

SPONTANEOUS CORONARY ARTERY DISSECTION AS THE CAUSE OF ACUTE MYOCARDIAL INFARCTION

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Spontaneous coronary artery dissection (SCAD) is a form of acute coronary syndrome. Better diagnostic tools and increased operator awareness have led to increase in the number of SCAD diagnosis. We present a case of a 62-year-old female diagnosed with acute inferior ST elevated myocardial infarction with spontaneous right coronary artery dissection in background, complicated by iatrogenic artery perforation. Owing to prompt coated stent deployment and successful sealing of the vessel rupture and additional stent implantation to cover dissection, the patient was stable and fully recovered.

Key words: coronary artery disease, acute myocardial infarction, spontaneous coronary artery dissection

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INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a non-iatrogenic, non-atherosclerotic intimal tear leading to false lumen and/or intramural hematoma formation compressing the true arterial lumen and compromising blood flow causing acute coronary syndrome (ACS) (1). In early studies, the incidence of SCAD was underestimated to account for 0.2%-1.1% of ACS patients, but wider use of coronary angiography in ACS and introduction of novel vascular imaging techniques such as intravascular ultrasound and optical coherence tomography has led to increase in the number of SCAD diagnosis (2-6). Recent studies report on the incidence of SCAD in ACS to be 22%-31% (6-10). SCAD predominantly affects female gender, usually below the age of 60, and with few or no traditional atherosclerotic risk factors (1,6-10). Many questions about SCAD causes and pathogenesis still remain unanswered. Various studies and early case reports associated SCAD with migraine headache, emotional or physical stress, pregnancy, arteriopathies, system inflammatory diseases, and connective tissue diseases (6-16). Pregnancy-associated SCAD is the most common cause of myocardial infarction in

the last trimester of pregnancy and early postpartum period (17). Although the reasons of this are not fully understood, alterations in arterial wall structure, possibly by the influence of estrogen and progesterone changes during pregnancy, may predispose vessel wall weakening *via* their receptors present in coronary arteries, leading to dissection or rupture (17). Several studies have also reported pregnancy-associated SCAD to have poorer prognosis than pregnancy unassociated SCAD, with more proximal artery dissections and larger infarcted area, lower left ventricle ejection fraction and higher complication rate (17). Current consensus considers that a combination of genetic and hormonal factors, arteriopathies, chronic inflammation, as well as physical or emotional stress exposure, increases the probability of SCAD development (1,6,12,18,19). Differentiating ACS caused by atherosclerotic plaque complications and SCAD is becoming important because of differences in treatment strategy. Stable SCAD is recommended to be treated conservatively, whereas unstable SCAD should undergo percutaneous coronary intervention (2,20). Our SCAD case presented with acute inferior ST elevated myocardial infarction (STEMI) and a complication of iatrogenic right coronary artery perforation.

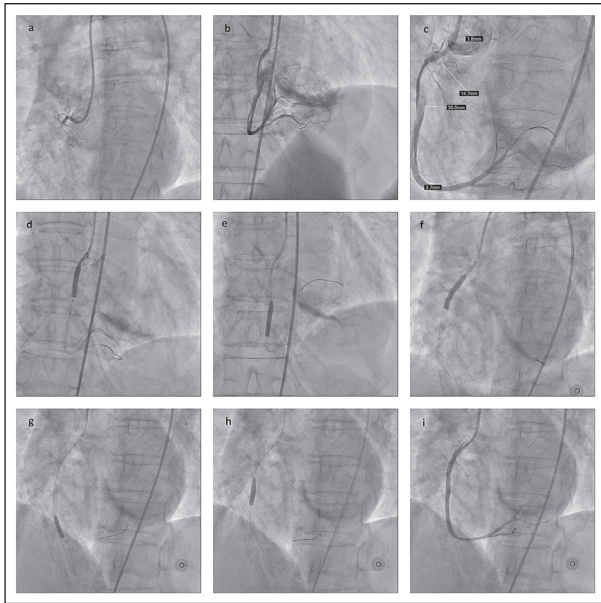


Fig. 1. Emergency coronary angiography

(a) acute occlusion of proximal right coronary artery; (b) wire passage showing dissection of the vessel and perforation of proximal right coronary artery; (c) measurement of vessel dimension; (d) placement of graft stent at the site of vascular perforation; (e) placement of drug eluting stent in the middle segment of the vessel; (f) placement of drug eluting stent in the ostium of the vessel; (g) placement of the drug eluting stent in the distal part of the vessel; (h) dilatation of the entire segment with non-compliant balloon; (i) final result.

CASE REPORT

A 62-year-old female presented to the emergency department for acute chest pain lasting for four to five hours. Electrocardiogram demonstrated acute STEMI of the inferior left ventricular wall. She was immediately admitted to catheterization laboratory. Blood pressure was 110/75 mm Hg, auscultatory heart sounds were normal and lungs were clear (Kilip stage I). The patient received acetylsalicylic acid 300 mg and ticagrelor 180 mg at the emergency department.

She was generally in good health, without prior history of cardiovascular diseases. Her family history was also negative for cardiovascular diseases, but the patient stated that her family had experienced a tragedy a couple of months before. Seventeen years before, she was investigated for inflammatory bowel disease without definitive confirmation of diagnosis. Those symptoms ceased without recurrence. Two years before, she was diagnosed with hypothyroidism caused by chronic lymphocytic thyroiditis and had been treated with thyroid hormone substitution. Eight days before admission, she had undergone laparoscopic cholecystectomy without complications.

Emergency coronary angiography was done with the following findings: left main coronary artery, left descending and circumflex arteries were without occlusions or significant stenosis; right coronary artery was occluded at proximal segment. After securing good guide catheter support, we crossed occlusion with floppy wire without significant resistance and obtained an angiogram that showed artery dissection beginning from the ostium and penetrating proximal and middle artery portions with contrast extravasation in the proximal segment area. The inserted wire was initially located in posterolateral artery; we moved it deep in the posterior descending artery securing good wire support, and placed a PK Papyrus 3.5/20 mm covered stent in the proximal artery segment, successfully sealing the vessel rupture. Further angiograms taken from multiple projections showed no contrast extravasation. In the continuation of the procedure, we implanted two additional stents in the middle and one in the ostial artery segments with final TIMI 3 flow. Bedside echocardiography done in catheterization laboratory revealed no significant pericardial fluid accumulation.

After the procedure, the patient was transferred to coronary unit; she was hemodynamically stable without chest pain recurrence. Echocardiography showed lower ejection fraction (EF) 37% with inferior and posterior left ventricular wall hypokinesia, first degree diastolic dysfunction and mild mitral regurgitation. Further investigations did not reveal the existence of cardiovascular risk factors; total cholesterol, LDL, triglycerides, blood sugar and blood pressure were all normal, and the patient was a nonsmoker. At the hospital, the patient was treated with acetylsalicylic acid and ticagrelor, angiotensin-converting enzyme (ACE) inhibitor, and beta blocker.

The patient was discharged from the hospital with a recommendation of cardiac rehabilitation. After rehabilitation, heart systolic function was significantly improved with EF 55%, only mild inferior wall hypokinesia remained.

DISCUSSION

This case represents a somewhat unexpected angiographic finding for SCAD, initially pointing to atherosclerotic coronary disease as the cause of ACS. The patient's age also contributed to this conclusion. After passing the wire into the coronary artery, the true nature of ACS background was revealed. Unfortunately, the wire was inserted in the false vessel lumen causing perforation. Contrast pressure also contributed to perforation. Quick wire placing in the correct lumen and sealing the vessel perforation with covered stent was

lifesaving. Additional stents were also placed to cover dissection. Although available studies and guidelines recommend conservative therapeutic strategy for hemodynamically stable SCAD patients, in this case the conservative strategy was not suitable due to vessel perforation and possible cardiac tamponade (1,20,21). Angiographic SCAD presentation varies from the most common (52%-67%) long diffuse smooth stenosis bordered by normal segments (type 2A), or extends to distal tip of the artery (type 2B), double lumen appearance in 20% (type 1), and rare types 3 and 4, focal tubular stenosis and distal artery occlusion, respectively (6,7,12,22,23). Rare type 4 dissection and the patient's age initially led us to consider atherosclerotic plaque rupture rather than SCAD as the cause of ACS. On the other hand, the absence of classic cardiovascular risk factors pointed to SCAD, but at the time of the patient's admission to catheterization laboratory, only the patient's medical history and blood pressure were available, without cardiovascular risk factors identified. Blood tests had not been finished yet and risk estimation was incomplete. Some studies suggest association of SCAD and coronary vessel tortuosity, but we did not find significant vessel tortuosity in this case (12,24). According to two published studies by Saw *et al.*, which included 168 and 750 SCAD patients, respectively, physical and emotional stress is identified as an important precipitant (12,25). In our opinion, emotional stress due to the recent family tragedy, as well as stress from recent surgery could be considered as precipitant factors in this case. No other risk factors previously described in various studies or case reports could be applied to our case.

Early autopsy studies have reported two underlying pathologic mechanisms for SCAD, i.e. intimal tear with false lumen formation and intramural hematoma with distal blood flow obstruction (26,27). The role of thrombus in SCAD pathogenesis is unclear. One study in 11 patients has reported the presence of minor thrombi in all patients by using OCT image technique, while many other studies did not find any thrombi present in coronary arteries (12,28).

Fibromuscular dysplasia represents disorganization and destruction of connective tissue affecting arterial wall and presenting with dissection, aneurysms, beading or tortuosity (29,30). It usually affects renal arteries, but can affect any arterial bed. The US FMD registry reports that out of 921 patients enrolled, 2.1% had SCAD (31). Although some centers recommend FMD screening for SCAD patients, general recommendation for routine screening needs further evaluation (1). At present, it is unlikely that screening will change the course of treatment (1). Left ventricular function is usually preserved in ACS caused by SCAD. Saw *et al.* found reduced EF below 50% in only 17.3% of patients

(12). Our patient had significantly reduced EF, which recovered after cardiac rehabilitation and administration of ACE inhibitor and beta blocker. Personalized cardiac rehabilitation is recommended after ACS caused by SCAD, similar to the atherosclerotic cause (17,32). The patient's cardiovascular risk was estimated to be at an intermediate level. Total cholesterol and LDL were within the normal range for risk level, 2.6 mmol/L and 2.01 mmol/L, respectively. Statin therapy was not initiated according to the guidelines and recommendations for SCAD (17,32). Because of stent implantation, dual antiplatelet therapy was introduced and aimed to continue for one year at least in the absence of complications.

In conclusion, although the number of diagnosed SCAD in ACS is increasing owing to better diagnostic tools and wider usage of coronary angiography, as well as increased awareness of SCAD possibility, interventional cardiologists should be vigilant for atypical and rare clinical angiographic SCAD presentation. In case of hemodynamic instability or iatrogenic complications, prompt application of appropriate therapeutic procedure is mandatory.

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SAŽETAK

SPONTANA DISEKCIJA KORONARNE ARTERIJE KAO UZROK AKUTNOG INFARKTA MIOKARDA

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Spontana disekcija koronarne arterije je oblik akutnog koronarnog sindroma. Bolji dijagnostički alati i povećana svijest operatora doveli su do povećanja broja dijagnosticiranja spontanih disekcija koronarnih arterija. Prikazujemo slučaj 62-godišnje žene s dijagnozom akutnog inferiornog infarkta miokarda s ST elevacijom uzrokovanog spontanom disekcijom desne koronarne arterije i kompliciranog jatrogenom perforacijom arterije. Zahvaljujući brzom postavljanju obloženog stenta i uspješnom zatvaranju puknuća žile te dodatnoj ugradnji stenta radi pokrivanja disekcije bolesnica se uspješno oporavila.

Ključne riječi: akutni koronarni sindrom, akutni infarkt miokarda, spontana disekcija koronarnih arterija