VIA MEDICA

CASE REPORT

Cardiology Journal
2010, Vol. 17, No. 6, pp. 625–627

ISSN 1897–5593

Multiple spontaneous coronary artery dissection associated with Trousseau's syndrome

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Abstract

Spontaneous coronary artery dissection (SCAD) remains a rare cause of acute coronary syndrome. SCAD has been observed in three groups of patients: those with coronary atherosclerosis, women in the peripartum period, and an idiopathic group. SCAD may also be associated with some other conditions. Herein, we present a 57 year-old man who developed SCAD concomitant with Trousseau's syndrome secondary to colon adenocarcinoma. (Cardiol J 2010; 17, 6: 625–627)

Key words: spontaneous coronary artery dissection, Trousseau's syndrome

Introduction

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome and sudden cardiac death [1]. SCAD has been described in young women during the peripartum period and in women using oral contraceptive pills [1]. Most cases of SCAD appear to be idiopathic, although dissections have been linked to Marfan's syndrome, atherosclerotic cardiovascular disease, blunt chest trauma, intense physical exercise, use of contraceptives, Kawasaki disease, systemic lupus erythematosus, and cocaine use [1]. Management of these patients has been controversial. Here, we present a 57 year-old man who developed SCAD concomitant with Trousseau's syndrome secondary to colon adenocarcinoma.

Case report

A 57 year-old man presented to our emergency department with chest pain, numbness, and coldness of the right arm of one week duration. He had no history of coronary artery disease, diabetes mellitus or hyperlipidemia. However, he had hypertension for five years and a 20 pack-year history of smoking. Medical history revealed two episodes of lower extremity deep vein thrombosis treated medically in the last ten months, in spite of regular warfarin therapy. He had also a history of weight loss of ten kilos in the last four months. International normalized ratio was 2.1 at presentation. Electrocardiogram showed pathologic q waves and deep s waves on precordial leads. Transthoracic echocardiography revealed hypokinesia of left ventricular anterior, septal, and apical walls with an ejection fraction of 45%. Concurrent biochemical markers were consistent with myocardial necrosis (TnI: 2.4 ng/dL, reference: 0–0.1 ng/mL) and complete blood count revealed mild anemia (Hb: 9.8 g/dL). Coronary angiography showed flap-like filling defect in the middle-third of the left anterior descending artery (Fig. 1) and in the right coronary artery after the acute marginal branch (Fig. 2). Cardiac positron emission tomography was performed after stabilization of the patient and revealed no viable myocardium. Beta-blocker, warfarin sodium, acetyl salicilic acid, statin and angiotensin converting

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Received: 30.09.2009 Accepted: 23.10.2009

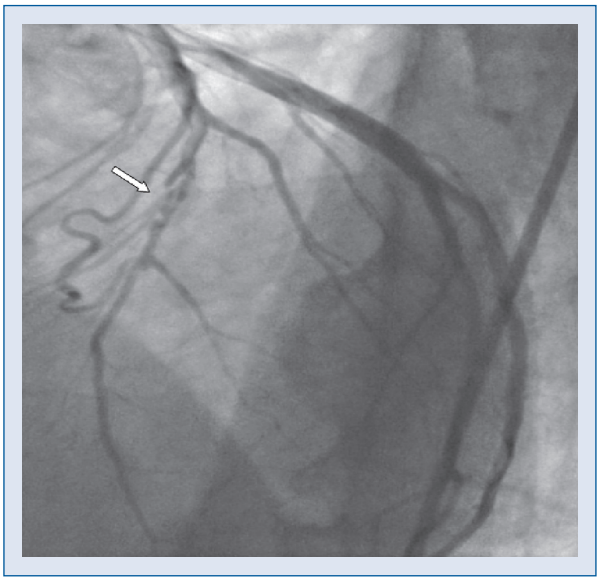

Figure 1. Coronary angiography showing flap-like filling defect in the middle third of the left anterior descending artery (arrow).

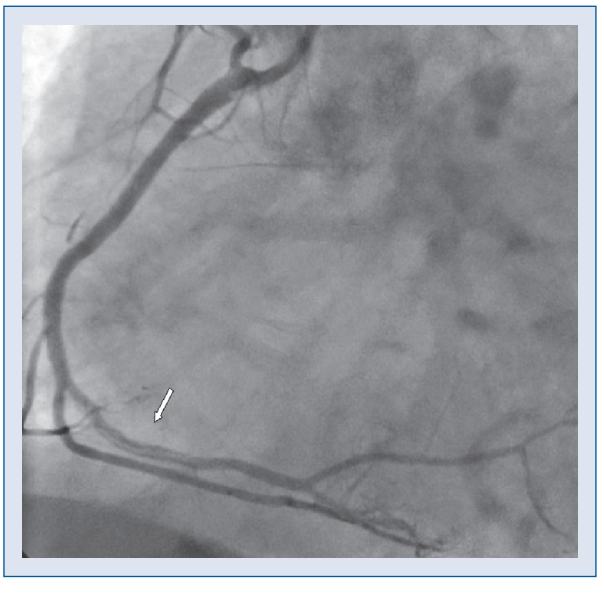

Figure 2. Dissection at right coronary artery after the acute marginal branch (arrow).

enzyme inhibitor therapies were chosen as a treatment option without percutaneous or surgical therapy because of non-viable myocardium and patient comorbidity. Due to the recurrent lower deep vein thrombosis, weight loss and low hemoglobin values, gastroenterology consultation was performed. Colonoscopy revealed colon adenocarcinoma. He was discharged with anti-anginal and anti-ischemic medication and referred to the oncology department. After three months, he died from pneumonia caused by febrile neutropenia secondary to chemotherapeutics.

Discussion

SCAD is a rare cause of myocardial infarction [1]. The incidence of SCAD has been estimated to range from 0.1% to 1.1% [1]. SCAD usually occurs as a consequence of hemorrhage within the outer third of the media or between media and the external elastic lamina of a coronary artery [2]. Expansion of the false lumen by further bleeding and separation of the dissected layers lead to compression of the true lumen, with subsequent myocardial ischemia or infarction [2]. Disruption and bleeding from vasa vasorum have been suggested as a possible mechanism. An underlying inflammatory process has been discussed by several authors [3]. In patients with atherosclerosis, plaque rupture that disrupts the intima–media junction with an intramural hematoma formation is believed to be causative. Additionally in women who develop dissection during pregnancy or puerperium, hormonal changes are thought to impair collagen synthesis, loosen the ground substance, and cause changes in the media of the coronary walls [1–3].

Clinically, SCAD may present with the entire spectrum of coronary syndromes, varying from unstable angina to myocardial infarction. The pattern and severity of presentation is primarily related to the vessel involved, the extent of dissection, its rate of development, and the presence of coronary artery disease [1].

SCAD has been described in young healthy women [4], among whom 25–30% were pregnant or in the peripartum period [4]. SCAD probably accounts for up to 30% of myocardial infarctions during pregnancy or during the early postpartum period [4]. It has also been described in the setting of atherosclerotic coronary artery disease [5], isolated fibromuscular dysplasia of the coronary arteries [6], hypertrophic cardiomyopathy [7], retching [8], oral contraceptive use [9], cocaine abuse [10], cyclosporine use [11], Marfan or Ehlers-Danlos syndrome [12], and sarcoidosis [13].

Finally, SCAD has also been described after physical exercise such as light or heavy aerobics, running, lifting heavy objects, snow shoveling, and swimming [14]. In rare cases, the occurrence of SCAD is a cause of acute myocardial infarction after heavy lifting in male patients who have coronary risk factors [14]. However, SCAD associated with Trousseau's syndrome has not been reported before.

The association between venous thrombosis and malignancy was first described by Trousseau in 1865 [15]. Trousseau's syndrome is a rare variant of venous thrombosis, characterized by a recurrent and migratory pattern [16]. It is usually associated with a tumour, generally of the gastrointestinal tract. Given the exuberance and recurrence of the symptoms in our patient, despite therapy with oral anticoagulants, and after excluding other causes of secondary venous thrombosis, the hypothesis of Trousseau's syndrome as a paraneoplasic event was raised [16].

SCAD predominantly occurs as single vessel disease. Left anterior descending artery is most often involved (in 75% of cases), followed by right coronary artery (RCA), the left main and circumflex artery [1–13]. Only a few cases of multiple vessel dissection have been reported [17]. Most patients with left coronary artery dissection sustain myocardial infarction (*vs* only 50% of patients with RCA dissection) [17].

Several treatment modalities (coronary artery bypass grafting, percutaneous transluminal coronary angioplasty and/or stenting) have been reported, with variable success [17]. Medical therapy alone in patients who completed their infarctions after dissections, and without residual ischemic symptoms, produced good long-term outcomes [17]. To the best of our knowledge, this is the first case reported with SCAD and Trousseau's syndrome. Although our patient presented with recent myocardial infarction, he was discharged with antianginal and anti-ischemic medication without percutaneous or surgical therapy, and referred to the oncology department. Stent implantation or coronary artery by-pass grafting was not considered because of short life expectancy and the stable cardiac condition of the patient.

Acknowledgements

The authors do not report any conflict of interest regarding this work.

References

1. Almeda FQ, Barkatullah S, Kavinsky CJ. Spontaneous coronary artery dissection. Clin Cardiol, 2004; 27: 377–380.
2. Mulvany NJ, Ranson DL, Pilbeam MC. Isolated dissection of the coronary artery: a postmortem study of seven cases. Pathology, 2001; 33: 307–311.
3. Robinowitz M, Virmani R, McAllister H. Spontaneous coronary artery dissection and eosiniphilic inflammation: A cause and effect relationship? Am J Med, 1982; 72: 923–928.
4. Roth A, Elkayam U. Acute myocardial infarction associated with pregnancy. Ann Intern Med, 1996; 125: 751–762.
5. Celik SK, Sagcan A, Altintig A et al. Primary spontaneous coronary artery dissections in atherosclerotic patients report of nine cases with review of the pertinent literature. Eur J Cardiothorac Surg, 2001; 20: 573–576.
6. Lie JT, Berg KK. Isolated fibromuscular dysplasia of the coronary arteries with spontaneous dissection and myocardial infarction. Hum Pathol, 1987; 18: 654–656.
7. Lette J, Gagnon A, Cerino M. Apical hypertrophic cardiomyopathy with spontaneous post partum coronary artery dissection. Can J Cardiol, 1989; 5: 311–314.
8. Velusamy M, Fisherkeller M, Keenan ME et al. Spontaneous coronary artery dissection in a young woman precipitated by retching. J Invasive Cardiol, 2002; 14: 198–201.
9. Azam MN, Roberts DH, Logan WF. Spontaneous coronary artery dissection associated with oral contraceptive use. Int J Cardiol, 1995; 48: 195–198.
10. Jaffe BD, Broderick TM, Leier CV. Cocaine-induced coronary--artery dissection. N Engl J Med, 1994; 330: 510–511.
11. Tsimikas S, Giordano FJ, Tarazi RY et al. Spontaneous coronary artery dissection in patients with renal transplantation. J Invasive Cardiol, 1999; 11: 316–321.
12. Bateman AC, Gallagher PJ, Vincenti AC. Sudden death from coronary artery dissection. J Clin Pathol, 1995; 48: 781–784.
13. Ehya H, Weitzner S. Postpartum dissecting aneurysm of coronary arteries in a patient with sarcoidosis. South Med J, 1980; 73: 87–88.
14. Cinzia C, Marco S, Simonetta P, Ornella L, Claudio C. Spontaneous coronary artery dissections. Cardiovasc Revascular Med, 2006; 7: 231–233.
15. Trousseau A. Plegmesia alba dolens. Lectures on clinical medicine. Delivered at the Hotel-Dieu, Paris 1865; 5: 281–322.
16. Candeias N, Lopez A, Costa L et al. Trousseau's syndrome as initial presentation of malignancy. Report of two clinical cases. Eur J Inter Med, 2003; Sl–S159: P109.
17. Mohamed HA, Eshawesh A, Habib N. Spontaneous coronary dissection. A case and review of the literature. Angiology, 2002; 53: 205–211.