Spontaneous coronary artery dissection is a rare condition that may produce severe myocardial ischemia. The growth of indications for cardiac catheterization have led to an increment in the number of cases identified in patients with acute coronary syndromes. Because the therapeutic approach and prognosis are uncertain, doubts often arise regarding the optimal management of these patients. We describe here the clinical and angiographic characteristics of 7 patients with spontaneous coronary artery dissection, as well as treatment and follow-up.

**Key words:** Myocardial ischaemia. Coronary angiography. Revascularization.

Full English text available at: www.revespcardiol.org

**INTRODUCTION**

Spontaneous dissection of the coronary artery is an uncommon condition that can lead to myocardial ischemia. In the earliest series, the usual clinical presentation was sudden death, with the diagnosis made at autopsy. In recent years, however, a number of authors have reported spontaneous coronary dissection in cases of acute coronary syndrome. Various therapeutic strategies have been proposed, from medical treatment alone to surgical or percutaneous revascularization, but standard therapy has not yet been established. Thus, the interventional cardiologist may face a dilemma when confronted with this condition, which has an uncertain prognosis and a high risk of associated ischemic events.

In this study we describe the clinical and angiographic characteristics, treatment, and clinical course in 7 patients with spontaneous coronary artery dissection diagnosed at our center.

**PATIENTS AND METHOD**

Among the 21 000 diagnostic coronary angiographies performed in our center from January 1989 to March 2003, a total of 7 patients with spontaneous coronary artery dissection were identified. Coronary dissection was defined as a double lumen within the artery separated by a radiolucent line corresponding to the intimal flap. The other associated signs assessed were retention of contrast medium in the false lumen, stenosis, and phase changes in coronary artery diameter. In addition, we carefully determined whether the abnormal images might be false dissections resulting from thrombi or filling defects caused by slow flow or a small amount of contrast material in the coronary artery. To establish the diagnosis of spontaneous dissection, other causes of coronary dissection (chest trauma, aortic
dissection, iatrogenic causes or prior surgery) were ruled out. Patients underwent periodic follow-up on an outpatient basis.

RESULTS

All 7 patients were women, with a mean age of 48±8 years. Table 1 summarizes the clinical characteristics, treatment and follow-up of the patients studied, and Table 2 shows the angiographic characteristics of the spontaneous coronary artery dissections.

The most frequent cardiovascular risk factor in the series was smoking, recorded in 5 patients. All except one patient had at least one risk factor. In one patient the first sign of coronary artery dissection was chest pain followed by ventricular fibrillation, whereas the remaining six patients presented with acute myocardial infarction (2 non-Q wave and 4 Q wave). Complications in these patients included postinfarction angina in 4, residual ischemia detected on the pre-discharge exercise test in one, and ineffective thrombolysis in one patient who was referred for rescue angioplasty.

The diagnosis was made on the basis of coronary angiography findings, which showed characteristic features of coronary dissection, including a double lumen and contrast retention in 7 patients, signs of true lumen compression with stenosis in 5 patients, total occlusion in 4 patients, and phase changes in artery diameter in 3 patients. Only 1 patient had atherosclerotic plaques in other locations. The dissections were extensive, affecting several coronary arteries in 3 cases and a single vessel in 4 cases.

None of the patients had a clinical history that would suggest secondary dissection (trauma, prior surgery, aortic dissection, etc.). The left main coronary artery was affected in 2 patients and in both of them the onset of symptoms occurred before diagnostic catheterization. The coronary angiography procedure was carefully reviewed to rule out iatrogenic causes during catheterization. On the basis of these factors, coronary dissection was considered to be primary or spontaneous in all the patients.

Percutaneous treatment was used in the 4 patients with dissections in a single vessel. One dissection required placement of 4 stents to completely seal the lesion (Figure 1), one resolved with one stent, and one with 2 stents; all involved long segments of the vessels. In one patient percutaneous treatment was attempted for a dissection affecting the medial and distal segments of the left anterior descending (LAD) artery. However, after insertion of a 0.014-inch guidewire and initial balloon inflation, proximal progression of the dissection to the LAD ostium was visualized and the patient was referred for surgery. The 3 patients with extensive dissections underwent
surgical treatment (Figure 2). Dissection was confirmed in all 3, coronary artery bypass grafting was performed, and the dissected media was sutured to the adventitia of the vessel.

Clinical follow-up lasted from 2 months to 9 years. The clinical course was satisfactory in all except one patient treated with stent implantation, who had occlusive stent restenosis (Figure 1).

DISCUSSION

Spontaneous coronary artery dissection is a rare cause of acute coronary syndrome. The real incidence of this condition is unknown; reported rates vary from 1 to 2.4 per 1000.\textsuperscript{3,7} The condition seems to affect young women most often\textsuperscript{1,9} although all the patients in our series were middle-aged women and no case was related to imminent labor or recent delivery.\textsuperscript{9} Furthermore, none of the patients had a history of medication or toxic substance consumption, other related conditions,\textsuperscript{15} or any factors that might substantiate a secondary dissection.

The cause of spontaneous coronary dissection is uncertain; histologic studies have cited primary vasculitis...
as the suggested origin.^{15} Spontaneous dissection can sometimes occur with the rupture of atherosclerotic plaques.^{4} An intimal flap may be present in up to 40% of complicated coronary plaques, but it is usually localized and associated with lumen irregularities, ulcerations, aneurysms and thrombotic material,^{17} which gives the lesion the appearance of a complex plaque. Such features were not seen in any of our patients. Therefore, we consider this cause improbable, even though all our patients had at least one cardiovascular risk factor and some had mild coronary lesions in other locations. Regardless of the mechanism that triggered the intimal disruption, an intramural hematoma would be produced and then progress along the artery, leading to arterial dissection.^{3} The hematoma could compress the true lumen, and this would explain the high incidence of angiographic stenoses and vessel diameter phase variations seen in our patients.

Formerly, spontaneous dissection was mainly diagnosed at autopsy in cases of sudden death.^{1} In recent years, however, a number of cases have been reported in patients with acute coronary symptoms.^{2,9} This may be attributable to the extended availability of cardiac catheterization and the increasing indication for its use in these patients.

Spontaneous coronary artery dissection is diagnosed in life by coronary angiography, a technique that allows visualization of the characteristic signs of this condition. All our patients presented a double lumen in these patients. The differential diagnosis includes other situations in which intraluminal filling defects are visualized, such as the presence of intracoronary thrombi, slow coronary flow, or complicated atherosclerotic plaques. Some authors have used intracoronary ultrasound to confirm the diagnosis or to determine proper guidewire positioning before stent placement.^{15} Intracoronary ultrasound was not used in our patients; the angiographic images were diagnostic and we avoided manipulations that could worsen the patient’s condition. To confirm cannulation of the correct lumen in patients receiving percutaneous treatment, we assessed certain indirect data, such as guidewire progression to the distal segment without difficulty or through lateral branches, or direct data obtained by contrast injection through a probe or coaxial balloon catheter.

Medical treatment alone, applied on the basis of certain observations, has proved to be successful in some studies, with spontaneous sealing of the dissections and no cardiovascular events at long-term follow up.^{8-11} The patients in our series were clinically unstable to some degree, and for this reason a conservative approach was not taken. Surgery has been used mainly in cases where a large anatomical territory is at risk, was the case of the 4 surgical patients in this study.

Percutaneous treatment has also been reported previously^{3} and has become widespread since the availability of stents.^{4,13-16} It is appropriate for many cases and is particularly indicated in patients with evolving acute myocardial infarction. Several considerations should be taken into account when using percutaneous therapy: a) It is essential to assure that the guidewire is advancing in the true lumen. Direct signs can be used for this purpose or indirect signs evaluated by experienced operators. b) When the stenosis is not fixed, pre-dilation with balloon angioplasty should be avoided because of the risk of worsening the dissection. In these cases direct stenting is preferable to seal the dissection. c) Some authors advocate stent placement only at the dissection entry point, whereas others propose sealing the entire dissection to avoid distal progression.^{4,15} d) As in the case of atherosclerotic lesions treated with stent placement, there is a risk of acute or subacute thrombosis, particularly when very long segments are involved. e) Lastly, even though the patient may have no arteriosclerosis, there is always a risk of restenosis.

REFERENCES


