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Acute Inferior Myocardial Infarction Caused By Spontaneous Coronary Artery Dissection: A Case Report

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Abstract

Spontaneous Coronary Artery Dissection (SCAD) is a disease caused by spontaneous tear of coronary artery intima or spontaneous bleeding in the vessel wall, which affects or blocks coronary artery blood flow. It is an important cause of non atherosclerotic acute coronary syndrome and acute myocardial infarction. In this case report the patient was treated with coronary angiography in the catheter room, temporary pacemaker implantation and intravascular ultrasound (IVUS). Subsequent workup using coronary angiography revealed spontaneous dissection of the distal left anterior descending artery. The patient's condition improved a few days later and was discharged. The patient was instructed to take aspirin and metoprolol for secondary prevention outside the hospital for a long time. The patient was followed up for 6 months without cardiovascular events.

Keywords: Spontaneous coronary artery dissection; Acute inferior myocardial infarction.

Introduction

Spontaneous Coronary Artery Dissection (SCAD) is a rare cardiovascular disease, mainly refers to coronary artery dissection caused by non iatrogenic injury and non atherosclerotic causes. At present, the pathogenesis is not completely clear. SCAD is most often associated with Acute Myocardial Infarction (AMI) in women during their third trimester of pregnancy or the early postpartum period [1]. The demographics, causes, and natural history of Acute Coronary Syndrome (ACS) associated with SCAD are different from those of ACS caused by atherosclerosis or plaque rupture. SCAD patients are typically young women who do not have risk factors for atherosclerosis. The average age of incident SCAD is 42 years, with reported cases from the age of 14 years to well into the 7th decade. Almost 80% of SCAD patients are female and, of those, 20% to 25% of cases occur in the peripartum period [2]. Coronary dissection is thought to develop from either an intimal tear or a spontaneous hemorrhage in the vessel wall, forming a hematoma underneath the arterial wall and compressing the true coronary lumen leading to AMI. Early recognition and treatment is critical, given the high mortality rate associated with SCAD [3]. This paper reports

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the treatment process of a rare SCAD patient with myocardial infarction, providing clinical reference for the treatment and management of SCAD patients in the future.

Case presentation

A 33 year old female patient was admitted to Chengdu Second People's Hospital for emergency treatment due to "sudden chest pain for 2 hours". At admission, the patient had tearing like chest pain, which was located under the sternum, with a sense of compression behind the sternum. She had sweating and dyspnea with no other discomfort. The patient's chest pain was not alleviated and it was continuous. Before admitted to our department, she went to the emergency department of our hospital and the ECG showed that the ST segment of II, III and aVF was elevated. Considering the acute inferior myocardial infarction she was transferred to our department. The cardiology department performed coronary angiography in the catheter room **(Figure 1A,1B)**, temporary pacemaker Implantation And Intravascular Ultrasound (IVUS) **(Figure 2A, 2B)**. During intervention, no obvious calcification and stenosis is found in the left main trunk, no obvious calcification and stenosis is found in the anterior descending branch, TIMI3 blood flow, no obvious calcification and stenosis is found in the circumflex branch, and TIMI3 blood flow. The proximal segment of the right coronary had severe stenosis (90%), with TIMI grade 3 blood flows. Intravascular ultrasound showed coronary artery dissection. The patient had a slow ventricular rate, which was a junctional escape, and underwent temporary pacemaker implantation. After operation, she returned to the ward and planned for further diagnosis and treatment of "spontaneous coronary artery dissection". The patient's recent mental health, diet and sleep were normal, her urine and stool were normal, and her recent weight did not drop significantly. She has a history of chronic gastritis and cholecystitis. She denied history of hypertension, diabetes, family history of hereditary diseases and she has normal menstruation. Her physical examination on admission reveled her breath sounds of both lungs was clear, and a few moist rales can be heard. The heart rhythm was uniform, and no abnormal heart sounds or heart murmurs were heard. The abdomen was soft, without tenderness and rebound pain. There was no obvious edema in both lower limbs, no abnormality is found in joints, and the pathological signs were negative. The patient returned to CCU after operation and rechecked the cardiac biomarkers which were: troponin 35.81ng/ml (reference value 0-0.25 μg/ml), CK-MB 39.6 μg/ml (reference value 0-4 ng/ml), myoglobin<10 μg/ml (reference value 0-70 ng/ml). Blood lipid sugar: G1u 6.2mmol/L, TG 3.24mmo1/L, TCho 5.5mmol/L, HDL-C 1.18mmol/L.There was no obvious abnormality in blood coagulation, electrolyte, fibrinolysis and nail function. After the surgical treatment her cardiac MRI show, the left ventricular posterior inferior wall may have acute myocardial infarction, bilateral ventricular chambers were slightly enlarged and left ventricular free wall myocardium became thinner. The CTA of thoraco-abdominal aorta shows the left kidney has accessory renal artery and the right renal artery branches were normal, no obvious abnormality is found in others. There is a little exudation and band focus under the pleura in the lower lobe of both lungs. The gallbladder, bilateral renal pelvis and ureter are of patchy high density. The transthoracic echocardiography examination reveled moderate coronary dissection of mitral valve. After the operation, the patient's vital signs were stable, and they were treated with aspirin+clopidogrel dual antiplatelet, metoprolol and other drugs. The patient's condition improved a few days later and she was discharged. The patient was instructed to take aspirin and metoprolol for secondary prevention outside the hospital for a long time. The patient was followed up for 6 months without cardiovascular events, chest tightness, chest pain and other discomfort.

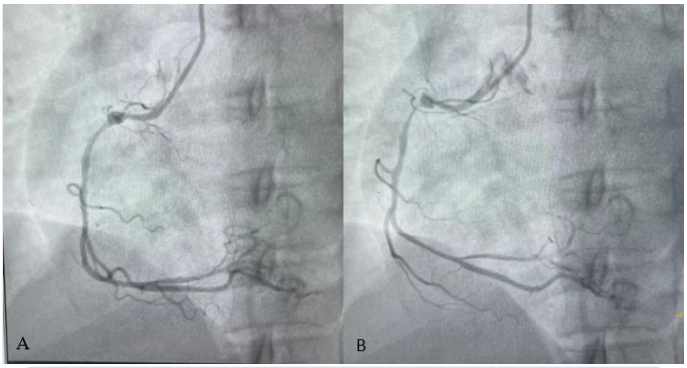

Figure 1: Coronary Angiography Results: Right Coronary Proximal Stenosis.

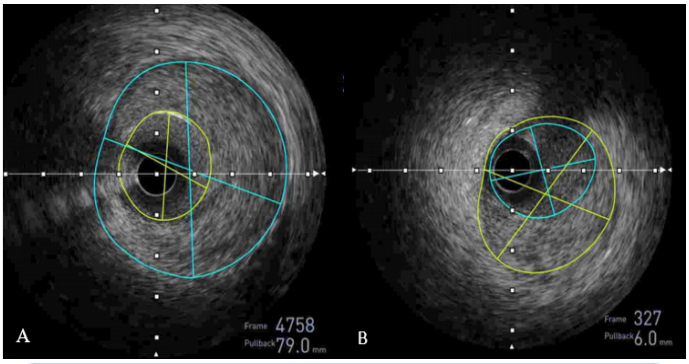

Figure 2: Coronary artery dissections by intravascular ultrasound.

Discussion

In 1931, Pretty, H.C. found SCAD for the first time when dissecting a 42 year old female coronary aneurysm. In recent years, acute coronary syndrome, myocardial infarction and sudden cardiac death induced by Spontaneous Coronary Artery Dissection (SCAD) are increasing, which are also important inducements of pregnancy related myocardial infarction, especially in young women. SCAD was initially defined as acute coronary syndrome in perinatal women, and women accounted for more than 90% of the SCAD population [3]. Most patients do not have traditional risk factors for cardiovascular disease. Its pathological characteristics are spontaneous formation of hematoma in the coronary artery wall and the formation of false lumen. The false lumen compresses the true lumen and leads to coronary artery blood flow obstruction, leading to myocardial ischemia and infarction. The main cause of false lumen formation is still unclear. At present, it is believed that there are two possible mechanisms: (1) the "inside out" hypothesis, that is, the intima "tears" so that blood passes through the intima and gathers in the middle membrane; (2) "From the outside to the inside" hypothesis, that is, spontaneous rupture of intramural microvessels leads to bleeding directly into the middle membrane [4,5].

Its etiology is multifactorial and may be related to potential arterial disease, genetic factors, hormone effects or systemic inflammatory diseases [6]. Current clinical studies have confirmed that the formation of SCAD is related to myofibrodysplasia (FMD), which is a non purulent sclerosing vascular disease that can lead to stenosis, dissection and aneurysm [7, 8]. In addition, Sun Y et al. recruited 85 SCAD cases and 296 non SCAD

controls from Chinese Han population, and found that the occurrence of SCAD was related to genes [9]. Of course, in addition to these causes, SCAD is also associated with postpartum status, multiple births, connective tissue diseases, systemic inflammation, hormone therapy and other factors.

The clinical manifestations of SCAD are similar to those of acute coronary syndrome. Chest pain is the most common symptom, and it is often accompanied by ST segment changes in myocardial zymogram and electrocardiogram [10]. There are also a few patients with neck and shoulder pain, dyspnea, atrial fibrillation, ventricular tachycardia and other symptoms. Therefore, SCAD patients are easily confused with acute myocardial infarction, unstable angina, heart failure, cardiogenic shock, ventricular arrhythmia, sudden cardiac death and other diseases caused by coronary atherosclerotic heart disease, with a high misdiagnosis rate.

At present, coronary angiography is still the main means to diagnose SCAD. The development of intravascular ultrasound (IVUS) and Optical Coherence Tomography (OCT) has improved the accuracy of early diagnosis of SCAD. IVUS has advantages in penetration depth and evaluation of the depth and scope of hematoma, while OCT is superior to IVUS in displaying the lumen intima boundary, the location of intimal tear, and the relationship between pseudolumen and lateral branches [11].

When the patient's condition is stable and there are no high-risk characteristics, conservative treatment strategies can be adopted [12]. Among 156 SCAD patients who were followed up by Hassan et al, 95% of them had spontaneous healing of coronary artery dissection [13]. Saw J [15] et al. followed up 327 non pathogenic sclerosing SCAD patients in 2016 and found that β receptor blockers reduced the risk of recurrent SCAD (HR=0.36, P=0.004). There is no guideline for the use and duration of antiplatelet therapy. Most studies advocate dual antiplatelet therapy (aspirin + clopidogrel) in acute phase, while anticoagulation should be used with caution in general [14]. Thrombolysis may lead to coronary artery rupture and cardiac tamponade, so thrombolytic therapy is contraindicated in the acute phase of SCAD [16]. When patients have high-risk characteristics, they should consider immediate revascularization [15], including percutaneous coronary intervention (PCI) and coronary bypass grafting (CABG). Some studies have shown that PCI will increase the risk of coronary artery complications [16]. Tweet et al, and others studied 87 SCAD patients and found that the failure rate of PCI surgery exceeded 50% [17,18]. However, PCI is still needed in some specific cases, such as repeated ischemia, unstable hemodynamic, and so on. CABG is optional for patients with PCI failure and complicated lesions, but the short-term success rate of CABG is high, but the long-term patency is poor [19].

Conclusion

Although the understanding of SCAD has made some progress, the etiology, diagnosis, management and prognosis of SCAD still need further research, and more research is expected to explore strategies to improve the cardiovascular outcome of SCAD patients.

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