Challenges in Clinical Electrocardiography

An Uncommon Cause of Acute Chest Pain

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Case Presentation

A patient in their 50s presented to the emergency department with 20 minutes of substernal chest pain radiating to the left shoulder and back, which was accompanied by shortness of breath and sweating. The patient's medical history was notable for poorly controlled hypertension and heavy smoking. On admission, the patient's blood pressure was 185/104 mm Hg in the right upper limb and 113/98 mm Hg in the left upper limb, their heart rate was 58 beats per minute, and their oxygen saturation level was 98% on room air. A faint early diastolic murmur was found in the aortic area, and the lungs were clear. Subsequent investigation revealed a D-dimer level of 3.914 µgm/L (normal value,0-0.3 µg/mL; to convert to nmol/L, multiply by 5.476), and a troponin I level within normal range. An electrocardiogram (ECG) obtained on admission is shown in the **Figure**.

Questions: What is the most likely cause of the patient's ECG changes? What would you do next?

Interpretation

The ECG on presentation (Figure) showed a sinus rhythm at a rate of 58 beats per minute, with ST-segment elevation (STE) in leads I, aVL, and V_1 through V_4, as well as ST-segment depression in leads II, III, and aVF. These ECG changes are consistent with the diagnostic criteria of the unprotected left main artery (ULM) occlusion, namely the left main disease without patent bypass graft to the left anterior descending or circumflex coronary artery.

Clinical Course

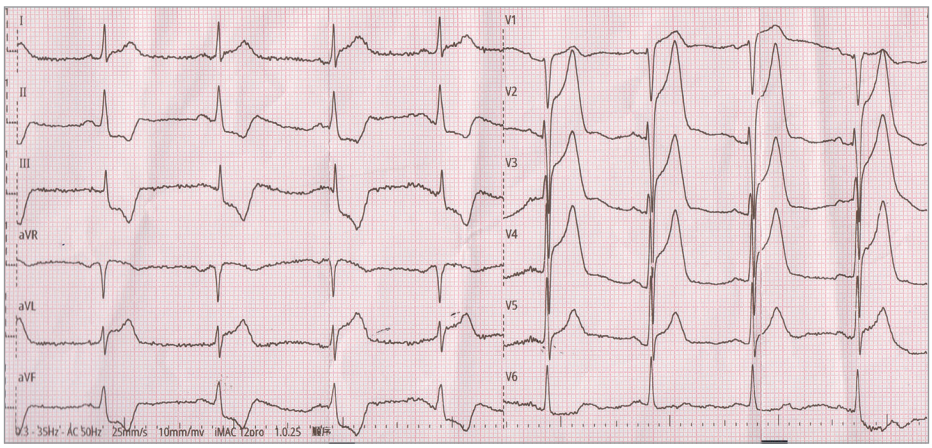
According to the patient's symptoms and the ECG findings, the initial diagnosis was ST-segment elevation myocardial infarction (STEMI). However, considering that this patient presented with substantial bilateral blood pressure asymmetry, severe back pain, a normal troponin I level, and a remarkably elevated D-dimer level, diagnosis of aortic dissection was strongly suspected. Therefore, the patient was quickly transferred for a contrast-enhanced aortic computed tomographic angiography examination. The computed tomography angiogram results showed a Stanford type A acute aortic dissection (AAD) arising from aortic sinus to the right common iliac artery and involving the ostia of the left and right coronary arteries. The patient was transferred to the cardiovascular surgery department and underwent aortic arch replacement concomitant with aortic valve replacement. Unfortunately, the patient died 2 days postprocedure.

Discussion

Differential diagnoses for acute chest pain mainly include pulmonary embolism (PE), acute coronary syndrome, and acute aortic syndrome (AAS). Acute aortic syndrome consists of acute aortic dissection (AAD), intramural hematoma, and penetrating atherosclerotic ulcer. Aortic dissection is defined as the disruption of the medial layer provoked by intramural bleeding, in which is followed by separation of the aortic wall layers and subsequent formation of a true lumen and false lumen. A case of AAD or intramural hematoma involving the ascending aorta and aortic arch is defined as type A aortic syndrome; if only the descending aorta is involved, it is type B aortic syndrome.[1] Typical chest pain of AAD usually presents with tearing chest pain that radiates to the back, and physicians should be aware of possibility of AAD in such cases.

Acute myocardial infarction (AMI) is a common cause of STE. However, AMI is not the only cause of STE. The differential diagnosis for STE includes conditions such as AMI, early repolarization, left ventricular hypertrophy, left bundle-branch block, acute pericarditis, hyperkalemia, Brugada syndrome, and PE.[2] Electrocardiogram changes for PE include sinus tachycardia, rightward shift in the QRS axis, complete or incomplete right bundle-branch block, precordial T-wave inversion, an S1Q3T3 pattern, and STE; STE of PE occurs predominantly in the anterior-septal and inferior distribution.[3] However, in this patient, the STE presented in leads I, aVL, and V_1 through V_4. In addition, the patient also had sinus bradycardia. These find-

Figure. Electrocardiogram (ECG) on Admission



The admission ECG showed a sinus rhythm at a rate of 58 beats per minute, with ST segment elevation in leads I, aVL, and V_1 through V_4, as well as ST segment depression in leads II, III, and aVF.

ings differ from the typical ECG changes observed in PE. In addition, AAS should be considered to be the cause of STE.

Type A AAD often shows ST-T abnormalities at presentation. Kosuge et al[4] reported that the prevalence of ECG patterns in type A acute aortic dissection cases was 51% for ST-T abnormalities (4% for STE and 47% for ST-segment depression and/or negative T waves), 30% for normal ECG findings or no significant ST-T changes, and 19% for ECG confounders, such as bundle branch block or left ventricular hypertrophy, whereas the ST-T changes in type A AAD are fairly nonspecific. It has been reported that when involving the ascending aorta, approximately 2.5% of patients with type A AAD may progress to STEMI.[1,5] In type A AAD, the dissected membrane occasionally extends to a coronary ostium, causing AMI associated with acute ischemic ST-T changes on ECG. Among ST-T abnormalities, STE was rare but was strongly associated with coronary ostial involvement, as well as the highest rate of in-hospital mortality (30%).

Acute total occlusion of the ULM is an uncommon but clinically catastrophic event. The ECG findings of the ULM in the setting of AMI are variable. Several ECG patterns have been reported to be associated with ULM occlusion[6]: ST-segment elevation starting in precordial leads V_2 to V_4 and continuing through lead V_6 and in the lateral extremity leads I and aVL and STE in lead aVR or aVR and aVL with widespread ST-segment depressions. However, some patients with acute ULM occlusion may present with STE in leads aVR or aVR and aVL. Liu et al[7] reported that these different ECG patterns were associated with the different collateral filling territories in ULM occlusion: (1) STE in the anterior leads indicated the absence of collateral flow; (2) the collateral flow from the right coronary artery into the left anterior descending artery territory attenuated ST elevation in the anterior region, causing patients to present with STE in leads aVR and aVL in this setting; (3) the collateral flow from the right coronary artery into the left anterior descending artery and left circumflex coronary artery territory attenuated STE in the anterior and lateral extremity leads, which was followed by STE in lead aVR only.

Symptoms of type A AAD and STEMI are similar. And the treatment of the 2 diseases is fundamentally different. Therefore distinguishing AAS from acute coronary syndrome is extremely important. When a patient with chest pain shows an ECG of STE, physicians should be aware of the possible of type A AAD.

Take-home Points

- Acute chest pain mainly includes pulmonary embolism, acute coronary syndrome, and acute aortic syndrome.
- The differential diagnoses of STE includes AMI, type A AAD, early repolarization, left ventricular hypertrophy, left bundle branch block, acute pericarditis, hyperkalemia, Brugada syndrome, and PE.
- Patients with classic tearing chest pain radiating to the back, an STE ECG pattern, and a normal troponin level should be highly suspected for type A AAD.

ARTICLE INFORMATION

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