A Rare Case of Boerhaave Syndrome with Bilateral Empyema

Parth Gupta, Naresh Patel**, Tushar Patel***, Rushi Patel****

Abstract

Effort rupture of the Esophagus or Boerhaave syndrome is a spontaneous perforation of the esophagus that results from a sudden increase in intra-esophageal pressure combined with negative intrathoracic pressure. A 25 year old male patient presented with complaint of dyspnea on exertion (MMRC grade-1), low grade fever, hemoptysis and dull type of chest pain for 2 months. Patient was previously underwent Upper GI Endoscopy which showed distal esophageal tear with perforation (Boerhaave syndrome). Esophageal stenting was done. After re-admission, Patient was successfully cured with medical management was done with antibiotics, supportive medication & adequate drainage with tube thoracostomy.

Key Words: Management, Oesophageal tear, Pleural effusion.

Introduction:

Effort rupture of the Esophagus or Boerhaave syndrome is a spontaneous perforation of the esophagus that results from a sudden increase in intra-esophageal pressure combined with negative intrathoracic pressure (e.g. severe straining or vomiting). Boerhaave syndrome usually occurs in patients with normal underlying esophagus; however, a subset of patient with Boerhaave syndrome has underlying eosinophilic esophagitis, medication induced esophagitis,Barrett's or infectious ulcers.[1] Most common anatomic location of tear is at the left postero-lateral wall of the lower third of the esophagus, 2-3 cm proximal to the gastro esophageal junction. [2,3] Incidence of Boerhaave syndrome is relatively rare, incidence of 3.1 per 1 lakh person per year. Esophageal rupture may lead to the development of septicemia, pneumomediastinum, mediastinitis, massive pleural effusion, empyema or subcutaneous emphysema. [2,3]

* Resident
** Professor & Head
*** Associate Professor, Department of Respiratory Medicine, GCS Medical College, Hospital & Research Centre, Ahmedabad, Gujarat, India
Correspondence : Dr. Parth Gupta
E-mail : guptaparth.0401@gmail.com

Case Report:

A 25 year old male patient presented with complaint of dyspnea on exertion (MMRC grade-1), low grade fever, hemoptysis and dull type of chest pain for 2 months. Patient was previously admitted in a private hospital where he had undergone Upper GI Endoscopy which showed distal esophageal tear with perforation (Boerhaave syndrome). Esophageal stenting was done. CT Chest with contrast showed subcutaneous emphysema of anterior chest wall and bilateral pleural effusion. USG chest showed mild free fluid in left pleural cavity with collapse. Left side intercostal drainage (ICD) insertion was done and fluid was sent for routine microscopy, cytology, gene expert and culture all of which were negative. Patient then underwent bronchoscopy. BAL culture showed Klebsiella pneumonia. Gene expert and cytology of BAL were negative. Klebsiella pneumonia was managed with sensitive antibiotics. ICD and esophageal stent were removed and patient was discharged with oral antibiotics in stable condition. Patient then came to GCS hospital after few days of discharge with redevelopment of bilateral pleural effusion. On examination the patient was vitally stable but febrile. There was no pallor / cyanosis/ edema/ clubbing/ icterus. On auscultation air entry was reduced in bilateral lower lung zone. There was no

hepatomegaly or splenomegaly. Chest X-ray was suggestive of bilateral pleural effusion right > left (fig-1).

Routine investigations were performed which showed mild leukocytosis (12300 /cumm) and hypoalbuminemia (2.5 mg/dl). Rest of blood investigations including PT-INR and APTT were normal. USG chest showed moderate pleural effusion on right and mild effusion on left side. Empirical antibiotics viz inj. Cefoperazone + Sulbactum 1.5 g iv b.d. and inj. Metronidazole 500 mg iv t.d.s. for both gram positive and gram-negative coverage were given and ICD was inserted on right side. Post ICD CT Chest contrast was done which was suggestive of bilateral pneumothorax, visualized portion of esophagus appeared normal, thick walled cavitory lesion in left lower lobe and subcutaneous emphysema (fig- 2).

Pig tail was done on left side to drain cavity. Pleural fluid from both sides was sent for routine microscopy, gene expert, cytology and culture and sensitivity. Pleural fluid reports from ICD were suggestive of transudative effusion while that of pigtail were suggestive of sterile empyema. Patient was then kept under observation and daily drain noted. After 2 days of pig tail insertion, it was removed as the purpose was solved. However due to persistent drain of around 50 ml from the right sided ICD; it could not be removed and the patient was discharged with oral antibiotics viz tab. Cefixime + Clavulanic Acid (200+125) b.d. and tab Metronidazole 400 t.d.s. and ICD care explained. Patient was kept in regular follow up of 5 days and chest x-rays were repeated at every visit to ensure proper drainage. On the chest x-ray of 2nd follow-up there was minimal haziness in right side and left side was clear. Patient was readmitted; USG chest showed 20 cc collection in right side and hence ICD was removed and patient was discharged the next day with regular weekly follow up and continuation of previously mentioned oral antibiotics. After one month x-ray showed complete radiological resolution.

Figure 1: X-ray suggestive of bilateral pleural effusion

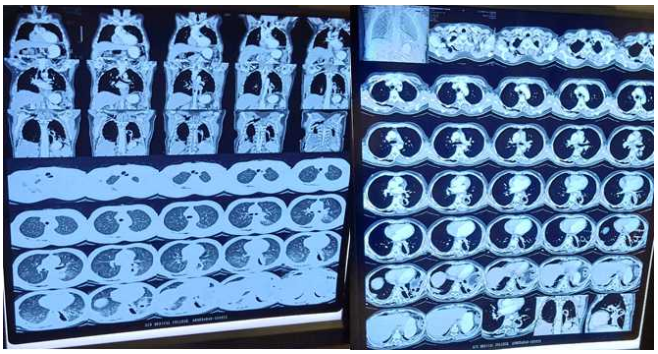
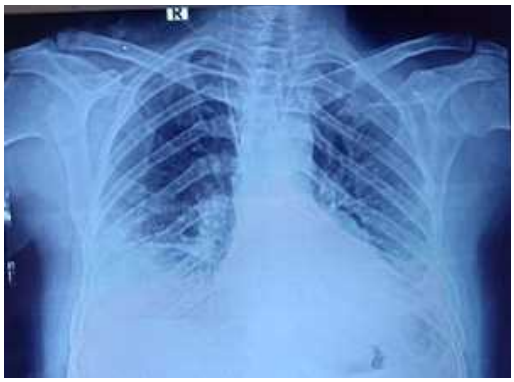


Fig 2: CT scan of right sided chest post-ICD



Discussion:

Boerhaave syndrome typically occurs after forceful emesis. Boerhaave's syndrome is a transmural perforation of the esophagus. Common causes of esophageal perforation include medical instrumentation, foreign-body ingestion, and trauma. Iatrogenic esophageal perforations have become more common due to the rapid increase and development of upper gastrointestinal tract endoscopies and now account for 63.9% of perforations. A further 16.7% can be attributed to foreign bodies, 13.9% to external trauma, and 5.5% to spontaneous rupture (Boerhaave's Syndrome). Other causes of spontaneous perforation include caustic ingestion, pill esophagitis, Barrett's ulcer and infectious ulcers in patients with AIDS, and following dilation of esophageal strictures.[4]

Perforation of the distal oesophagus usually results in pleural effusion or hydropneumothorax on the left, while perforation of the midoesophagus tends to produce pleural effusion or hydropneumothorax on the right. Spontaneous perforation (Boerhaave's syndrome) usually occurs in the distal oesophagus, with resultant hydropneumothorax on the left. The clinical

picture depends on the level of the perforation and the time interval from rupture to presentation. The mid oesophagus lies next to the right pleura while the lower oesophagus abuts the left pleura. Once a perforation occurs, saliva, retained gastric contents, bile and acid enter the mediastinum, resulting in mediastinitis, pneumo-mediastinum and pleural collections. Initially, the patient presents with pain at the site of perforation usually in the neck, chest, epigastric region, or upper abdomen.[5] The gold standard for diagnosis is to perform a contrast enhanced esophagogram, initially with water-based contrast such as gastrograffin. If this study is negative and the index of suspicion is still high, diluted barium contrast should be performed to confirm the diagnosis and localize the perforation. Due to the nonspecific nature of its presentation, diagnosis is often delayed, since it is a rare disease and is usually confused with other equally serious but more prevalent pathologies such as acute myocardial infarction, perforated peptic ulcer and acute pancreatitis.[6] Management of oesophageal perforation is either surgical or medical. Surgery has been the most common treatment. Barrett described the first successful surgical repair of the oesophagus in 1947.[7] Surgical repair within 12 to 24 hours is associated with a mortality rate of 34% while a delay of 24 hours is associated with 64% mortality.[8] The present case managed medically consisting of intravenous fluids, broad-spectrum antibiotics, nasogastric suction, no oral intake, and adequate drainage with tube thoracostomy. Early use of nutritional supplementation was helpful.

Conclusion:

Oesophageal perforation is a serious condition with a high mortality rate. Successful therapy depends on the size of the rupture; the time elapsed between rupture and diagnosis, and the underlying health of the patient. The diagnosis (as in our current case) is often missed initially because this syndrome is rare and the conditions that can mimic it are more common. Direct repair of the rupture and adequate drainage of the mediastinum and pleural cavity provide the best survival rates.

References:

1. Brauer RB, Liebermann-Meffert D, Stein HJ, et al. Boerhaave's syndrome: analysis of the literature and report of 18 new cases. Dis Esophagus 1997; 10:64.
2. Ckorn O, Onate JC, Lopez R. Anatomy of the Boerhaave syndrome. Surgery. 2007 Feb. 141 (2). 222 8 (Mediline).
3. D E Skipper JP, Pull ter gunne af, oostovogel Hj, Van Laarhoven CJ. Spontaneous rupture of esophagus Boerhaave syndrome in 2018. Literature review and treatment algorithm. Dig Surg. 2009. 26 (1) 1-6 (Medilink).
4. Eroglu A, Can Kürkçüogu I, Karaoganogu N, Tekinba C, Yimaz O, Ba og M. Esophageal perforation: the importance of early diagnosis and primary repair. Dis Esophagus. 2004;17(1):91–4.
5. Katsetos MC, Tagbo AC, Lindberg MP, Rosson RS. Esophageal perforation and mediastinitis from fish bone ingestion. South Med J. 2003;96(5):516–20
6. Fell SC. Esophageal perforation. In: Pearson FG, Cooper JD, Deslauries J, Ginsberg RJ, Hiebert C, Patterson GA, editors. Esophageal Surgery. New York: Churchill Livingstone; 2002. pp. 615–36.
7. Chu HN, Chang CH, Hui CK, Hui YL. Anesthetic management of Boerhaave's syndrome. Acta Anaesthesiol Taiwan. 2004;42(2):103–6.
8. Janjua KJ. Boerhaave's syndrome. Postgrad Med J. 1997;73(859):265–70.