not recordable and the pulse was between 100 and 130/min. The patient was transfused with seven units of blood and continued to require inotropic support. At laparotomy no obvious oesophageal tear was found. Oesophagoscopy revealed a huge oesophageal tear at 26 cm into the right chest. In view of the patient’s very poor condition thoracotomy was contraindicated. Oesophageal transection was carried out through an abdominal approach and a gastrostomy tube and mediastinal drain were inserted.

Postoperatively the patient continued to be hypotensive and anuric in spite of inotropic support.

He died 56 hours after admission.

The coroner’s necropsy concluded that the cause of death was bleeding from oesophageal varices, ruptured oesophagus, and liver cirrhosis.

Discussion

Tamponade with the Sengstaken-Blakemore tube as a sole method or following sclerotherapy is a life saving procedure for stopping profuse haemorrhage from oesophageal varices. This procedure is used in accident and emergency departments as a temporary emergency measure aiming to arrest bleeding, allow volume replacement, and prevent further blood loss in order to optimise the patient’s condition before definitive management.

Though oesophageal tamponade with the Sengstaken-Blakemore tube is in many circumstances a life saving procedure, it also carries a risk of complications; this has been reported to be 13%. The most common complications are aspiration pneumonia and minor oesophageal injuries. Oesophageal rupture is a major but rare complication of the procedure, and carries a high risk of death.

In the cases described in published reports, oesophageal perforation followed difficult tube insertion with repeated inflation of the gastric balloon in the oesophagus, or retching and vomiting, leading either to spontaneous oesophageal rupture or to dislocation of gastric balloon to the oesophagus which is already obstructed by the inflated oesophageal balloon, creating pressure sufficient to tear the oesophageal wall.
This case involved an unsuspected massive oesophageal rupture following sclerotherapy and inflation of the Sengstaken-Blakemore tube. It is an unusual case, as massive rupture occurred in the thoracic part of the oesophagus, whereas oesophageal rupture usually involves the lower oesophagus. Tube insertion was easy and there was no associated retching or vomiting as in the above mentioned case. Factors precipitating oesophageal rupture in this case could have included sclerotherapy and repeated endoscopy, contributing to the weakening of the oesophageal wall which was subsequently ruptured by inflation of the gastric balloon in the oesophagus. The Sengstaken-Blakemore tube was inserted and the gastric balloon inflated without confirming the position with chest radiographs.

Inflation of the gastric balloon in the oesophagus is one of the factors leading to oesophageal rupture, and it has been suggested that the position of the Sengstaken-Blakemore tube should be checked with a chest radiograph before the balloon is inflated.

In this case, a large rupture of the oesophagus into the right mediastinum, with the resulting lung compression by the gastric balloon of the Sengstaken-Blakemore tube, was not suspected, as the clinical features were thought to be due to ongoing haemorrhage. The diagnosis of the oesophageal rupture became obvious only on the basis of the chest x ray which was done several hours after inflation of the gastric balloon.

This case illustrates an unusual fatal complication of sclerotherapy and oesophageal tamponade with the Sengstaken-Blakemore tube. It shows the importance of radiography in checking the position of the tube before and after inflation of the gastric balloon, to ensure that the balloon is not inflated in the oesophagus. Timely use of chest radiographs will not only prevent accidental inflation of the gastric balloon in the oesophagus but will also detect dislocation of the balloon proximally to the oesophagus.

Heterotopic bone formation within a missile track

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Abstract
A case is presented which is thought to be the first described example of heterotopic ossification occurring within the path of a bullet. Although the information was not available from prior medical records, the bullet presumably passed through bone or periosteum, thereby seeding the permanent cavity and facilitating ossification within the surrounding muscle and soft tissue.

Key terms: heterotopic ossification; bullet wound

Heterotopic ossification following blunt or operative trauma has been well described in published reports. The development of ectopic lamellar bone was recognised over a century ago when Binnie noted calcium deposition and stroma within muscular tissue. Myositis ossificans, or ectopic bone formation originating in muscle, represents the most common type of heterotopic ossification. It typically occurs in an active person and may follow a single blunt force to an extremity. Ossified haematomas have also been described. Heterotopic ossification may also occur in areas exposed to repeated trauma, such as the ischial area in horseback riders or in the shoulder in field marksmen. There are various clinical situations, each with specific aetiologies, which have led to ectopic bone formation, and although the pathogenesis is uncertain, the entity is well described. The following case represents an unusual cause of pathologic bone formation—penetrating trauma to the lower extremity.

Case report
While crossing a street, a 28 year old black male was struck on the left hip/flank by an automobile that was making a right hand turn. The patient was brought to the Loyola University Medical Center trauma unit by local paramedics. He arrived on a stabilising backboard and wearing a cervical collar. The patient denied loss of consciousness and was alert and well oriented. He was haemodynamically stable and afebrile. He complained most significantly of left upper, inner thigh pain. The patient held his left lower extremity with the knee in near complete flexion and his hip partially abducted and fully flexed.

On physical examination he was noted to have a palpable hard mass beneath the skin of
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