Perplexing Epigastric Pain-Coincident Myocardial Infarction and Acute Pancreatitis

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Abstract

Acute myocardial infarction (MI) complicated with acute pancreatitis has been rarely reported. A 68-year-old man presented to our department 15 hours after development of epigastric pain. In addition to his symptoms, the elevated serum pancreatic enzymes and the image study on abdominal computerized tomography all led to the diagnosis of acute pancreatitis. Elevated cardiac biomarkers and a standard 12-lead electrocardiogram (ECG) demonstrating ST-segment elevation in 5 of the 6 precordial leads suggested an attack of MI. Oral intake was resumed after medical management for his acute pancreatitis and acute MI. Coronary angiogram on day 11 revealed total occlusion of the middle segment of the left anterior-descending coronary artery. Subsequently, angioplasty with stenting was done. The patient was discharged without significant complications. It is critical to make a rapid but detailed differential diagnosis of abdominal pain. Even though acute pancreatitis-associated ECG abnormalities have been reported previously, any ECG abnormalities in a patient presenting abdominal pain should be evaluated and treated cautiously. Thorough clinical evidence, including history, physical findings, ECG, image studies and serum biomarkers, are informative in seeking and analyzing possible etiologies.

Key words: acute myocardial infarction, acute pancreatitis, electrocardiography

Introduction

Epigastric pain is common but varies greatly in severity. Acute and severe pain is almost always a symptom of intra-abdominal disease. However, neighboring pathology such as intra-thoracic lesions can also be the potential causes.

Acute pancreatitis typically presents with severe and constant upper abdominal pain, which are often associated with nausea and vomiting. Confirmatory tests are required in ascertainment. Several scoring systems have been developed for the prediction of the severity of acute pancreatitis. In 70% to 80% of patients with acute pancreatitis, the disease is managed by supportive measures (1). Meanwhile, the diagnosis of acute myocardial infarction (MI) depends primarily on symptomatology, elevation of cardiac enzymes and electrocardiographic changes. Electrocardiography (ECG) plays a pivotal role in differentiating the ST-segment elevation and non-ST-segment elevation groups, which are managed differently in prompt coronary angiography. In addition to chest pain, acute MI presents pain in the neighboring areas such as epigastrium even with non-pain syndromes. Around 50% victims of pancreatitis present ECG abnormalities due to electrolytes (2, 3). However, simulated acute myocardial infarction in acute pancreatitis has been described in the literature (4-6). In the literature, acute MI combined with acute pancreatitis has been only documented in one case, which had angiographic evidence of high-grade coronary lesions (7). We report a patient with acute MI combined with acute pancreatitis with the initial presentation of epigastric pain.
A 68-year-old man was transferred to our service 3 hours after being diagnosed with acute MI and acute pancreatitis. He denied a habit of alcohol drinking and had no significant risk factors of coronary artery disease except for impaired fasting glucose, which was noted for years. He went to the emergent service because of severe epigastric pain lasting for 12 hours. He denied an attack of chest pain or shortness of breath. On admission to our service, his vital signs showed blood pressure of 106/72 mmHg with a pulse rate of 110 beats/min, a respiratory rate of 20 breaths/min and a temperature of 36.4°C. Initial physical examination revealed no respiratory wheezes or crackles. Cardiac auscultation demonstrated no significant murmur or gallop. Only localized tenderness was developed over the upper abdomen without muscle guarding. Laboratory tests showed elevated leucocyte count (21,230 /per cubic millimeter), normal hematocrit (45%), impaired renal function as blood urea nitrogen/serum creatinine (28/1.8 mg/dL). Serum liver enzymes elevated [alanine aminotransferase (ALT): 717 U/L; aspartate aminotransferase (AST): 554 U/L, lactate dehydrogenase (LDH): 958 U/L] along with elevated amylase/lipase (2,810/9,660 U/L). Non-fasting blood sugar was 361 mg/dL and plasma low-density/high-density lipoprotein and triglyceride were 96/23 and 66 mg/dL. C-reactive protein was 10.8 mg/dL at admission. Chest radiograph showed mild pulmonary edema on admission (Fig. 1). ECG demonstrated atrial fibrillation, ST-segment elevation and Q wave in precordial leads as well as lead I and aVL with reciprocal ST-segment depression over lead III/aVF (Fig. 2A). Elevated cardiac biomarkers, including creatine kinase (CK) 1,167 U/L, CK-MB 49 U/L, troponin I 59 ng/mL, were noted. An echocardiography revealed akinesia and middle- to apical ventricular septum and anterior wall of left ventricle with preserved systolic function with estimated ejection fraction of 47%. Abdominal sonography was performed at the same time, which showed a swollen pancreas head without dilatation of the common bile duct. Ranson’s score was 5 at admission. Because of the high initial Ranson’s score, delayed transfer to our service (15 hours after presentation of first symptom) and stable hemodynamics, emergent coronary catheterization was not arranged.

Initially, only low-molecular weight heparin and parenteral nutrition with daily fluid 2,200 mL and sodium load 112 mEq were given. Epigastric pain gradually subsided. Forty-eight hours after admission, hematocrit was 42%; blood urea nitrogen was 31 mg/dL; serum free calcium was 0.91 mmol/L adding another point to Ranson’s score. Dual antiplatelets along with beta-blocker and angiotensin-converting enzyme inhibitor were given. Table 1 shows the timeline of cardiac biomarkers and pancreatic enzyme variations. Abdominal computerized tomography (CT) 6 days after admission demonstrated mild peri-pancreatic fat stranding without abscess or phlegmon formation in the pancreas with a CT severity index of 4 (Fig. 3). Oral intake was started 8 days later. Coronary angiography ten days later revealed total occlusion of the middle left anterior-descending coronary artery with organized thrombi. A paclitaxel-eluting stent was placed successfully with good distal coronary flow (Fig. 4). The patient was discharged uneventfully on day 14.
Abdominal pain has always been a diagnostic dilemma. Lesions of neighboring or distant visceral organs are equally possible etiologies. Localization of pain and detailed history description are always helpful. Signs and accompanying symptoms and the absence or presence of physical findings facilitate the differential of possible causes. Rapid identification of the etiologies of abdomen pain enables earlier implementation of specific therapies.

**Diagnosis and management of acute pancreatitis**

Acute pancreatitis is a clinical diagnosis which is made by clinical available history, including physical findings, serum levels of amylase and lipase and images of typical pancreas swelling or necrosis (8). The disease is generally mild and promptly subsides after supportive management. It is noteworthy that in 20% of patients severe pancreatitis is accompanied by complications as infection, organ failure and even profound shock (9). Alcoholism and gallstone account for major etiologies. However, idiopathic pancreatitis has been reported in 20 to 40% (10).

The principle of the initial treatment of acute pancreatitis are monitoring vital signs, adequate fluid and electrolyte replacement and nutritional support. Abdominal CT demonstrates a complete view of the pancreas and peripancreatic tissues. With contrast medium enhancement, the possible necrosis of pancreas can be carefully evaluated (11). Further, the recently developed CT severity index and scoring systems using clinical biochemical criteria have been adapted for the prediction of severity. These include the 11 criteria described by Ranson, the Glasgow score and the acute physiology and chronic health evaluation (APACHE II) score (12). However, some biochemical parameters, such as LDH and AST, are confounded by coexisting medical conditions. In the present case, acute necrosis of myocardium results in abundant release of intracellular enzyme. Ranson’s and Glasgow scores overestimate the severity of acute pancreatitis. The CT severity index, as in the present case, provides a better prognostic prediction.

**Differentiation of acute MI**

For diagnosing acute MI, symptomatology of angina or angina equivalents, elevation of cardiac biomarkers and ECG changes are fundamental. However, chest pain does not necessarily present in every patient suffering from acute MI. In the report of Culic et al, 16.1% of men and 13.3% of women with acute MI had epigastric pain; 71.2% of men and 84.2% of women presented with non-pain symptoms such as nausea, vomiting or sweating (13). Those symptoms are indiscernible to acute abdomen emergencies such as acute pancreatitis. Electrocardiographic abnormalities in acute pancreatitis have been described as ST-segment devia-
Figure 4. Pre-angioplasty coronary angiography showed total occlusion over the middle segment of the left anterior descending coronary artery with thrombi (left); post-angioplasty and stenting coronary angiography showed TIMI 3 flow after paclitaxel-eluting stent deployment (right).

tions and T-wave inversions, which mimic acute MI (14). Several theories, such as cardiobiliary reflex related to the vagal nervous system and toxic effects of pancreatic enzymes on the myocardium, have been proposed to elucidate the mechanisms of electrocardiographic abnormalities. The administration of thrombolytic agents in such occasions might lead to catastrophic consequences. In such conditions, it is a diagnostic and therapeutic challenge for the clinicians. Cardiac-specific biomarkers and an ECG facilitate the diagnosis in specific circumstances (15). Electrocardiographic changes resembling acute MI are a rare phenomenon in acute pancreatitis. Several mechanisms have been presented as a possible explanation. Only two cases in the literature have been documented as coexistent acute MI and acute pancreatitis (7). In the present case, the index symptom developed 15 hours prior to arrival to our department. Cardiac biomarkers activities showed CK-MB and troponin I peaking at roughly the 25th hour. In general, cardiac biomarkers peak within one day at MI (16). Emotion or physical stress-related increased catecholamine may predispose ST-segment elevation acute coronary syndrome and Takotsubo cardiomyophay (17). Also, evidence suggests that heavy exercise and emotional stress precipitates MI (18). Therefore, coincident MI may be related to the high level of catecholamine released soon after abdominal pain. In the report of Qazi et al a 52-year-old diabetic patient with hypertriglyceridermia presented mid-abdominal pain initially then the subsequent ECG revealed ST-segment elevation at inferior and RV4 leads. The patient had CT-confirmed pancreatitis and was treated with immediate thrombolytic therapy then a subsequent coronary angiography revealed 90% stenosis of proximal right coronary artery.

Acute MI combined with acute pancreatitis is a very rare condition. Symptomatology and electrocardiographic changes may not sufficiently differentiate the two. A perfunctory decision of management might lead to detrimental results. Cardiac-specific biomarkers and image studies facilitate not only the diagnosis but also the possible prognosis. An overestimated disease severity due to complex clinical conditions leads to the delay of necessary therapy.

References


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