

Treatment of spontaneous coronary artery dissection with drug-eluting stents

Samad Ghaffari, MD, Jahanbakhsh Samadikhah, MD.

ABSTRACT

Spontaneous coronary artery dissection (SCAD) is a rare condition which may result in sudden coronary occlusion, acute myocardial infarction and sudden cardiac death. The prognosis of spontaneous coronary artery dissection is uncertain and optimal treatment is unknown. We describe a case of SCAD in a 29-year-old male with acute inferior wall myocardial infarction (MI) and post-MI angina that was treated with drug-eluting stents.

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From the Cardiovascular Research Department of Tabriz University of Medical Sciences, Tabriz, Iran.

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Address correspondence and reprint request to: Dr. Samad Ghaffari, Department of Cardiology, Madani Heart Hospital, Tabriz, Iran. Tel. +98 (411) 3357771. Fax. +98 (411) 3344021. E-mail: ghafaris@gmail.com

Spontaneous coronary artery dissection (SCAD) has been most commonly described in middle-aged, otherwise healthy women in the peripartum period, with no coronary atherosclerosis and no apparent risk factors for atherosclerosis or coronary artery disease (CAD).¹ Although the lesion is often fatal and the diagnosis established postmortem,² in recent years there have been reports of successful medical, percutaneous and surgical interventions.³ In this study, we describe a case of an extensive spontaneous right coronary artery dissection that led to inferior wall myocardial infarction (MI) and post MI angina and was treated with drug-eluting stents (DES).

Case Report. A 29-year-old man was admitted with sudden onset of retrosternal pain lasting for more than one hour. His past medical history was negative for recent chest trauma, intense physical exercise and cocaine abuse. He had history of smoking 10-15 cigarettes per day for the last 5 years. On examination, the patient was still complaining of chest pain, and had profound diaphoresis. His arterial pressure was 110/80 mm Hg, and his heart rate was 78 beats/minute; heart auscultation was unremarkable except for an audible

S4. Jugular vein pressure was normal and both lungs were clear. An electrocardiogram showed ST-elevation of the inferior leads with <1 mm ST change at right precordial leads. The troponin I and CK-MB levels showed remarkable elevation. Initial management included intravenous morphine, nitroglycerin, aspirin, low dose metoprolol and streptokinase infusion (1.5 million units/1 hours). Echocardiography showed inferoapical hypokinesia with global left ventricular ejection fraction of about 45%. There was a mild mitral regurgitation with no right ventricular enlargement or significant dysfunction. He was stable for the next 48 hours but he suffered from 2 episodes of retrosternal pain at third day and was immediately transferred to the catheterization laboratory for coronary angiography. The left coronary system was normal but right coronary artery (RCA) showed a long linear dissection flap involving proximal and mid segments (**Figure 1**). There was no evidence of any atherosclerotic coronary artery disease. Two drug eluting stents 3.0 x 28 mm and 3 x 18 mm Cypher™ stents (Cordis Corp, Miami, Florida) were deployed consequently at mid and proximal RCA with overlap and inflated up to 16 atm. Postdilatation with a noncompliant balloon (3.0 x 20 mm) was undertaken. At the end of the procedure, a complete sealing of the dissection flap was achieved with TIMI 3 distal flow (**Figure 2**). His hospital course was uneventful and he was discharged home on aspirin, metoprolol, plavix and captopril in a stable condition at seventh day following admission. At 6 months post MI visit he was asymptomatic and maximal exercise test was negative for ischemia.

Discussion. Spontaneous coronary artery dissection was first described at postmortem examination in a 42-year-old woman >70 years ago.⁴ Up to now >250 cases of SCAD have been reported in the literature. Sudden death has been reported commonly, and nearly 69% of cases are diagnosed in post-mortem studies.² The most

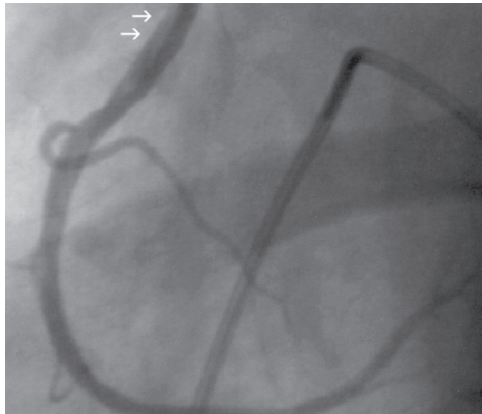


Figure 1 - Right coronary artery dissection flap extending from proximal to mid segment. Temporary pace maker was inserted to prevent severe bradycardia.

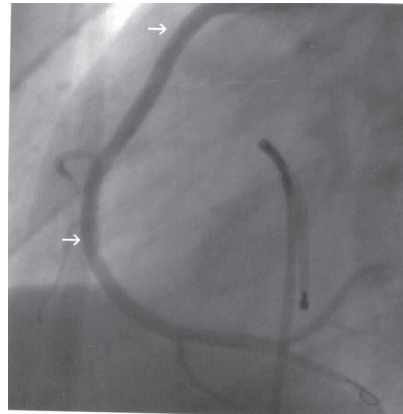


Figure 2 - Complete sealing of dissection flap after stent implantation.

frequently involved vessel is the LAD, though left main and multivessel dissections have been described.⁵ Spontaneous dissection of bilateral internal mammary arterial grafts has also been observed in a single patient.⁵ In general, this disease is associated with a high mortality.² Most reported patients are apparently healthy, young to middle-aged women (78%; mean age 40 years)² without overt risk for CAD and without severe coronary atheromatosis. A review by Jorgensen et al² of the reported cases depicts poor survival (30%) and frequent postmortem diagnosis (69%). In women with SCAD, there is a predilection for the left coronary system (84%), whereas in men, the right coronary artery is usually affected (67%).⁶ The etiology remains uncertain, although a significant proportion of these women (25%) are in the puerperium. The use of oral contraceptives and the exceptional hormonal balances in the peripartum period are supposed to weaken the arterial wall and to predispose it to rupture or dissection.⁶ Cystic medial necrosis of the coronary arteries is a rare finding, and few case reports have been published illustrating SCAD due to this arterial wall anomaly.⁷ Other reported possible causes of SCAD are cocaine abuse,⁶ connective tissue diseases, systemic hypertension and intense physical exercise.⁵ The pathogenesis is still unclear and subject to debate. In the peripartum period, there is an altered hemodynamic status with an increase in cardiac output and blood volume, both augmenting the mechanical stress on the arterial wall. Coupled with an adaptive endocrine metabolism, this may lead to smooth muscle cell hypertrophy, loosening of ground substance and fragmentation of reticular fibers. An accumulation of eosinophils, with liberation of lytic proteases in the media, has been shown to contribute to vessel wall damage.⁵ Furthermore, excess progesterone has been postulated to induce a weakness in the tunica

media by structural modification in the coronary vessel wall. Disruption of the vasa vasorum with intramedial hemorrhage and dissection has been found in autopsy studies. The use of oral contraceptives is also considered a contributor since high estrogen levels may be responsible for low collagen synthesis and subsequently predisposes women to spontaneous dissection.⁵ Koul et al,³ reported an overall mortality for spontaneous coronary dissections of 38%, mostly occurring within the first few hours from the onset of symptoms. There are no guidelines available on the best treatment of patients with coronary artery dissection. Conservative treatment in the form of aspirin, nitrates, beta-blockers, and antiplatelet agents has been associated with clinical and angiographic resolution.¹ Low-molecular weight heparin has been found to be beneficial.¹ In theory, thrombolytic therapy could lyse a compressing intramural clot, but it could also contribute to the expansion of an intramural hematoma.^{6,8} Percutaneous coronary interventions have also had favorable outcomes. Cardiac transplantation, and total artificial heart implantation¹ have been attempted to stabilize patients with catastrophic presentations. Medical treatment appears to be appropriate if the patient has no hemodynamic instability and chest pain quickly disappears.⁹ In such cases, the dissection occasionally can completely heal, as confirmed by follow-up angiography.⁵ Surgical or percutaneous revascularization is mandatory for patients with persistent or recurring symptoms, since the 3-year mortality rate in this setting for the untreated patients is 20%.⁵ Coronary stenting for SCAD was first reported in 1996,¹⁰ and has become the treatment of choice for preventing further propagation of dissection. There are now over 30 case reports of bare metal stenting for SCAD in the literature; however, in-stent stenosis has been problematic.¹¹ Porto et al¹² reported the first

case of DES implantation in a patient with SCAD. At 2 years follow up the patient was asymptomatic with patent stent and no evidence of neointimal hyperplasia at angiographic and IVUS studies.¹¹ Coronary artery bypass grafting is recommended for patients with dissection involving the left main stem and for patients with multivessel involvement and ongoing myocardial ischaemia refractory to medical treatment that are not amenable for percutaneous intervention.⁹ In the case of severe heart failure, bridging to heart transplantation can be the most reasonable choice.⁴ In our patient, reappearance of symptoms at third day may be related to streptokinase infusion and expansion of intramural hematoma as previously was discussed by Buys et al.¹² In the absence of atheromatous coronary artery disease, reports of DES use for SCAD are rare, and as previously discussed there is only one report of long-term angiographic outcomes with this approach.¹⁰

In conclusion, in the absence of ongoing ischemia or hemodynamic instability, a trial of medical therapy for SCAD appears to be supported by the literature. Otherwise stenting appears to be the treatment of choice. Despite limited reports of DES for SCAD and lack of long term results, our case demonstrates the utility of DES in providing a good clinical outcome at 6 month follow up in the absence of atherosclerotic coronary artery disease.

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