VIA MEDICA

CASE REPORT

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Spontaneous healing of spontaneous coronary artery dissection

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Abstract

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome and sudden cardiac death. It should be suspected in every healthy young woman without cardiac risk factors, especially during the peripartum or postpartum periods. It is important to check for a history of drug abuse, collagen vascular disease or blunt trauma of the chest. Coronary angiography is essential for diagnosis and early management. We wonder whether thrombolysis might aggravate coronary dissection. All types of treatment (medical therapy, percutaneous intervention or surgery) improve the prognosis without affecting survival times if used appropriately according to the clinical stability and the angiographic features of the involved coronary arteries. Prompt recognition and targeted treatment improve outcomes.

We report a case of SCAD in a young female free of traditional cardiovascular risk factors, who presented six hours after thrombolysis for ST elevation myocardial infarction. Coronary angiography showed a dissection of the left anterior descending and immediate branch. She had successful coronary artery bypass grafting, with complete healing of left anterior descending dissection. (Cardiol J 2010; 17, 1: 92–95)

Key words: coronary dissection, spontaneous, thrombolysis, female

Case report

A 38 year-old woman, who had been experiencing chest pain for more than six hours, was referred for rescue percutaneous coronary intervention (PCI) to our interventional cardiac department after treatment with thrombolysis for an anterolateral ST elevation myocardial infarction (STEMI).

She had no medical history of collagen tissue disease, no history of blunt trauma to the chest, was not on any medication and was not known to be a drug abuser. She had no cardiac risk factors, she was not pregnant and was not in a peripartum period.

In the catheterisation theatre she presented with a Killip class II, hypotension and tachycardia. The electrocardiogram revealed ST elevation in the anteroseptal leads and inverted T waves in V1, V2 (Fig. 1). The cardiac biochemical markers were elevated. Rheumatoid factor and atrial natriuretic peptide were negative. The coronary angiography showed a dissection of the proximal left anterior descending (LAD) and intermediate branch, with probable coronary spasm of a small circumflex artery and a diagonal branch (Fig. 2). The right coronary artery was normal, as were the small lateral branches of the circumflex. Echocardiography confirmed anterolateral, septal and apical akinesia of the left ventricle. An intra-aortic-balloon-pump was introduced to sustain the labile hemodynamic situation. Confronted with PCI and hemodynamic instability, we referred her for immediate surgery with an off-pump coronary artery by-pass grafting a left interior

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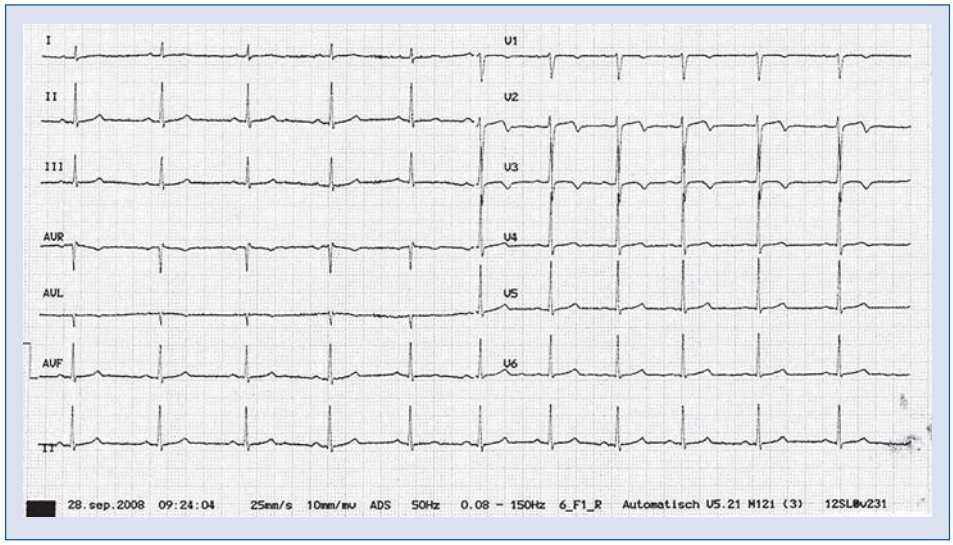

Figure 1. Electrocardiography shows ST elevation with inverted T waves in V1, V2.

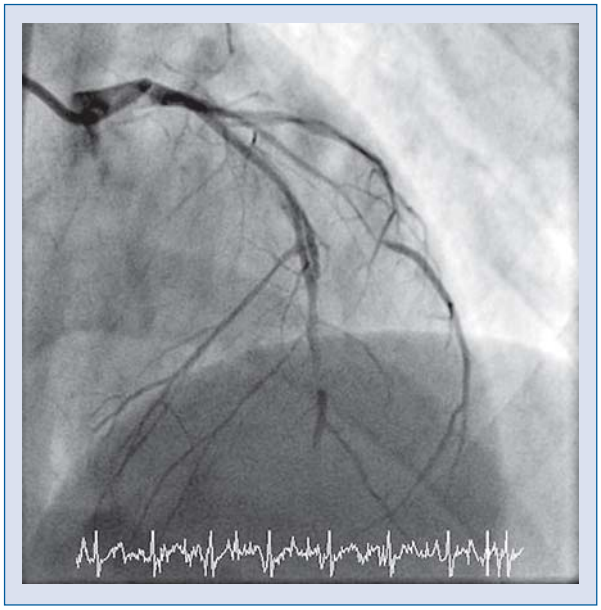

Figure 2. Coronary angiography on presentation shows dissection of the left arterior descending and intermediate branch with spasm of the circumflex.

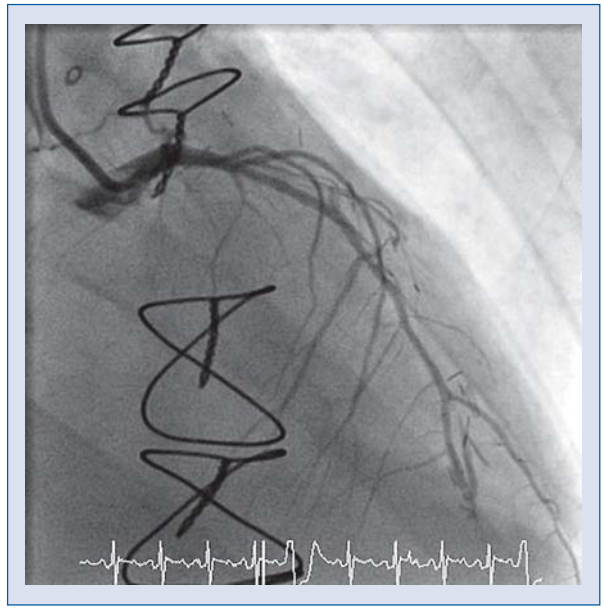

Figure 3. Coronary angiography post coronary artery bypass grafting shows healing of the left arterior descending dissection and the left interior mamary artery bypass graft.

mammary artery was placed on to the LAD and venous graft to the intermediate branch (Fig. 3).

One week after coronary artery bypass grafting (CABG), we performed a new coronary angiography to re-evaluate the coronary status. We found a healed LAD and intermediate branch. The spasms on the other vessels had disappeared.

Discussion

Spontaneous coronary artery dissection (SCAD) as a cause of acute coronary syndrome and sudden cardiac death is uncommon and has a poorly--understood pathophysiology. Dissection results in separation of the layers of the arterial wall leading

to intramural haematoma and haemorrhage into the false lumen, impairing blood flow and causing acute coronary syndrome and sudden cardiac death.

Approximately 300 cases of SCAD have been reported in the literature since the first description in 1931. A review of published reports showed that 69% of cases were diagnosed at autopsy. The angiographic incidence ranges from 0.28% to 1.1%. It most often appears in young women free of traditional risk factors: about 70% of cases occurred in women without traditional cardiac risk factors, more than 30% in the peripartum period. LAD is the most frequent location: about 60% in autopsy and angiographic series. Dissection of the right coronary artery is more frequent in men, whereas dissection of the left anterior descending coronary artery appears more common in women [1].

The pathogenesis of SCAD is not well established but can be understood through the two most common associated conditions: atherosclerosis and the peripartum period. Atherosclerotic plaque inflammation and rupture causes disruption of the intimal-medial junction. In pregnancy and peripartum and especially during labour, eosinophilic infiltrates and an increased level of collagenase and other lytic enzymes result in medial-adventitial dissection [2].

Variant angina and cocaine use may be associated with SCAD, due to an increase of shear stress on coronary arteries, which can lead to dissection. Eosinophils may also play a role in the pathophysiology of coronary spasm.

There are other conditions reported in association with SCAD: hypertension and cystic medial necrosis; diseases that cause vessel wall abnormalities including Marfan's Syndrome; Ehler-Danlos syndrome. Polyarteritis nodosa and systemic lupus erythematosus can involve the coronary arteries. SCAD has been associated with antiphospholipid syndrome and inflammatory bowel disease.

Medications (such as the oral contraceptive, cyclosporine, 5 FU, fenfluramine) have been reported as factors precipitating SCAD. Intense physical exercise precipitating coronary artery dissection has also been described.

Patients with SCAD may present with acute coronary syndrome and sudden cardiac death. The diagnosis is usually made by coronary angiography, computer tomography angio or intravascular ultrasound (IVUS). The angiographic diagnosis of SCAD by selective angiography depends on the visualisation of two lumina separated by a radiolucent intimal flap [3].

There are three well known treatments (conservative medical therapy, PCI, bypass surgery) to manage SCAD. The decision as to which one depends on clinical and angiographic factors.

When there is no persistent ischemia, hemodynamic instability or small and single vessel involvement, medical therapy alone may be suitable. This includes unfractionated heparin, low molecular weight heparin, aspirin, clopidogrel, beta-blockers and nitrates. Calcium channel blocker can be used in setting coronary spasm. GP IIb/IIIa inhibitors have been used to treat SCAD in both conservative and adjuvant therapy during PCI. There is a theoretical risk of haematoma expansion, but more data is needed [4].

Patients with STEMI due to dissection are at risk from the harmful effects of thrombolysis, which may promote extension of intramural haematoma and eventually dissection [5].

Where there is persistent ischemia, hemodynamic instability or the involvement of larger coronary arteries, percutaneous intervention or surgery may be options. Single vessel dissection is usually treated with PCI with stenting. If placed in the correct lumen, stenting shows restoration of flow, healing and prevention of further expansion of dissection. IVUS-guided procedure can ensure the placement of the guide wire, length of dissection and size of the vessel stent opposition [6].

Multivessel and left main dissection are indications for CABG. Surgery remains a technical challenge because of the fragility of the vessel wall, the involvement of long segments and the search for the true lumen [7].

In a single center study of 42 people with SCAD, 24 were treated with stenting, seven with balloon angioplasty, eight with CABG and three with medical therapy. During a mean follow-up period of 13.5 ± 9.9 months, two patients died and 35 patients remained entirely asymptomatic, including all patients who were treated with CABG. Restenosis developed in three patients after stent implantation (restenosis rate: 12.5%). Following primary PTCA, spontaneous coronary artery dissection recurred in two patients, one of whom subsequently died [1].

Contradicting earlier publications describing a mortality rate of 50%, recent analysis of reported cases describes a survival rate approaching 95% and the rate of recurrent dissection at 5% [8].

Wishing to investigate how the coronary state had evolved, we performed a new angiogram ten days after the CABG. We found complete healing of the dissection of the LAD and intermediate branch. We suggest that bypass placement on the correct lumen, with retrograde flow and intravas-

cular pressure augmentation, explains the healing of this dissected vessel.

Conclusions

SCAD is an important yet uncommon cause of acute coronary syndrome and sudden cardiac death. Classically, it occurs in young healthy women without traditional cardiac risk factors. Pregnancy and the postpartum period can be predisposing factors. Screening for possible other associated diseases remains necessary to prevent recurrence. Coronary imaging is mandatory for diagnosis. The selection of medical as well as interventional treatments depends on the extension of the dissection. In young people, especially women lacking in risk factors and with an infarction, thrombolysis should be avoided until swift coronary imaging excludes dissection.

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References

1. Hering D, Piper C, Hohmann C, Schultheiss HP, Horstkotte D. Prospective study of the incidence, pathogenesis and therapy of spontaneous, by coronary angiography diagnosed coronary artery dissection. Z Kardiol, 1998; 87: 961–970.
2. Borczuk AC, Van Hoven KH, Factor SM. Review and hypothesis: The eosinophil and peripartum heart disease (myocarditis and coronary artery dissection) coincidence or pathogenetic significance? Cardiovasc Res, 1997; 33: 527–532.
3. Kamran M, Guptan A, Bogal M. Spontaneous coronary artery dissection: Case series and review. J Invasive Cardiol, 2008; 20: 553–559.
4. Zampieri P, Aggio S, Roncon L et al. Follow up after spontaneous coronary artery dissection: A report of five cases. Heart, 1996; 75: 206–209.
5. Zupan I, Noc M, Trinkaus D, Popovic M. Double vessel extension of spontaneous left main coronary artery dissection in young women treated with thrombolytics. Catheter Cardiovasc Interv, 2001; 52: 226–230.
6. Moukarbel GV, Alam SE. Spontaneous coronary artery dissection: Management options in the stent era. J Invasive Cardiol, 2004; 16: 333–335.
7. Kamineni R, Sadhu A, Alpert J. Spontaneous coronary artery dissection: Report of two cases and a 50-year review of the literature. Cardiol Rev, 2002; 10: 279–284.
8. Thompson EA, Ferraris S, Gress T, Ferraris V. Gender differences and predictors of mortality in spontaneous coronary dissection. A review of reported cases. J Invasive Cardiol, 2005; 17: 59–61.