Primary spontaneous coronary artery dissections in atherosclerotic patients. Report of nine cases with review of the pertinent literature

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Summary

Primary spontaneous coronary artery dissection is one of the rare causes of acute myocardial infarction. Previous studies reports 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 severity related to extent and development rate of dissection. In the last 2 years, nine non-pregnant primary spontaneous coronary artery dissection cases were found in coronary angiography among 3750 patients prediagnosed as coronary artery disease. The cases were presented and discussed with review of the pertinent literature. © 2001 Elsevier Science B.V. All rights reserved.

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1. Patients and methods

Nine patients with primary spontaneous coronary artery dissections (PSCAD) were diagnosed and treated among 3750 patients who underwent coronary angiography in the last 2 years. Seven of them were male and two were female, and mean age of the patients was 55.7 years (range 45–73 years). All patients had one or more than one risk factors for coronary artery disease such as chronic smoking, hyperlipidemia, diabetes, hypertension and obesity (Table 1). Coronary vasodilators, aspirin, oral antidiabetics and antihypertensive agents were the drugs that patients had been using. Clinical presentations of the patients were different at the admission to hospital: seven patients had unstable (UAP) and two patients had stable angina pectoris (SAP). One patient with UAP was also in congestive heart failure due to ischemic cardiomyopathy. Four patients had suffered acute myocardial infarction (AMI) and three of them got thrombolytic treatment before coronary angiography was done. In coronary angiography, dissections were located in LAD, RCA and LMCA in five, two and two cases, respectively. (Figs. 1 and 2) Atherosclerotic coronary artery lesions were accompanying to dissections in all cases in dissected and non-dissected coronary arteries.

Seven patients were operated by coronary bypass surgery with conventional cardiopulmonary bypass and coronary artery stenting were applied to one patient (Fig. 3). Left internal mammarian artery for all LAD arteries and saphenous vein grafts for other vessel bypasses were used. The patient with dissection of LMCA could not be operated due to congestive heart failure and he was medically treated.

2. Results

Eight patients other than the medically treated one were symptom free and had negative stress tests at the 6th and 12th months after coronary bypass surgery and stenting. Control angiography of the stented patient at the 12th month after intervention revealed full patency of coronary artery stent and no recurrent dissections in the same and other coronary arteries (Fig. 3).

3. Discussion

Coronary artery dissection can occur iatrogenically as a complication of cardiac catheterization, coronary angiography, coronary angioplasty, coronary artery surgery and dissections of ascending aorta. Incidence of primary spontaneous coronary artery dissection is rare but it has been accepted an increasing reason of AMI in young woman having low risk for atherosclerotic coronary artery disease.
Pretty described the first case in 1931 and up to date more than 150 cases were reported in the literature. There are some characteristic features of PSCAD: (1) 75% of all cases are woman, (2) pregnant women in the peripartum period make one-third of patient population, (3) it is the disease of young people and mean age of onset in 35 years, (4) clinical presentation is related to extent and arterial location of dissection, (5) dissection can be seen in any coronary artery, (6) LAD dissection is more in woman while RCA involvement is more in men, (7) in living cases, dissection of RCA is more common than LAD dissection [2].

In spite of these characteristics of PSCAD collected in the literature, there was no pregnancy and all patients had not only PSCAD but also coronary atherosclerosis in this group. This might be the reason of differences in gender (M:7, F:2) and ages (mean age: 55.7 years) of patients in this group.

Usual location of dissection is in the proximal portion of a single coronary artery but simultaneous and recurrent involvement of multiple arteries can also be seen [3]. Coronary angiography of the patients did not show multiple coronary artery involvement and more than one dissections in the same vessel, but all dissected coronary arteries had atherosclerotic plaques, as well.

PSCAD can present itself as SAP and UAP, AMI and sudden death, and mostly it is diagnosed at autopsy [4]. Clinics of PSCAD is related with the coronary artery dissected, location and extension of dissection in the vessel. None of the patients were admitted to hospital with acute myocardial infarction. Four patients had prior myocardial infarction, and infarcted area of three of them in ECG (two anterior and one inferior MI) was fitting to artery dissected (two LAD and one RCA dissection), but another patient with chronic inferior MI had dissection in LMCA.

Coronary artery spasm accompaniment to dissection may worsen myocardial damage by causing the extension of dissection [5]. We did not observe coronary artery spasm during coronary angiography nor made any test that could induce coronary vasospasm, besides all patients got nitro-
glycerine infusion until therapeutic interventions. Rupture of dissected coronary artery can lead to pericardial tamponade or formation of ventricular pseudoaneurysm [6]. Before coronary angiography, all patients were examined by echocardiography and no pericardial effusion and ventricular aneurysm were detected.

Fate of PSCAD in living patients when untreated can be in two opposite ways: spontaneous regression and persistence of dissection flaps as seen in coronary angiographies made years after diagnosis [7,8]. Mortality of PSCAD is pretty high. In their series, Koller et al. found 48.8% mortality and 28% sudden death as the first sign of disease in the review of a patient group consisting of 42 peripartum patients [9]. All our patients were alive at the 1st year of their treatment and free of coronary disease symptoms except the patient medically treated that still had complaints of heart failure. The zero mortality rate among nine cases may be due to absence of pregnant patients, and could consider that PSCAD have a more benign course when developed in atherosclerotic basis.

Although PSCAD is said to be seen especially in women in peripartum period it can be found in various kinds of disease. Immune system disease like SLE, type IV Ehler Danlos and Marfan syndrome, Kawasaki disease and rheumatoid arthritis with coronary arteritis can cause PSCAD [10–13]. Dissection may exist after vigorous exercises and blunt chest traumas [14]. Atherosclerosis, coronary vasospasm and coronary artery ectasy were also thought as the main reasons for dissection [15]. In two cases, apical hypertrophic cardiomyopathy and cryoglobulinemia related with HCV were blamed for the development of PSCAD [16]. Oral contraceptives, cocaine and cyclosporin were found as the common drugs that patients with PSCAD had been using [17–19].

In this group, patients did not have any coexisting disease that could cause dissection except atherosclerosis. Four patients had chronic myocardial infarction and all had one or more risk factors of atherosclerotic coronary artery disease (Table 1). Inflammatory markers were not measured due to negative history and physical examination of patients for immune system disease. We think that etiology of PSCAD can be divided primarily in two groups: atherosclerotic and non-atherosclerotic coronary diseases. Importance of etiology of coronary dissection is its direct relation with prognosis of disease. Patients with atherosclerotic dissection could have less ratio of mortality that might be due to improved collateral circulation developed in chronic atherosclerotic basis.

Local inflammatory process rather than primary vasculitis is thought as the main reason of the pathogenesis of PSCAD [20]. Differences in content of mucopolisaccharides and proteins, and degeneration of collagen in the media layer of arteries were held responsible for dissection in pregnant women and women in postpartum period. Also, tissue fibroblast culture of postpartum PSCAD patients revealed decreased collagen synthesis.

In autopsy studies, accumulation of eosinophilic infiltrates having collagenetic and cytotoxic activity in adventitia and cystic medial necrosis were showed histologically in the 25–40% of reported PSCAD cases, and they were counted as the actual pathogenetic factor of dissection.

In atherosclerotic arteries, increased density of vaso vasorum due to atherosclerotic plaque may cause bleeding and rupture of nidus that can lead to dissection of adventitia from media and subsequent rupture of intima or primary event is an intimal tear progressing into media by the force of arterial pressure [21].

Coronary angiography is the standard tool for diagnosis of disease, and false lumen is pathognomonic for dissection.

Treatment of PSCAD is controversial, and depends on the location and extension of dissection, leading or coexisting diseases and clinical situation of patients. Coronary bypass surgery, PTCA, coronary artery stenting, thrombolytic agents and medical therapy are the treatments carried out for PSCAD. Nitrates and calcium channel blocking agents may prevent extension of dissection by reducing coronary spasm, and make patients gain time for the main therapy. Chest pain of seven patients with unstable angina pectoris that got nitroglycerine and standard heparin intravenously was mostly resolved before coronary angiography. Successful immunosuppressive therapy such as cytoxan with prednisolon was experienced in non-atherosclerotic postpartum patients that had surgical contraindication [8]. Outcome of medical treatment of PSCAD is controversial. Symptom free patients without recurrent dissections and subsequent myocardial infarctions, intractable angina pectoris and even sudden deaths were reported in follow-up of medically treated patients. Thrombolytic agent use is also a discussed issue. Some studies reported extension of dissection due to bleeding in the vessel wall would be increased by throm-
bolistic therapy [22]. Three of four patients in this series with chronic myocardial infarction had thrombolytic treatment in the acute phase. Thrombolytic treatment does not cure dissection, but it can prevent extension of dissection by holding the actual coronary lumen open. We think it would be better to delay thrombolytic treatment until coronary angiography, but thrombolytic therapy will be beneficial for PSCAD patients with AMI treated in centers does not have coronary angiography. Except in pregnant patients, primary PTCA and coronary artery stenting is being successfully applied to dissections even in left main coronary artery, but risk of coronary perforation by guidewire and angioplasty balloon is markedly more in a damaged and weakened artery. The authors think that PTCA could be applied to dissections having limited extension up to 1 cm and causing narrowing of lumen less than 50%. We suggest that coronary artery must be stented after PTCA to prevent extension and recurrence of dissection. Coronary bypass surgery with or without cardiopulmonary bypass is the most preferred treatment of PSCAD if patients do not have any contraindication for anesthesia and surgery. Indications and techniques of surgery for PSCAD are the same as for patients with isolated atherosclerotic coronary disease. Pregnant patients with PSCAD can be safely operated by off-pump coronary artery bypass surgery. We used our standard technique for distal anastomoses by starting the first stitch from just off the angle of graft and continued suture line beyond the proximal angle of arteriotomy. All dissections we met at the operations were lying through the epicardial half-circle of coronary arteries, for this reason arteries proximal to anastomoses were not ligated, and full layer anastomoses were carefully made by interrupting the continuity of dissection proximally and distally to anastomoses.

In conclusion, we think that PSCAD is not only the disease of middle aged women that do not carry any known risk factors but can be seen in middle and older aged males having risk factors for atherosclerotic coronary artery disease, as well. Prognosis of PSCAD may be more favorable in atherosclerotic patients than non-atherosclerotics due to improved collateral circulation in chronic coronary artery atherosclerosis.

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