Spontaneous coronary artery dissection (SCAD) is a rare cause of myocardial infarction (MI). We report a 66-year-old Japanese man who had had an anterior wall MI caused by SCAD of the left anterior descending coronary artery, developed left ventricular aneurysm 5 years later, with depressed left ventricular function and thrombus observed on echocardiography. Left endoventricular circular patch plasty according to Dor’s technique was performed without coronary artery bypass grafting, because of the absence of significant coronary artery stenosis on the preoperative coronary angiogram. The clinical course of SCAD in the late phase is generally favorable, but because the prognosis of SCAD is uncertain, patients with SCAD should be carefully followed. (Circ J 2002; 66: 972–973)

Key Words: Aneurysm; Dor’s procedure; Left Ventricle; Spontaneous coronary artery dissection

Spontaneous coronary artery dissection (SCAD) is a rare cause of myocardial infarction (MI). A 66-year-old Japanese man, who had had an anterior wall MI caused by SCAD of the left anterior descending (LAD) coronary artery and who later developed a left ventricular aneurysm (LVA) despite the dissection having completely disappeared.

Case Report

A 61-year-old Japanese man was admitted to hospital after an anteroseptal myocardial infarction (MI), which had occurred at rest. The coronary angiogram taken at that time revealed SCAD of the LAD without significant stenosis (Fig 1), and catheter intervention was not performed. He was discharged after rehabilitation, and was followed with medication at the outpatient clinic. Five years later, echocardiography revealed a mural thrombus at the apex of the left ventricle (LV) accompanied by an aneurysm. Anticoagulant therapy with warfarin sodium was started, but the LV ejection fraction (EF) decreased and he was admitted to hospital for surgical treatment of the aneurysm and thrombus.

Physical examination revealed a healthy man, 166 cm tall and weighing 69 kg. His blood pressure was 106/74 mmHg, and his pulse was 59 beats/min and regular. There were no stigma of connective tissue disorder. The electrocardiogram (ECG) confirmed the previous anteroseptal MI.

Although the preoperative coronary angiogram (CAG) did not show any stenosis in the LAD and the dissection had completely healed, the left ventriculogram (LVG) showed LVA and a dilated LV with an EF of 21.3% (Fig 2).

During surgery, we observed an old thrombus (3 g) in the...
LV, and performed left endoventricular circular patch plasty (EVCPP) according to Dor’s technique for the LVA. However, we did not perform coronary artery bypass grafting (CABG) because of the absence of significant stenosis. His postoperative course was uneventful and the improvement in LV function was confirmed by echocardiography and LVG (Fig 3).

Discussion

SCAD is a rare condition that was first described in 1931 by Pretty, and at least 140 cases have been described in the English literature. However, most reported cases were diagnosed on postmortem examination. Various causes of myocardial ischemia and sudden cardiac death have been increasingly reported with the popularization of CAG and were diagnosed on postmortem examination. Various causes of myocardial ischemia and sudden cardiac death have been increasingly reported with the popularization of CAG and advances in the imaging equipment, but nevertheless the etiology of SCAD remains unclear.

DeMaio classified SCAD into 3 groups from a clinical perspective: those with atherosclerotic coronary artery disease, a postpartum group, and those without any identifiable predisposing factor. The changes in connective tissue that occur during pregnancy are probably caused by the high estrogen level; but SCAD also occurs in men, leading to the necessity of formulating a multifactorial theory of pathogenesis. Angitis has been implicated in the pathogenesis and Kearney et al reported autopsy cases with eosinophilic infiltration of the adventitia of the involved coronary artery. They postulated that proteins, isolated from the granules of eosinophils, might have damaged the collagen, elastin or smooth muscle of the coronary artery, resulting in dissection.

The clinical presentation of SCAD is somewhat different from that of patients with atherosclerotic coronary artery disease. The average age is younger and risk factors may be absent. In addition, patients with SCAD often have chest pain that is weaker and of longer duration (days). Therefore, SCAD should always be considered in the differential diagnosis of any young person sustaining acute precordial pain, especially postpartum women.

The prognosis of patients with SCAD varies widely among the reports. There have been case reports of conservative therapy from Japan; Osaki et al reported a case in which the dissection had not changed on follow-up CAG after 10 years and Oka et al reported a case in which the dissection had not changed on follow-up CAG and advances in the imaging equipment, but nevertheless the etiology of SCAD remains unclear.

DeMaio classified SCAD into 3 groups from a clinical perspective: those with atherosclerotic coronary artery disease, a postpartum group, and those without any identifiable predisposing factor. The changes in connective tissue that occur during pregnancy are probably caused by the high estrogen level; but SCAD also occurs in men, leading to the necessity of formulating a multifactorial theory of pathogenesis. Angitis has been implicated in the pathogenesis and Kearney et al reported autopsy cases with eosinophilic infiltration of the adventitia of the involved coronary artery. They postulated that proteins, isolated from the granules of eosinophils, might have damaged the collagen, elastin or smooth muscle of the coronary artery, resulting in dissection.

Thus, considering these results, we suggest the indication for intervention in this condition is the same as for ischemic heart disease from athelosclerosis, not only in the acute phase but also in the chronic phase. Our patient had been carefully followed up conservatively for 7 years and we only performed EVCPP for indicated for the LVA and a mural thrombus, but did not perform CABG because this dissection had completely healed and there was no preoperative stenosis in the coronary artery. To our knowledge, there have been no other reports in which complete disappearance of SCAD was confirmed. Although the present patient is doing well 1 year postoperatively, regular follow-up is necessary because the outcome of SCAD is still uncertain.

References