

VIDEO SESSION

Multivessel Disease in a Patient Presenting With ST-Elevation Myocardial Infarction

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INTRODUCTION

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Patients with multivessel coronary artery disease are common among patients presenting with ST elevation myocardial infarction (STEMI). The way to treat the rest of the lesions after treating the culprit lesion is not well defined yet. In this article we present a patient with inferior STEMI, who had also an ostial left anterior descending (LAD) coronary artery stenosis.

CASE REPORT

KEY WORDS: *myocardial infarction; percutaneous coronary intervention; primary angioplasty; multivessel coronary disease*

A 43-year-old patient was admitted to the emergency department of our hospital due to chest pain from two hours earlier. The patient had hypercholesterolemia treated with 20 mg of atorvastatin daily. The electrocardiogram showed ST segment elevation in leads II, III, AVF, without right ventricular involvement. The patient was still in pain, but clinically stable and his blood pressure was 120/70 mmHg. After receiving loading doses of clopidogrel (600 mg), aspirin (325 mg) and unfractionated heparin (5000 IU), the patient was transferred to the catheterization laboratory to perform emergency coronary angiography and primary PCI. The angiogram showed two-vessel-disease, with total occlusion of the right coronary artery in the mid segment (which was the culprit lesion) and an ostial 80% stenosis of the LAD. We decided to perform primary PCI in the right coronary artery. Intravenous bivalirudin was administered to the patient. The transradial approach was chosen by the treating physician. The right coronary artery was engaged with a JR4 6F catheter and a soft PCI wire easily penetrated the occlusion, establishing TIMI III flow at the distal part of the right coronary artery. Two bare metal stents (Liberte 3.0 × 20 mm and Liberte 3.0 × 16 mm) were implanted in the mid segment of the right coronary artery with an excellent angiographic result. The sheath was withdrawn in the laboratory and a radial compression device (TR band) was deployed over the puncture site.

ABBREVIATIONS

PCI = percutaneous coronary intervention

LAD = left anterior descending

STEMI = ST elevation myocardial infarction

The patient was transferred to the cardiac care unit where he had an uncomplicated post infarct recovery. The ultrasound performed showed inferior wall hypokinesia with a preserved ejection fraction of 50% and no significant abnormalities from the cardiac valves.

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Six days after the infarction we decided to proceed with planned PCI of the ostial lesion of the LAD. The procedure was performed again from the right radial approach. Unfractionated heparin was administered into the sheath. The left coronary artery was

engaged with a JL 3.5 6F catheter. After wire crossing of the lesion, a direct stenting of the stenosis was performed with a drug eluting stent (Promus Element 3.5 × 24 mm), with no residual stenosis left. The patient was discharged home the next day.

DISCUSSION

We presented a case of inferior STEMI treated with primary PCI of the culprit lesion at the acute phase and scheduled PCI of the ostial LAD lesion before patient discharge, without patient having any documented residual angina or pathological stress test.

Treatment of patients with multivessel disease presenting with STEMI was addressed in the recent ESC Guidelines for patients with STEMI,¹ where an indication of IIa was given to culprit lesion only PCI during the primary procedure, with the exception of patients with cardiogenic shock or documented ischemia.

Recently, the PRAMI trial² compared preventive PCI for all lesions more than 50% during the initial procedure with an ischemia-driven staged approach after hospital discharge. The study was early discontinued due to ethical reasons, because of the obvious superiority of the preventive strategy. The primary endpoint of the study, which was the combined endpoint of death from cardiac causes, non fatal myocardial infarction and refractory angina was met in 21 patients in the preventive PCI group and in 53 patients in the non preventive PCI group ($p < 0.001$). This was driven mainly by a reduction in the refractory angina secondary outcome (12 patients vs 30 patients, $p = 0.002$), but the other composites of the primary endpoint were also significantly reduced (non-fatal myocardial infarction: 7 patients vs 20 patients, $p = 0.009$ and cardiac death: 4 patients vs 10 patients, $p = 0.07$).

This is a very important study because it gives evidence that perhaps a more aggressive treatment of patients with STEMI and multivessel disease might be beneficial for them. However, there are several issues to be discussed. A big percentage of patients in PRAMI trial had an inferior STEMI (66% in the preventive PCI group and 55% in the non preventive PCI group). This may be interpreted that a significant proximal LAD stenosis, with prognostic significance, may be left untreated in the non preventive PCI arm of the study. Patients with STEMI and more than one culprit lesions, who are approximately 10% of all patients presenting with STEMI, may also be a problem in the non preventive PCI group.

The study also received criticism about its design. The non

preventive PCI group is normal to have more refractory angina reported, since both the patient and the physician knew that the patient has untreated significant lesions. The secondary endpoint of non-fatal myocardial infarction may also be affected since patients who underwent staged PCI have approximately 30% possibility of asymptomatic troponin elevation after the procedure, a fact that would increase the non-fatal myocardial infarction cases in the non preventive PCI group. On the other hand, those troponin elevations could not be diagnosed in the preventive PCI group, since they would be covered by the troponin elevation due to the initial STEMI.

Are any of the arms followed in PRAMI trial according to the routine followed in our institution? In the present case we followed a strategy different from both arms in the PRAMI trial. We performed a staged procedure before hospital discharge and without losing time waiting for angina or ischemia documentation. The current policy in our institution is to treat the culprit lesion only during primary PCI according to the ESC guidelines for patients with STEMI. The treatment of the other stenoses is determined by the location and the severity of them. Obviously significant proximal LAD stenosis are treated before hospital discharge, while significant lesion in the right and in the circumflex coronary arteries are treated with scheduled PCI two months after STEMI. Patients without angina and borderline lesions, ischemia documentation is necessary before a new intervention is decided. The fractional flow reserve could be a reasonable tool distinguishing significant from non significant lesions, but the high cost is a considerable issue.

CONCLUSION

Treatment of patients with STEMI and multivessel disease is challenging and the treatment can be individualized according to the lesion severity and location.

REFERENCES

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