e - ISSN - 2349 - 8005

INTERNATIONAL JOURNAL OF ADVANCES IN CASE REPORTS

Journal homepage: www.mcmed.us/journal/ijacr

OESOPHAEAL PERFORATION SECONDARY TO PORK BONE

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Article Info

Received 15/01/2015
Revised 27/02/2015
Accepted 15/03/2015

Key words:
Oesophageal Perforation; Food Bolus; Pleuritic chest pain; Non-operative Management; Pleural Effusion; Mediastinitis; Boerhaave syndrome; CT Contrast Study.

ABSTRACT

We present the case of a 53 year old male, who presented with oesophageal perforation that occurred post a choking on a food bolus with bone. Initially, it was misdiagnosed to be a cardio-respiratory event. On day 2 post presentation, it was confirmed to be a lower oesophageal perforation, with minimal contamination which was managed non-operatively. A case report and review of the literature is presented. This case report highlights the importance of having a high degree of suspicion for oesophageal perforations.

INTRODUCTION

Oesphageal perforation is usually iatrogenic (at therapeutic endoscopy) or due to 'barotrauma' (spontaneous perforation). In contrast to spontaneous perforations, many instrumental perforations can be managed conservatively. Spontaneous perforation is often a life-threatening condition that regularly requires surgical intervention. Perforations can be caused by bones ingested as a part of a food bolus [1]. We shall be presenting a case of oesophageal perforation in a 53-year-old male secondary to food bolus with bone, and followed by successful non-operative conservative treatment.

Case report

A 53-year-old gentleman otherwise fit and well, presents to the Emergency Department in the evening with the complaints of Odynophagia and Pleuritic chest pain. He had choked on a bolus of pork, and developed chest pain. He was brought into the hospital by the ambulance, haemodynamically stable and afebrile. Since the episode, he was unable to swallow any solid food at all. Based on the presentation, a provisional diagnosis of cardio-respiratory pathology was formulated, and the patient was investigated on the Acute Coronary Syndrome (ACS) pathway. All the cardio-respiratory investigations were unremarkable, and the patient was discharged home in the morning after being observed overnight. [Fig 1].

The patient was re-referred by his General practioner to the Emergency Department the next day with similar complaints. On presentation to the hospital, the patient was haemodynamically stable, although febrile at 38.3. This time, a surgical consultation was requested from the Emergency Department. On this presentation, the patient had leucocytosis 12.8x109/L, and a CRP of 107. The patient was able to swallow water but with little discomfort in the epigastric region. On examination, mild tenderness was noted in the epigastric region on deep palpation, otherwise unremarkable examination.

The chest X-ray revealed a small left sided pleural effusion [Fig-2]

The patient was reviewed by the surgical registrar, and was noted to be stable. He was referred to the medical team, with a provisional diagnosis of pleurisy, which was negated by the Medical Consultant. Finally, the patient was admitted under the General Surgery team, and had a CT scan with contrast. The CT showed contrast extravasations at the distal oesophagus, along with bilateral pleural effusion Fig-3.

The patient was transferred to a tertiary centre for the management of this lower oesohageal perforation. An urgent gastrscopy was performed, and a food bolus with bone was retrieved from the lower oesophagus. He was managed with a non-operative conservative approach on intravenous antibiotics, TPN, Nil by mouth and intravenous fluids. He underwent a contrast swallow test a week post admission to the tertiary hospital, which was unremarkable. Free fluids were commenced and the diet was advanced over next few days. The patient was discharged in a stable condition on the 10th day of admission on oral proton pump inhibitors.

Fig 1. Unremarkable Chest X –ray

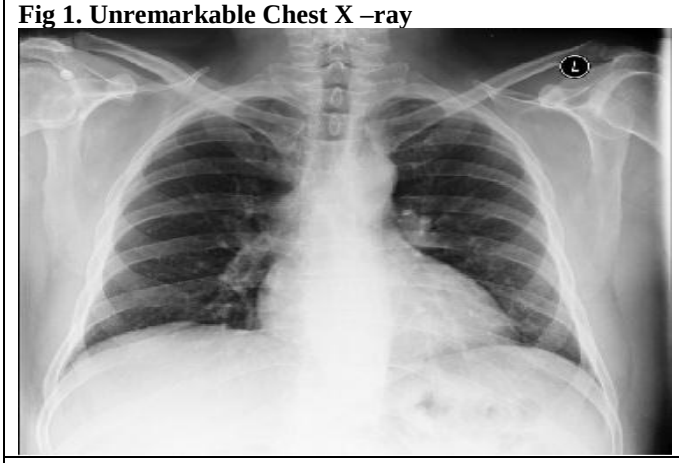


Fig 2. Chest X-ray with small left sided pleural effusion

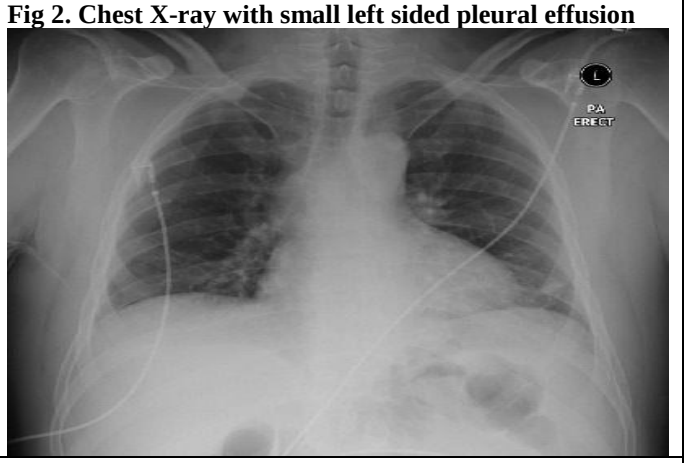


Fig-3(B). Lower oesophageal peforation with bilateral pleural effusion, and contrast extravasation

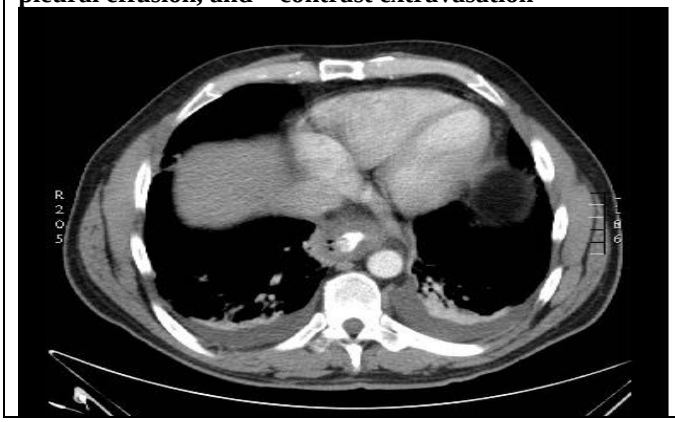
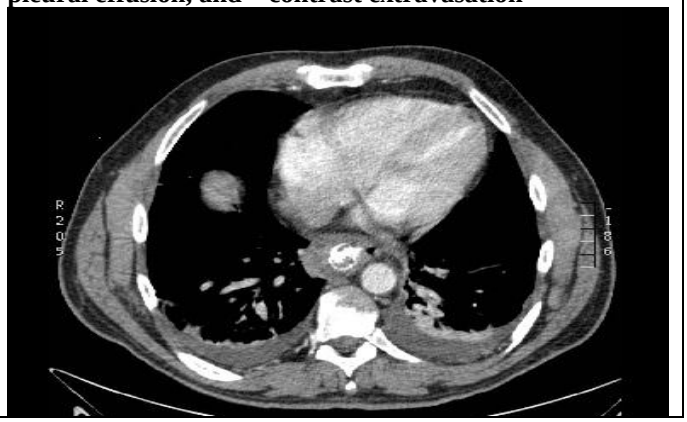


Fig 3(A). Lower oesophageal peforation with bilateral pleural effusion, and contrast extravasation



DISCUSSION

Oesophageal injuries can be managed with conservative management or operative intervention, with supportive measures to control sepsis and provide adequate nutrition [2, 3].

Treatment selection should be based on patient condition and performance status, timing of diagnosis, resources available, oesophageal pathology, and presence/absence of complications, local phlegmon, and/or sepsis [4].

This condition was first reported by Boerhaave, who reported the case of a grand admiral of the Dutch fleet who was a glutton and practised autoemesis. Due to the large volune of material that is released under pressure as a result of perforation, Boerhaave syndrome is the most serious type of perforation. This leads to a rapid chemical irritation in the mediastinum and pleura followed by infection if left untreated.

Foreign bodies, usually bones: chicken, fish, pigeon, rabbit and pork causes. Oesophageal perforations [5]. They puncture the oesophageal wall directly or can cause perforation by pressure necrosis ultimately leading to perforation. Other notable foreign bodies include button batteries, which require urgent retrieval because of their alkaline contents [6,7].The diagnosis of spontaneous perforation can be a clinical dilemma, which at times can be misdiagnosed due to a low degree of suspicion. History and associated clinical features should prompt the clinician

to consider oesophageal perforation as a part of the diagnosis. The history is usually of severe pain in the chest or upper abdomen following a meal or a bout of drinking. Associated shortness of breath is common. There may be a surprising amount of rigidity on examination of the upper abdomen, even in the absence of any peritoneal contamination.

A chest X-ray is often confirmatory with air in the mediastinum, pleura or peritoneum. Pleural effusion occurs rapidly either as a result of free communication with the pleural space or as a reaction to adjacent inflammation in the mediastinum. A contrast swallow or CT is nearly always required to guide management.

The appropriate management of oesophageal perforation is a controversial issue [8

The decision between operative and non-operative management rests on four factors, which are:

1 the site of the perforation (cervical vs. thoracoabdominal oesophagus);

2 the event causing the perforation (spontaneous vs. instrumental);

3 underlying pathology (benign or malignant);

4 the status of the oesophagus before the perforation (fasted and empty vs. obstructed with a stagnant residue). [9].

Conservative management might be appropriate in patients who have remained clinically stable despite diagnostic delay. The principles of non-interventional management involve hyperalimentation, preferably by an enteral route, nasogastric suction and broad-spectrum intravenous antibiotics.

Surgical management is required whenever patients:

• are unstable with sepsis or shock;

• have evidence of a heavily contaminated mediastinum, pleural space or peritoneum;

• have widespread intrapleural or intraperitoneal extravasation of contrast material

Patients with small well-defined tears and minimal extra - oesophageal involvement may be better managed by non-operative treatment [10, 11].

Adequate analgesia including narcotic analgesia should be provided to control pain and discomfort, but it should be used judiciously in hypotensive patients. Intercostal chest tube should be placed to decompress the chest as and when necessary.

Percutaneous gastrostomy may also be considered. Recently endoscopic placements of removable covered oesophageal stents have been described in the care of patients with oesophageal perforation with excellent results [12-13].

Major prognostic factors determining mortality are the cause and location of the injury, the presence of underlying oesophageal pathology, the delay in diagnosis and the method of treatment.

CONCLUSION

In conclusion, oesophageal perforation in adults is a highly morbid condition with an associated higher mortality. Mortality rates mainly depend on time of presentation and etiology of perforation.

The overall mortality is 20-25%, while iatrogenic instrumental perforation has a lower mortality of 10%, and postemetic perforation has a higher reported mortality rate of 60-70%. The reported mortality from treated oesophageal perforation is 10% to 25%, when therapy is initiated within 24 hours of perforation, but it could rise up to 40% to 60% when the treatment is delayed beyond 48 hours. The mortality rates are also higher in patients with thoracic and abdominal rupture and underlying oesophageal disease like malignancy and benign stricture. Hence, a high index of suspicion is necessary to diagnose the oesophageal perforation.

Competing interests

Nil

Author's contributions

HM-data collection and writing the article, AH- proof reading and the consultant in charge

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