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Spontaneous Coronary Artery Dissections: Three Case Reports and Review of Literature

Bora Farsak,[1] Mehmet Oc,[1] Bahar Oc,[2] Murat Simsek,[1] Hakan Akbayrak,[1] Ahmet Avcı[3]

Selcuk University, Selcuklu Faculty of Medicine, Departments of [1]Cardiovascular Surgery, [2]Anaesthesiology and Reanimation, and [3]Cardiology, Selcuk University, Selcuklu Faculty of Medicine, Konya, Turkey

ABSTRACT

Primary spontaneous coronary artery dissection is one of the rare causes of acute myocardial infarction and is mostly fatal. Previous studies report that it is mostly seen in middle-aged women in the last trimester of pregnancy and early postpartum period. Clinical presentation of the disease is variable in pattern, and its severity is related to extent and development rate of dissection. Herein we present three cases of primary spontaneous left main coronary artery dissection. Two of the patients are men and the third patient is a non-pregnant 69-year-old woman. The cases were presented and discussed with review of the pertinent literature.

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare, but it has received increasing attention recently with 51 publications since the year 2000, nearly 500 up to date. Spontaneous coronary artery dissection is an uncommon case of sudden cardiac death and acute coronary syndrome [Butler 2005]. Several diseases and conditions have been associated with SCAD, so it therefore probably constitutes a heterogeneous entity. Risk factors for SCAD include pregnancy, Ehlers-Danlos disease, Marfan's Syndrome, intensive exercise, or cocaine abuse [Jorgensen 1994; Hering 1998; Butler 2005]. Coronary artery dissection is often fatal, with approximately 75% of cases diagnosed at autopsy [De Maio 1989; Zampieri 1996]. The clinical presentation depends on the extent and the flow-limiting severity of the coronary artery dissection and ranges from asymptomatic to unstable angina, acute myocardial infarction, and ventricular arrhythmias to sudden cardiac death. Coronary angiography is frequently used in the evaluation of patients with acute coronary syndromes. Thus, most cases with SCAD are detected by angiography. Moreover, intracoronary imaging techniques such as intravascular ultrasound (IVUS), optical coherence tomography (OCT), and multidetector computed tomography (MDCT) have been used in evaluation of patients with SCAD. There is no consensus about the method of treatment including medical therapy, interventional treatment with percutaneous coronary intervention, or surgery. We present three cases of SCAD treated surgically.

CASE REPORTS

Case 1

A 49-year-old man presented to the emergency room with sudden onset of chest pain. He had no prior history of cardiovascular disease, trauma, or collagen tissue disease. He smoked 1 pack of cigarettes per day and drank alcohol minimally. He was taking no medications. The patient had no drug allergies. Additionally, no history of coronary artery disease existed within his family. Vital signs included blood pressure, 122/64 mm Hg; pulse, 80 beats/minute; respirations, 19 breaths/minute; and weight, 86 kg. Physical examination was normal.

Electrocardiogram was indicative of an acute anterior myocardial infarction. Initial cardiac enzyme studies included CK-MB = 55.7 ng/mL and troponin-T = 0.19 ng/mL. The patient was treated with clopidogrel and glycerin trinitrate (GTN) in the emergency room. He was urgently transported for cardiac catheterization, which showed evidence of dissection of the LMCA with involvement of the circumflex artery (Figure 1) and has 80% narrowing in the right coronary artery (RCA). He was transported to the operating room in urgent basis and underwent bypass grafting to the left anterior descending artery (LAD), posterior descending artery, and circumflex artery. He had no postoperative complications and was discharged on the fifth postoperative day. On follow-up examination, he was without complications, with normal exercise tolerance.

Case 2

A 63-year-old man presented with sudden onset of severe chest pain at rest, with progressively worsening shortness of

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Correspondence: Mehmet Oc, MD, Associate Professor, Selcuk University, Selcuklu Faculty of Medicine, Department of Cardiovascular Surgery, Konya, Turkey; 00 90 332 2415000 / 45156 (e-mail: mehmetoc@hotmail.com).

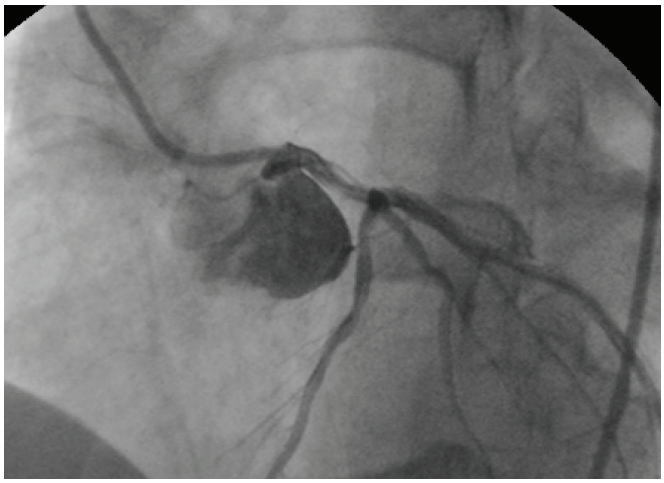

Figure 1. Coronary angiography shows dissection of the LMCA

breath. He was a smoker, and his only past medical history was hypertension. On examination, he had signs of heart failure. Electrocardiography (ECG) showed widespread T-wave inversion, and a blood test revealed raised levels of troponin and creatine kinase. He suffered ongoing angina despite being treated with continuous intravenous heparin and GTN. In view of his unsettling cardiac symptoms and worsening heart failure, cardiac catheterization was performed, showing spontaneous left main coronary artery (LMCA) dissection. Echocardiogram revealed poor left ventricular systolic function with ejection fraction of 25% to 30% and inferior apical hypokinesia. Following insertion of an intraaortic balloon pump, he was referred for surgical treatment, and emergency coronary artery bypass grafting was performed. At operation, the heart was found to have globally poor ventricular function. There was clear evidence of dissection with bruising around the proximal part of the LAD. The LAD was opened distally to the site of bruising. The false lumen with hematoma in between the tunica media and adventitia and also the dissection flap were observed in LAD. The true lumen was then carefully identified for conduit anastomosis. The anastomosis was performed in routine fashion. The left internal mammary artery (LIMA) was used as the conduit for LAD, and the sapheneous vein graft (SVG) was used for the first obtuse marginal artery (OM1) anastomoses. Postoperatively, he needed prolonged inotropic support, and the transthoracic echocardiogram showed that the ventricular function remained poor. He had a slow but progressive recovery before being discharged from the hospital 3 weeks postoperatively. On follow-up examination, he was doing well with minimal symptoms.

Case 3

A 69-year-old woman with cardiovascular risk factors experienced acute onset of chest pain while walking. Pain did not resolve at rest. The patient was admitted to the emergency room, and the ECG showed slightly depressed ST–T waves in V2–6. Initial troponin and creatine kinase assessment was normal, and the patient was free of symptoms. The patient was given clopidogrel, unfractionated heparin, and a beta-receptor blocker. Six hours later, there was a marked rise troponin and creatine kinase. Echocardiography showed normal left ventricular ejection fraction without regional wall motion abnormalities. Angiography revealed a dissection of LMCA (Figure 2) with normal coronaries. She has undergone bypass grafting to the LAD and circumflex coronary arteries. She had no postoperative complications and was discharged on the seventh postoperative day. On follow-up examination, she was doing well and asymptomatic.

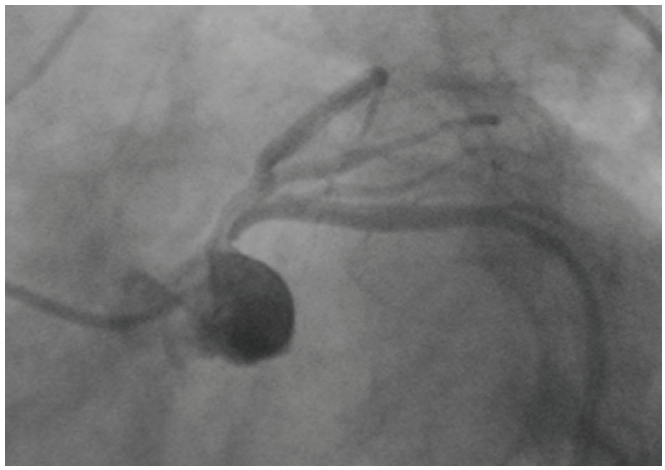

Figure 2. Coronary angiography shows dissection of the LMCA

DISCUSSION

Although the first presentation by Pretty was in 1931 [Pretty 1931] at autopsy, spontaneous coronary artery dissection is still a rare cause of acute coronary syndrome [De Maio 1989; Jorgensen 1994; Koul 2001; Mohamed 2002]. Approximately 50% of patients with coronary artery dissection die abruptly, and 18% to 20% die within hours [Jorgensen 1994]. In consecutive coronary angiographies, overall incidence of SCAD has been reported to vary from 0.1% to 1.1% [Jorgensen 1994; Hering 1998; Celik 2001]. We suggested that the true number of cases will be underestimated due to a large amount of SCAD leading to sudden death without being diagnosed.

Overall, there is a striking predominance of SCAD in the female gender [De Maio 1989; Dhawan 2002]. The mean age at presentation is 35 to 40 years, but SCAD has also been reported in older patients [Dhawan 2002]. The LAD is the most common location of dissection overall and especially in women, whereas the right coronary artery is involved more often in men [De Maio 1989; Dhawan 2002; Kamineni 2002]. There are also reports about dissections of the LMCA and multivessel dissections [Atay 1996; Rensing 1999; Zupan 2001], which much more frequently occur in women [Kamineni 2002]. These are not in accordance to the results of our study. Two of our patients are men ages 49 years and 63 years. The third patient was a 69-year-old woman. In all of our patients, the predilection site was the LMCA.

SCAD in patients with underlying coronary artery disease probably is more frequent than usually thought. The authors

emphasized that newer diagnostic tools such as IVUS allow finding underlying atherosclerotic plaques even in cases of only minor atherosclerosis [Hering 1998]. There are several reports about SCAD during pregnancy and the early puerperium [Rensing 1999; Koul 2001; Dhawan 2002; Mohamed 2002; Butler 2005]. More than two-thirds of cases present in the postpartum period, usually within two weeks of delivery [Koul 2001]. Multiparty and advanced age were found to be associated with SCAD [Koul 2001]. The very high percentage of multivessel involvement and the striking predominance of SCAD in pregnant women support the hypothesis of arterial wall changes during pregnancy under hormonal influence.

The third heterogeneous group includes patients with different predisposing pathologies or without obvious reasons, such as Marfan's syndrome, Ehlers–Danlos syndrome, polyarteritis nodosa, and lupus erythematosus [Eltchaninoff 1997; Chu 1998; Aldoboni 2002; Mohamed 2002]. The disease can be associated with use of cocaine, cyclosporine, and oral contraceptives [Azam 1995; Tsimikas 1999; Steinhauer 2001]. There are also reports of SCAD in young to middle-aged people occurring during or after heavy exercise such as running [Hering 1998; Choi 2002], weight lifting [Hong 1996], aerobic exercise [Almahmeed 1996], baseball playing [Vale 1998], or even after prolonged sneezing [Da Gama 1999]. Patients investigated in our study were two men and one woman. Only Case 1 had risk factors for atherosclerosis. We found neither signs of connective tissue disorder nor one of the previously mentioned risk factors; Cases 2 and 3 were found to be idiopathic.

SCAD can result in a broad spectrum of presentation ranging from unstable angina pectoris to cardiogenic shock due to extensive myocardial infarction or sudden cardiac death [Choi 2002; Dhawan 2002; Mohamed 2002]. Diagnosis was made postmortem in most cases in the past [Jorgensen 1994; Dhawan 2002]. Recently, with widespread use of coronary angiography, SCAD is more often diagnosed antemortem. It is important that clinicians are aware of this rare condition. All of our patients presented with angina pectoris and were diagnosed early with angiography.

Therapy depends on several factors such as site of dissection, single or multivessel involvement, coronary blood flow, and hemodynamic state. In cases of single vessel involvement without lesion of the LMCA, lacking signs of persisting ischemia, and hemodynamic stability, medical treatment is the therapy of choice [Jorgensen 1994; Zampieri 1996; Sarmento-Leite 2003]. Healing of dissection at control angiography has been reported by several authors [Mohamed 2002; Sarmento-Leite 2003].

In patients presenting with acute coronary syndrome, medical treatment including heparin, antiplatelet therapy, and beta-receptor blockers is usually initiated before the diagnosis of SCAD is known. All our patients received unfractionated heparin before coronary angiography. Choi and Davidson [Choi 2002] reported on a 37-year-old woman suffering from multivessel SCAD. After 7 months of treatment with aspirin and clopidogrel, control angiography revealed complete healing of the multivessel dissection. The successful use of a glycoprotein IIb/IIIa inhibitor was reported only in a single case [Cheung 2000]. The use of beta-receptor blockers is rare in patients with SCAD. It could be beneficial to continue beta-receptor blocker therapy in all patients without coronary artery bypass surgery, since we could not be sure that the dissection would heal in all patients, and extension of the dissection was not excluded, so patients therefore would probably benefit from anti-ischemic therapy. This practice is somewhat supported by two published case reports [Mauser 2003; Tsiamis 2003]. Nitrates and calcium antagonists are sometimes given to prevent coronary spasm [Jorgensen 1994; Koul 2001]. The role of thrombolysis is somewhat unclear. There are several reports about its successful use [Dhawan 2002; Kamineni 2002]. Thrombolytic agents are thought to restore the flow of the true lumen by lysing thrombi of the false lumen with a consecutive reduction of compression of the true lumen. However, there are reports about patients whose clinical symptoms and electrocardiograms deteriorated immediately after the start of thrombolysis [Buys 1994; Zupan 2001]. In clinical practice, thrombolysis will be given according to the current criteria because SCAD is a very rare event in contrast to the majority of patients with myocardial infarction due to acute thrombotic occlusion of coronary arteries. Interventional procedures are the therapy of choice in single vessel dissections with persistent impairment of blood flow distal to the site of dissection and ongoing signs of ischemia. Percutaneous dilatation without stent placement should be avoided, whereas coronary stenting has been successfully performed in several cases [Hong 1996; Klutstein 1997; Hering 1998; Vale 1998; Leclerc 1999].

Indication for coronary artery bypass grafting is reported as follows: (i) left main dissection, (ii) multivessel disease, (iii) failure of interventional procedures [De Maio 1989; Baron 1998; Vale 1998; Dhawan 2002]. However, in selected cases, left main dissection and multivessel involvement may be treated with stenting as well [Togni 1999]. Some authors recommend coronary artery bypass grafting for any SCAD due to underlying coronary atherosclerosis [Celik 2001]. Of course, our group is too small to permit general considerations. Each case will undergo an individualized evaluation in the light of the clinical and angiographic presentation.

Prognosis of SCAD was thought to be catastrophic for a long time because the majority of cases were diagnosed at autopsy. In the series of De Maio et al [De Maio 1989], 22 of 27 patients (81%) survived a mean follow-up of 41 months. Kamineni et al [Kamineni 2002] reviewed 152 cases of SCAD and reported higher survival rates in men compared to women (93% versus 74%). Younger people without sufficient collateral circulation may have a worse outcome due to larger areas of infarcted myocardium [Celik 2001].

CONCLUSIONS

Idiopathic SCAD is a rare cause of acute coronary syndrome. The initial treatment includes heparin, anti-platelet therapy, and beta-receptor blocker in the absence of contraindications. A high grade of suspicion should lead to an early invasive strategy. Optimal treatment depends on the site of dissection, number of affected vessels, coronary blood flow,

and hemodynamic state. In case of single vessel dissection and normal blood flow of the dissected vessel, medical treatment with acetyl salicylic acid, clopidogrel, and beta-blockade may lead to complete angiographic resolution within a few months. In other cases, stenting or bypass surgery should be considered.

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