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Institutional report - Esophagus

Boerhaave's syndrome: a review of management and outcome

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Abstract

Spontaneous oesophageal rupture (Boerhaave's syndrome) is an uncommon but serious condition. A retrospective review was undertaken of the management of 34 patients (age range 17–85 years) presenting between 1991 and 2006. Contrast swallow was possible in 22 patients, confirming the diagnosis in 17. Five patients showed pleural effusion on chest X-rays, with subsequent aspiration or chest drain insertion, confirming the diagnosis. Eleven needed CT scan, four of which showed evidence of a leak. Whilst some patients were referred immediately with the diagnosis, some waited up to 12 days (median delay four days). Whilst most patients were treated by operation on the day of diagnosis, diagnostic delay >24 h and delay in referral resulted in treatment delays of up to 24 days. Fifteen (44%) patients were suitable for primary surgical repair, ten were treated by aggressive conservative management with thoracotomy performed to visualise the perforation and assess suitability for primary repair, and hemithorax being debrided and drainage tubes and nasogastric tubes being accurately positioned under direct vision. One patient required an emergency oesophagectomy and eight patients were suitable only for surgical debridement, their initial diagnosis being delayed (median 2 days, range 2–18 days). The major factor determining treatment was the condition of the patient following initial resuscitation, there being a tendency for delayed referrals to be unsuitable for primary repair ($P=0.03$). Combined 30-day and in-hospital mortality was 17.6% ($n=6$). Median ICU stay was 1.5 (range 1–50) days with those with delayed diagnosis needing an average of 6.5 days (range 1–45). Median hospital stay was 21 (range 4–210) days.

Keywords: Boerhaave's syndrome; Spontaneous oesophageal rupture

1. Introduction

Boerhaave's syndrome, or spontaneous oesophageal rupture [1], is a rare but serious condition. The pathology involved is a complete, transmural laceration of the oesophagus. Patients who sustain this injury can be a rather heterogenous group. They can be very well clinically when initially seen or present moribund and with no obvious diagnosis. It is uncommon for patients to present with the classic triad of retching, sudden epigastric pain and shock, hence causing delayed diagnosis. However, the outcome relies heavily on prompt diagnosis and treatment.

The aim of this paper is to review the management of Boerhaave's syndrome in our institution from 1991 to 2006. We specifically reviewed the delay surrounding diagnosis and treatment, mode of treatment, mortality and morbidity.

2. Patients and methods

A retrospective review was undertaken of 53 patients presenting with oesophageal perforation to our unit. Fourteen were excluded as instrumental perforation, one excluded as an upper oesophageal perforation following traumatic and misplaced endotracheal intubation, one had initial surgical repair elsewhere and three sets of casenotes could not be obtained. We present data on 34 cases of Boerhaave's syndrome. These patients were aged between 17 and 85 years. We reviewed the casenotes focusing on clinical presentation, investigations performed, management selected and outcomes in terms of morbidity and mortality. We considered any diagnostic delay of more than 24 h to the probable event, to be delayed presentation.

3. Results

Of the 34 patients treated for Boerhaave's syndrome, 22 had initial history of nausea and vomiting and eight a history of gastro-oesophageal reflux disease. The presence of a hiatus hernia was found at operation in these eight. Surprisingly the remainder had no specific features in their history or initial examination to suggest a diagnosis of perforation.

Diagnosis was delayed in 20 patients, nine of whom sought medical attention only after a delay of 24 h, and eleven patients were diagnosed late whilst in hospital often following a CT performed in the absence of a diagnosis. Four of these showed convincing evidence on CT of an oesophageal leak, the remaining seven showing pneumomediastinum, pleural effusion, subcutaneous emphysema or a pneumothorax. Contrast swallow was performed in 22 patients, 17 of which definitely confirmed the pathology. The others were non-conclusive. Five patients had chest X-rays which

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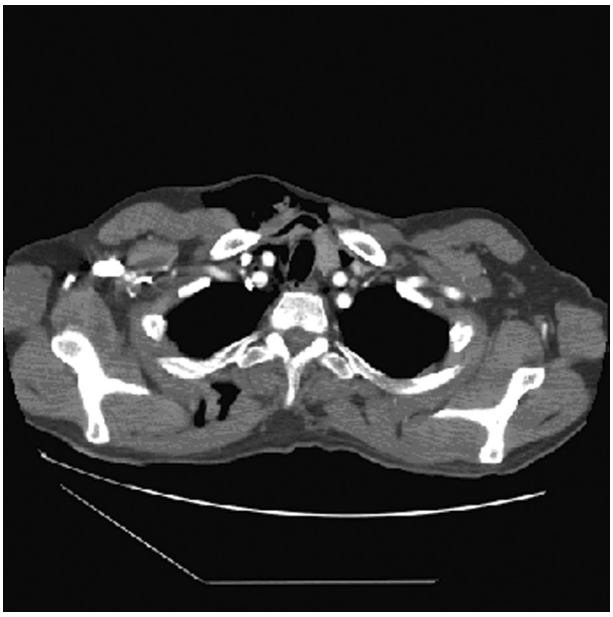

Fig. 1. CT showing subcutaneous emphysema.

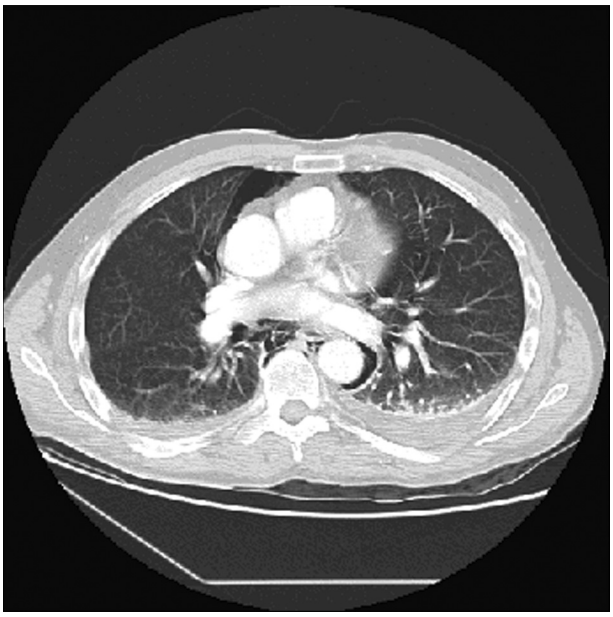

Fig. 3. CT showing right pneumothorax, pneumomediastinum and pleurisy.

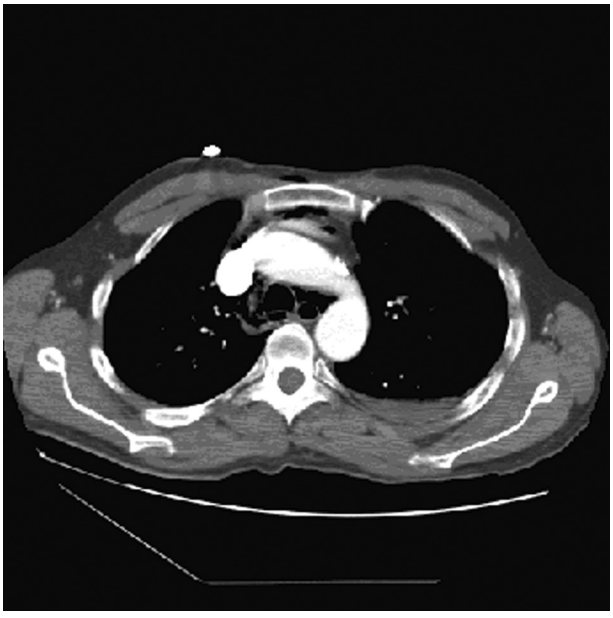

Fig. 2. CT showing pneumomediastinum.

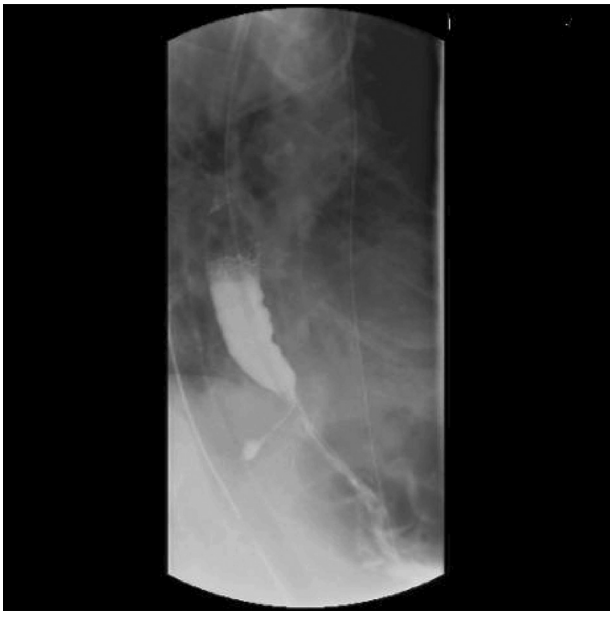

Fig. 4. Contrast swallow showing a small oesophageal leak.

showed pleural effusion. Diagnosis was only confirmed when gastrointestinal fluid was drained from the intercostal chest drain (Figs. 1–4).

Of the 34 patients, fifteen (44%) patients underwent primary repair. Median time interval from injury was 1 day (range 1–24 days), five of whom were diagnosed late (i.e. time from onset of injury to treatment in more than 24 h). A further 10 patients were treated by aggressive conservative management with functional isolation of the oesophagus and stomach using draining nasogastric and feeding nasojejunal or jejunostomy tubes, drainage of pleural cavities and antibiotics, as well as hyperalimentation, but no attempt at primary repair. Median time interval from injury to referral was four days (range 1–12 days). The remaining eight patients required surgical debridement (i.e. aggressive conservative plus either surgical debridement of mediastinum or pleural cavity) (median 2 days; range 2–18), and only one required oesophagectomy due to the extent of injury with no attempt at reconstruction. The modality of management was not found to be significantly influenced by the time of injury to transfer to our unit ($P=0.3$) but on the overall condition of the patient following initial resuscitation (see Table 1).

The combined 30-day and in-hospital mortality was 17.6% ($n=6$). The mortality rate of patients with delayed diagnosis was slightly higher at 23.8% ($P=$n.s.). 58.8% of

Table 1
Modality of management not influenced by time of injury to transfer

	Conservative	Primary repair	Debridement
<24 h	5	3	3
>24 h	5	12	5

$P=0.03$.

Table 2
Major co-morbidities were not greater when diagnosis was delayed

	<24 h to presentation	>24 h to presentation
No major co-morbidities	7	7
Major co-morbidities	5	15

$P=0.13$.

patients were complicated by major co-morbidity, compared to 68.2% in patients whose diagnosis was delayed (P=n.s.). Five patients had no associated co-morbidity. Thirteen patients had respiratory failure. The most common associated co-morbidities are sepsis, respiratory failure, cardiac complications, empyema and acute renal failure (Table 2).

Those who had early diagnosis and treatment had a median ICU stay of 1.5 (range 1–50) days, whereas in the group of patients with delayed diagnosis, ICU stay was 6.5 days (range 1–45). Median hospital stay was 20.5 (range 4–210) days compared with 22.5 (range 4–78) days in the patients with delayed diagnosis.

Median time to oral intake was 13 days (range 6–28 days) in patients who underwent primary repair, 8.5 days (range 3–25 days) in those treated conservatively and 25 days (range 18–56 days) in those who had surgical debridement.

4. Discussion

Due to the rarity of this condition and the absence of the classic triad of symptoms in presentation, Boerhaave's syndrome often presents a diagnostic challenge [2–4]. Various modalities of imaging can be used as diagnostic tools. Chest X-ray is a good preliminary test although it is quite often normal. Contrast oesophagogram is the procedure of choice [5, 6]. Our experience would support the view that CT is emerging as a very useful tool and has recently been advocated as a routine diagnostic work-up [7–10]. It has been difficult to come up with a consensus to agree on treatment protocol. Past series had been small in terms of numbers, hence it has been difficult to achieve any significant statistical analysis and outcome.

Factors that influence the prognosis of this condition are the time interval between onset of injury and primary repair as well as the underlying physical status of the patient. Generally, there are four aims in management strategy.

1. Direct repair if possible. If this is not possible, functional or surgical isolation of oesophagus from the stomach.
2. Adequate drainage.
3. Appropriate antibiotics.
4. Adequate feeding.

Unfortunately, diagnosis is often delayed due to reasons previously mentioned and there is currently no consensus with regards to the optimal management, whether surgical or conservative [11–17]. Primary repair is usually advocated, if presentation is not delayed longer than 24 h. The longer the delay, the more tissue necrosis and oedema are present, perhaps preventing successful repair. However, recently Jougon et al. [17] suggested that primary repair still yielded satisfactory results regardless of the time interval from injury to repair. Whilst we would not disagree with this view, our experience has been that few of our patients who presented late were fit for attempted primary surgical repair and we were less optimistic that repair would be successful.

Another factor adding to the difficulty in achieving a consensus view to treatment is the heterogeneity of this group of patients. As can be seen from our series, the age range can be rather wide. The severity of illness was also quite varied, regardless of time interval between injury and treatment. Some patients presented in frank septic shock requiring intubation and inotropic support, whilst on the other hand, they can appear really well with minimal signs of sepsis. Even when diagnosis had been delayed, some can be critically ill whilst others can have contained collection (Table 3).

It was shown with this review that the mortality and morbidities were not significantly affected by the treatment modality or when diagnosis was delayed. It may be that those who had survived to get to our care represented the fittest, having survived the initial injury. In fact, our

Table 3
Patients who died

Age	Time delay (days)	Treatment	Morbidity	Intubation (days)	Inotropes (days)	AICU stay (days)	Hospital stay (days)
85*	4	Conservative	Mild sepsis	No	No	0	7
78	7	Conservative	Sepsis, respiratory failure	No	No	0	49
77	7	Primary repair	Sepsis, AF, empyema, DVT, respiratory failure	16	8	16	16
75	2	Surgical debridement	Sepsis, AF, ARF, cardiac	1	2	10	10
84	<1	Primary repair	AF, sepsis	20	28	31	31
77	8	Primary repair	Sepsis, empyema, respiratory failure, DIC	1	1	2	7
61	24	Primary repair	Sepsis, respiratory failure, GI bleed	No	No	16	74

*Unrelated death.

Table 4
Outcome according to treatment modality

	Mortality (%)	Intubation (median/days)	Inotropes (median/days)	AICU stay (median/days)	Hospital stay (median/days)	Time to oral intake (median/days)
Primary repair	27	16	8	16	16	28
Conservative	20	0	0	0	18.5	32.5
Surgical debridement	13	6	3.5	9	38	28
Oesophagectomy	0	13	6	14	68	N/A

mortality is on the lower range compared to previously quoted mortality by other series. Most other series did not mention the time taken from injury to oral intake. Lawrence et al. [11] reported that in patients treated conservatively for contained leak, healing occurred in one week. In patients treated with primary repair (and one oesophagectomy), the tear was healed in 1 week in 13 patients but was 2 weeks in 4 patients (Table 4).

The success of our management can be attributed to the fact that we individualised the therapy to each patient. Generally, we aim to perform primary repair when time of injury was <24 h and if the condition of the patient permitted this. However, when this is exceeded, we treat them conservatively, but aggressively, with surgical drainage. During thoracotomy, we assess the suitability for primary repair.

5. Conclusion

Boerhaave's syndrome is indeed a rare and serious clinical condition. It has a high demand for critical care services and high mortality rate. We were fortunate to experience a fairly low mortality rate. It may be appropriate to manage patients aggressively with surgical debridement when patients present late.

Acknowledgments

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