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Transmyocardial Laser Revascularization: Histopathological Findings

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Transmyocardial laser revascularization (TMLR) is a new surgical technique clinically tested in patients with advanced severe coronary arteriosclerosis when classic routine treatment by medicaments, percutaneous transluminal coronary angioplasty (PTCA), or aorto-coronary bypass surgery does not improve symptoms of ischemic heart disease. During the procedure high-energy CO_2 laser performs 35–40 transmyocardial channels via left-sided thoracotomy. Channels are drilled from the epicardial side of the heart through the myocardium into the left ventricle cavity. Impulses are synchronized with EKG (diastole), the channel diameter is about 1 mm. Transmural laser penetration is confirmed by intraoperative transesophageal echocardiography (TEE). This technique is based on a theory that channels allow blood supply from left ventricle directly into the intramyocardial vessels (possibly capillaries) and so improve oxygenation of ischemic myocardium. Presented are gross and microscopic findings in a 75-year-old woman who suffered from classic class IV angina with shortness of breath. She had a history of an inferior myocardial infarct, ventricular tachyarrhythmia, aorto-coronary bypass, and mitral valvuloplasty. Her ejection fraction by echocardiography was 25%. Angiographically, she had multiple occlusions of native coronary arteries and diffuse distal stenosis in the graft of the left internal mammary artery (LIMA) to the left anterior descending coronary artery (LAD). Thirty six of forty laser pulses were confirmed by TEE as transmural. The patient died suddenly of ventricular fibrillation 5 days after TMLR surgery. The autopsy was performed 6 hours after death. After cross-sectioning of the heart all the laser-bored channels were found partially or completely filled by fibrin and cell infiltrate composed mainly of polymorphonuclear leukocytes. Patent channels were found within myocardial scars, channels performed through viable myocardium appeared to be partially collapsed and occluded. *Cardiovasc Pathol 1998;7:63–67* © 1998 by Elsevier Science Inc.

Case Report

In July 1994, a 75-year-old woman was admitted to Texas Heart Institute at St. Luke's Episcopal Hospital in Houston, Texas, with severe ischemic heart disease—angina and shortness of breath. Her ejection fraction by multigated acquisition radionuclide ventriculography (MUGA) was 40%, by intraoperative transesophageal echocardiography (TEE) 25%. The patient had a history of aorto-coronary bypass with mitral valvuloplasty (1993). By angiography, she had multiple occlusions of native coronary arteries and stenosis of the left internal mammary artery (LIMA) to the left anterior descending coronary artery.

Operative Summary

Transmyocardial laser revascularization (TMLR) was performed with the Heart Laser CO_2 system (Laser Engineering, Inc., Milford, MA), 40 laser pulses were provided,

Manuscript received July 16, 1996; accepted June 2, 1997.

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655 Avenue of the Americas, New York, NY 10010
1054-8807/98/$19.00
PII S1054-8807(97)00061-6

Cardiovasc Pathol Vol. 7, No. 2
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36 pulses were confirmed as transmural by transesophageal echocardiography. Laser pulses were performed in the anterolateral portion of left ventricle; the procedure was completed without any complications.

The patient died suddenly of intractable ventricular fibrillation 5 days after TMLR surgery.

Pathological Findings

The consent for unlimited autopsy was obtained and the autopsy was performed 6 hours after death.

The heart showed hypertrophy of both right and left ventricle (the weight was 545 g). There were extensive pericardial adhesions, probably as a result of previous heart surgery in 1993. Grossly, we were able to identify 10 rusty-brown puncture marks in the anterolateral left ventricle wall corresponding with the area of laser pulses. There were also fibrin deposits and a few surgical sutures found in epicardial area. After cross-sectioning of the ventricles, tiny reddish strips were seen grossly in the TMLR surgery area. In the close vicinity, softer light brown irregular areas of possible myocardial necrosis were evident.

Native coronary arteries showed severe multifocal stenoses of all major branches by arteriosclerotic plaques partially calcified. No arteriosclerotic or thrombotic arterial occlusion was found. Three bypass grafts were identified: from aorta to the right coronary artery and left circumflex and from left internal mammary artery to the left anterior descending coronary artery. The lumen of the bypasses was focally moderately stenosed by thin arteriosclerotic plaques, but no total occlusion was found. Distal anastomoses showed minimal arteriosclerotic stenoses.

Tricuspid, pulmonic, and aortic valves were unremarkable, the mitral valve was repaired by a prosthetic supporting ring (valvuloplasty).

There was bilateral hydrothorax (300 ml of serous fluid on the left side, 150 ml on the right side). Findings in other parenchymatous organs were unremarkable—lungs with minimal pleural anthracosis, liver, spleen, and kidneys with minimal congestion.

Microscopic examination of the heart was performed, serial sections from paraffin blocks were carefully examined, stained by routine hematoxylin and eosin and by Masson trichrome stain. Ten laser formed channels were identified on microscopy. Channels were lined either by coagulated fibrous tissue in areas of pre-existing myocardial scars (Figure 1) or by coagulated cytoplasm of cardiac myocytes in other myocardial areas (Figure 2). There were amounts of fibrin intermixed with large amounts of polymorphonuclear leukocytes within the lumina of the intramyocardial parts of channels as well as in the channel orifices (Figure 3). Partial

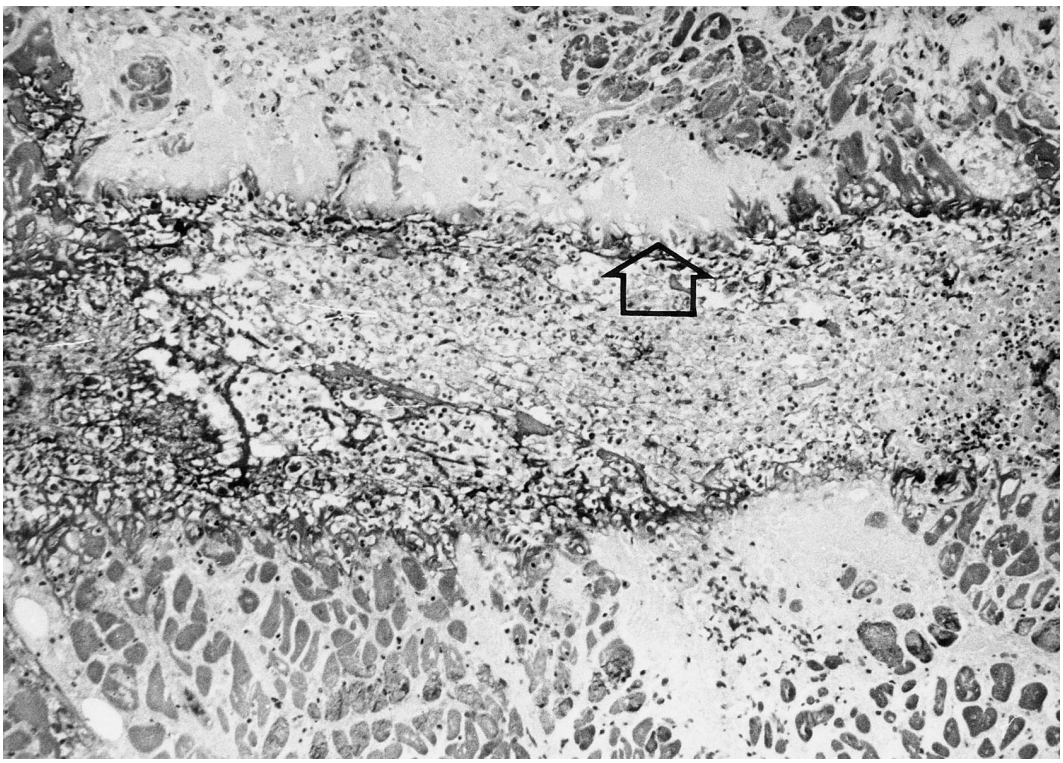


Figure 1. Laser formed channel performed through hyalinized scar, lined by coagulated fibrous tissue (*arrow*). Fibrin and polymorphonuclear leucocytes inside the lumen. (Masson trichrome stain, ×100.)

channel patency was found only in areas where channel was formed through hyalinized rigid myocardial scar.

Relatively extensive areas of recent myocardial necrosis with leukocytic infiltrates were found in anterolateral left ventricle wall in both close and remote neighborhood of the laser formed channels (Figure 4). No relationship between the necroses and the channels was identified.

Discussion

Coronary artery arteriosclerosis still remains a major medical therapeutical problem all over the world. Classic noninvasive medical treatment often fails in severely stenosed and calcified coronary arteries where surgical techniques are widely used.

For the treatment of coronary arteriosclerosis two main surgical procedures are used—coronary artery bypass graft surgery (CABG) and percutaneous transluminal coronary angioplasty (PTCA), recently transmyocardial laser revascularization (TMLR) has been added. This technique is based on the theory that laser bored channels allow blood (oxygen) supply directly from the left ventricle to the ischemic myocardium and consequently to the intramyocardial capillary network. After laser boring, the epicardial channel orifice is closed either by digital pressure or by ligation. The endocardial orifice and the whole channel lumen is supposed to remain patent.

From a brief review of the laser surgery history we learn that experimental trials were provided in 1965 (1) when authors performed transmural myocardial channels by needles. Needle-formed channels were immediately thrombosed so additional techniques were used (T tubes, stents, etc.) but unfortunately with poor results.

Development of the laser beam brought a new light and renaissance of these theories (2). Low-energy lasers were not able to bore the channel in the short time during diastole so trials were performed on cardioplegically arrested hearts.

New powerful laser CO_2 machines developed in the last few years use higher energy and they drill a channel through the whole myocardial wall during the end-diastole (10 to 60 milliseconds). Clinical studies are now conducted under an Investigational Device Exemption approved by the United States Food and Drug Administration.

According to the literature, results of this technique remain controversial especially as to whether they improve oxygenation of acutely ischemic myocardium and whether the channels remain permanently patent and therefore serve as real conduits for myocardial revascularization.

Acutely ischemic canine myocardium cannot be probably revascularized by TMLR—measured by the amount of adenine nucleotides in ischemic areas (3) or by measure-

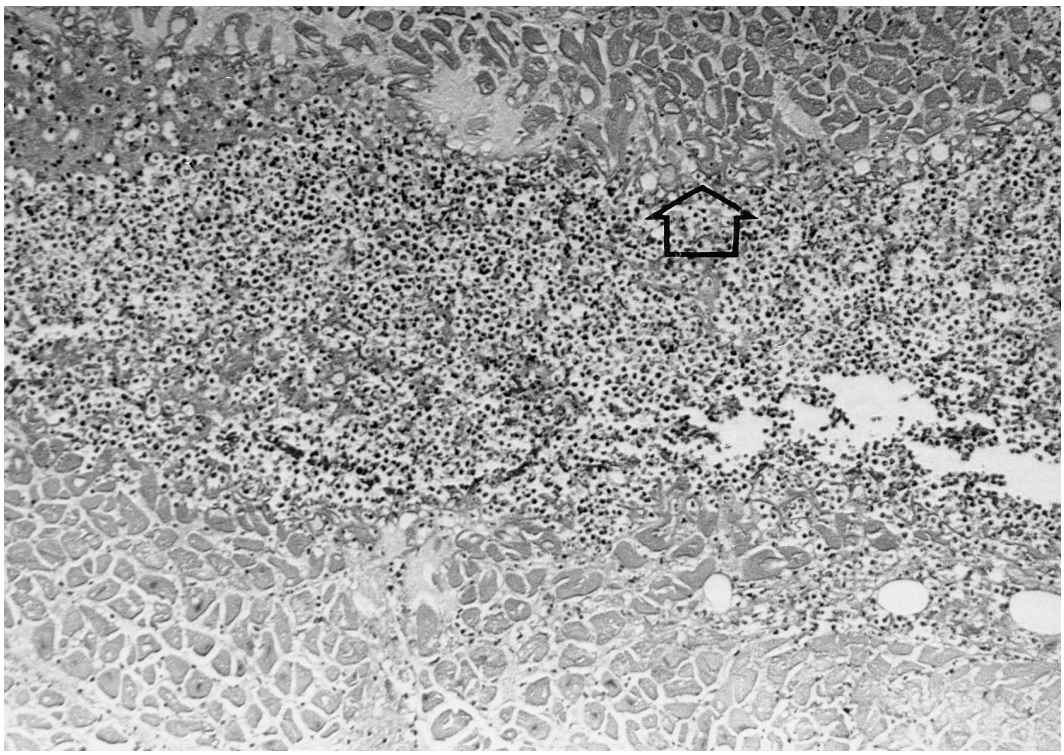


Figure 2. Laser formed channel within heart muscle, lined by coagulated cardiac myocytes (*arrow*). Extensive number of leucocytes within the lumen. (Hematoxylin-eosin stain, ×100.)

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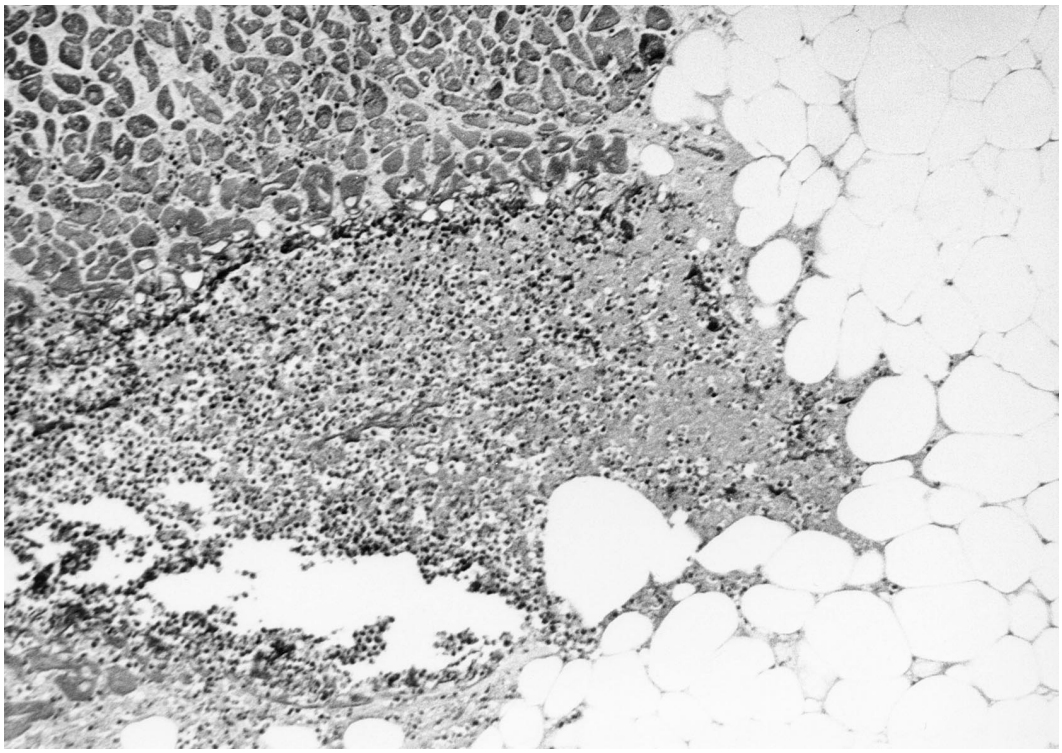


Figure 3. Epicardial channel orifice. (Masson trichrome stain, ×100.)

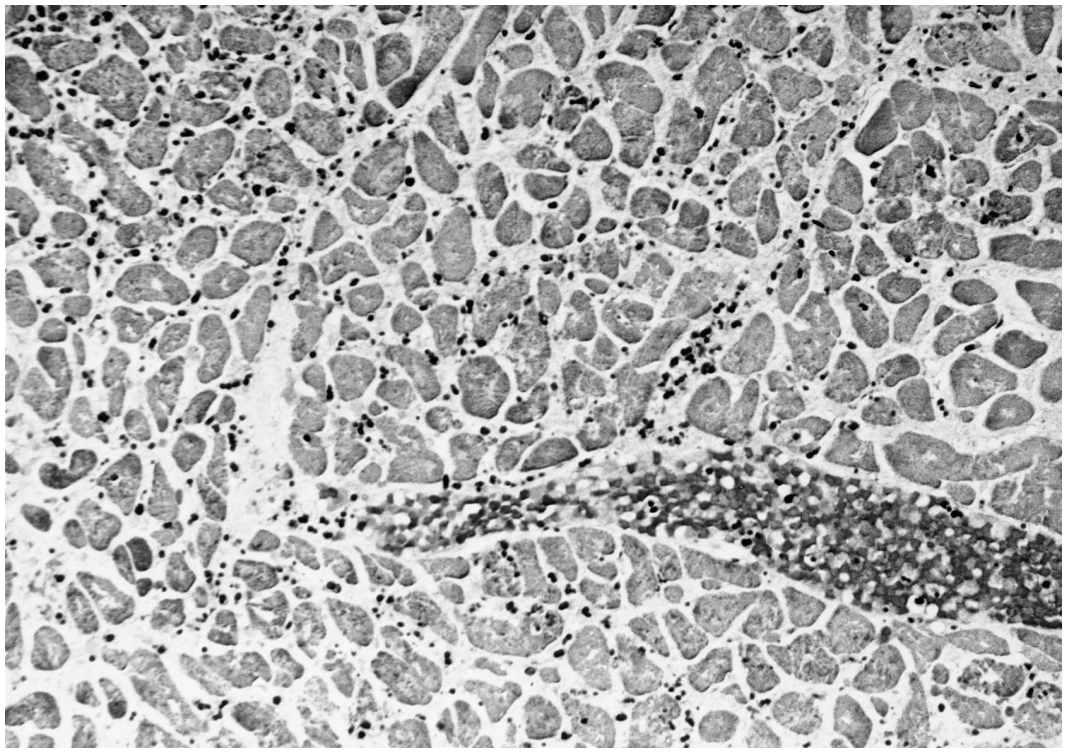


Figure 4. Areas of recent myocardial infarction. (Hematoxylin-eosin stain, ×200.)

ment of blood flow using radiolabeled microspheres (4). On the other hand, some authors confirmed improved contractility of ovine cardiac myocytes and diminishing of acutely ischemic area after TMLR surgery (5).

Chronic ischemia in patients with symptomatic coronary disease refractory to interventional or medical treatment show better results with the usage of TMLR. Clinical trials show improvement of mean wall motion score measured by dobutamine echocardiography in lased myocardium (6) and of relative subendocardial perfusion by positron emission tomography (7).

Permanent channel patency in animal models was proved by some authors (8) and also in humans—longer term histology may be different than the acute picture due to endothelization of the channel lumen. Angiogenetic effect of laser irradiation may also play a role. This result was confirmed by the autopsy (9).

In our case we cannot confirm patency of laser formed channels. Questions remain regarding the possible negative role of extensive areas of necrosis in neighboring anterolateral left ventricle wall.

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