Descending necrotizing mediastinitis mimicking acute ST segment elevation myocardial infarction

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Descending necrotizing mediastinitis (DNM) is a rare but lethal disease. Despite prompt use of broad-spectrum antibiotics and proper surgical drainage, the mortality is still high. High vigilance for disease presentation is helpful to early diagnosis. A 59-year-old female with a history of hypertension, diabetes and dyslipidemia came to our emergency room. Her initial presentation of chest pain, elevated troponin-I level and ST segment elevation in electrocardiogram (ECG) mimicked acute ST segment elevation myocardial infarction. However, DNM was suspected by her atypical clinical presentation and imaging studies. The ST segment elevation in ECG resolved immediately after pigtail catheter drainage. Finally, DNM was proved by right thoracotomy with mediastinal exploration. Klebsiella pneumoniae was isolated from pus and necrotic tissue.

Key Words: Coronary artery disease • Descending necrotizing mediastinitis • Myocardial infarction

INTRODUCTION

Descending necrotizing mediastinitis (DNM) is a rare but lethal disease. This disease results from odontogenic, pharyngeal, or cervical infection extending downward into the mediastinum via pretracheal, parapharyngeal and retrovisceral spaces of the neck. The infection is often polymicrobial in etiology, with gas-producing organisms. Despite prompt use of broad-spectrum antibiotics and proper surgical drainage, the mortality is still high. We report a case of descending necrotizing mediastinitis with initial presentation of chest pain, elevated troponin-I level and ST segment elevation in electrocardiogram (ECG), mimicking acute ST segment elevation myocardial infarction, so we couldn’t recognize it at the first time.

CASE REPORT

A 59-year-old woman presented at the emergency room of our institution with progressive, substernal dull chest pain for three days and mild drowsiness for one day. Within the previous two weeks, she had experienced non-productive cough and snuffle. She had history of hypertension, type 2 diabetes and dyslipidemia, but she denied smoking. On physical examination, she was afebrile. Her heart rate was 108 beats per minute, blood pressure was 100/64 mmHg and respiratory rate was 24 breaths per minute. She had regular and rapid heart rhythm, S4 gallop without audible murmur or pericardial friction rub. She had clear breathing sounds, no jugular vein distention or peripheral edema. Chest radiography demonstrated cardiomegaly, slight widening of the superior mediastinum and clear lungs field. The ECG showed sinus rhythm with complete right bundle branch block and 1- to 2-mm ST segment elevation in leads III, aVF, and V3-V6 (Figure 1A). The results of laboratory studies were as follows: troponin-I 2.5 ng/dL, creatine kinase (CK) 133 U/L, CK-MB isoenzyme 12 U/L, glucose 569 mg/dL, blood urea nitrogen 34 mg/dL, creatinine 1.3 mg/dL, sodium 126 mmol/L, potassium 123
4.4 mmol/L, white blood count 12,900/uL with 88% segmented neutrophils, hemoglobin 15.4 g/dL and platelets 207,000/uL. Arterial blood gas sampling showed metabolic acidosis (pH 7.34, HCO₃ 14 mmol/L, PaCO₂ 27 mmHg, PaO₂ 110mmHg), and ketone body test was strongly positive. Bedside echocardiography performed in the emergency department revealed mildly generalized hypokinesis of the left ventricle, with an ejection fraction of 45%. There was no evidence of aortic dissection intima flap, pericardial thickening or effusion. Under the impression of acute myocardial infarction and type 2 diabetes with impending ketoacidosis, the patient was treated as acute coronary syndrome with intravenous nitroglycerin, subcutaneous low-molecular heparin, oral aspirin, clopidogrel, carvedilol and intravenous insulin pump, then transferred to the intensive care unit. By the second day of hospitalization, blood glucose level was under control. The CK and CK-MB levels remained within normal limits. However, the patient’s consciousness remained drowsy and her body temperature elevated over 38 °C, producing suspicion of uncertain source of infection. On the other hand, she also experienced intractable cough. Hoarseness and dysphonia had been noted for days, which led us to perform a lateral view chest X-ray, showing subtle air bubbles over the anterior superior mediastinum (Figure 2A). Contrast-enhanced cervicothoracic computed tomography revealed diffuse inflammation with air intermixed tissue in para-tracheal, pre-cardiac, and bilateral para-vertebral region, indicating descending necrotizing mediastinitis (Figures 2B-D). Tracing back for the possible cause of this disease, the patient stated that she had undergone dental procedures many times in the recent six months for full-mouth dental bridges. Coronary angiography was per-

**Figure 1.** (A) ECG done in emergency room shows sinus rhythm with complete right bundle branch block and 1- to 2-mm ST segment elevation in leads III, aVF, and V₇-V₉ (V₈ and V₉ leads were reversed incautiously). (B) ECG done immediately after pigtail catheter drainage on the seventh day of hospitalization shows resolution of ST segment elevation.
formed on the third day of hospitalization. Although triple-vessel disease was seen, no culprit lesion could support the diagnosis of acute myocardial infarction. Empirical antibiotic therapy of piperacillin, tazobactam and levofloxacin were started. Two pigtail catheters were inserted into the mediastinal space and yellowish pus material was drained. Although we initially thought about acute myocardial infarction, ECG did not disclose evolutional change. After pigtail catheter drainage, ECG showed resolution of ST segment elevation immediately (Figure 1B), which suggested pericardial involvement of DNM. After one week of antibiotic treatment and pigtail catheter drainage, she received right thoracotomy with mediastinal exploration and drainage.

*Klebsiella pneumoniae* was isolated from pus and necrotic tissue. The patient’s post-operative course was uneventful.

**DISCUSSION**

Acute mediastinitis is an uncommon disease. The majority of mediastinitis results from esophageal perforation or infection following median sternotomy. Rarely, odontogenic, pharyngeal, or cervical infection may extend downward into the mediastinum via pretracheal, parapharyngeal and retrovisceral spaces of the neck, described as DNM, the most lethal form of mediastinitis. In 1938, Pearse reported the first series of DNM; overall mortality rate was 55% (86% in patients treated medically and 35% in patients treated surgically). Criteria for the diagnosis of DNM were established in 1983 by Estrera. They include (1) clinical manifestation of sepsis, (2) characteristic roentgenographic features, (3) documentation of the necrotizing mediastinal infection at operation or at postmortem examination, and (4) establishment of the relationship of oropharyngeal infection and the mediastinal process. Endo classified DNM into three groups. Type 1 DNM is localized to the upper mediastinal space above the carina. Type 2A DNM extends into the lower anterior mediastinum and type 2B DNM extends into both the anterior and posterior lower mediastinum. Bacteriologically, DNM is most frequently a polymicrobial process, with anaerobes playing a major role. Because of its rapid development and severe complication such as purulent pericarditis, abscess or thoracic empyema leading to multiple organ dysfunction, the mortality is reported to be 40-50%, even with the proper use of computed tomography scanning, aggressive drainage, and antibiotic treatment. Delay of diagnosis and delayed or inappropriate drainage of the mediastinum are the main causes for the high mortality in this life-threatening condition.

To date, DNM mimicking acute ST segment elevation myocardial infarction has rarely been reported. With our patient’s presentation of dull chest pain, elevated troponin-I level and ECG change, acute myocardial infarction was initially impressed, but clinical presentation and persistent ST-T elevation made this diagnosis doubtful. These findings could be explained by pericardial involvement of DNM. Hoarseness and dysphonia indicated paratracheal region or recurrent laryngeal nerve involvement. As demonstrated by this DNM mimicking acute myocardial infarction case, it is very important to organize clinical data explaining any other possibilities of myocardial infarction, and to not consider only ECG or serum troponin-I in diagnosis.
REFERENCES