Spontaneous mid-oesophageal rupture

S. HOLT
M.B. Ch.B., M.R.C.P. (U.K.)

R. C. HEADING
M.B. Ch.B., M.R.C.P. (U.K.)

J. W. MILLAR
M.B. Ch.B., M.R.C.P. (U.K.)

R. G. CHARLES*
M.B. Ch.B., M.R.C.P. (U.K.),
M.R.C.S. Eng., L.R.C.P. Lond.


The University Department of Therapeutics, the Royal Infirmary, Edinburgh, and
*The Liverpool Regional Cardiac Centre, Sefton General Hospital, Liverpool

Summary
The clinical presentation and management of spontaneous rupture of the middle third of the oesophagus is described in two patients. Early presentation and treatment in one case led to uncomplicated recovery. In the other patient late presentation and diagnosis resulted in delayed surgical intervention with an unsuccessful outcome. The nature of this rare lesion is discussed and nine previously described cases are reviewed.

Introduction
We have a surprising observation given us by the celebrated Boerhaave, which is perhaps the only one published, namely, the illustrious Baron Wassenaer, Lord High Admiral to the Republick, after intense straining in vomiting, broke asunder the tube of the oesophagus, near the diaphragm, so that, after the most excruciating pains, the aliments which he swallowed passed, together with air, into the cavity of the thorax, and he expired in twenty-four hours'. (Van Swieten, 1765)

Although Boerhaave (1724) is credited with the first account of oesophageal rupture the earliest critical review of this subject was presented by Fitz in 1877. In fact, Fitz considered Boerhaave's report to 'lack so much that is desired, that the diagnosis is not only not proven, but may well be regarded as incorrect'. Fitz quoted a clinical description by a Dr Allen which is the first recorded example of rupture of the middle third of the oesophagus. The patient was a 31-year-old alcoholic who experienced a small haematemesis during an episode of forced vomiting and later developed subcutaneous emphysema and died after seven-and-a-half days. During this period 'he was constantly turning his head from side to side, his countenance at the same time expressing mingled pain and terror'.

Spontaneous rupture of the healthy oesophagus is a rare event with high morbidity and mortality. The rupture usually involves the left postero-lateral aspect of the distal third of the organ, within 6 cm of the cardia. Less frequently, rupture may occur in the cervical (Russell, 1953), abdominal (Barrett, 1946), and middle third of the organ (Fitz, 1877). Since the first description of spontaneous middle third rupture by Fitz in 1877, only eight further cases have appeared in the literature (Moynihan, 1954; Wachtel and Gengins, 1955; Ross, 1961; Hamilton, 1967; Tidman and John, 1967; O'Leary, Hennessy and Brady, 1975).

The present authors have encountered two patients with rupture of the middle third of the oesophagus, in one of whom early diagnosis led to prompt surgical repair and uncomplicated recovery. In contrast, the other patient presented late in the course of his illness with resultant delayed diagnosis and surgical treatment. These two patients, and those previously reported, illustrate the varied presentation of oesophageal rupture, emphasizing the importance of early consideration of the diagnosis in patients with a history suggesting acute abdominal or thoracic catastrophe.

Patient no. 1
A previously healthy male, aged 28 years, was admitted to hospital 4 hr after a sudden onset of severe central chest pain. During a meal he had
attempted to swallow a piece of fatty steak and experienced a sensation of this sticking in the middle of his chest. Repeated attempts to swallow resulted in sudden sharp chest pain. No vomiting or retching occurred. The pain became continuous and radiated through to his back and epigastrium. Pethidine afforded only minimal relief. Clinical examination at the time of admission revealed pallor, tachypnoea and a pyrexia of 38.4°C. Examination of the cardiovascular and respiratory systems was normal. There was no surgical emphysema but the abdomen was rigid, with striking absence of tenderness and normal bowel sounds. A chest X-ray showed mediastinal emphysema (Fig. 1), which prompted emergency contrast radiography of the oesophagus. Lipiodol was seen to extravasate posteriorly from the mid oesophagus just below the aortic arch on the left (Fig. 2). An ECG demonstrated the incidental finding of inversion of T waves in V1-V5, a short PR interval (100 msec) and delta waves, characteristic of Wolff-Parkinson-White syndrome.

Emergency oesophagoscopy performed 16 hr after the onset of symptoms revealed a linear, longitudinal abrasion 3 cm in length on the posterior wall of the mid oesophagus, the mucosa being otherwise normal.

At thoracotomy there were 500 ml of blood-stained fluid in the left hemithorax, intense mediastinitis and a small defect, 0.5 cm in diameter, at the site corresponding to the abrasion observed at oesophagoscopy. The tear was repaired and a feeding gastrosotomy constructed. The postoperative course was complicated by an episode of supraventricular tachycardia, which was controlled by digoxin. Repeat contrast radiography 3 weeks postoperatively confirmed successful closure, and the patient was discharged from hospital on the twenty-seventh postoperative day. He remains well 4 years after the incident.
Patient no. 2

A 62-year-old male was admitted to hospital with a 2-day history of severe epigastric pain. Forty-eight hours before admission he had experienced sudden, severe epigastric pain which resolved spontaneously within 1 hr. Twenty-four hours later the pain recurred, immediately following a meal, and gradually became more severe. It was exacerbated by movement and deep inspiration, and radiated to the right hypochondrium and anterior chest wall. He experienced nausea, thirst and profuse sweating but did not retch or vomit. He had gained significant relief from pethidine, administered by his general practitioner 2 hr before hospital attendance. He gave a 5-year history of mild post-prandial lower chest discomfort.

Clinical examination revealed pallor, mild respiratory distress and a normal temperature. There was abdominal rigidity and epigastric tenderness; bowel sounds were normal. The pulse was regular at 100/min, blood pressure 120/75 mmHg and heart sounds normal. Examination of the chest demonstrated dullness to percussion at the right lower zone posteriorly, with decreased air entry and bronchial breath sounds. A chest X-ray showed opacification at the right base, with some inflammatory change (Fig. 3). Plain abdominal X-ray was normal. Haemoglobin, white cell count, urea and electrolytes, serum amylase and ECG were within normal limits. A clinical diagnosis of pneumonia was made and treatment commenced with intravenous ampicillin and flucloxacillin but his condition deteriorated. Seventeen hours after admission, examination of the chest revealed signs of a right pleural effusion. Chest X-ray showed a right hydropneumothorax and early subcutaneous emphysema in the neck (Fig. 4). Diagnos-

Fig. 3. Chest X-ray demonstrating opacification right lower zone (Case 2).

Fig. 4. Chest X-ray showing right hydropneumothorax and subcutaneous emphysema (Case 2).
tic thoracocentesis was performed and 1·5 litres of purulent fluid were aspirated. Contrast radiography using gastrograffin demonstrated a leak from the right postero-lateral wall of the oesophagus, 2·5 cm below the carina. The oesophagus below the site of rupture appeared narrow with an irregular outline, suggesting the presence of a stricture (Fig. 5).

Right thoracotomy revealed an empyema with suppurative mediastinitis, and a 2-cm longitudinal tear was found in the right postero-lateral aspect of the azygos vein. No other oesophageal disease was detected. The tear was repaired and chest drainage instituted. Initial postoperative progress was good but copious purulent fluid continued to drain from the chest. Repeat contrast radiography performed on the seventh postoperative day indicated a persistent oesophageal fistula. On the twenty-first day after operation, because of continuing drainage from the chest, cautery of the tear was performed at oesophagoscopy. The oesophageal mucosa surrounding and below the defect was noted to be normal. Closure of the oesophageal fistula was not achieved and a feeding gastrostomy was constructed.

The patient's general condition deteriorated, he developed staphylococcal septicemia, and died 10 weeks postoperatively. Permission for post-mortem examination was refused.

**Discussion**

The pathogenesis of spontaneous rupture of the oesophagus has been explained in terms of a sudden rise of intra-oesophageal pressure. The average pressure required to cause rupture is probably of the order of 5 lb/in² (Mackler, 1952). It is believed that a high intraluminal distending pressure consequent upon inappropriate contraction of the upper oesophageal sphincter leads to rupture (Mackler, 1952). This mechanism is in keeping with the clinical observation that 80% of cases of oesophageal rupture are preceded by vomiting or retching (Abbott et al., 1970).

Both in clinical practice and in experimental models a longitudinal rupture occurs, usually in the left postero-lateral wall of the distal third of the oesophagus. Derbes and Mitchell (1956) produced experimental rupture of the oesophagus in ten fresh corpses in a manner that closely simulated ante-mortem conditions. In all cases longitudinal rupture occurred in the lower portion of the oesophagus. Various anatomical explanations have been proposed to explain this preferential site of rupture. Resistant tissue, extending between the vena cava and the azygos vein, buttresses the right, anterior and posterior sides of the oesophagus, leaving the left side vulnerable to dilatation and rupture. Mosher (1930) considered intrinsic weakness in the lower oesophagus to be important, indicating that lower oesophageal smooth muscle is weaker than stomach musculature. He also suggested that points of entry of neurovascular bundles, and the presence of segmental defects in the circular muscle layer, are factors further decreasing the strength of the lower oesophageal wall.

Using a cadaveric stomach-oesophagus preparation, Tidman and John (1967) were able to produce three middle third tears in fifteen separate experiments, most ruptures being located in the distal third of the organ. These middle third tears affected the right wall, regardless of whether the experimental preparation was isolated or in situ. This finding led them to suggest that this was a further site of intrinsic structural weakness within the oesophagus, as they were able to produce two of the three mid-right-sided ruptures in stomach-oesophagus preparations outside of the body. Tidman and John's findings match the clinical observation that most middle

**FIG. 5.** Contrast medium is seen to leak from the right postero-lateral wall of the oesophagus (Case 2).
third ruptures occur on the right side of the oesophagus since six out of the nine cases of middle third rupture of the oesophagus reviewed affected the right side (Table 1). One of the present two patients also had a rupture into the right hemithorax but the other had a posterior tear with soiling of the left pleural cavity.

Antecedent vomiting is common in reported cases of mid oesophageal rupture and in those examples where vomiting is not associated, other conditions potentially leading to a sudden rise in intra-oesophageal pressure may be present. Spontaneous lower oesophageal rupture has been described in association with childbirth (Kennard, 1950), convulsions (Klein and Grossman, 1943), weight-lifting (Griffith, 1932), sudden gastric distension with air (Kerr, Sloan and O'Brien, 1953), and defaecation (Beal, 1949). Although vomiting was present in seven out of the nine reported cases of mid third rupture (Table 1), in the present patients this symptom was notably absent. In the first patient, forceful attempts to swallow a temporarily impacted bolus of food appeared to be an important aetiological factor, and may have been responsible for the mucosal abrasion observed at oesophagoscopy. Conte (1966) described two similar cases where a bolus of food was felt to stick substernally, and swallowing resulted in severe chest pain due to distal third rupture. However, those two patients gave a previous history of heartburn and dysphagia, a feature absent in case no. 1 reported here. The only example of middle third rupture associated with attempts to clear a bolus of food is that described by Fitz (1877). This patient after 'a concentration of his entire muscular energy' succeeded in vomiting a piece of hard, tough, gristly meat. It is of interest that patient no. 1 experienced

<table>
<thead>
<tr>
<th>Author</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Position of rupture</th>
<th>Main presenting features</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Misdiagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fitz, R.H. (1877)</td>
<td>31</td>
<td>M</td>
<td>Right anterior</td>
<td>Alcoholic, forced vomiting of impacted bolus of food. Haematemesis. Swelling left lower jaw.</td>
<td>—</td>
<td>Death at 7½ days</td>
<td>—</td>
</tr>
<tr>
<td>Moynihan, N.H. (1954)</td>
<td>32</td>
<td>M</td>
<td>Right posterolateral</td>
<td>Alcohol excess. Sudden severe chest pain. Dyspnoea, swelling of face.</td>
<td>Conservative</td>
<td>Death at 10 days</td>
<td>Spontaneous pneumothorax</td>
</tr>
<tr>
<td>Wachtel, F.W. and Gengins, G. (1955)</td>
<td>80</td>
<td>M</td>
<td>Left posterior (9-5 cm from cardio-oesophageal junction)</td>
<td>Vomiting, epigastric and lower substernal pain.</td>
<td>Conservative</td>
<td>Death at 13 days</td>
<td>Bilateral broncho-pneumonia</td>
</tr>
<tr>
<td>Ross, J.G. (1961)</td>
<td>73</td>
<td>F</td>
<td>Right posterolateral</td>
<td>Vomiting, small haematemesis, persistent epigastric pain.</td>
<td>Pleural aspiration</td>
<td>Death at 12 days</td>
<td>Perforated peptic ulcer</td>
</tr>
<tr>
<td>Tidman, M.K. and John, H.T. (1967)</td>
<td>60</td>
<td>F</td>
<td>Right side (7-5 cm below the vena azygos arch)</td>
<td>Vomiting, right lower chest and upper abdominal pain, dyspnoea.</td>
<td>Repair</td>
<td>Survived</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>59</td>
<td>M</td>
<td>Right posterior</td>
<td>Nausea, straining at stool followed by severe chest pain and breathlessness.</td>
<td>—</td>
<td>Death within 24 hours</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>81</td>
<td>F</td>
<td>Right posterior (below aortic arch)</td>
<td>Vomiting, pain in upper abdomen, radiating to posterior chest.</td>
<td>Repair</td>
<td>Death within 3 days</td>
<td>Basal pneumonia</td>
</tr>
<tr>
<td>O'Leary, J.F., Hennessey, T.P.J. and Brady, M.P. (1975)</td>
<td>45</td>
<td>M</td>
<td>Left lateral</td>
<td>Attempted suppression of vomiting, sudden onset of severe pain in chest and upper abdomen.</td>
<td>Repair</td>
<td>Survived</td>
<td>Nil</td>
</tr>
</tbody>
</table>
Oesophageal rupture whilst attempting to swallow fatty steak.

Opinion has varied about the suitability of the term 'spontaneous', as pre-existent, possibly asymptomatic, oesophageal disease may be present, especially in cases where vomiting is absent. Oesophagitis may be an important predisposing factor, and a history of preceding heartburn and dysphagia is not uncommon in patients with oesophageal rupture. Experimentally, it can be shown that the animal oesophagus is more likely to burst if acute oesophagitis is present (Brackney et al., 1955). The second patient gave a 5-year history of post-prandial indigestion but as in the first case, no oesophageal disease was identified.

The clinical presentation of spontaneous middle third oesophageal rupture may mimic other common medical emergencies, so that delayed or erroneous diagnosis is common. Middle third ruptures have been initially misdiagnosed as pneumonia, spontaneous pneumothorax, and perforated peptic ulcer (Table 1). In the second case the chest signs were initially considered to represent pneumonia and the correct diagnosis became apparent only with the development of a right hydropneumothorax over a period of 17 hr. The clinical features of previously reported cases of middle third rupture do not differ significantly from those associated with distal rupture. The diagnostic triad of rapid respiration, abdominal rigidity and subcutaneous emphysema, emphasized by Barrett (1946), is not always present. Abdominal rigidity and rapid respiration were present in both the present patients, but clinically detectable surgical emphysema was not, despite there being radiological evidence of it in the second patient. Mackler (1952) regarded vomiting, low thoracic pain and emphysema of the neck as useful pointers in diagnosis. Considering the reports of mid-oesophageal rupture collectively, vomiting was present in seven out of nine, and emphysema of the neck in two out of nine. It is clear that this condition has no pathognomonic features and diagnosis depends mainly on its early consideration.

Accurate diagnosis is aided by radiology, including contrast radiography and chest aspiration. An erect chest radiograph is the most useful initial investigation and may detect early mediastinal emphysema, hydropneumothorax or thoracic visceral displacement, all suggesting oesophageal rupture. The V sign described by Naclerio (1957) is sometimes seen on plain chest X-ray and represents air dissecting fascial planes of the mediastinum. It may easily be overlooked but is often present before physical signs of air in the mediastinum are detectable. A lateral chest radiograph is advisable as abnormalities may be more obvious with this projection. Diagnostic thoracocentesis is valuable when an effusion is present, for acid fluid, food particles or purulent exudate, in late presenting cases, may be aspirated. Localization of the site of rupture is best established by contrast radiography, the extravasation of contrast medium into the mediastinum or pleural space being easily seen. Accurate determination of the site of rupture is important for planning the appropriate thoracotomy incision. It is of interest in patient no. 2 that the oesophagus distal to the site of rupture was considered radiographically to be abnormal. This appearance may have been due to mediastinitis and peri-oesophagitis (Margulis and Burhenne, 1967).

The necessity for early diagnosis and surgical intervention in the avoidance of fatality and complications following spontaneous oesophageal rupture has been strongly emphasized (Moynihan, 1954; Keighley et al., 1972). Only 11% of untreated patients can be expected to be alive at 48 hr after the event and all will die within 1 week (Flavell, 1963). Following the first successful operative repair in 1946 (Barrett, 1947), it became clear that surgery offered the only secure hope of recovery, although a small number of patients will survive with conservative treatment (Hamilton, 1967). Derbes and Mitchell (1956) reported a 36% mortality rate in surgically treated patients and although Keighley et al. (1972) reported mortality at 8%, postoperative complications occurred in 84% of patients. Three out of nine reported cases of mid oesophageal rupture have survived. From present information there is no reason to suspect that the mortality rate is different in patients with middle third as compared with distal rupture.

Survival depends upon early thoracotomy with closure of the tear and chest drainage. In patient no. 1 early surgery was undertaken with resulting uncomplicated recovery. The second patient, owing to late presentation, already had suppuration throughout the mediastinum with an empyema and, despite adequate drainage, postoperative staphylococcal septicaemia resulted.

A review of previously reported cases of middle third rupture of the oesophagus and study of the two patients illustrates the lack of uniformity in clinical presentation of this condition. Only a high index of clinical suspicion for the disorder will lead to prompt treatment with consequent reduction in mortality and morbidity.

Acknowledgment

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