Occasional review

Intramural oesophageal dissection

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Intramural oesophageal dissection is a rare cause of sudden severe retrosternal pain. It was first recognised by Williams in 1957 and since then a further 39 cases have been reported. The injury is intermediate between oesophageal rupture (Boerhaave’s syndrome) and a Mallory Weiss tear of the lower oesophagus. All three conditions are caused by a sudden increase in pressure in the lower oesophagus.

Spontaneous rupture of the oesophagus was first recognised by Boerhaave in 1724. It usually occurs as a result of violent retching or vomiting and typically presents with severe retrosternal pain, surgical emphysema in the neck and mediastinum, and a pleural effusion, which is more common on the left side. Diagnosis is confirmed radiologically by contrast swallow, the contrast leaking into the pleural cavity. Early thoracotomy with suture of the oesophageal defect offers the best chance of survival.

Mallory and Weiss reported upper gastrointestinal haemorrhage in association with vomiting and mucosal laceration of the lower oesophagus in 1929. This is typically painless and resolves with conservative treatment. Diagnosis is confirmed endoscopically when a mucosal tear is seen in the lower oesophagus.

Presentation

Intramural oesophageal dissection occurs when a haematoma forms between the mucosal and muscular layers of the oesophagus and then extends in the submucosal plane, stripping off the oesophageal mucosa. Review of the 40 reported cases and one of our own (see below) shows that it is more common in women than men, women in their sixth decade being most commonly affected (fig 1). In 32% of the 41 cases intramural oesophageal dissection followed an episode of retching or vomiting. In 29% the injury followed a meal, during which some patients described an impacted food bolus being forcefully swallowed. Other causes were sneezing (two patients), drinking (two), and falling (one). In a quarter of cases the precipitating factor was not recorded. The most common presenting symptoms were sudden, severe retrosternal pain (in 83% of cases), haematemesis (71%), odynophagia (41%), and dysphagia (32%). Diagnosis was confirmed by barium swallow in 38 of the 41 patients. In 53% of these barium entered the submucosal haematoma cavity, producing the “mucosal stripe sign”—a sharply defined lucent linear stripe where the true oesophageal lumen is separated from the submucosal haematoma cavity. In 42% the oesophageal lumen appeared compressed by the submucosal haematoma. Two patients had both radiological features. One patient had a normal barium swallow and in this case the diagnosis was made at thoracotomy. Two patients did not undergo barium swallow. In both cases the diagnosis was confirmed by oesophagoscopy when the submucosal haematoma was seen bulging into the oesophageal lumen.

Prognosis

Thirty one of these patients (75%) recovered fully with conservative treatment. In 26 patients this included fasting and intravenous fluids for 24 hours to 16 days (median seven days); intravenous feeding was used in four cases and prophylactic antibiotics in three.

Five patients were allowed liquid diets from the onset of symptoms. One patient died three days after the onset of symptoms while being treated conservatively but the cause, massive haemorrhage from a duodenal ulcer, was unrelated. Nine patients underwent surgery. Five had an exploratory right thoracotomy in each case the oesophagus was mobilised and found to be intact. All recovered fully. Two patients underwent laparotomy, during which a tube gastrostomy was inserted and pyloroplasty performed. The aim of this was to decompress the stomach, thereby reducing reflux and the risk of peptic

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Figure 1. Histogram showing age and sex distribution in 41 cases of intramural oesophageal dissection (40 previously reported and one reported in this article).
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Figure 2 A tube of oesophageal mucosa hanging from the patient's mouth.

digestion of the oesophagus. Both patients recovered fully. In one patient barium swallow showed blood clot obstructing the mid oesophagus. Laparotomy was performed and at operation a large tube was passed down the oesophagus and into the stomach without difficulty. As this indicated the absence of appreciable oesophageal obstruction no other procedure was undertaken. This patient also recovered fully. None of these eight patients underwent a definitive procedure to repair the oesophagus. The patients may therefore have recovered without surgical intervention. One further patient underwent laparotomy. A high gastrotomy was performed and the lower oesophagus was palpated from below. Blood clot was present in the oesophageal lumen. An oesophagoscope was passed from above but this unfortunately perforated the oesophagus. Thoracotomy was performed and the oesophageal perforation repaired. The patient developed an infection after surgery and emergency total oesophagectomy was performed eight days later. The patient subsequently died. This is the only death attributable to intramural oesophageal dissection in this series. With hindsight it seems possible that this patient would have survived if treated conservatively from the onset of symptoms.

New case
A further case of intramural oesophageal dissection is reported here, which documents the mechanisms of injury in this condition as the dissected tube of mucosa was recovered from the patient. A 45 year old woman developed severe retrosternal pain and a sensation of food sticking while she was hurriedly eating a sandwich. She forced herself to vomit and brought up a small amount of blood and a 15 cm tube of oesophageal mucosa. This remained attached to the upper oesophagus and was protruding from her mouth (fig 2). She was not shocked and her chest radiograph was normal. A barium swallow examination was performed. Views of the thoracic oesophagus showed a thin line of barium apparently lying outside the oesophageal lumen (fig 3). This appearance, which is diagnostic of intramural oesophageal dissection, is caused by barium collecting in the haematoma cavity between the dissected oesophageal mucosa and oesophageal muscle. To remove the tube of oesophageal mucosa from the patient's mouth gentle traction was applied, and the mucosa was easily avulsed from its attachment.

Histological examination of the specimen confirmed that it was a complete tube of oesophageal mucosa. The plane of dissection was in the lamina propria just beneath the epithelium (fig 4). For treatment the patient fasted for five days. A liquid diet was then reintroduced uneventfully and she started having solids two days later. Gastroscopy was performed eight weeks later to assess oesophageal healing. The oesophageal mucosa appeared friable in places with contact bleeding but was otherwise intact. It is now 20 months since the injury. Further endoscopic examinations have not been performed as the patient has remained symptom free. The proposed mechanism of injury in this patient
is illustrated in figure 5. A haematoma formed in the mid oesophagus between the mucosal and the muscular layers as a consequence of forced swallowing. The haematoma tracked longitudinally, dissecting a tube of oesophageal mucosa off the underlying muscle. The haematoma was then decompressed into the oesophageal lumen, producing haematemesis. During vomiting part of the tube of dissected mucosa was expelled through the patient's mouth (fig 2).

Diagnosis
When intramural oesophageal dissection occurs an intramural haematoma forms, which dissects the oesophageal mucosa from the underlying muscle. The haematoma may compress the oesophageal lumen, causing dysphagia, and may rupture through the dissected mucosa into the oesophageal lumen, producing haematemesis. Intramural oesophageal dissection typically presents with sudden, severe retrosternal pain and may initially be confused with myocardial infarction. It should be suspected when there is a history of retching, vomiting, or the forced swallowing of an impacted food bolus and when dysphagia or haematemesis develops. The absence of surgical emphysema in the neck and a left basal pleural effusion argue strongly against complete oesophageal rupture. When intramural oesophageal dissection is suspected the best initial investigation is barium swallow, performed as a matter of urgency. This is less traumatic than endoscopy and will determine whether the oesophagus is intact. The diagnosis will be confirmed when oesophageal views show either compression of the lumen by submucosal haematoma or barium entering the haematoma cavity and causing the “mucosal stripe sign.” If barium studies prove inconclusive endoscopy should be performed. The diagnosis will be confirmed when a submucosal haematoma is seen bulging into the oesophageal lumen. Evidence from previous reports suggests that intramural oesophageal dissection should be treated conservatively and that the prognosis is good.

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