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# *Boerhaave Syndrom: Management of uncommon presentation*

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**ABSTRACT:** Boerhaave syndrom or spontaneous oesophagus rupture is a rare clinical entity of difficult diagnosis. It is due to a sudden increase in intra-thoracic and intra-abdominal pressure transmitted to the non-pathogenic oesophagus. The Triad Makler involving vomiting, chest pain and subcutaneous emphysema is evocative. Positive diagnosis is based on oesophageal transit of hydrosoluble and thoracic scann after opacification by oral water- soluble. The treatment of perforation is surgical. However the success of the surgery involves the earliness of and quality of peri-operative intensive care. In this paper, we report an unusual presentation of transmural oesophageal perforation - located at the third lower- with severe abdominal pain and massive bilateral haemothorax. The interest of this paper focuses on diagnostic difficuties in unfavorable environment and on the earliness of the treatment

**Keywords**: *spontaneous rupture of the oesophagus, septic shock, mediastinitis.*

## Introduction

Boerhaave's syndrome was first described in 1724 by Hermann Boerhaave. It is rare and serious nosological entity that carries significant risks of diagnostic errors. This syndrome is a spontaneous rupture of œsophagus in the absence of pre-existing affected œsophagus [2]. There are no specific clinical signs. So Symptoms may vary. However, the triad of Mackler are evocating. They consist on: Vomiting, thoracic pain and sub-cutaneous emphysema [1,3]. In this paper we repport an uncommon presentation of transmural oesophagus perforation -on the third bottom end- with intense abdominal pain and bilateral haemothorax. The interest of this case is about the difficulties of diagnostic and which are the better care in underprivileged environment.

## Observation

It is about a 49 years old chronic alcoholic male patient, admitted in the emergency department of the Essos hospital center for intense abdominal pains associated to nauseas and vomiting.
The onset of symptoms is traced back to 10 hours prior admission. It was marked by premature postprandial food vomiting following a copious meal. Followed up an intense thoracic pain like a muscle tear. The pain extended to the abdomen. The patient's history did not mention other symptoms suche trouble of abdominal transit and fiever. The clinical examination at the admission noted a conscious and apyretic patient, and do not detect any signs of shock. The abdomen was tender in all area with "peritoneal signs". Biological check-up was normal. The plain abdominal X-ray was not contributive. Presumptive diagnosis was peritonitis due to gastric perforation, and the differencial diagnosis was in favor of mesenteric ischemic syndrom. Exploratory laparotomy was performed with the patient under general anesthesia do not observe effusion of peritoneal fluid. Immediate postoperative care was marked by respiratory destress and hypoxemia.

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The thoraco-abdominal CT scan performed at the 12th hour after admission revealed a bilateral fluid pleural effusion with medium abundance on the right side and a high abundance on the left side, a pneumo-mediastinum with cervicothoracic cutaneous emphysema suggestive of a hollow organ rupture intra-mediastinal. The CT injected sequence revealed a transmural oesophageal perforation of the lower third level. That by showing the passage of the contrast medium through the fistulous pathway. Pleural drainage was performed by the resuscitator with respect to chest imaging.

Progression to a septic shock state at the 15th hour post admission has justified mechanical ventilatory support and haemodynamic support by synthetic catecholamines. The patient were reoperated for a left postero-lateral thoracotomy at the level of the seventh intercostal space. He also profited of oesophageal repairs. The surgical exploration showed a purulent potion collected in the left postero-latero-basal pleura.

There was also a thickening of the parietal pleura and basal atelectasis. In mediastinum, we finded multiple adherences, necrotic and food debris with perforation 5 cm from the oesophageal hiatus.

The repair of the osophagus was realised after pleuro-mediastinal toilet.

The patient was transfered to ICU (intensif care unit).

The patient died on the second day post-operative care.

**Discussion**

Oesophageal perforation is considered as the most fatal of all digestive tract perforations [1]. Boerhaave syndrome is a spontaneous rupture of the esophageal wall. It concerns man in 80% of cases, with an age of predilection between 40 and 60 years old [1]. Despite surgical evolution of techniques and resuscitation measures, the prognosis remains dark and depends on the early diagnosis.

The mortality rate is of 100% in the absence of treatment, and is up of 50% in the series of patients treated. This syndrome was first described by Herman Boerhaave, who reported case of a perforation that occurred at the grand admiral of the Dutch fleet Baron van Wassenaer. The perforation appeared after major vomiting after a copious meal and well watered [2]. The triggering element is usually an episode of vomiting although other causes have been reported such as the Hemlich maneuver or uplift efforts that grossly increase intra-esophageal pressure [1]. The perforation is most often located on the left postero-lateral border of the lower thoracic esophagus at 3 to 5 cm above the diaphragmatic hiatus [2, 3]. It is noted that there are no pathognomonic signs of spontaneous rupture of the esophagus [1, 3, 4]. This situation is contributing to making late diagnosis especially in front of an array of mediastinitis. Howevr Mackler's triad are represented by vomiting after a large meal, thoracic chest pain and subcutaneous emphysema is strongly suggestive of the syndrome. Water-soluble oesophageal transit helps to confirm the diagnosis by revealing leakage of the contrast material into the pleura and / or the mediastinum. Computed tomography with water-soluble opacification shows extra-luminal digestive leakage [1, 5, 6]. Treatment of the oesophageal breccia is surgical [6, 7,8].

Intensive care measures must always frame the surgical procedure. They include broad-spectrum probabilistic antibiotic therapy that will be reorientedregarding the bacteriological study of intraoperative specimens, correction of hemodynamic and ventilatory disorders, as well as disorders of the acid-base balance.

In our observation, we note that abdominal pain symptomatology simulating a peritoneal syndrome or mesenteric ischemia syndrome and a clinico-biological context not very specific. prognosis of our patient.

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All that had contributed to delay the diagnosis of spontaneous rupture of the esophagus. This misguided initial diagnosis also led to delaying the IC measures and thus to burden.
The management of Boerhaave syndrome has long been the subject of much controversy. Indeed some medical teams arguing that medical treatment may be a therapeutic option and that surgical treatment depends on the severity of oesophageal lesions.
It is now recognized that multidisciplinarity is very important. Surgery represents the radical sanction, but its success remains closely linked to the importance of initial IC.
The occurrence of death at the furst 48th hour postoperative, in a septic shock case suggests that the perioperative IC occupies a preponderant place in the management of Boerhaave syndrome.

## Conclusion

Boerhaave syndrom is a rare clinical entity.
This diagnostis remains difficult. The prognosis is related to the precocity of the diagnosis and the initial intensive care **[2, 7,8].** The treatment of the oesophageal perforation is surgical.
The peroperative IC is the main point of surgical succes.

## Conflict of interest

The authors declare no conflict of interest.

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