


# Improved myocardial perfusion after transmyocardial laser revascularization in a patient with microvascular coronary artery disease

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## Abstract

We report the case of a 59-year-old woman who presented with symptoms of angina that was refractory to medical management. Although her cardiac catheterization revealed microvascular coronary artery disease, her symptoms were refractory to optimal medical management that included ranolazine. After undergoing transmyocardial revascularization, her myocardial ischemia completely resolved and her symptoms dramatically improved. This case suggests that combination of ranolazine and transmyocardial revascularization can be applied to patients with microvascular coronary artery disease.

## Keywords

Transmyocardial laser revascularization, microvascular angina, coronary artery disease, ranolazine

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## Introduction

A subset of patients with coronary artery disease (CAD) who are not ideal candidates for coronary revascularization remain symptomatic despite receiving optimal medical therapy. Transmyocardial revascularization (TMR) offers another therapeutic option for these patients. Although some studies suggest that TMR does not improve myocardial perfusion, results have consistently shown an improvement in angina symptoms when TMR is used as sole therapy.<sup>1-4</sup> Here, we present a case report of a patient who demonstrated significant improvement in myocardial perfusion and symptoms of angina after undergoing TMR.

## Case report

The patient was a 59-year-old African-American female with a medical history of type II diabetes mellitus, dyslipidemia, hypertension, obesity and a previous history of smoking. The patient had progressively worsening shortness of breath in 2008 and early 2009.

In January 2009, the patient underwent a thallium-MIBI myocardial perfusion test with dobutamine which revealed left ventricular ejection fraction (LVEF) of 28%,

hypokinesis of the left ventricle at the apex, septum and inferior walls. There was a moderately sized perfusion defect in the mid-anteroseptum which was 50% fixed and 50% reversible. Normal perfusion was noted in the remainder of the left ventricle (Figure 1(a)-(d)). Of note, the patient had never been diagnosed with acute myocardial infarction (MI) in the past.

A cardiac catheterization in February 2009 showed a normal left main coronary artery, diffuse but mild luminal

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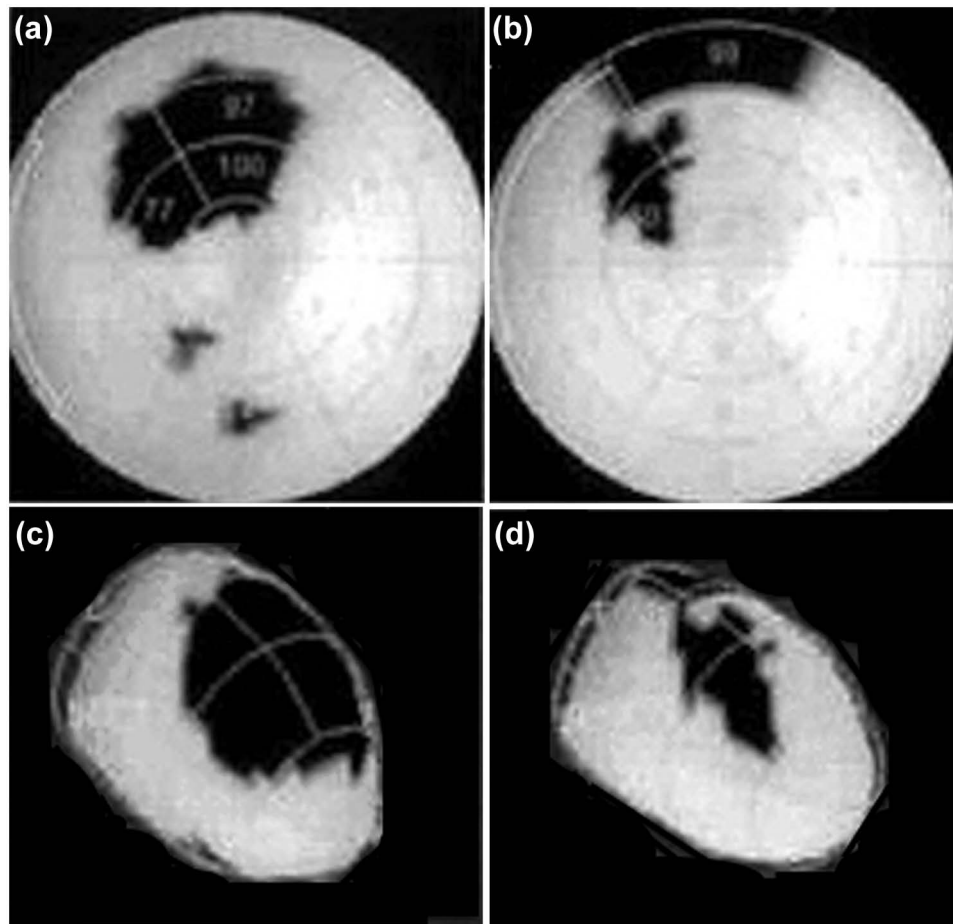
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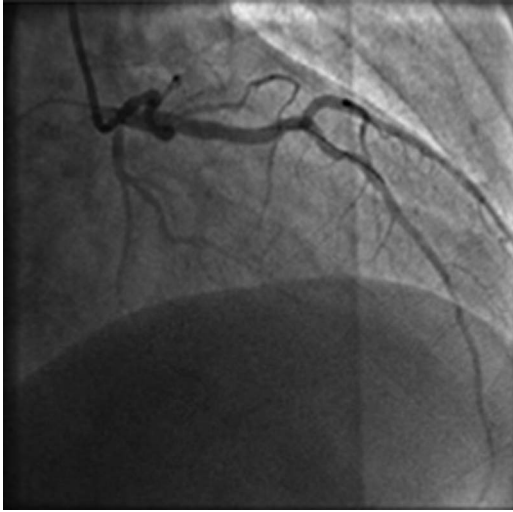
**Figure 1.** Thallium/MIBI myocardial perfusion test with dobutamine performed prior to TMR shows a moderately sized perfusion defect in the mid-anteroseptum which was 50% fixed and 50% reversible post stress. The perfusion defect with stress (dobutamine) in (a) and (c) is considerably larger compared to (b) and (d). A quantity of 3.6 mCi of Thallium 201 was used. SPECT imaging was then performed. The patient was then administered dobutamine IV followed by 31.5 mCi of Tc-99m MIBI. SPECT and gated SPECT imaging were then performed. Patient exercised to a maximum heart rate of 127: (a) Bull's eye view of the heart during stress. (b) Bull's eye view of the heart during rest. (c) Vertical long axis view of the heart during stress. (d) Vertical long axis view of the heart during rest. TMR: transmyocardial revascularization; SPECT: single-photon emission computed tomography; Tc-99m: Technetium-99m.

irregularities in the left anterior descending (LAD), 30% stenosis in the proximal and middle portions of the left circumflex coronary artery (thrombolysis in myocardial infarction (TIMI) III flow), a dominant right coronary artery with 30% stenosis in its middle section (TIMI III flow) and an LVEF of 30%.

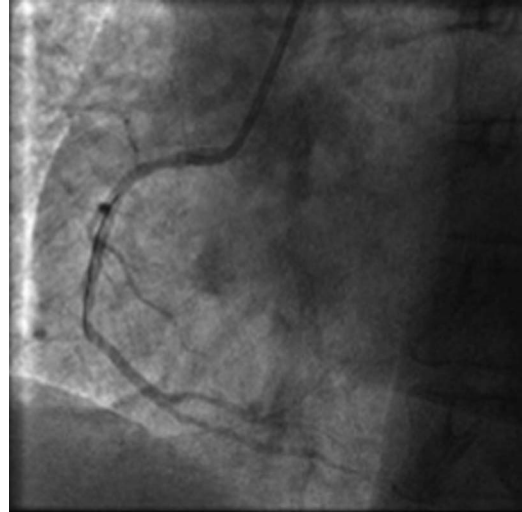
In May 2009, the patient complained of greatly diminished exercise capacity and dyspnea on exertion after walking one block. She was no longer able to participate in water aerobics classes because of her symptoms. Consequently, she was started on ranolazine 1000 mg twice daily and was continued on isosorbide mononitrate 60 mg daily, nebivolol 10 mg daily and sublingual nitroglycerin as needed for chest pain.

In June 2009, the patient reported no change in her symptoms despite the addition of ranolazine. Given the presence of ischemia on the stress test, the patient's complaints

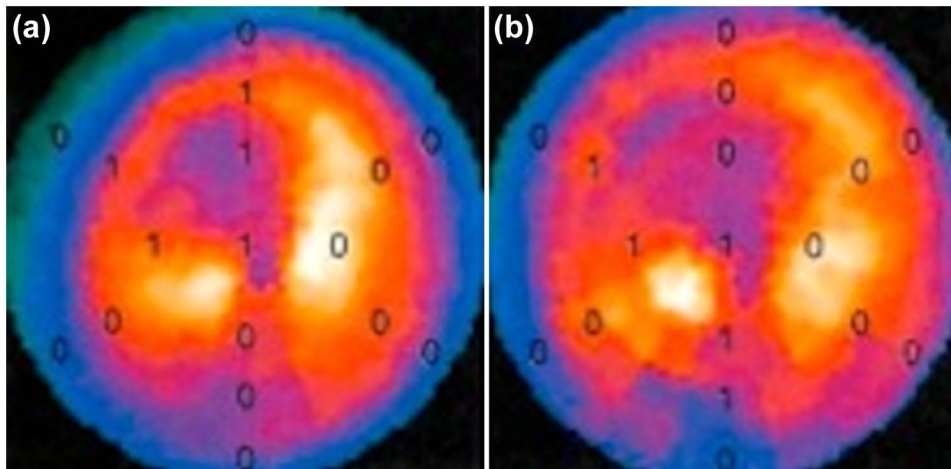
consistent with ischemic heart disease, and the presence of only luminal coronary artery irregularities, microvascular disease (also referred to as syndrome X) was highly suspected. In June 2009, the patient underwent another cardiac catheterization that included an assessment of coronary flow reserve (CFR) (Figures 2 and 3). Given her abnormal CFR of 1.4 isolated to the LAD, she was referred to cardiothoracic surgeons for TMR of the anterior wall. In August 2009, using the holmium:yttrium–argon–garnet (Ho:YAG) laser device, 41 transmyocardial channels were created (11 channels in the lateral wall, 16 in the anterior wall, 9 in the apex and 5 in the inferior wall). The TMR procedure was completed without complications. In November 2009, the patient reported experiencing a milder degree of angina symptoms. Her dose of ranolazine was adjusted to 500 mg twice daily. A sestamibi treadmill stress test revealed normal myocardial perfusion with no evidence of ischemia or scar, LVEF of 33% with



**Figure 2.** Cardiac catheterization performed in June 2009 prior to undergoing TMR revealed a coronary flow reserve of 1.4 in the left anterior descending coronary artery.  
TMR: transmyocardial revascularization.



**Figure 3.** Cardiac catheterization performed in June 2009 prior to undergoing TMR revealed a coronary flow reserve of 2.7 in the right coronary artery.  
TMR: transmyocardial revascularization.



**Figure 4.** Sestamibi treadmill stress test performed in December 2009, approximately 3 months after TMR, showed normal myocardial perfusion post stress. An apical thinning artifact is noted. The perfusion image on the left was obtained at rest, and the image on the right was obtained post stress. A quantity of 8.6 mCi of Tc-99m sestamibi was used. Patient exercised for 10 min and 11 s to a heart rate 137 (84% maximum heart rate). After IV administration of 27.34 mCi of Tc-99m sestamibi at peak exercise, post-stress images were obtained: (a) normal perfusion at rest and (b) normal perfusion post stress.  
TMR: transmyocardial revascularization; IV: intravenous; Tc-99m: Technetium-99m.

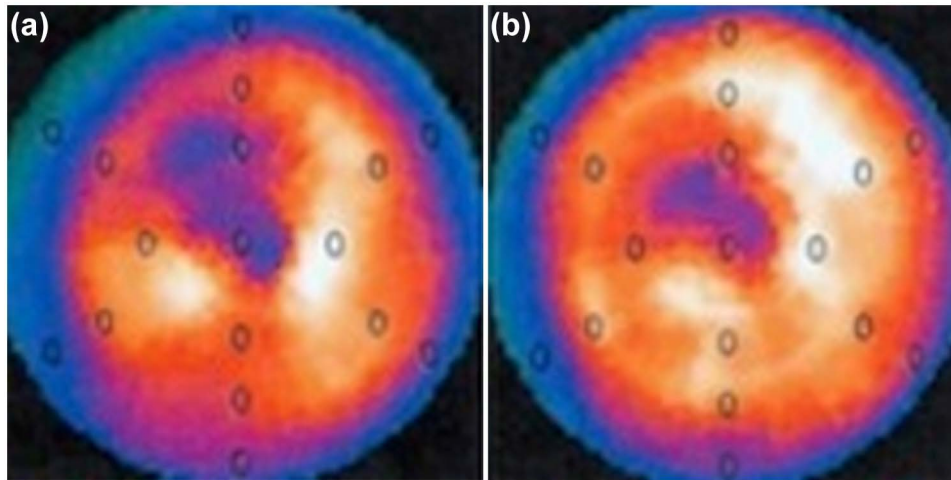
global left ventricular hypokinesis (Figure 4(a) and (b)). In January 2010, the patient reported significantly improved exercise tolerance as well as decreased frequency and severity of exertional chest pain. By that time, she was able to participate in water aerobics classes two or three times weekly.

Since January 2010, the patient's symptoms have been progressively improving and her exercise tolerance increasing. She underwent an additional sestamibi treadmill stress test in December 2010 which again showed no evidence of

ischemia or scar, LVEF 34%, with global left ventricular hypokinesis (Figure 5(a) and (b)).

## Discussion

Despite great advances in myocardial revascularization techniques, there still remains a subset of patients with CAD in whom complete coronary revascularization cannot be achieved. Some of these patients continue to suffer from lifestyle-limiting angina despite best efforts at medical



**Figure 5.** Sestamibi treadmill stress test performed in December 2010, approximately 15 months after TMR, showed normal myocardial perfusion post stress. An apical thinning artifact is noted. The perfusion image on the left was obtained at rest, and the image on the right was obtained post stress. A quantity of 8.6 mCi of Tc-99m sestamibi was used. Patient exercised for 8 min and 25 s to a heart rate of 134 (82% maximum heart rate). After IV administration of 22.51 mCi of Tc-99m sestamibi at peak exercise, post-stress images were obtained: (a) normal perfusion at rest and (b) normal perfusion post stress.

TMR: transmyocardial revascularization; IV: intravenous; Tc-99m: Technetium-99m.

therapy. TMR serves as an additional therapeutic option in the armamentarium of physicians taking care of patients with refractory angina.

TMR is performed by direct application of laser energy to ischemic regions of myocardium. TMR can be performed by one of two available techniques. The epicardial technique involves direct application of laser to the exposed epicardium via a mini left thoracotomy site. Percutaneous myocardial laser revascularization (PMR) is an alternative technique, which delivers laser energy to the endocardium and utilizes fluoroscopy. It should be noted that two randomized controlled trials have shown no improvement in angina class nor exercise capacity in patients undergoing PMR in addition to optimal medical management when compared to optimal medical management alone.<sup>5,6</sup> In addition to the available techniques, there are three available laser types that include CO<sub>2</sub>, Ho:YAG and excimer laser. For the patient described in this case report, we used the endocardial approach with a Ho:YAG laser.

The exact mechanism of action by which TMR improves angina is unknown. Although it is possible for TMR to initially increase myocardial perfusion by creating micron-sized channels at the sites of laser application, convincing histologic evidence suggests that these channels do not remain patent chronically. Instead, the transmyocardial channels become filled with necrotic debris and ultimately are replaced by scar tissue.<sup>7</sup> Others have proposed that the myocardial denervation that occurs secondary to laser therapy may improve angina.<sup>8</sup> However, the evidence for this hypothesis is contradictory.<sup>9</sup> The third hypothesis involves the histologic evidence demonstrating formation of new capillary vessels in the areas surrounding the laser application

site.<sup>10</sup> Finally, angina relief after TMR may be a placebo effect or caused by a combination of the previously mentioned mechanisms.

In a meta-analysis of patients with CAD who underwent TMR while receiving optimal medical management, Cheng et al.<sup>11</sup> discovered a statistically significant improvement in the number of patients experiencing severe angina, readmission rates after 1 year and the need for reintervention secondary to coronary ischemia. Despite significant symptomatic improvement, the authors did not find a difference in mortality rates, MI or stroke between the two groups. The effect of TMR on myocardial perfusion was not investigated by Cheng et al. However, four of the available six randomized clinical trials on TMR have shown no improvement in myocardial perfusion after TMR.<sup>1-4</sup> Although our patient's symptoms were refractory to optimal medical management even after ranolazine was added, her symptoms and myocardial perfusion dramatically improved after undergoing TMR. It may be possible that the combination of the late sodium channel inhibitor ranolazine and TMR improves myocardial perfusion in patients with microvascular CAD. To the best of our knowledge, there are no randomized clinical trials that have investigated the effects of combination ranolazine and TMR in patients with microvascular CAD.

## Conclusion

TMR improves angina in patients with CAD who are not amenable to myocardial revascularization. Although existing evidence does not demonstrate an improvement in myocardial perfusion after TMR, our patient experienced complete resolution of myocardial ischemia and symptoms after

undergoing TMR while concurrently receiving optimal medical management, including ranolazine.

### Declaration of conflicting interests

None.

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