J Med Sci 2009;29(4):225-228
http://jms.ndmctsgh.edu.tw/270225.pdf

Boerhaave Syndrome Secondary to Renal Colic

Wen-I Liao[1], Chia-Chen Liu[2], Tsai-Wang Huang[3], and Ko-Chiang Hsu[1*]

[1]Department of Emergency Medicine; [2]Department of Radiology; [3]Division of Thoracic Surgery, Department of Surgery, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, Republic of China.

Boerhaave syndrome (BS) or spontaneous esophageal rupture represents a special instance of baro-trauma to the lower esophagus; this syndrome is rare and potentially fatal if treatment is delayed or there is a missed diagnosis. The triad of chest pain, vomiting and subcutaneous emphysema is critical for the diagnosis. However, difficulties occur when the diagnosis takes place at the emergency department (ED) and may arise as a result of an atypical presentation; this can lead to a challenging emergency. Here we report a case of patient at an ED who sustained BS and was initially diagnosed as having left renal colic. We emphasize that a high index of suspicion as well as a thorough systematic evaluation using imaging approaches promptly together with a judicious emergency procedure, in this case tubal thoracostomy, are the key points to identify the associated life-threatening pathology in these patients.

Keywords: Boerhaave syndrome, esophageal rupture, renal colic, multidetector computed tomography

INTRODUCTION

Boerhaave, a Dutch physician, described a case of sudden chest pain after forceful vomiting in 1724; that patient sustained shock and death within 18-hours.[1] During autopsy, Boerhaave found a tear in the patient ' s distal esophagus with food particles and fluid in the pleural cavities. The spontaneous (non-traumatic) rupture of the esophagus was therefore named Boerhaave syndrome (BS). In the emergency department (ED), the clinical diagnosis of a BS may not always be easy, mainly due to the fact that the condition mimics a variety of other emergency clinical entities including acute coronary syndrome, congestive heart failure and pulmonary edema.[2] Unfortunately, a significant delay in correct diagnosis can result in substantial morbidity and mortality.[3] Here we present a case of BS that presented secondary to a left renal colic. The patient came to our ED with his chief complaint being severe left flank pain with dyspnea and fever.

Received: January 13, 2009; Revised: April 6, 2009; Accepted: May 19, 2009
*Corresponding Author: Ko-Chiang Hsu, Department of Emergency Medicine, Tri-Service General Hospital, National Defense Medical Center, No. 325, Sec. 2, Cheng-gong Rd., Taipei 114, Taiwan, Republic of China. Tel: +886-2-87923311 ext. 16877; Fax: +886-2-87927034; E-mail: kc0611@gmail.com

CASE REPORT

A 43-year-old man was brought to our ED with severe left flank pain for one hour before his presentation. He had undergone many episodes of left renal colic based on his medical record at our ED from one month previous. The pain was described as severe, cramping in quality, located in the left lateral and lower chest wall to the inguinal region, with an exacerbation after two episodes of forceful vomiting following an abundant lunch. There had been no event that involved trauma.

On physical examination, his vital signs included a blood pressure of 130/68 mmHg, a temperature of 36.1°C, a pulse rate of 115 beats per minute, and a respiratory rate of 18 per minute with an oxygen saturation of 98% under O_2-cannula 4 liters per minute. He was alert, but obvious distressed due to the severe left flank pain. There was tenderness to percussion over the left costo-vertebral angle and no palpable subcutaneous emphysema in the skin. Clear breathing sounds were noted in auscultation on arrival. There was no heart murmur and the abdomen was soft with normoactive bowel sound. The EKG revealed sinus tachycardia and the cardiac isoenzymes were normal. There was microhematuria (red blood cell 12-14 per high power field) noted on urine analysis. The plain abdominal radiograph shows a faint area (about 0.3 cm) of calcification just below the left sacroiliac joint. The abdominal ultrasonography revealed a tiny left upper third ureter stone about 2.8 mm with mild hydronephrosis. A diagnosis of left renal colic was made.

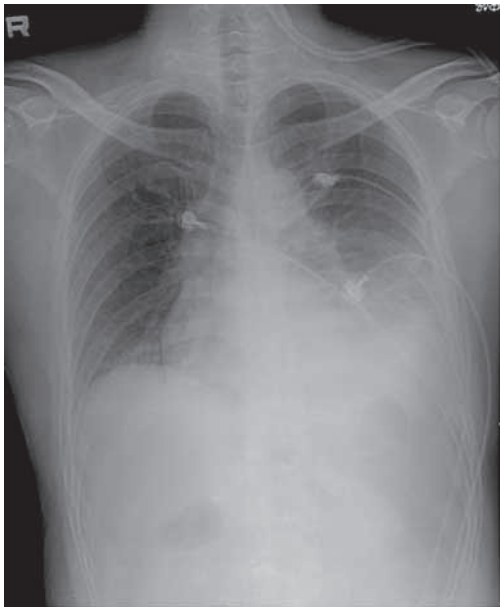

Fig. 1 Chest radiograph reveals a massive pleural effusion of the left hemithorax.

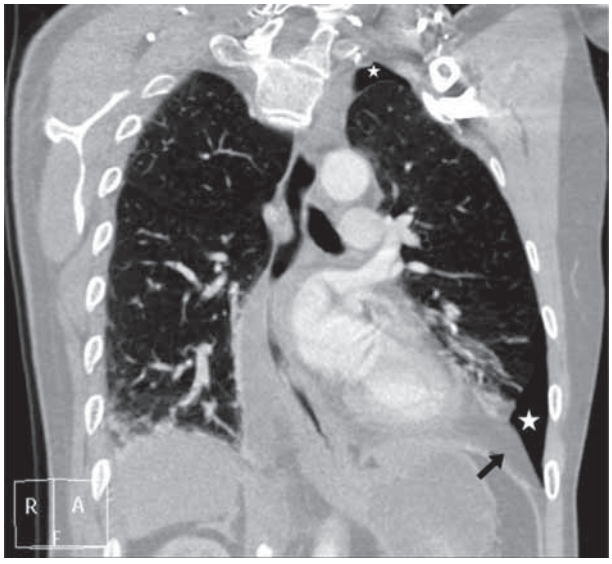

Fig. 2 MDCT of the chest with oblique reformation demonstrated a considerable amount of left-sided pleural effusion (arrow) along with a mild pneumothorax (stars).

Nevertheless, an excruciating left flank pain propagating to the left lower chest could not be ameliorated by analgesics. While the patient was still under emergency observation, he developed dyspnea and fever (38.7°C). A slightly decreased breath sound and a crackling sound on the left hemithorax were noted during re-evaluation. Accordingly, upright chest radiography was performed, which revealed a massive pleural effusion of the left hemithorax (Figure 1). The laboratory tests revealed white blood cell count of 17,100/mm^3 and a hemoglobin of 12.2 g/dl. The remaining tests, including serum concentrations of glucose, urea, creatinine, sodium, potassium, lipase, bilirubin and liver functions were normal.

Multidetector contrast-enhanced computed tomography (MDCT) of the chest demonstrated a considerable amount of left pleural effusion along with a mild pneumothorax (Figure 2). A tubal thoracostomy was inserted into the left pleural space, which allowed the extraction of turbid fluid (Figure 3A) and partially digested food particles (Figure 3B); this was similar to the fluid aspirated from a nasogastric tube. The turbid fluid was sent to the laboratory and the results were a pleural fluid protein of 3.2 g/dL (serum protein level, 5.3 g/dL; ratio 0.6), a LDH of 164 U/L (serum LDH level, 256 U/L; ratio 0.64), a glucose of 95 mg/dL, a pH of 7.12 and elevated amylase at 986 U/L; these results are compatible with exudative pleural effusion. A diagnosis of BS was made.

The patient received empirical antibiotic treatment with ampicillin plus sulbactam and metronidazole intravenously. In addition, an emergency surgical consultation took place and exploratory thoracotomy with thoracic debridement and primary repair of the perforation (about 2 cm) present in the lower esophagus (Figure 4), just above the hiatus, was accomplished within six hours. The patient was admitted to hospital under the surgical services and stayed for 3 weeks. Before his discharge, he received an esophagography, which showed no evidence of contrast leakage.

DISCUSSION

BS is a serious but rare condition in the ED, and many physicians may be unfamiliar with its clinical features; on this basis, the diagnosis may be initially missed or delayed.[4] Furthermore, an unusual presentation may confuse the emergency physician and also result in a misdiagnosis. Here we present a patient who sustained BS and was initially diagnosed in the ED as having left renal colic. Numerous causes of BS, including defecation, parturition, coughing and seizure, have been proposed in previous reports.[5-7] A review of the medical literature has not revealed any relationship between BS and renal colic. This is probably a reflection of the fact that the above combination of events is extremely rare.

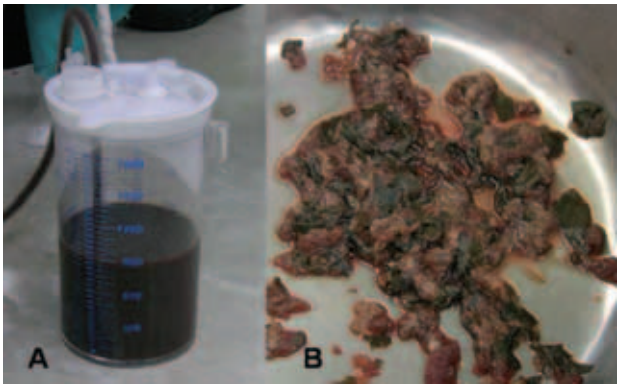

Fig. 3 Extraction of turbid fluid (A) and partially digested food particles (B); these were drained from the left-sided tubal-thoracostomy.

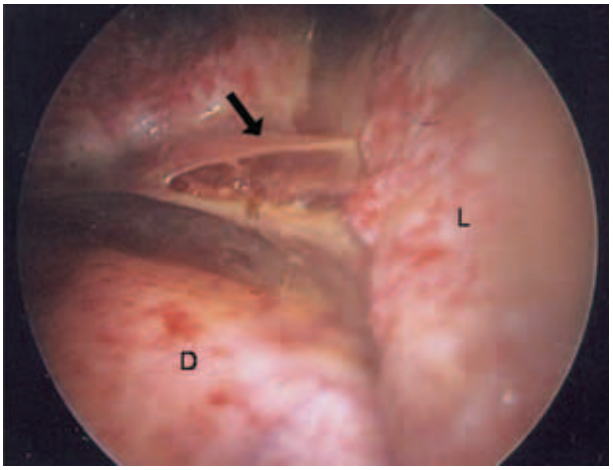

Fig. 4 The esophageal perforation is about 2-cm in length (arrow) and present in the lower esophagus just above the hiatus. (D: diaphragm, L: Lung parenchyma).

The classical presentations of BS are identified as vomiting, chest pain and subcutaneous emphysema, which also called the Mackler's triad.[8] Vomiting, particularly after alcohol or abundant food intake, may increase intraluminal pressure, which can result in a rupture of the distal left lateral wall of the esophagus. This is the most common anatomical weak-point involved in BS. In our case, the patient underwent forceful vomiting, the most important symptom of the triad; this is not uncommonly induced by renal colic.[9] Without detailed history taking or a physical examination, we are unable to present a complete picture of the clinical correlation between BS and renal colic. Nonetheless, based on the initial history of nausea and vomiting noted in 22 out of 34 patients from an institutional report, there were still 20 patients who underwent a delayed diagnosis.[10] In this context, a previous episode of forceful vomiting happens in less than 80% of all BS cases from a large series report, even though the event is emphasized as a major hint for the diagnosis.[11]

In addition to the above, an analysis of the pleural effusion is also helpful when diagnosing an esophagus perforation because it reveals the exudative process, the low pH level and the high amylase level. However, a low pH level is nonspecific and a high amylase level may secondary to malignancies and pancreatitis.[12] Esophagoscopy has little role in the evaluation of a suspected BS or as a means of investigating the location of perforation directly; this is because it can be difficult to perform on a critically ill patient.[13] However, an elaborate clinical imaging study may offer important clues that clarify the key features of BS. Conventional computed tomography can detect extraluminal air, periesophageal fluid, wall thickening and extraluminal contrast as well as providing additional information about the level and size of the perforation.[14,15] Recently, MDCT, which has a shorter acquisition time and superior image resolution than conventional CT, has been able to detect the smaller periesophageal air collections that indicate esophageal perforation.[16,17] In our case, we used MDCT for the differential diagnosis of the unusual presentation of a left flank pain along with vomiting. This approach finally confirmed the diagnosis from tubal thoracostomy by demonstrating exudative pleural effusion and food particles that were the same as obtained from the naso-gastric tube.

From the point of view of examining the management strategy for BS, there are four aims currently. These include direct repair if possible (<24-hours), adequate drainage, appropriate antibiotics, and adequate feeding.[10] The mortality rate can be as high as 92% if BS goes untreated and 60% if such treatment is delayed.[18] This indicates that the time course from the event to the diagnosis is very important. Thus, it can be seen that the prognosis mainly depends on the length of the delay between diagnosis and treatment. Therefore, we should keep a high level of suspicion relative to this syndrome, even in cases such as renal colic where it is a rare event. The diagnosis of spontaneous esophageal rupture should be kept in mind if a patient with renal colic combines this with other atypical symptoms such as dyspnea or pleuritic pain.

CONCLUSION

BS should be considered in patients who have renal colic symptoms with unusual presentations. We highlight that any patient who presents with abruptly left flank pain with a propagating pain to the left lower chest, dyspnea and fever following forceful vomiting secondary to a renal colic should be aggressively evaluated in order to rule out BS. MDCT and tubal thoracotomy should be performed judiciously in the ED as soon as BS is suspected. Adequate drainage of the turbid fluid, appropriate antibiotics and an early surgical repair of the perforated esophagus, are substantially helpful in lowering mortality and mobility.

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