Misleading Chest Pain

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Abstract

Chest pain is a common complaint in a patient presents to the emergency department(ED) and it caused by several life threatening conditions such as esophageal perforation. A 78 years old man known case of ischemic heart disease came into the ED with complaint of chest pain, physical examinations were normal on arrival. Electrocardiogram and chest x ray were no specific. Eventually he was admitted in CCU with acute coronary syndrome. During admission the patient got fever and productive cough. Despite antibiotic administration, patient was deteriorated and in this stage chest CT scan showed pulmonary abscesses. Having no results in spite of utilizing board spectrum antibiotic, abscess drainage under guide of sonography was performed. A large amount of pus and food particle was drainage. Gasterographin study indicated esophageal perforation and entrance of contrast to mediastan and right sided pleura. With chronic perforation he was admitted in intensive care unit (ICU) with conservative management. After two weeks, the patient was discharged from hospital with good recovery and acceptable outcome. Chest pain is a common complaint in the emergency visits. Perhaps putting wrong the first step, begin a series of measures to be unnecessary in patients and delay the correct diagnosis. Esophageal perforation is a life-threatening condition that must be identified and treated early to minimize morbidity and mortality.

Keywords Chest pain; Acute Coronary Syndrome; Esophageal perforation

Introduction

Chest pain is one of the most common complaints of patients referring the emergency unit of the hospitals and it has a variety of differential diagnoses, and most of its causes are important and life-threatening, including: acute coronary syndromes, pulmonary emboli, dissection of aorta and perforation of esophagus [1-3].

Case Report

Patient is male 78 years old and has come to emergency unit with a complaint of chest pain. His symptoms have begun last night suddenly and during resting.

Patient’s pain is severe, intermittent and constrictive and it was lasting a few minutes each time. The pain worsened by eating or exercise. Patient also had a complaint of epigastric pain and mentioned 2 times non-bloody vomiting’s before the onset of the pain. There were no other sign and symptoms such as diaphoresis, hemoptysia, loss of consciousness, dyspnea, fever and orthopnea. The patient mentioned a history of multiple hospitalizations in cardiac care unit (CCU) because of heart diseases, but since two years ago so far he stopped using all of his medications. There was no pathologic point during the physical examinations on the time of entering of the patient.

The patient’s Electrocardiogram (ECG) has no specific point except left bundle-branch block and there was no new changes compared with his previous ECGS. In chest x ray (CXR), Cardiomegaly and a mild pleural effusion was seen (Figure 1). Cardiac enzymes and clotting tests were normal. After half-an-hour patient’s symptoms became slightly better by Nitroglycerin, and finally the patient was hospitalized in CCU with the diagnosis of unstable angina. Next day an angiography was applied and there was no evidence of involvement of coronary arteries. But during this time the patient became febrile and has a complaint of productive coughs with sputum and epigastric pain. Auscultation of lungs showed a reduction in respiratory sounds in right side as well as tenderness in epigastrium and right upper quadrant (RUQ) in examination of abdomen. After taking consults from lung and infectious diseases specialists, with diagnosis of pneumonia Salbutamol, Atruvant, Ceftriaxon and Azitromycin were administered, regarding the infiltrations in lung. In surgery consulting, regarding the ultrasound imaging of the abdomen which showed a slight dilation of biliary sac and a few little stones and by noticing an Increase in serum Amylase by 3 times, nothing per ose (NPO) was applied to patient with suspicion to cholecystitis and pancreatitis and Metronidazole was added to current medications (Figure 2).

2 days later, general condition of the patient became better and abdominal pain was lessened. But already there was chest pain and fever and finally the patient was sent from the unit of cardiology and surgery to the unit of infectious diseases in order to continue the treatment. Despite of beginning of broad-spectrum antibiotics, clinical condition of the patient worsened. At this time CT-Scan of thorax showed many pulmonary abscesses as well as right-sided effusion and proper result didn’t came out of the treatments, aspiration of abscess with guiding of ultrasound was applied After putting the Catheter on the location of largest abscess, approximately 50cc overt sputum as well as food particles were sucked up. In this step, with a suspicion to perforation of esophagus, a study by gastrographin is applied which
showed discharge of food particles from esophagus to the pleura and mediastinum and this indicated the perforation of esophagus (Figure 3).

Figure 1: Cardiomegaly and plural effusion

Immediately, patient was sent to thorax surgery unit and was hospitalized in ICU. Since the perforation was chronic, sustaining treatment was applied including beginning of total parental nutrition by central vein (CV) line and administration of broad-spectrum antibiotics. In endoscopy there were evidences of hiatal hernia and severe esophageal Candidiasis, but there was no proof indicating perforation and the patient was treated and followed with diagnosis of micro-perforation. After two weeks, discharges from the location of aspiration and patient’s fever were no longer lasted. A diet of transparent liquids begins and the patient was sent to the unit of surgery. After 3 weeks, regarding absence of fever, tolerance of PO and absence of discharges from the location of aspiration, patient was permitted to go home with prescription of medications.

Discussion and Conclusion

This patient was a case of perforation of esophagus and his symptoms was demonstrated as a chest pain following a vomiting. Although the most known title for this condition is spontaneous perforation, but in fact it never happens and there is an underlying factor such as vomiting or any factor which triggers valsalva maneuver.

This disease was discussed at first by a Dutch doctor named Hermann Boerhaave in 18th century after the death of his patient Baron Jan von Wassenear. That patient had severe chest pain and dyspnea following a vomiting and autopsy showed food particles in left pleura and perforation in 5cm of distal of esophagus and emphysema in mediastinum [1]. Boerhaave syndrome are most commonly seen in men with 40-60 years old and it’s among the male individuals 205 times more common than the female ones. The most common location (90%) of perforation is 1/3 distal of esophagus which is weaker than other parts [3,4] and in contrast with Mallory-Weiss syndrome in which the perforation is limited in mucosal layer, this syndrome is transmural and in 80% of cases, is associated with left pleura [5]. The demonstration of this syndrome can be acute, subacute and chronic. In acute form, the patient will be symptomatic 24 hours after perforation In subacute form, this period would be 24h-2 weeks after perforation. In chronic form, it is more gradual and it vary from weeks to months [6,7] classic signs and symptoms of this syndrome is vomiting, chest pain under xiphoid bone and subcutaneous emphysema [8,9] which are known as Mackler’s triad. Legminas and lemke [10] reported that toxic appearance, pleural effusion, pneumothorax, leukocytosis azetomia and hypoxia strongly indicate this syndrome and these findings vary, depending on the grade of peritoneum or pleura involvement and they could be mild. Physical examination specially in primary stages doesn’t help (Coetaneous emphysema or cryptus is a key finding) and has a low sensivity (27%) labatory findings also poorly helps and maybe the most common finding is leukocytosis which is nonspecific. Pate et al by a study of 24 patients during 30 years reported that their CXR in %97 of cases is abnormal. CXR findings include pneumome diastinum, lung effusion specially in left side, pneumothorax, hydronemothorax, sub cutaneous emphysema and mediastinum widening. Existence of air also can be seen as lucent line which are divided in two parts because of cardiac fascia and has a V-shape appearance [11,12].

In this patient at first visit a mild right-sided effusion was the only pathologic finding. Other diagnostic tools to detect perforation of esophagus include esophagography with Barium and CT-Scan of thorax [13] and their application is to determine discharge of food particles and billium from pleural space into mediastinum [14]. If there is a high clinical suspicion for this syndrome, then endoscopy is valuable, but it is time-taking and is not applicable for a patient in a severe unstable condition and is usually applied for detecting the
location of perforation or ruling out the other probable causes. However in 1/3 of cases, perforation of esophagus may not be seen by endoscopy, like this patient that endoscopy did not show the location of esophageal perforation.

Immediate diagnosis and treatment of this life-threatening condition is in some cases hard and in chronic form of the disease it may be mixed up with other diseases (Pneumonia, MI, aneurism of aorta). Based on the study of Abott, et al. [14] only in 21% of cases, in first 12 hours the correct diagnosis is made.

So diagnosis of this disease should be kept in mind in every case which has a great clinical suspicion, even if the specific signs and symptoms of the disease are absent [15]. Choosing the proper sort of treatment is mentioned by Altorjay, et al. [16] in 1977 and by Cameron, et al. [17] in 1979, which are already acceptable. These suggestions depend on the cause of the disease and the location of perforation and when the time gap between onset of damages and clinical diagnosis is short or very late (more than 24 h) sustaining treatment is beneficial. Also if the disease is totally treated by sufficient aspiration of pleural fluid, there will be no need for surgery since in this condition a esophage-coutaneous fistula forms and is repaired like the most of gastro-esophageal fistulas [18]. But in sepsis with unstable vital signs, intra-abdominal perforation, absence of contraindication for surgery, severe emphysema, an underlying disease in site of perforation such as a malignancy, occlusion and stenosis in site of perforation and leakage of stuffs into the mediastinum (extravasations contrast to nearby cavities) surgery is necessary. The purpose for presenting this disease was expressing our experience in diagnosis and treatment of a patient with subacute signs and symptoms. This patient at the time of entering the emergency unit had evidences of them in ultrasound imaging but didn't match the patient's symptoms. Furthermore, CXR indicated a right-sided pleural effusion, which in this disease effusion is typically seen in left side and right-sided effusion is rare. It can be said for explanation of patient's symptoms that dry coughs originated by irritation of pleura following esophageal perforation, but there were no leakage and involvement of mediastinum and apparent symptom of the disease, and in first grades of the disease, radiological findings or pleural effusion was because of activation of pleural sympathetic system, not mediastinal pleura involvement. But after a few days, fever, tachycardia, leukocytosis and reduction in lung sounds in right side became more apparent and because the massive pleural effusion, chest tube was put for the patient and existence of food particles indicated the possibility of this disease, and he has been under sutaining treatment because of his age and the time gap more than 24h until the confirmed diagnosis.

Conclusion

In this patient and by reviewing the previous papers it is obvious that he has different and atypical demonstration [19] and the diagnosis of this disease should be reserved in every patient with a toxic appearance who refers with a sudden chest pain and a history of vomiting and an abnormal CXR.

References

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