

Spontaneous rupture of the oesophagus

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Background: The aim of this study was to evaluate the diagnosis, management and outcome of patients with spontaneous rupture of the oesophagus in a single centre.

Methods: Between October 1993 and May 2007, 51 consecutive patients with spontaneous oesophageal rupture were evaluated with contrast radiology and flexible endoscopy. Patients with limited contamination who fulfilled specific criteria were managed by a non-operative approach, whereas the remainder underwent thoracotomy.

Results: The median time to diagnosis was 24 (range 4–604) h. Initial diagnosis was by contrast swallow in 18 of 24 patients, computed tomography in 15 of 17 and endoscopy in 18 of 18. There were no deaths among 17 patients who were managed non-operatively with targeted drainage, intravenous antimicrobials, nasogastric decompression and enteral nutrition. Of 31 patients who underwent primary thoracotomy and oesophageal repair (over a Ttube in 29), 11 died in hospital. Three patients could not be resuscitated adequately and did not have surgical intervention.

Conclusion: Spontaneous oesophageal rupture represents a spectrum of disease. Accurate radiological and endoscopic evaluation can identify those suitable for radical non-operative treatment and those who require thoracotomy.

Paper accepted 4 July 2008

Published online 25 July 2008 in Wiley InterScience (www.bjs.co.uk). DOI: 10.1002/bjs.6294

Introduction

Spontaneous rupture of the oesophagus, first described by Boerhaave in 1724¹, is characterized by barogenic disruption of the distal oesophagus and contamination of the mediastinum and pleural cavity with gastric contents. Surgery has traditionally been the treatment of choice, although it is now recognized that non-operative management can be employed according to the degree of contamination^{2–7}. The rarity of this condition means that even specialist centres have limited exposure to the problem and current evidence is based on small case series. Some series have inappropriately included patients with iatrogenic perforation or focused only on those who survive to surgery^{7–9}.

The aim of this study was to evaluate the diagnosis, management and outcome of all patients presenting with spontaneous rupture of the oesophagus over a 13-year period with a consistent approach to management.

Methods

A prospective database was analysed to identify all patients with spontaneous rupture of the oesophagus treated at the Northern Oesophago-gastric Unit between October 1993 and May 2007. Clinical presentation, diagnosis, management and outcome were evaluated. Patients with iatrogenic rupture of the oesophagus, perforation from swallowed foreign bodies or other barogenic trauma (for example blast injuries) were excluded from the study.

Spontaneous rupture was defined as complete disruption of the oesophageal wall occurring in the absence of pre-existing pathology^{1,3,10}. The series included tertiary referrals from all regional hospitals with a suspected diagnosis of spontaneous oesophageal rupture. The diagnosis of perforation was made by the appearance of a full-thickness defect on flexible upper gastrointestinal videoendoscopy or extravasation of contrast during a radiological swallow (performed with water-soluble contrast followed by barium if no

leak was demonstrated) or contrast-enhanced computed tomography (CT).

Following initial resuscitation, all patients were investigated in the specialist unit to confirm the diagnosis and further evaluate the site of perforation and degree of contamination. Stable patients underwent a combination of a contrast swallow (to determine the site, extent of leakage and communication with the pleural cavity), endoscopy (to determine the site of leakage and length of tear) and CT (to determine the site and extent of contamination). If the patient had undergone a contrast swallow or CT before transfer these investigations were repeated if the imaging was suboptimal. Endoscopy was performed only in the specialist unit. Unstable or ventilated patients underwent initial endoscopic evaluation in the critical care unit to confirm the diagnosis, followed by CT if non-operative management was contemplated. It was recorded whether patients met predefined criteria for systemic inflammatory response syndrome (SIRS) at the time of admission¹¹.

A multidisciplinary approach was adopted, involving oesophagogastric surgeon, intensivist, interventional radiologist, dietician, physiotherapist and nursing staff. Patients with limited contamination or who had demonstrated tolerance were managed using an intensive non-operative approach if they met the criteria outlined in Table 1, whereas those with more extensive contamination underwent thoracotomy (Fig. 1).

Patients managed non-operatively received supportive care in a critical care facility. They were kept nil by mouth with intravenous broad-spectrum antimicrobials, antisecretory agents, nasogastric decompression, enteral nutrition and targeted mediastinal/pleural drainage. Contrast radiology, endoscopy and CT were used to monitor the perforation, with a low threshold for re-evaluation if there was evidence of sepsis or failure to improve. Subsequent collections were drained under radiological guidance and surgical intervention considered only if this failed.

Patients who did not satisfy these criteria underwent thoracotomy. A left posterolateral (eighth intercostal space) or right posterolateral (sixth intercostal space) thoracotomy was used for access depending on the site of perforation. Solid food and contamination were removed,

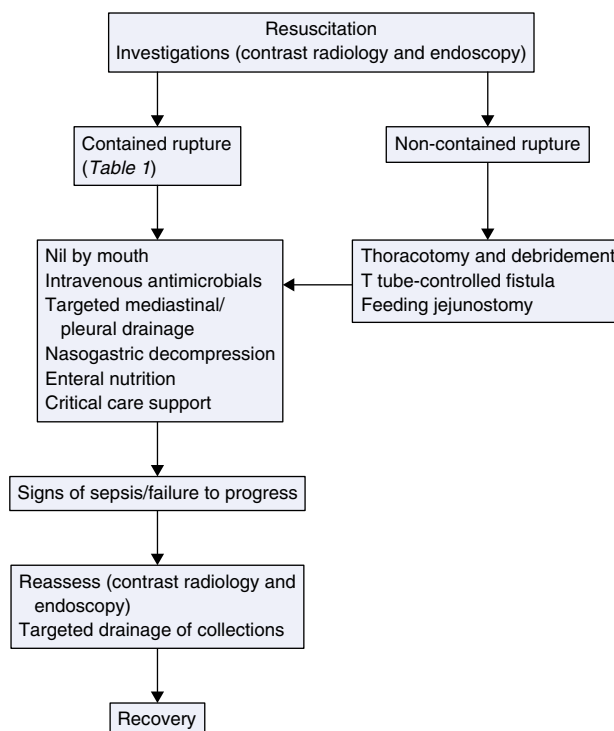


Fig. 1 Management of spontaneous rupture of the oesophagus

with debridement of necrotic tissue. The defect was opened to allow repair of all oesophageal layers, including mucosa, over an 18-G T tube^{8,10}. Drains were placed in the mediastinum adjacent to the repair and in the pleural cavity. Patients were monitored in the same way as those receiving non-operative treatment. The T tube was left *in situ* for a minimum of 3 weeks or until thoracic sepsis was fully controlled and a formal oesophagocutaneous fistula had been established.

Statistical analysis

Statistical analysis was by χ^2 test for categorical data and Mann–Whitney *U* test for continuous data. $P < 0.050$ was considered statistically significant.

Results

Fifty-one patients with confirmed spontaneous rupture of the oesophagus were identified, 37 men and 14 women with a median age of 64 (range 18–88) years. Thirty-eight patients were referred from outlying hospitals and 13 patients from other units within the hospital.

All patients had a history of vomiting before presentation. Thirty-eight complained of chest pain and 22 had

Table 1 Criteria for non-operative management

Perforation contained by the mediastinal pleura
Flow of contrast back into oesophagus on contrast swallow
No clinical evidence of mediastinitis
No solid food contamination of mediastinum
or
Demonstrated tolerance to pleural contamination (radiologically drained) for > 72 h before transfer

abdominal pain. Clinical signs of peritonitis had led to a negative laparotomy in three patients before referral. Only seven patients had Mackler's triad of vomiting, chest pain and surgical emphysema at the time of presentation¹². Forty-two patients exhibited SIRS at presentation.

Seventeen patients were suspected of having spontaneous rupture of the oesophagus at the time of initial hospital admission. A variety of provisional diagnoses were made for other patients (Table 2). Forty-six patients had an abnormal chest radiograph, and a pneumomediastinum or hydropneumothorax was present in 36. The emergency interpretation of the chest radiograph differed from the reported result in 27 instances owing to failure to identify mediastinal, thoracic or subcutaneous air. The median delay from admission to confirmation of the diagnosis was 24 (range 4–604) h. Delay in diagnosis of more than 24 h had no effect on mortality rate (six of 23 *versus* eight of 28 patients with a shorter delay; $\chi^2 = 0.039$, 1 d.f., $P = 0.843$).

The diagnosis of oesophageal perforation was made initially by a contrast swallow in 18 patients, contrast-enhanced CT in 15 and flexible videoendoscopy in 18. Eight of 18 patients diagnosed by endoscopy had undergone previous radiological investigations (contrast swallow in six, CT in two) that had failed to identify a perforation.

Endoscopy was ultimately performed in all 51 patients, and identified the oesophageal tear in every patient. The median length of mucosal tear at endoscopy was 3 (range 1–11) cm. The perforation was left sided in 39 patients

and right sided in 12. At endoscopy a nasogastric tube was placed across the perforation to decompress the stomach.

Three patients with established multiorgan failure could not be resuscitated adequately for surgical intervention and all died within 24 h.

Seventeen patients who met the criteria for non-operative management were managed without surgery. Sixteen had limited contamination and one patient with a delayed diagnosis had demonstrated tolerance to prolonged pleural drainage. Nutrition was maintained enterally via a nasojejunal tube placed at the time of endoscopy (12 patients) or via a surgically placed jejunostomy (five). No patient required surgery, although eight required percutaneous drainage of collections under radiological guidance. One complex, loculated collection required repeated drainage facilitated by instillation of streptokinase. Two patients ultimately had a T tube placed across the perforation and brought out to establish a controlled oesophagocutaneous fistula using a percutaneous–endoscopic approach, avoiding thoracotomy¹³. The median hospital stay was 14 (range 6–113) days. There were no in-hospital deaths in this group (Table 3).

Thirty-one patients were managed surgically, 21 via a left and ten via a right thoracotomy. Two patients early in the series underwent primary oesophageal repair and the remaining patients had repair over an 18-G T tube. All had a surgical jejunostomy inserted via a minilaparotomy. One patient required repeat thoracotomy to drain a complex collection 12 days after T tube repair. There were 11 in-hospital deaths, a median of 23 (range 2–71) days after surgery, including one patient who had a primary oesophageal repair. Five early deaths (within 5 days of surgery) were from multiorgan failure. The main factors contributing to the six later deaths were sepsis and multiorgan failure (two patients), cardiac failure, bronchopleural fistula, bleeding duodenal ulcer and small bowel obstruction. Patients managed surgically had a median hospital stay of 44 (range 3–147) days. For those who left hospital this was 57 (range 37–147) days. All patients required intensive care support, with a median stay of 19 (range 2–75) days.

The proportion of patients with SIRS on admission was higher in the surgical group ($P < 0.001$), but there was no difference in the delay to diagnosis between groups (Table 3). The length of intensive care stay ($P < 0.001$), overall hospital stay ($P = 0.033$) and the in-hospital mortality rate ($P = 0.005$) were greater in the surgical group than in the

Table 2 Initial diagnosis and admitting specialty

Specialty and initial diagnosis	No. of patients
Medicine	20
Pneumonia	11
Spontaneous pneumothorax	4
Myocardial infarction	2
Oesophageal varices	1
Mallory–Weiss tear	1
Gastroenteritis	1
General surgery	15
Spontaneous oesophageal rupture	6
Peritonitis	2
Food bolus obstruction	1
Mesenteric ischaemia	1
Pancreatitis	1
Oesophagitis	1
Perforated duodenal ulcer	2
Leaking aortic aneurysm	1
Intensive care	15
Spontaneous oesophageal rupture	11
Sepsis of unknown origin	4
Cardiothoracic surgery	1
Thoracic aortic dissection	1

Table 3 Comparison of clinical data and outcome in patients who had non-operative or surgical management

	Management		All patients (<i>n</i> = 48)	<i>P</i>
	Non-operative (<i>n</i> = 17)	Surgical (<i>n</i> = 31)		
Age (years)*	64 (18–87)	64 (47–78)	64 (18–87)	0.382†
Delay to diagnosis (h)*	22 (4–500)	24 (4–604)	24 (4–604)	0.657†
SIRS on admission	9	30	39	< 0.001‡
Hospital stay (days)*	14 (6–113)	44 (3–147)	41 (3–147)	0.033†
ICU stay (days)*	0 (0–30)	19 (2–75)	9 (0–75)	< 0.001†
Death	0	11	11	0.005‡

*Values are median (range). SIRS, systemic inflammatory response syndrome; ICU, intensive care unit. †Mann–Whitney *U* test; ‡ χ^2 test.

non-operative group. The overall in-hospital mortality rate for the entire series was 27 per cent (14 of 51).

Discussion

A management algorithm for spontaneous oesophageal rupture based on radiological and endoscopic findings was used to select patients for radical non-operative management or thoracotomy.

The diagnosis of spontaneous oesophageal rupture was frequently delayed, probably reflecting its rarity and varied presentation. This condition represents a spectrum of disease from contained leaks that can be managed by an intensive non-operative strategy to widespread mediastinal and pleural contamination requiring thoracotomy.

Atypical presentations were common; although all patients in this series had a history of vomiting, only seven had chest pain and subcutaneous emphysema as described by Mackler¹². Subclinical air escape is a common early feature on chest radiography but seems frequently to be missed by admitting staff, resulting in admission to the wrong specialty and delayed diagnosis as seen here and in other series¹⁴.

Rapid diagnosis and treatment has repeatedly been associated with a better outcome in spontaneous oesophageal perforation, with 24 h being arbitrarily set as a divide for diagnostic delay^{4,6,15–17}. In this series, the median delay to diagnosis was 24 h and a longer delay was not associated with increased mortality. Although unexpected, this finding is likely to reflect selection bias; some patients with a lesser degree of contamination and those who demonstrated tolerance experienced longer delays yet had a better outcome than those who were moribund at presentation but whose diagnosis had been made rapidly.

Although the clinical picture and fluid from thoracocentesis may suggest oesophageal rupture, further investigations are needed to confirm the diagnosis and plan

management. A contrast swallow was the most common initial investigation for stable patients in this series. It is important to note that there were a number of false-negative results, a limitation identified in previous studies^{18,19}. Advances in contrast-enhanced CT mean that it is increasingly effective for patients who are unstable or in whom the diagnosis is in doubt²⁰.

Flexible endoscopy was used to confirm the diagnosis in all patients in the present series, including those with negative radiological examinations, and allowed further evaluation of the tear. When the perforation was contained by mediastinal pleura it allowed insertion of nasogastric and nasojejunal feeding tubes to facilitate non-operative management. For ventilated patients it was vital to confirm the diagnosis and provide information required to plan surgical management, including the site, level and extent of the mucosal tear. In the authors' experience the mucosal tear, which must be included in any repair, is invariably longer than the muscular injury. Endoscopy can also exclude oesophageal perforation and prevent unnecessary thoracotomy for patients with a similar presentation resulting from spontaneous mediastinum from ruptured bullae²¹, mediastinal haematoma and primary chest sepsis. Historically there were concerns that endoscopy caused further damage but there is no evidence to support this belief. It has also been used without additional morbidity to examine the oesophagus in penetrating thoracic trauma and following oesophagogastric surgery^{22,23}.

This study has confirmed that there is a spectrum of contamination^{2–7} that can be classified by radiological, endoscopic and clinical findings, and used to tailor management. The concept of a rupture contained by the mediastinal pleura was introduced by Barrett²⁴ in 1947. The intensive non-operative strategy described seems ideal for this situation and was successful for all such patients.

This non-operative approach depends on a rapid, accurate initial assessment and focused multidisciplinary approach to managing complications. Continuous reassessment and reinvestigation is required with a low threshold

for intervention. Two patients required further control of the oesophageal leak. This was achieved by a combined endoscopic–radiological technique, which has been reported separately¹³. Those with a delayed diagnosis who demonstrate tolerance to pleural drainage may also be suitable for non-operative management^{7,25–27}.

Surgery remains the mainstay of treatment for those who present early with widespread pleural contamination. These patients invariably exhibit a systemic inflammatory response and are acutely unwell. If there has been a long delay before diagnosis, a primary oesophageal repair is prone to leak owing to local sepsis and tissue oedema^{15,17}. The preferred technique in this study was repair over a T tube to create a controlled oesophagocutaneous fistula^{8,10}. This was successful in minimizing mediastinal and pleural sepsis, with only one patient requiring repeat thoracotomy. Of equal importance to the type of repair are thorough lavage and debridement, and careful placement of mediastinal and pleural drains. No patient in this series required oesophageal resection, which would have been considered as a salvage procedure if oesophageal repair had not been possible. Good results have been reported in small series using oesophagectomy and exclusion for selected patients with severe mediastinal sepsis^{28,29}, although such patients subsequently require further major surgery for oesophageal reconstruction.

The overall hospital stay, intensive care stay and in-hospital mortality rate in the operative group were higher than those in patients managed non-operatively, and in some smaller series³⁰. This may be partly because all the surgical patients were at the severe end of the disease spectrum, reflected by five early deaths from multiorgan failure despite aggressive supportive treatment. The overall mortality rate of 27 per cent is comparable with published values^{3,6,27} despite there being no case selection in this series, which included even moribund patients in whom supportive care only was instituted.

Recent studies have proposed the insertion of self-expandable stents to treat oesophageal disruption^{31,32}. Although immediate stent insertion following iatrogenic perforation of advanced oesophageal carcinoma may be appropriate³³, a delay to stent insertion and the presence of contamination are inevitable with spontaneous rupture and may explain why such patients had poor outcomes in recent reports^{31,32}. In this situation the stent may prevent adequate drainage of sepsis, delay healing and potentially erode adjacent structures if it cannot be removed. There is also a high risk of stent migration in the absence of a mechanical stricture to hold it in place. The authors strongly recommend avoiding the use of stents in these situations.

Although surgery remains the mainstay of treatment, intensive non-operative management is possible in carefully selected patients with spontaneous oesophageal rupture. Success depends on a multidisciplinary approach with continuous reassessment and a low threshold for intervention.

Acknowledgements

The authors acknowledge Dr Alastair Gascoigne (Consultant in Intensive Care and Respiratory Medicine) and Mr Shaun Preston (Consultant Upper Gastrointestinal Surgeon) for their help with the clinical management of patients in this series.

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