CASE REPORT

Spontaneous coronary artery dissection in a patient with bacterial meningitis

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SUMMARY

A 40-year-old man was admitted to our hospital because of the acute onset of fever and headache, which were attributed to bacterial meningitis. Antibiotic treatment was initiated and his condition gradually improved. On day 5 after admission, immediately after masturbation, he developed abrupt onset of severe chest pain and cold sweat and the ECG suggested acute anterior myocardial infarction. Immediate coronary angiography revealed spontaneous dissection of the left anterior descending artery. After conservative management, his cardiac function improved. Acute coronary syndrome may be rarely caused by spontaneous coronary artery dissection. Sepsis was considered as a probable trigger for spontaneous coronary artery dissection, possibly through vascular damage from increased nitric oxide and sympathetic nervous over-activation.

BACKGROUND

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome identified in young patients, mostly pregnant women.¹ In addition to peripartum patients, there are other predisposing conditions for SCAD such as Marfan syndrome,² Ehlers-Danlos syndrome,³ fibromuscular dysplasia,⁴ vasculitis,⁵ exercise,^{6–8} oral contraceptives,⁹ cocaine use,¹⁰ chest wall trauma,¹¹ prolonged sneezing¹² and atherosclerotic coronary disease.¹³ Thus, functional or structural vessel wall abnormalities have been suggested in causes for SCAD, but to the best of our knowledge there have been no reports describing SCAD in patients with sepsis. We reported a case of a patient who developed SCAD immediately after masturbation during admission for bacterial meningitis.

Figure 1 ECG was obtained when the patient developed chest pain.

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Figure 2 Coronary angiography revealed dissection of the left anterior descending artery (the black arrow indicates the site of dissection).

CASE PRESENTATION

A 40-year-old man presented with a 4-day history of headache and fever. He had a medical history of bronchial asthma and atopic dermatitis. He had no history of ethanol drinking or smoking. The temperature was 40.6°C, the blood pressure 124/90 mm Hg and the pulse rate 110 bpm. He had no neck stiffness, but jolt accentuation of headache¹⁴ was positive. He had extensive cutaneous eczema over the upper extremities and neck. Cardiac examination was unremarkable. The lungs and abdomen were normal.

In laboratory data, the peripheral leucocyte count was 9400/ μ L (band 14%, segmented 71%, monocyte 7%, lymphocyte 8%). The cerebrospinal fluid analysis showed an opening pressure of 22 cm H₂O, cell count of 23/ μ L (mononuclear lymphocyte 9, polymorphonuclear leucocyte 14), protein 50 mg/dL and glucose 67 mg/dL. The Gram stain of cerebrospinal fluid showed no organisms and PCR tests for bacterial pathogens were not performed.

Owing to the severe clinical condition despite the negative bacterial findings for cerebrospinal fluid, bacterial meningitis was suspected and ceftriaxone (4 g/day) and vancomycin (2 g/day) were administered. The blood culture grew methicillin-sensitive *Staphylococcus aureus*, although the cerebrospinal fluid culture did not grow any organisms. After being admitted to the department of medicine, his condition improved and fever subsided. On day 5, immediately after masturbating in a toilet of the ward, he developed abrupt onset of severe chest pain and cold sweat. The blood pressure was 195/61, pulse rate 96 bpm and pulse oxymetry oxygen saturation of 98% while breathing ambient air. On examination, chest and heart were unchanged.

INVESTIGATIONS

ECG showed ST segment elevations in V1–V5, I, aVL, and ST segment depression in II, III, aVF (figure 1). The heart-type fatty acid-binding protein was positive. Since acute coronary syndrome was suspected, emergency coronary angiography was performed.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis before coronary angiography included atherosclerotic or vasospastic coronary diseases, SCAD, acute myocarditis and Takotsubo-type acute cardiomyopathy. Coronary angiography revealed a sharp-edged oblique defect in the left anterior descending (LAD) artery (figure 2). Intravascular ultrasound showed that there were false lumens occupying a large part of the vessel lumen, indicating coronary dissection (figure 3).

TREATMENT

Owing to the difficulty in inserting a guide-wire into the true lumen, angioplasty was not conducted and conservative management was chosen thereafter.

OUTCOME AND FOLLOW-UP

Five hours after he developed acute coronary syndrome, ventricular tachycardia associated with Torsade de Pointes was identified (figure 4) and treated by cardioversion. Serum potassium concentration was 2.7 mEq/L, and thus potassium and magnesium were added to the intravenous drip infusion. The peak

Figure 3 Intravascular ultrasound confirmed coronary artery dissection. D1, first diagonal branch; FL, false lumen; LAD, left anterior descending artery; TL, true lumen.

Figure 4 ECG telemetry showing Torsade de Pointes and ventricular tachycardia.

creatinine kinase was 1343 unit/L. The course under conservative management was uneventful and echocardiography showed only mild wall motion abnormality in the apex on the day of discharge (day 21).

DISCUSSION

Investigation of the epidemiology of SCAD¹⁵ indicated that it is a relatively rare disease causing acute coronary syndrome; compared to plaque rupture or vasospasm, it is more frequently identified in women than in men. The higher incidence among women of this disease may be due to the fact that many cases with SCAD occur in peripartum women. The minority of cases with SCAD are caused by connective tissue diseases, trauma, drugs and exercise.

Several exercises were considered as causes for SCAD. They included weightlifting and cycling. The pathophysiology of exercise-related SCAD may be multifactorial, including increased stress and shear forces exerted on the coronary endothelium leading to intimal tear with subsequent intramural haematoma and arterial dissection.¹⁰

Regarding the causes for SCAD in our patient, impairment of vascular integrity by increased vascular stress may have been induced by the increased level of nitric oxide from a systemic inflammatory response syndrome and sympathetic reflex discharge associated with bacterial meningitis.¹⁶ However, since sepsis was relatively mild in our patient and he was already in the recovery phase of the sepsis, this possible causative hypothesis is still awaiting further research.

As regards the masturbation process, penile erection also involves nitric oxide, while the ejaculation process requires a sympathetic reflex discharge.¹⁷ Although ejaculation may lead to increased stress to the vascular endothelium in a way similar to heavy exercise regarding sympathetic nervous over-activation, this link is still speculative based only on our single case report.

Further, comedications might have influenced the development of SCAD in our patient. β -Blockers might have caused vasoconstriction by unopposed α -receptor stimulation and heparin anticoagulation and antiplatelets might also have led to the risk for SCAD. In terms of bacterial entry in our patient, extensive eczema over the upper extremities and neck might be a potential source of *S aureus* bacteremia.

In our patient, there were no other predisposing factors for SCAD, such as Marfan syndrome, Ehlers-Danlos syndrome, vasculitis, cocaine use, chest wall trauma, prolonged sneezing or atherosclerotic coronary disease, although the possibility of Ehlers-Danlos syndrome was not completely ruled out because we did not perform a test for transforming growth factor- β and other related molecules. Therefore, causes for SCAD in our patient could be considered as related to meningitis-associated sepsis.

As regards the therapeutic approach to SCAD in our patient, conservative management was chosen with fortunately good

recovery. However, coronary artery bypass surgery might have been considered as an option for treating an extensive LAD dissection if an elective follow-up coronary angiogram had been performed before the patient was discharged.

To the best of our knowledge, there have been no reports of cases with SCAD associated with sepsis. Acute coronary syndrome induced by SCAD should be considered in patients with acute onset of chest pain and ischaemic change in ECG during the course of sepsis.

Learning points

- Rare causes for acute coronary syndrome include spontaneous coronary artery dissection.
- Spontaneous coronary artery dissection can be induced by Marfan syndrome, Ehlers-Danlos syndrome, vasculitis, oral contraceptives, cocaine use, chest wall trauma, prolonged sneezing or atherosclerotic coronary disease.
- Sepsis could also be related to spontaneous coronary artery dissection, possibly through vascular damage from increased nitric oxide and sympathetic nervous activation.

Contributors KK, YT, SW and YT cared for the patient and also contributed significantly to the writing of the manuscript.

Competing interests None.

Patient consent Obtained.

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