Intramural rupture of the oesophagus

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Summary

Two cases of intramural rupture of the oesophagus are described. In both cases the diagnosis was made endoscopically and confirmed radiologically. Both patients were successfully managed conservatively but were later found to have disordered oesophageal motility. The clinical presentation of severe retrosternal chest pain followed by haematemesis and then dysphagia is stressed, and the diagnostic features on barium swallow examination and endoscopy are described. It is suggested that the diagnosis should be made on clinical presentation and barium swallow, and that the possible hazards of endoscopy in the acute stage should be avoided.

Transmural oesophageal rupture following violent vomiting has been recognized since the original description by Boerhaave in 1724. Less commonly it follows any abrupt increase in intrabdominal or intrathoracic pressure such as occurs in epilepsy or during childbirth, defaecation or weight-lifting. Early surgery (within 12 hours) offers the only hope of survival in this potentially lethal syndrome. Less well recognized is the intramural variety of oesophageal rupture where the striking feature is the dissection of blood into the submucosa while the surrounding muscle remains intact. As in transmural rupture, pain is the dominant clinical feature but the picture is often complicated by intraluminal bleeding from an associated mucosal tear. Intramural rupture of the oesophagus is classified as being acquired following oesophageal instrumentation or foreign body perforation, or as spontaneous. Intramural rupture generally settles on conservative management.

The purpose of this article is to describe 2 cases of intramural rupture of the oesophagus and to re-emphasize the characteristic clinical history and diagnostic findings on barium swallow and endoscopy.

Case reports

Case 1

A previously healthy 64-year-old White woman suddenly developed retrosternal chest pain while watching television. The pain initially came in waves for about an hour but then became continuous and radiated up into her neck and jaw and to her back. This was followed by nausea, sweating and haematemeses of about one cupful of fresh blood. Her past history was non-contributory and specifically there was no dyspepsia, heartburn or dysphagia. She took ibuprofen (Brufen) intermittently for mild osteo-arthritis and an aspirin, codeine and paracetamol preparation (Veganin) occasionally for headaches.

On examination she was pale but not shocked; other than epigastric tenderness there were no abnormalities. Investigations revealed a haemoglobin concentration of 11 g/dl and the ESR was 30 mm/1st h. The ECG and cardiac enzyme levels were normal. Radiography of the chest showed a normalized heart and clear lung fields with no air in the mediastinum.

Treatment consisted of pentazocine intramuscularly and transfusion of 2 units of blood. The next morning she was submitted to upper gastro-intestinal endoscopy. Through an Olympus D2 panendoscope a blue sausage-shaped swelling was noted on the posterior wall of the oesophagus starting at 25 cm from the lower incisors and increasing in width until at the oesophagogastric junction at 35 cm it almost occluded the lumen. No mucosal tear was noted but some fresh and altered blood was seen in the fundus of the stomach. The rest of the stomach and duodenum were normal. A barium swallow examination was then carried out which showed a large smooth filling defect causing a degree of obstruction of the lower oesophageal lumen (Fig. 1). No leakage of barium into the mediastinum was noted.

The subsequent course was uneventful. She was given nothing by mouth for 48 hours and then a liquid diet until her dysphagia and pain settled. Daily chest radiographs failed to reveal air in the mediastinum. A further barium meal examination on the 7th day showed considerable resolution of the filling defect in the oesophagus. She was discharged on the 9th day completely asymptomatic.

Fig. 1. Initial barium swallow in case 1 showing the intraluminal filling defect.
She was followed up at the Oesophageal Clinic and on cine radiography was noted to have developed a localized ballooning of the lower oesophagus (Fig. 2) with gross incoordination on motility studies.

Case 2

Two hours after a light meal of a toasted cheese sandwich, this 64-year-old White woman suddenly experienced retrosternal pain radiating across the chest. She described the pain as stabbing and tearing in nature. The pain was immediately followed by nausea and then haematemesis of about 1 unit of fresh blood. She had no previous history of dyspepsia, dysphagia or heartburn. She took ibuprofen occasionally for mild osteoarthritis but had not taken any tablets on the day of admission.

On examination she was pale but not shocked. There was epigastric tenderness, but no other abnormalities were found. Investigations revealed a haemoglobin concentration of 6.5 g/dl with a normal white cell count, platelet count and prothrombin index. The ECG and chest radiograph were within normal limits.

She was transfused with 4 units of blood and at upper gastrointestinal endoscopy the following morning a blue haematoma was noted on the posterior wall of the oesophagus, starting at 20 cm and extending down to the oesophagogastric junction where it took up most of the lumen (Fig. 3). Some fresh bleeding was noted at the lower end. The stomach was not entered. A diagnosis of intramural haematoma was made and this was confirmed radiologically when a smooth, long intraluminal filling defect was noted (Fig. 4, top, left) with extension into the stomach causing an unusual filling defect in the fundus (Fig. 4, bottom).

She was treated conservatively with intravenous fluid for 48 hours and then oral fluids until her painful dysphagia settled. A repeat barium meal examination on the 9th day showed longitudinal swollen folds and a ridge dividing the lumen, giving the bizarre appearance of a double channel (Fig. 4, top, right). After 4 weeks in hospital her symptoms settled and she was discharged on a soft diet. She was followed up at 6 and 10 weeks and resolution of the haematoma was noted endoscopically (Fig. 5) and radiologically (Fig. 6). Three months after her initial presentation manometric studies were carried out which revealed gross incoordination of the body of the oesophagus.
Discussion

In 1957 Williams first drew attention to an oesophageal injury resulting in an intramural haematoma following remote trauma. His patient was a 77-year-old woman who fell from her bed onto her outstretched hand. Within a short while she developed dull retrosternal chest pain and a sensation of fluids sticking 'halfway down'. This was followed by a small haematemesis of bright red blood. A barium swallow revealed a filling defect in the mid-oesophagus which reverted to normal in 11 days. In 1965 Benjamin and Hanks described 3 cases of submucosal dissection of the oesophagus due to intramural haemorrhage. This was associated with foreign body impaction and instrumentation in 2 cases and 'spontaneous haemorrhage' in the 3rd patient, who was on anticoagulants. In 1967 Thompson et al. described a case of oesophageal dissection. The patient presented with epigastric pain and haematemesis and a complete perforation was produced by oesophagoscopy, necessitating oesophagectomy. The surgical specimen revealed an intramural oesophageal dissection starting as a mucosal laceration.

In 1968 Marks and Keet became the first to describe intramural rupture of the oesophagus as a distinct entity. They reported on a 63-year-old woman who developed retrosternal chest pain and the sensation of something sticking in her lower gullet during the course of a hurriedly eaten meal. There was no haematemesis but the patient was in shock and the diagnosis was made on barium swallow, which showed a longitudinal intraluminal filling defect and a tear in the lower oesophagus. They suggest that once complete transmural rupture has been excluded conservative treatment is indicated. Since then a further 13 cases have been reported, including another from Groote Schuur Hospital. Eleven of the 13 cases were treated successfully by conservative measures. Two patients were treated surgically.

In all the cases reported, with the exception of the patient on anticoagulants described by Benjamin and Hanks, the event was precipitated by vomiting or stifling a sneeze, or occurred while eating or immediately after eating. To classify this type of injury as a spontaneous rupture is therefore not strictly correct, as there is invariably a precipitating factor. It has been suggested that this type of injury should be classified as part of the emetogenic spectrum. However, a history of vomiting may not be obtained; in fact in both our cases the pain preceded the retching and vomiting. This suggests that factors other than a sudden rise in intragastric or intra-oesophageal pressure may be operative. Smith et al. described 3 cases in females in their 7th decade, all with disorders of the cardia and lower oesophagus. Both our patients had these abnormalities on manometric testing, as did the only other patient in the literature on whom manometry was performed, but it is purely conjectural whether the oesophageal inco-ordination predated or postdated the event.

We wish to draw attention to the clinical presentation of this rare but important syndrome. It needs to be included in the differential diagnosis of severe chest pain, particularly if followed by dysphagia and haematemesis. In this clinical setting with the radiologist alerted the condition is readily diagnosed on barium swallow examination when a smooth elongated filling defect is noted. If barium enters the dissection space, duplication of the oesophageal lumen separated by a radiolucent stripe — the mucosal stripe sign — will be noted. Endoscopy, if carried out, usually shows a blue sausage-shaped haematoma taking up the lumen of the oesophagus and a mucosal tear may be present. The importance of an exact diagnosis is that conservative treatment is indicated and the prognosis is good. The diagnosis can be confirmed by fibre-optic endoscopy, but with the characteristic clinical history and diagnostic barium swallow the possible hazards of endoscopy in the acute stage should be avoided.
Iron prophylaxis in pregnancy — is it useful?

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Summary

Iron deficiency anaemia in pregnancy is not a common problem among Black patients in the Durban area, and prophylactic iron supplements do not lead to an increase in haemoglobin values. Prophylactic iron therapy should give way to investigation and appropriate treatment of patients with low haemoglobin values.

Haemoglobin values in normal pregnancy

Patients and methods

Haemoglobin concentrations, determined on a routine basis for 1,051 Black patients attending the booking antenatal clinic at the King Edward VIII Hospital, Durban, were recorded together with the duration of pregnancy and parity.

Results

Initial haemoglobin concentrations are shown in Table I. Differences in values between women of various parities were not statistically significant. The duration of pregnancy at booking was similar for all parity groups (Table II), and is therefore unlikely to invalidate a comparison between the haemoglobin values in women of different parities. The overall prevalence of low haemoglobin values (<10.0 g/dl) was 7%.