

Angiographic evidence of myocardial channels after percutaneous transmyocardial laser treatment

Alessandro Colombo, Paolo Danna, Maurizio Viecca

Cardiology Department, L. Sacco Hospital, Milan, Italy

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The available clinical reports on percutaneous transmyocardial laser revascularization do not provide data on the visibility of the channels after the procedures. We present the case of a patient who had clear angiographic evidence of the presence of myocardial channels immediately after percutaneous transmyocardial laser revascularization.

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Address:

Dr. Alessandro Colombo

*Divisione di Cardiologia
Ospedale L. Sacco
Via G.B. Grassi, 74
20157 Milano
E-mail:
colombo_alessandro@
hotmail.com*

Percutaneous Holmium:yttrium-aluminum-garnet (YAG) laser treatment of the left ventricle (PTMR) is aimed at the creation of myocardial channels¹, which are instrumental if the patient is to derive any benefit from the procedure^{2,3}. As assumed from experimental studies, laser-induced myocardial channels approximate 1 mm in width and 5 to 8 mm in depth and thus should be visible at left ventricular angiography¹. We routinely perform left ventricular angiography immediately after PTMR, but the angiogram usually fails to confirm the presence of the channels. Here we report on the clinical history of a patient in whom myocardial channels were readily visible after the procedure.

Case report

A 59-year-old male patient with a history of effort angina and left anterior descending coronary artery disease had successful coronary artery bypass grafting with the left internal mammary artery on the middle left anterior descending artery and a saphenous vein graft on an occluded large diagonal branch. After 1.5 years of symptomatic relief, the patient again presented with angina despite diltiazem 60 mg 3 times daily and isosorbide mononitrate 40 mg twice daily (CCS class III). A Bruce exercise stress test during therapy induced angina and a 1.5 mm ST-segment depression on the infero-lateral leads at step II. However, a subsequent myocardial stress/rest perfusion scintigraphy revealed no

ventricular perfusion defects. A negative scan was not considered sufficient to rule out an intervention because the exercise stress test was clearly positive and the history of angina fully convincing. Coronary angiography showed saphenous graft occlusion. At transthoracic echocardiography the left ventricle had a normal volume and function, the wall thickness being 9 mm and the ejection fraction 66%, without regional systolic dysfunction. Neither a percutaneous nor a surgical coronary intervention was judged to be clinically indicated; hence the patient agreed to undergo the experimental laser therapy.

On May 30, 2000 the patient underwent PTMR with the Eclipse Surgical Technologies device (Sunnyvale, CA, USA). After heparin (5000 IU i.v.), and under fluoroscopic guidance Holmium:YAG laser impulses (three 3.5 W pulses per shot) were delivered in ten different spots of the distribution territory of the occluded diagonal branch. Control left ventricular angiography at the end of the procedure disclosed three full-width myocardial channels with contrast spilling into the pericardium (Fig. 1), the patient being completely asymptomatic. The partial thromboplastin time was promptly set back to normal with i.v. protamine sulphate. A slight pericardial effusion did not require drainage and gradually disappeared at serial post-procedure echo examinations.

In the following 1 month angina steadily improved to CCS class I. The time to 1 mm ST-segment depression during the Bruce exercise stress test was found to in-

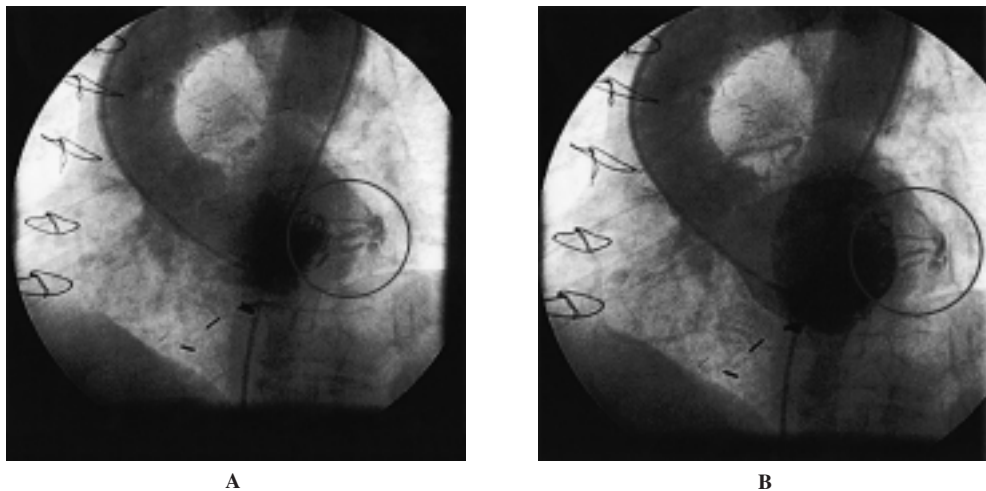


Figure 1. Systolic (A) and diastolic (B) pictures of the left ventricle in the 60° left anterior oblique view showing three myocardial channels in the lateral wall immediately after percutaneous transmyocardial laser revascularization (circles).

crease from 345 s (baseline) to 408 s, in the absence of any modifications in the patient's pharmacological treatment regimen. Three months after the intervention the patient's clinical status and stress test results were similar.

At 6 months of follow-up the patient reported worsening effort angina, approaching that at baseline, albeit the drug regimen had not been modified. The ischemic threshold at the exercise stress test was found to be unchanged compared to that observed at 1 month of follow-up.

Left heart catheterization was performed, and it showed no significant difference in the coronary picture, the myocardial channels being no longer visible. The overall left ventricular systolic function and that in the laser-treated region were found to be normal.

Discussion

In most of PTMR cases why don't laser impulses to the left ventricle result in the creation of visible laser channels? We have no obvious explanation for that. One reason could be a less than optimal contact between the tip of the catheter and the left ventricular wall, that consequently leads to dissipation into the blood of a part of the laser energy. Another biological aspect to be taken into account is a possible anisotropy of the ischemic myocardium. The fact that all the channels were found to be occluded at follow-up angiography is not surprising and is in line with studies demonstrating early channel thrombosis in laser-treated animals⁴.

This case depicts a typical pattern of marked symptomatic effectiveness of PTMR associated with only slight changes in the results of stress testing. This leaves the issue of whether and how PTMR benefits patients open. The possibility that transmyocardial laser therapy is a highly technological "placebo" cannot be discarded⁵.

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