[CASE REPORT]

We Should Pay Attention to “Referred Pain” - A Case of Acute Myocardial Infarction that Masked and Delayed the Diagnosis of Esophageal Perforation

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Abstract:

We herein report a 93-year-old woman diagnosed with acute myocardial infarction (AMI) based on typical laboratory findings of severe chest pain accompanied by throat pain. This condition was initially interpreted as referred pain of cardiac origin. However, the patient had persistent throat pain after successful percutaneous coronary intervention. Upper esophageal perforation with life-threatening acute mediastinitis was unexpectedly identified by a further examination. Clinicians should have a high index of suspicion in cases with persistent symptoms thought to be referred pain among AMI patients, as these symptoms may not be of cardiac origin but rather a sign of another concomitant critical disease.

Key words: diagnosis, esophageal perforation, myocardial infarction, referred pain, throat pain

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Introduction

Acute myocardial infarction (AMI) is a common but fatal disease that should not be overlooked in our daily clinical care. Pain in the neck and head areas observed with relatively high frequency in patients with AMI is known as “referred pain” of cardiac origin (1-3). Indeed, chest pain in addition to pain radiating to these areas increases the likelihood of AMI (4, 5). The mechanism underlying referred craniofacial pain have been described in multiple clinical and basic studies, which revealed the key involvement of the vagal nerve (2, 6). Due to these backgrounds, clinicians may be prompted to misinterpret craniofacial pain in AMI exclusively as referred pain of cardiac origin.

We herein report a case of AMI in which throat pain initially considered to be referred pain delayed the diagnosis of esophageal perforation.

Case Presentation

A 93-year-old woman with a medical history of hypertension, diabetes mellitus and breast cancer presented to our emergency department with severe chest pain and throat pain.

On an examination, she was alert and afebrile (35.8 °C). Her blood pressure was 80/46 mmHg, heart rate 84 beats per minute, and oxygen saturation 98% with ambient air. An electrocardiogram (ECG) showed significant ST-segment elevations in leads II, III, and aVf (Fig. 1). Laboratory data on admission showed a white cell count of 17,700/μL, troponin-I level of 212.9 pg/mL, CK level of 930 U/L, CK-MB level of 100.6 U/L and C-reactive protein (CRP) level of 21.27 mg/L. Her history and these data indicated acute inferior myocardial injury with referred craniofacial pain of cardiac origin. Emergent coronary angiography showed a completely occluded right coronary artery (RCA, #1). Subsequently, percutaneous coronary intervention (PCI) was

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Figure 1. An electrocardiogram obtained at the emergency department. It showed significant ST-segment elevations in leads II, III and aVf.

Figure 2. (a) The enhanced CT image. The red asterisk indicates an abscess in the posterior mediastinum adjacent to the esophagus (yellow arrow). (b) The thoracoscopy image. The black star indicates an abscess with purulent exudate in the upper posterior mediastinum between the trachea and esophagus.

performed. The ECG findings promptly normalized after successful revascularization.

However, her throat pain did not subside despite improve-

ment of other clinical indices, including the amelioration of her chest pain, and the continued improvement of laboratory results, including white blood cell counts, which peaked at day 2, as well as the improvement of her ventricular function on an echocardiogram obtained immediately after PCI. Her throat pain and odynophagia worsened without a fever, pharyngitis or laboratory findings suggestive of infectious etiology. A careful history taking revealed that she had first noted throat pain while eating fish a few days prior to her admission. Accordingly, we suspected penetration and trauma to the oropharyngeal mucosa by fishbones.

Subsequent enhanced computed tomography of the neck and chest revealed an abscess in the posterior mediastinum adjacent to the esophagus and mediastinal soft-tissue infiltration as well as stranding of the paraesophageal posterior mediastinal fat (Fig. 2a). Accordingly, she was diagnosed with esophageal abscess and acute mediastinitis due to esophageal perforation secondary to direct trauma from an ingested fishbone.

Conservative treatment over aggressive surgical management was chosen due to concerns made following a preoperative surgical risk assessment, including based on the patient's age and cardiac risk due to her recent AMI diagnosis, hemodynamic stability and the risk of further esophageal perforation. Empiric antimicrobial therapy with ampicillin-sulbactam (9 g/day) was initiated, but no improvement in her symptoms was noted.

After careful consideration of her unresponsiveness to conservative treatment, we performed surgical thoracoscopic abscess drainage on day 6. Thoracoscopy showed an abscess with purulent exudate in the upper posterior mediastinum

between the trachea and esophagus (Fig. 2b). Cervical exploration for an abscess behind the trachea and esophagus did not identify any obvious esophageal laceration or fishbones. The microbiologic culture of the exudate revealed *Streptococcus anginosus* that was sensitive to ampicillin-sulbactam. Although she required additional thoracoscopic intervention for complete drainage of the abscess on day 15, her symptoms gradually resolved, and she was eventually transferred to a rehabilitation facility.

Discussion

Referred pain in the craniofacial area is a common presentation of acute myocarditis, although awareness of craniofacial pain as a symptom of cardiac ischemia is low (1). Multiple studies have shown that craniofacial pain is experienced in up to 40% of ischemic heart disease cases (1-3). In addition, diabetes is reported to increase the incidence rate of craniofacial pain during myocardial ischemia (2). Women tend to experience craniofacial pain more frequently than men (2, 3). Furthermore, the involvement of the RCA in AMI may play an important role in the development of referred pharyngeal pain (2, 6). These data in addition to the patient's physical and laboratory findings strongly supported AMI as the origin of the throat pain in this patient. However, we were bewildered by the persistent throat pain that did not abate despite improvements in other clinical signs and symptoms and nearly overlooked a critical disease.

Another factor associated with the diagnostic dilemma in the present case may have been how uncommon coexisting posterior mediastinum lesions are in determining the origin of throat pain. Esophageal perforation is a rare but fatal injury found in 3.1 per 1 million per year, and is frequently misdiagnosed when it is unrelated to cardiac or thoracic surgery (7-10). Persistent throat pain after PCI and probable fishbone indigestion determined by a thorough medical interview provided clues resulting in the diagnosis of this rare condition.

The diagnosis of esophageal perforation and subsequent mediastinitis is generally difficult due to the nonspecific symptoms (10). The most typical symptom, chest pain, is reported in 85% of cases (11). In our case, the expected resolution of chest pain after PCI allowed us to conclude that the pain was of cardiac origin, so we did not suspect any other etiology. The spread of a contagion to the mediastinum through the retropharyngeal space is generally moderate after cervical perforation of the esophagus (8), which might have resulted in the limited presentation of our patient.

However, it has been reported that esophageal perforation can cause ST-elevation on an ECG and mimic AMI (12). Thus, esophageal perforation can be considered as a differential diagnosis when ST-elevation is observed on an ECG. This highlights the significance of taking a thorough history and performing a comprehensive physical assessment to make a precise diagnosis.

Conclusion

In summary, we encountered a rare case in which probable life-threatening esophagus perforation compromised with abscess formation and mediastinitis hid behind the typical presentation of AMI. The clinical pearl in this case is that "referred pain" in AMI patients may not be of cardiac origin but rather a sign of another concomitant critical disease. An atypical presentation of AMI after PCI should not be underestimated and receive a thorough exploration of other potential etiologies. Clinicians should have a high index of suspicion and take a comprehensive history with a thorough physical examination to ultimately obtain a correct diagnosis.

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