

C072

A rare cause of myocardial infarction

A 29-year-old female was admitted to emergency department for continuous chest pain of three hours at 13:27 pm on October 17th 2016. The patient felt sudden chest pain at 10:00 am when she was doing aerobic exercises, locating in the retrosternal, accompanying sweating, without cough, hemoptysis or fever. She was admitted to the nearest hospital at 10:30 am, initial blood test of myocardial necrosis was negative, ECG demonstrated that ST segment of lead AVR elevated 0.2 mV, ST segment of lead I, II, avL, V2-V9 depressed 0.1-0.6 mV. The patient was diagnosed of acute coronary syndrome, and treated with aspirin, clopidogrel, papaverinen and omeprazole. At 11:30 am the patient's symptoms were reduced by about 50%, the second ECG demonstrated that lead I, avL, V1 and V3 form Q wave, ST segment of lead V1 - V5 elevated 0.1-0.2 mV. The patient was subsequently transferred to our hospital.

The patient had experienced several syncope in the past 12 years. She was diagnosed of possible viral myocarditis in our hospital. She denied hypertension, diabetes, dyslipidemia, stroke, and didn't have habit of smoking, alcohol and illicit drugs. She was unmarried, not pregnant, with regular menstrual cycle, without siblings. She hadn't family history of cerebrovascular disease. The patient's temperature was 36.5 °C, pulse was 80 bpm, respiration rate 18 breaths per minute, blood pressure was 104/70 mmHg. The patient had normal consciousness, auscultation of lung was clear, heart rate was 80 bpm, the rhythm was regular, and there were no murmurs. Laboratory examination demonstrated that cardiac troponin I was 37.0 ng/ml (ULN< 0.020 ng/ml), myoglobin was 646 ng/ml (ULN< 46.4 ng/ml), CK - Mb was 467 ng/ml (ULN< 4.99 ng/ml), CK was 4800 u/L, LDH was 621 u/L, white blood cells was 19.39 x 10⁹ / L, neutrophils was 92.3%, hemoglobin was 125 g/L, platelet was 354 x 10⁹ / L. Bedside echocardiography demonstrated that left ventricular end-diastolic diameter was 42 mm, left ventricular ejection fraction was 66%, the heart structure and blood flow at resting state hadn't seen obvious abnormality.

The patient subsequently underwent emergency coronary angiography which demonstrated that the proximal left main was compressed by outside. IVUS examination revealed that there was no atherosclerosis plaque or stenosis in left main coronary artery, left anterior descending, left circumflex and right coronary artery.

Subsequent coronary CTA demonstrated that the left main origin anomalously from right coronary sinus at the root of ascending aortic, the proximal segment was in the aortic wall, and was severe stenosis.

Cardiac MRI demonstrated that there were acute, subacute, myocardial infarction, involving in the interventricular septum and left ventricular anterior wall and side wall to apex, basically focused in the endocardium.

The patient was treated with dual antiplatelet, statins, beta blockers and ACEIs, and hadn't chest discomfort any more. The patients underwent surgery of correction of anomalous original coronary artery on November 10th, 2016. The surgery confirmed that the left coronary artery originated from the right coronary sinus, the proximal segment was in the aortic wall. Autologous pulmonary artery patch reshaped the ostium of left coronary artery, autologous pericardial patch repaired pulmonary artery, transesophageal echo demonstrated that the aortic valve function was normal after surgery, the diameter of ostial left main coronary artery was 3.7 mm.

Post-operative coronary CTA demonstrated that the outside vascular compression of the proximal left main opening disappeared.