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Acute Myocardial Infarction Secondary to Aortic Dissection

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Abstract

A 50-year-old man with a history of hypertension awakened with acute retrosternal chest pain accompanied by dim consciousness. He was transferred to cardiac catheterization laboratory for emergent percutaneous coronary intervention (PCI) but diagnostic coronary angiography was difficult and unsuccessful. Therefore, he was immediately conducted the thoracoabdominal computed tomography angiography (CTA). The CTA showed aortic dissection (Stanford type A), involving the left main coronary artery (LMCA) and left anterior descending (LAD). Emergency surgery was executed. Unfortunately, this patient eventually died of multiple organ dysfunction syndromes.

Keywords: Aortic dissection; Acute myocardial infarction; Misdiagnosis

Introduction

Aortic dissection is known as a breach in the intima responsible for the creation of a neo-channel, which splits media between its third means and its third extern with the spontaneous mortality at 48 hours of 50% [1]. The typical patient presents with abrupt onset of chest pain, which is sharp (more often than constrictive), and moves toward the back if there is distal extension. The rare clinical features can be presented as syncope with or without chest pain, renal failure, or as in this case, an acute myocardial infarction related to occlusion of the left main artery etc. The diagnosis of an acute Stanford type A aortic dissection remains a challenge for the emergency physician.

Case Report

A 50-year-old man with a history of hypertension (non-therapeutic) awakened with acute chest pain accompanied by dim consciousness. On the electrocardiogram (ECG) at emergency room, signs of acute myocardial infarction in the anterior territory were presented with ST elevation in leads V1 to V6 and ST depression in leads II, III and aVF. Q-waves were noted from leads V1 to V6, I and avL (Figure 1). Troponin I concentration was 32.28 ng per milliliter (reference range, 0 to 0.04 ng per milliliter). The transthoracic echocardiogram demonstrated the anterior wall and right ventricular apical wall motion abnormality, aortic valve mild regurgitation, no signs of aortic dissection. Emergency personnel considered the diagnosis of acute myocardial infarction with routine protocol for Acute ST-levation myocardial infarction (STEMI) including loading doses of aspirin (300 mg), ticagrelor (180 mg) and rosuvastatin (20 mg) followed by intravenous injection of tirofiban hydrochloride (Initial 30 minutes drip rate: 0.4 μg/kg/min) and unfractionated heparin (5000 U). Then he was transferred to the coronary care unit. Following a review by the cardiology team, 5-F multipurpose catheter was repeatedly tried into the left and right coronary sinus unsuccessful, non-selected angiography showed the aortic root flow divided into double cavity (close to the right side of tube cavity contrast agents filling well, the left side of tube cavity contrast agents slightly enhanced), left and right coronary artery no imaging (Figure 2). Therefore we immediately stopped the operation and treatment of anticoagulation antiplatelet drugs.

The blood pressure and heart rate were within control by intravenous injection labetalol and nicardipine. Laboratory examination showed D - dimer 13.509 ug/mL; Troponin I 272.308 ng/mL; the N-terminal pro-B-type natriuretic peptide 2590 ng/L; Routine blood: WBC count 15.47 × 10^9/L, neutrophil 0.88, hemoglobin 90.5 g/L, platelet count 57.2 × 109/L; Liver function: alanine aminotransferase (ALT) 73.1 U/L, aspartate aminotransferase (AST) 430 U/L; Renal function: serum creatinine 226 μmol/L, serum cystatin C 1.18 mg/L; Inflammatory factor: tumor necrosis factor alpha (TNF alpha) 98.20 ng/L, interleukin 6 (IL - 6) 52.00 ng/L, high-sensitivity c-reactive protein (hsCRP) 21.84 mg/L; Urine red blood cell count to 2152.9/μL. Thoracoabdominal CTA was performed because of an aortic dissection suspicion. The CTA showed aortic dissection (Stanford type A), involving LMCA and LAD and Right innominate artery, bilateral common carotid artery, bilateral subclavian artery, coeliac trunk artery, splenic artery, right renal artery, superior mesenteric artery (Figure 3). After obtained consent of the family and related medical records signature, he was underwent emergency surgery. Pathologic biopsy showed aortic

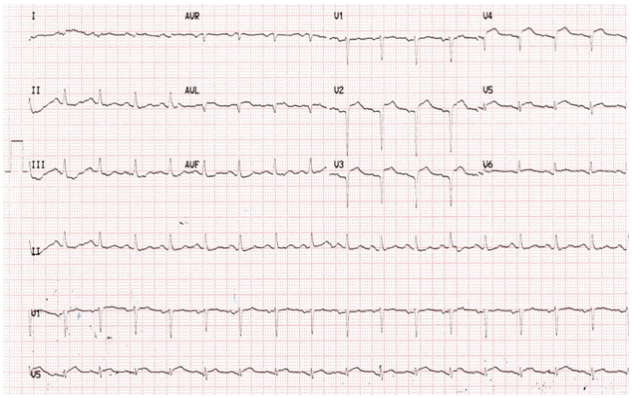

Figure 1: On the electrocardiogram, signs of acute myocardial infarction in the anterior territory were present, with ST elevation in leads V1 to V6 and ST depression in leads II, III and aVF. Q-waves were noted from leads V1 to V6, I and avL.

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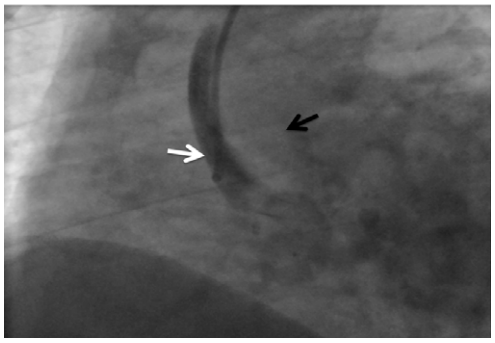

Figure 2: With a 5-F multipurpose catheter was repeatedly tried into the left and right coronary sinus unsuccessful, non-selected angiography showed the aortic root flow divided into double cavity (close to the right side of tube cavity contrast agents filling well (white arrow), the left side of tube cavity contrast agents slightly enhanced (Black arrow)), left and right coronary artery no imaging.

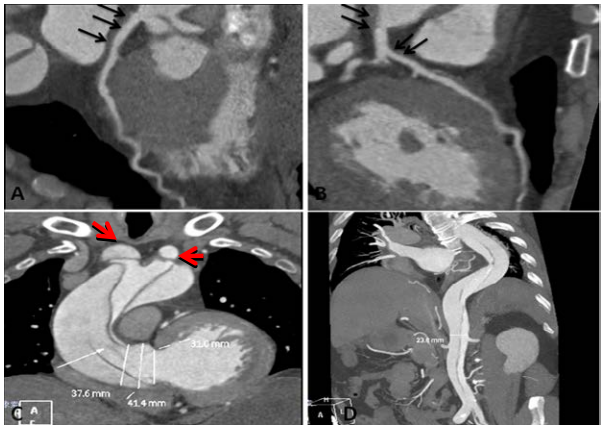

Figure 3: Aorta CTA: aortic dissection (Stanford type A), involving LMCA and LAD(Black arrows) and Right innominate artery(Red arrows), bilateral common carotid artery(Red arrows), bilateral subclavian artery(Red arrows), coeliac trunk artery, splenic artery, right renal artery, superior mesenteric artery.

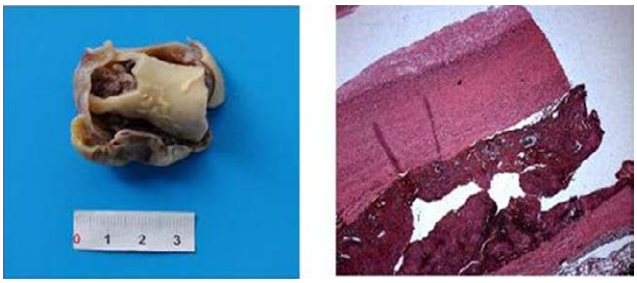

Figure 4: HE, EVG dyeing microscopically shows: fibrous thickening of the endothelium, atheromatous plaque formation, membrane in laminar necrosis, tearing the membrane, dissection, outer membrane interstitial edema.

dissecting aneurysm (layered media necrosis, atherosclerosis) (Figure 4). He was transferred to intensive care unit for further treatment, but eventually died of multiple organ dysfunction syndromes.

Discussion

Aortic dissection is known as a breach in the intima responsible for the creation of a neo-channel, which splits media between its third means and its third extern with the spontaneous mortality at 48 hours of 50% [1]. The typical patient presents with abrupt onset of chest pain, which is sharp (more often than constrictive) and moves toward the back if there is distal extension? The rare clinical features can be presented as syncope with or without chest pain, renal failure, or as in this case, an acute myocardial infarction related to occlusion of the left main artery etc. The diagnosis of an acute Stanford type A aortic dissection remains a challenge for the emergency physician. Abrupt-onset chest pain occurs in 80% of patients and is frequently sharp and severe in quality [2]. Although an ECG commonly shows nonspecific ST-segment or T-wave changes, signs of acute myocardial infarction occur in only 5% of patients [2]. When dissection is complicated by acute myocardial infarction, it might be extension of dissection into a coronary artery or coronary ostial occlusion from the dissection flap, often affecting the RCA [3]. The higher incidence of right coronary artery (RCA) involvement is attributed to dissection originating more commonly from the right anterior aspect of the ascending aorta above the right sinus of Valsalva.

This case illustrates the difficulty in the diagnosis of an aortic dissection, where the clinical symptoms resemble acute myocardial infarction accompanied by dim consciousness. Aortic root dissection involving the LM is a rare cause of ST-segment elevation myocardial infarction [4]. Myocardial infarction or ischemia complicating aortic dissection represents a critical situation with reported incidence of 3% and 5%, respectively [5]. The differential diagnosis of an acute aortic dissection should always be considered because more delay will result in a higher mortality rate. However, when confronted with a patient presenting with the suspicion of an occluded LMCA or proximal LAD based on the ECG, no time has to be wasted, and urgent transport with intent to primary PCI is crucial [6]. Eventually, emergency angiography identifies signs of an aortic dissection, and prevents further risks. Diagnosing acute Stanford type A aortic dissection with the uncommon involvement of the LMCA or proximal LAD is difficult because it can resemble acute myocardial infarction. Eventually, the dynamic pattern of signs of acute myocardial ischemia and unaltered control coronary angiography should immediately point to the probability of extrinsic coronary compression. Although emergency angiography identifies signs of an aortic dissection, parallel emergency vascular surgery, but eventually died of multiple organ dysfunction syndromes. In the current era of primary PCI with emphasis on door-to-balloon time, aortic dissection should be part of the differential diagnosis of STEMI. Difficulty with catheter engagement or significant aortic regurgitation or patient's dim consciousness should raise the suspicion for acute type A aortic dissection.

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