Heart Vessels (1996) 11: 165-168



A rare indication for stenting: Persistent coronary artery spasm

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Summary. A 34-year-old man presenting with angina both at rest and on exertion was investigated. He developed severe ST segment elevation and a brief period of ventricular tachycardia during an exercise tolerance test. On coronary angiography, 60% fixed luminal narrowing was observed in the proximal left anterior descending coronary artery and a severe spasm developed at this site, leading to temporary total occlusion of the vessel. Successful coronary angioplasty (PTCA) was performed on this lesion, with a residual 15% narrowing. However, the patient had a recurrence of angina 3 weeks later, despite being administered high doses of nitrate and calcium antagonist. During control angiography, the lesion severity was unchanged, but spasm developed again following contrast injection. At this time, a Palmaz-Schatz stent was implanted. Calcium antagonist, nitrate, Ticlopidine and low molecular weight heparin therapy was started. There was no recurrence of symptoms during a 3-month follow-up. The exercise tolerance test, and myocardial perfusion scintigraphy findings were normal and the stent was patent without restenosis at the end of the 3-month follow-up. Intracoronary stent implantation for persistent coronary spasm refractory to conventional medical therapy can be considered a feasible and attractive treatment modality for the control of symptoms.

Key words: Variant angina – Coronary stent

Introduction

Coronary spasm and subsequent variant angina, described in 1959 by Prinzmetal et al., is an important presentation in the clinical spectrum of coronary artery

Received February 1, 1996; accepted March 1, 1996

disease [1]. Coronary angiographic findings in patients with variant angina range from normal coronary arteries to severe multivessel disease. Coronary artery spasm, presenting either with normal vascular anatomy or superimposed on atherosclerotic plaque, is the main pathogenetic mechanism in the development of variant angina.

Nitrates and calcium channel blockers in high dosages and in combination are usually effective in the management of patients with coronary artery spasm. Coronary angioplasty (PTCA) and coronary bypass surgery (CABG) are also alternative treatment modalities in patients refractory to medical treatment, in whom spasm is usually superimposed on a fixed coronary lesion, as development of a spasm may lead to complete obstruction of the vessel, even in the presence of a mild lesion.

After considering the above-mentioned facts and following vigorous medical treatment and PTCA, we finally implanted an intracoronary stent in a young patient with a persistent left anterior descending artery (LAD) spasm.

Case report

A 34-year-old man presenting with angina, both at rest and on exertion, was referred to our clinic for coronary angiography. An exercise tolerance test (ETT) had been carried out at another institution, 2 weeks before this admission. During ETT, the patient developed ST segment elevation and brief ventricular tachycardia attacks with chest pain (Fig. 1). Although he was put on high-dose Ca antagonist and nitrate therapy (diltiazem 240 mg/day, nitrate 60 mg/day) the anginal attacks continued. Coronary angiography at our institution revealed a mild-to-moderate degree proximal LAD lesion (60% luminal diameter narrowing) with normal right coronary and left circumflex arteries. The lesion was

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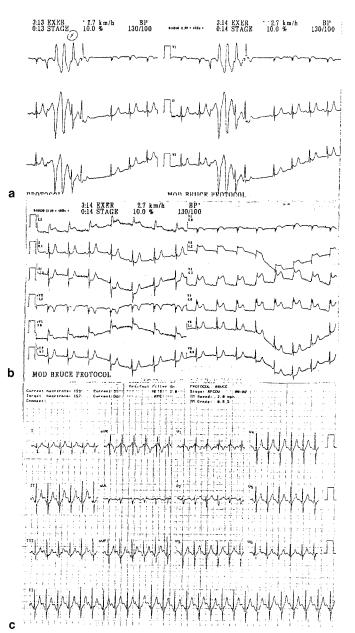


Fig. 1. a Short episodes of ventricular tachycardia during the exercise test. **b** ST segment elevation during exercise. **c** Normal exercise test result at 3-month follow-up

evaluated carefully in multiple orthogonal views, with particular regard to the presence of a thrombus. Left ventricular ejection fraction and wall motion was normal (EF, 65%). However, following the second contrast injection to the left coronary system, a severe spasm developed in the proximal LAD at the level of the organic lesion, compromising the coronary flow to TIMI 0 grade (Fig. 2a,b,c). The spasm was relieved by intracoronary nitrate injection. Considering the insufficiency of the high-dose Ca antagonist and nitrate combination therapy and, believing that the reduction or elimination of this narrowing might relieve the focal spasm and symptoms, we decided to perform PTCA on this lesion. The stenosis rate was reduced to 15%. The patient was discharged 2 days later and was again administered high-dose Ca antagonist, nitrate, aspirin, and dipyridamole. He did not have any chest pain for the following 3 weeks, but angina at rest and on exertion subsequently recurred. On control angiography, the vessel was seen to be patent and the lesion severity had not changed. However, following the first two injections, severe coronary spasm developed again at the same site. Significant hypokinesia was observed in the anterior wall and the left ventricular ejection fraction decreased to 45%. At this time, we decided to implant a coronary stent to prevent the persistent coronary spasm and keep the vessel patent. A 3-mm Palmaz-Schatz stent was deployed successfully, using a 3-mm balloon dilatation catheter. High-dose Ca antagonist, nitrate, aspirin, Ticlopidine, and low molecular weight heparin was started. The patient was discharged 7 days after implantation without any chest pain and EKG changes. During a 3-month follow-up period, there were no complaints or EKG changes. At the end of the follow-up period, an exercise tolerance test finding was negative (Fig. 1). Myocardial perfusion scintigraphy with TI 201 revealed no ischemia. Control coronary angiography demonstrated normal left ventricular function and a patent coronary stent without narrowing (Fig. 2d,e,f).

Discussion

Variant coronary angina is caused by severe coronary spasm. Although the cause of the coronary spasm is not clear, it is usually intimately associated with organic lesions and can be reproduced pharmacologically [2].

Serious ventricular arrhythmias can also be induced by episodes of coronary spasm and can be fatal [3]. Sudden death has been reported in 2%–16% of patients with variant angina [4]. The absence of a severe fixed organic stenosis may increase the likelihood of fatal arrhythmias developing due to acute ischemia, since the protective effect of collateral development from chronic myocardial ischemia may not be seen in these patients [3]. Abrupt deterioration of left ventricular function during recurrent episodes of coronary spasm was another consideration in our making the decision for stent implantation in this patient.

Nitrates and Ca antagonists at high dosages are used routinely in such patients. Different Ca antagonists can be combined to control coronary spasm [5]. Coronary spasm may be refractory to medical treatment, as illustrated by our patient. A second Ca antagonist, nifedipine, was added to the therapy but was stopped as the patient developed severe hypotension. In patients

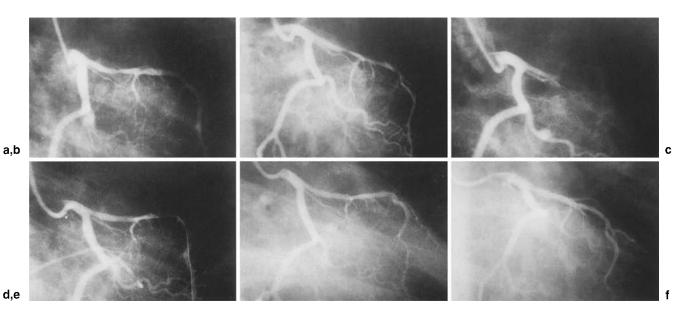


Fig. 2. a Proximal left anterior descending artery (LAD) lesion at first injection. b Beginning of spasm at lesion. c Worsening of spasm and complete occlusion of LAD. d Ap-

with moderate fixed coronary lesions and superimposed spasm, coronary angioplasty can be performed, since spasm may lead to complete obstruction of the vessel in the presence of a subcritical lesion. Subsequent reduction of stenosis by PTCA may relieve the symptoms. Although the incidence of acute complications related to PTCA may not be higher in patients with variant angina than in other patients undergoing PTCA, the clinical and angiographic recurrence rate is higher in patients with variant angina [6-9]. Repetitive coronary artery spasm may play a role in the development of coronary atherosclerosis [10, 11]. Coronary bypass surgery has also been performed in this group of patients. Similarly, following bypass grafting, coronary spasm may recur, even in the presence of patent grafts, and the long-term clinical benefit is less satisfactory [12].

To our knowledge, this is the first report of Palmaz-Schatz stent implantation for medically refractory coronary spasm. Lopez et al. [13] recently reported Gianturco-Roubin stent implantation for a coronary spasm refractory to medical treatment. Although there was a 60% renarrowing in the stented portion at 3month follow-up, the patient remained asymptomatic and exercise myocardial perfusion scintigraphy findings were normal. In our patient, stent patency without restenosis was present 3 months following the procedure. The patient remains free of symptoms with normal exercise test and myocardial perfusion scintigraphy findings.

In conclusion, intracoronary stent implantation for persistent coronary spasm that is refractory to medical treatment may be a feasible alternative treatment modality to maintain coronary patency. Further evaluation pearance of proximal LAD after coronary angioplasty. e Appearance of proximal LAD after stent implantation. f Patent stented LAD segment at follow-up

of this treatment strategy in a larger group of patients appears warranted.

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