Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary events or sudden cardiac death. It most commonly occurs in young women in the peri-partum period. The etiology remains uncertain. Possible mechanisms are hormonal changes, hemodynamic stress and modifications in the immune system during pregnancy. Treatment strategies include medical management, percutaneous coronary intervention (PCI) and surgical revascularization. We describe the case of a young woman presenting with spontaneous coronary artery dissection of the left anterior descending coronary artery, which was successfully treated with PCI. Spontaneous coronary artery dissection is a rare but important cause of myocardial ischaemia and infarction in young, healthy women, without classical coronary risk factors. Immediate coronary angiography is essential to establish an early diagnosis and allowing a therapeutic decision.

Key Words: Acute coronary syndrome; dissection


Anahtar Kelimeler: Akut koroner sendrom; diseksiyon

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manifestations.\textsuperscript{1} Pathogenesis and optimal management of SCAD are still controversial.\textsuperscript{6,7} Treatment strategies include medical management, percutaneous coronary intervention and surgical revascularization.\textsuperscript{7-10}

We report the case of a young woman who presented with anterior ST elevation myocardial infarction due to SCAD of LAD and underwent successful percutaneous coronary intervention (PCI).

\section*{CASE REPORT}

A 32-year-old woman with no significant medical history was admitted with sudden onset of substernal chest pain. Ten days earlier she underwent an uncomplicated cesarean delivery performed at 38 weeks' gestation and this was her third baby (G3P3). Her pregnancy and delivery was uneventful. On the day of presentation she was breastfeeding her infant when her chest pain recurred and persisted. Initially mild, it became progressively more severe over the course of several hours, with radiation to her back and left shoulder. It was sharp, substernal, associated with shortness of breath (SOB) and diaphoresis. She also noticed left arm numbness and tingling. She had never experienced this pain before. She had no known risk factors for atherosclerotic disease and no history of any connective tissue disorder. She did not smoke and did not use alcohol or drugs. There was no family history of premature coronary artery disease.

On admission, physical examinations were unremarkable. Initial electrocardiography (ECG) demonstrated ST-segment elevation at V1-6 (Figure 1). She was given 300 mg aspirin and 600 mg clopidogrel, unfractionated heparin and treated with antiischemic drugs. The patient underwent emergency cardiac catheterization which revealed long, linear filling defect in whole LAD, beginning from the LAD ostium and extending to the end of LAD and involving Diagonal-2 branch (Figures 2, 3). Otherwise the coronary arteries were free of angiographically visible atherosclerosis. The patient was still having pain at the time of catheterization and ST elevation on ECG. The LAD dissection was treated with primary stenting without predilation. A 2.5x26 mm drug eluting stent (DES) (Scimed, Boston Scientific, Natick, MA) was deployed dis-
tally. A second 2.75x24 mm DES (Scimed, Boston Scientific, Natick, MA) was then deployed proximally to the first stent with an overlap of 1 mm. The third stent 3.0x24 mm DES (Scimed, Boston Scientific, Natick, MA) was deployed finally for proximal LAD. After deployment, the last stent was postdilated with a 3.5x15 mm non-compliant balloon (Brosmed Medical, Netherlands). An excellent angiographic result was achieved (Figures 4, 5). She had no pain after the catheterization and the ECG after angiography was show no ST elevation (Figure 6). A serum lipid profile was normal. During hospitalization there was no further ischemic chest pain. To assess the cardiac function transthoracic echocardiography was performed that showed left ventricular apical hypokinesis with an ejection fraction (EF) of 40% to 45%.

The patient was discharged after fourth day on an antiplatelet regimen of aspirin 100 mg and clopidogrel 75 mg and anti-ischemic drugs including ACE inhibitory and beta blocker. After 3 mo of follow-up, the patient remains well, with no recurrence of ischemic symptoms.

**DISCUSSION**

Spontaneous coronary artery dissection is one of the rare causes of acute coronary syndrome, particularly among young healthy individuals. Its incidence ranges from 0.07 to 1.1% in angiographic series. The most of the cases (70-75%) are diag-
nosed post mortem. More frequently spontaneous coronary dissection is observed in women, particularly during the post-partum period or in women on oral contraception. The etiologies of spontaneous dissection may include hypertension, Marfan syndrome, connective tissue disease or immune system disease and sometimes, a recent chest trauma is related to a spontaneous dissection. Intense physical exercise, prolonged sneezing, cyclosporine and cocaine abuse are rarer causes. Dissection is commonly observed at LAD (75%) and with multivessel and left main coronary artery (LMCA) involvement in 20% and 12% of cases. Also, spontaneous dissections of right coronary artery (RCA) were reported in literature. The initial event leading to dissection is not clear, and no single factor has been found to be causative. Many of the proposed mechanisms pertain to changes in vascular wall properties that lead to weakening of the media and connective tissue. These include changes in smooth-muscle-cell metabolism, the effect of proteases released from eosinophilic infiltrates, and pregnancy related changes in connective tissue.

Given the rarity of the condition, there is no prospective randomized controlled data available to guide management of SCAD. Management decisions are generally based on clinical presentation, angiographic characteristics, and to prevent potential adverse clinical outcomes. Conservative medical therapy, percutaneous coronary revascularization, and coronary artery bypass grafting (CABG) are available options. The treatment selection should be made on the clinical symptoms, extent and location of dissection, as well as the preservation of distal coronary flow.

Conservative management has yielded positive results. Medical treatment can be considered in asymptomatic patients. Medical management includes antithrombotic therapy with heparin or low molecular weight heparin, aspirin, clopidogrel and glycoprotein IIb/IIIa inhibitors, and anti-ischemic therapy with beta-blockers and nitrates. However, it should be borne in mind that while potent antithrombotic therapy decreases thrombus formation in the false lumen, enhancing blood flow in the true lumen, which can also increase bleeding into the false lumen, causing extension of the dissection. This is especially true for fibrinolytics and their use should generally be avoided. Medical treatment in stable patients without evidence for

FIGURE 6: ECG after stenting shows no ST elevation and keep R wave progression was preserved on anterior leads with biphasic T wave in V1-V3 and d1-aVL.
ongoing ischemia has a good overall prognosis with a majority of them showing spontaneous healing of the SCAD on follow-up angiography.14

If there is an evidence of ongoing ischemia such as cardiogenic shock, hemodynamic or electrical instability, revascularization should be considered with CABG or PCI.15 In single-vessel dissection not involving the LMCA, with persistent impairment of blood flow and signs of ongoing ischemia, PCI with stenting is the procedure of choice.1,3,8 However, PCI in patients with SCAD have been reported to have higher initial technical failure rates in approximately one-third of the patients, requiring additional stenting due to extension of dissection or intramural hematoma.3,15 So, intravascular ultrasound and optical coherence tomography may be of value in identification of the true lumen.8 The use of drug-eluting stents (DES) in SCAD is questionable. While the frequent need for long stented segments in these cases may justify the preferential use of DES, on the other hand, these stents may delay vessel wall healing. The choice between baremetal stents and DES remains subject of disagreement.8 We used DES for our case because of long dissection. CABG is usually reserved for patients with LMCA dissection, multivessel involvement and failed PCI.1,8 Nevertheless, in selected cases LMCA and multivessel dissections may be treated by stenting as well.8,10

The treatment selection should be made on the clinical symptoms, extent and location of dissection, as well as the preservation of distal coronary flow. Our case illustrates that intracoronary stenting may prove to be the treatment of choice for patients with single-vessel SCAD not involving the left main coronary artery and chest pain with ST elevation on ECG.

Spontaneous coronary artery dissection is a rare but important cause of myocardial ischaemia and infarction in young, healthy women, without classical coronary risk factors. Immediate coronary angiography is essential to establish an early diagnosis and allowing a therapeutic decision. We describe a case report diagnosed with ST elevation myocardial infarctus due to SCAD which treated with primer percutaneous coronary intervention.

REFERENCES