Hypopharyngeal perforation is mainly reported in association with high velocity road traffic accidents, or with low velocity direct blows to the neck. We report a case of hypopharyngeal perforation following a low velocity motorcycle accident where neither of these mechanisms of injury was apparent. A 52 year old man was referred from the emergency department (ED) with a sore throat and dysphagia. He had fallen from his motorcycle, following a low speed side impact accident. He was ambulant at the scene, complaining only of a painful ankle and a sore throat. His motorcycle helmet was undamaged.

Physical examination in the ED was normal. In particular, there was no evidence of head or cervical spine injury, no evidence of external neck trauma, and no subcutaneous emphysema. He complained of a persistent sore throat but did not have a hoarse voice. Lateral cervical spine radiographs demonstrated retropharyngeal free air. Flexible nasendoscopy was normal. A gastrograffin swallow demonstrated a posterior pharyngeal wall tear. After 11 days of conservative hospital treatment, he recovered and was discharged. The presumed mechanism of injury in this case was cervical spine hyperextension without cervical compression.

Hypopharyngeal perforation, a rare complication of non-penetrating blunt neck trauma, is mainly reported in association with high velocity road traffic accidents. The proposed mechanism of injury is neck hyperextension associated with airway closure due to cervical compression against the steering wheel. Low velocity direct blows to the neck have also been associated with pharyngeal perforation. We report a case of hypopharyngeal perforation following a low velocity motorcycle accident where neither of these mechanisms of injury was apparent. The presumed mechanism of injury in this case was cervical spine hyperextension without cervical compression.

CASE REPORT
A 52 year old man was referred from the emergency department (ED) with a sore throat and dysphagia. He had fallen from his motorcycle, following a low speed side impact accident. He was ambulant at the scene, complaining only of a painful ankle and a sore throat. His motorcycle helmet was undamaged.

Physical examination in the ED was normal. In particular, there was no evidence of head or cervical spine injury, no evidence of external neck trauma, and no subcutaneous emphysema. He complained of a persistent sore throat but did not have a hoarse voice. Lateral cervical spine radiographs demonstrated retropharyngeal free air. Flexible nasendoscopy was normal. A gastrograffin swallow demonstrated a posterior pharyngeal wall tear. He was admitted under the care of the otolaryngologists, kept “nil by mouth”, and given intravenous cefuroxime, metronidazole, and fluids. A gastrograffin swallow on day 4 demonstrated a persistent pharyngeal tear. Nasogastric feeds were commenced and intravenous antibiotics continued for a further week. Following radiological confirmation of a healed tear, the patient was discharged on day 11.

DISCUSSION
Oesophageal perforation following external blunt trauma is rare. A review in 1962 identified only 30 reported cases over a 62 year period. Pharyngeal perforations are even rarer, accounting for <2% of all perforations in the pharyngoesophageal region. Recognised causes of pharyngeal perforation include iatrogenic causes, blunt and penetrating trauma, spontaneous perforation, swallowed foreign bodies, blast injury, and emesis.

The most common cause of pharyngeal perforation is iatrogenic, usually secondary to instrumentation. Hypopharyngeal perforation secondary to endotracheal intubation is more likely to occur with difficult intubations, or when performed by inexperienced anaesthetists. Neonatal perforations are associated with rigid suction catheter use, nasogastric and endotracheal tube placement, and digital trauma during resuscitation. Diagnostic/therapeutic endoscopy has a perforation incidence of between 0.01% (flexible) and 1% (rigid).

Blunt trauma occurs with acceleration/deceleration injuries, direct blows to the neck and strangulation. Perforation occurs when laryngeal cartilage is compressed against the vertebral bodies. An anatomical weakness at the hypopharyngeal–oesophageal junction (Killian’s dehiscence) predisposes this area to perforation. During compression, the upper airway closes at the level of the hyoid bone. Concurrently, the thorax empties of expired air. If the force of expiration against the closed airway exceeds the “bursting pressure” of the hypopharyngeal–oesophageal junction, perforation occurs with compressed air being forced through the perforation into the cervical soft tissue. Most post-traumatic pharyngoesophageal perforations occur at this level.

The presence of hypertrophic anterior cervical vertebral osteophytes increases the risk of perforation.

Presentation
Signs and symptoms of acute pharyngoesophageal perforation include subcutaneous emphysema (extending proximally or distally), chest or neck pain, odynophagia, dysphagia, hoarseness, and haemoptysis. Later presentation with sore throat, dysphagia, swelling, and pyrexia suggests the presence of a retropharyngeal abscess and the associated risk of carotid artery pseudoaneurysm formation, mediastinitis, pyopneumothorax, septic shock, and death.

Diagnosis
This is often difficult, and a high index of suspicion is necessary to prevent mortality and morbidity. Unfortunately, the best diagnostic approach is unclear. Plain radiography, laryngoscopy, contrast studies, and computed tomography scans have all been used alone or in combination to confirm clinical suspicion.

The retropharyngeal space (extending from base of the skull to T2–T6) contains fatty tissue and lymph nodes and is, therefore, not usually visible on plain cervical spine radiographs. If air is present, the retropharyngeal space becomes visible in front of the prevertebral soft tissue. Visible retropharyngeal air on a lateral cervical spine radiograph...
should arouse suspicion of pharyngeal rupture. The differential diagnosis includes retropharyngeal abscess (usually associated with soft tissue swelling), dislocation of the atlanto-occipital joint, and any other cause of pneumomediastinum. Additionally, mediastinal, pericardial, or subcutaneous emphysema 20–23 on a plain chest radiograph, in the presence of the symptoms described above, should also arouse suspicion of pharyngeal rupture. In some instances, the presence of air, in association with a good clinical history, is critical to make a definitive diagnosis. 4, 8

Pharyngeal examination is difficult in the presence of free blood. Fibreoptic examination is the diagnostic investigation of choice for obtaining information about the presence, site, and extent of a pharyngeal tear. Additionally, pharyngeal oedema and haematomas may be visualised. 1, 9–11 Rigid endoscopy gives a superior view but requires a general anaesthetic and is impractical in a patient with an immobilised cervical spine. Contrast studies may demonstrate the site and extent of any rupture, through extravasation of contrast media. 29, 30 Lateral or supine decubitus positioning and using barium, rather than water soluble contrast, increases the sensitivity. 29, 30

Adjunctive use of contrasted CT scans may assist the surgeon preoperatively by demonstrating the exact position and extent of the injury, as well as detecting any other post-traumatic complications including haematomas and laryngeal injury. 35 This investigation may be preferable in ill patients, as no specific positioning is required. 36

Management

Although the morbidity and mortality of untreated perforations is high, optimum management has been controversial, due to their rarity. Early diagnosis and treatment is essential in order to avoid the most frequent complications: mediastinitis, fistulas, and abscesses. 1, 29 Traditionally, surgical closure of the perforation was the preferred approach, but over the last 40 years, a number of case series have advocated conservative medical management. 17–19

A literature review in 1990 suggested that pharyngeal lesions <2 cm should have conservative medical management, 17–19 consisting of keeping the patient nil by mouth, the insertion of a nasogastric tube, the administration of intravenous antibiotics and fluids, and total parenteral nutrition where necessary. Oesophageal lesions or pharyngeal lesions >2 cm should be managed surgically: drainage; 24 alone or primary surgical closure involving surgical exploration, debridement where necessary, and closure of the perforation. 12, 40 41

CONCLUSION

Hypopharyngeal rupture following blunt external trauma remains rare. A high index of suspicion is required to prevent morbidity and mortality. Any patient suspected to have a hypopharyngeal perforation (patients with trauma and associated sore throat or dysphagia) should have plain lateral cervical spine and chest radiographs, and a flexible nasendoscopy. Contrast studies and CT scans should be reserved for patients in whom the diagnosis is suspected but unconfirmed by initial investigations. Medical management is appropriate for small lesions limited to the pharynx. Surgical repair and drainage is appropriate for larger perforations and perforations involving the oesophagus.

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