Case Reports

Left Internal Thoracic Artery Graft

to Left Anterior Descending Coronary Artery after Blunt-Chest-Trauma Myocardial Infarction: 14-Year Outcome

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In 2005, we reported an acute myocardial infarction secondary to a left anterior descending coronary artery injury sustained in a motorcycle accident. The treatment was late myocardial revascularization with in situ left internal thoracic artery-to-left anterior descending coronary artery anastomosis. There is little information available about the natural history of acute myocardial infarction after blunt chest trauma, especially when treated in this manner. This present communication reports the 14-year outcome in our patient. **(Tex Heart Inst J 2017;44(3):214-8)**

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Although blunt chest trauma is one of the nonatherosclerotic mechanisms leading to acute myocardial infarction (AMI) in young adults, coronary artery injuries after nonpenetrating thoracic trauma are little recognized in the medical literature. In 2005, we reported an AMI secondary to a left anterior descending coronary artery (LAD) injury during a motorcycle accident.[1] The treatment was late myocardial revascularization with in situ LAD–left internal thoracic artery (LITA) anastomosis. Little information is available about the natural history of AMI after blunt chest trauma, and even less has been published about late LITA revascularization with in situ anastomosis. Therefore, the present communication reports the 14-year outcome in this patient.

Case Report

In August 2002, a 29-year-old man with no personal history of smoking, arterial hypertension, diabetes mellitus, or angina pectoris, and with no family history of coronary artery disease, was admitted to a community hospital after a motorcycle collision. He reported diffuse thoracic pain, and his physical examination revealed pain only upon palpation of his superficial right shoulder and superior anterior chest. His enzyme levels were: creatine kinase, 863 U/L; CK-MB, 68.8 U/L; and lactate dehydrogenase, 1,080 U/L. No other examinations were performed. Because of doubt regarding the presence of AMI or myocardial contusion, no thrombolytic agent was administered. At that time, the patient's thoracic pain was still being treated with analgesics only. His enzyme levels normalized with clinical improvement. However, an echocardiogram obtained 5 days after the accident had revealed septal–apical and anterior–apical hypokinesia, probably caused by myocardial contusion. Because the patient's electrocardiogram (ECG) suggested anterior myocardial infarction or contusion, he was transferred to a tertiary-care hospital, where optimized medical therapy was implemented.

First Presentation at Our Hospital. Eleven months after the accident, the patient presented to us with clinical conditions and laboratory results that indicated the need for intervention. In view of his thoracic pain and effort symptoms, we performed myocardial scintigraphy, which revealed anterior ischemia. Coronary angiograms showed an extensive, isolated proximal LAD lesion, with no apparent evidence of atherosclerotic coronary artery disease (Fig. 1). We therefore chose to treat the patient by
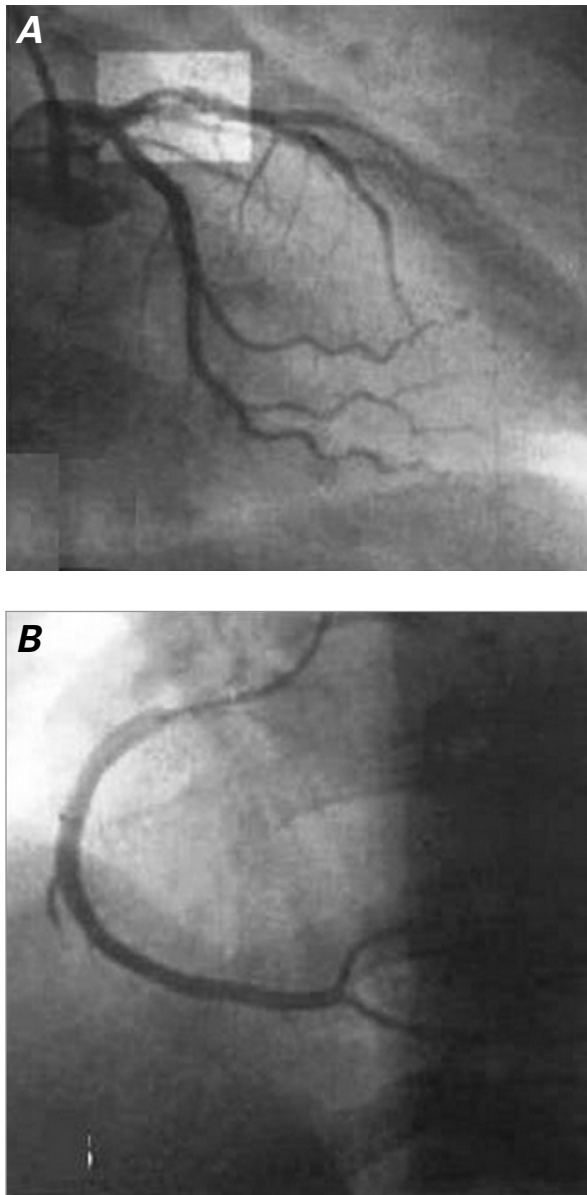
Fig. 1 Coronary angiograms obtained one year after blunt chest trauma show **A**) a proximal, nonatherosclerotic lesion (highlighted) of the left anterior descending coronary artery; and **B**) a nonatherosclerotic right coronary artery.

From: Evora PRB, Oshiro MS, de Souza DG, Menardi AC. Late revascularization of the anterior descending coronary artery in a young patient with previous myocardial infarction caused by blunt chest trauma. Eur J Trauma 2005;31(2):168-9.[1] Permission to reprint granted by Springer.

samples for a study of his blood chemistry, and we performed chest radiography, conventional ECG, myocardial scintigraphy, dobutamine stress echocardiography, and 64-slice multidetector computed tomography (CT).

The ECG registered sinus rhythm and advanced right bundle branch block, with right ventricular (RV) overload. The two ECG records showed that, in 10 years, there had been no change in either pattern (Fig. 2). In addition, the 2015 chest radiograph showed normal results (Fig. 3).

The scintigraphy performed with exercise stress testing (maximum 10 metabolic equivalents of task) showed 1) moderately viable myocardium along a great extent of the LAD, and, to a minor extent, nontransmural fibrosis in association with mild ischemia; 2) transmural fibrosis involving, again to a small extent, the anteroapical, apical septal, and apical segments; 3) viable and nonischemic myocardium involving, to a large extent, the left circumflex coronary artery/right coronary artery (LCx/RCA) territories; and 4) discrete depression of the left ventricular ejection fraction (LVEF) (0.42).

A stress echocardiogram with dobutamine showed no evidence of myocardial ischemia or ventricular dysfunction (Fig. 4). The left atrium was 37 mm wide, the end-
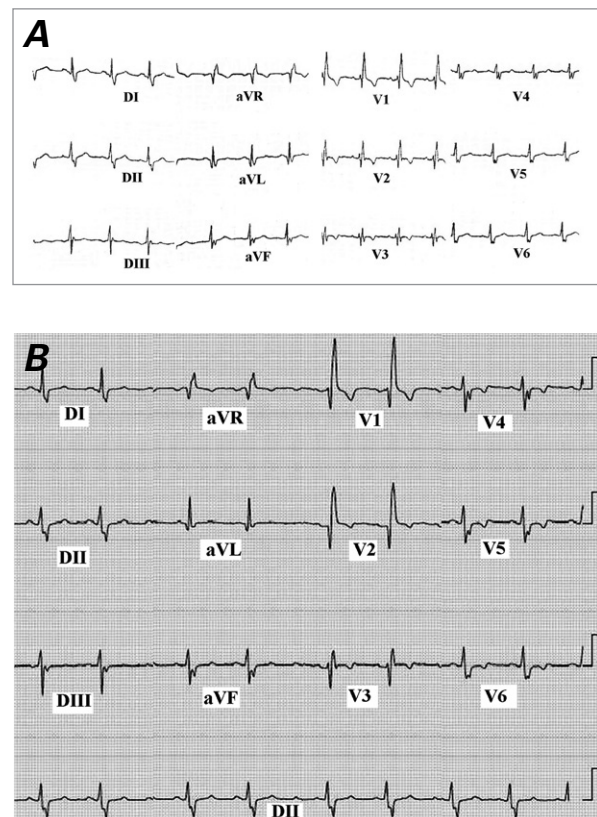
Fig. 2 Electrocardiograms recorded in **A**) December 2005 and **B**) November 2015 show sinus rhythm, advanced right bundle branch block, and right ventricular overload.

means of surgical revascularization. We performed an uneventful in situ anastomosis of the LITA to the LAD, with the patient under cardiopulmonary bypass (CPB). He underwent his follow-up examinations elsewhere.

Second Presentation at Our Hospital. After an absence of 12 years, our former patient lost his medical insurance and returned to our public institution, asymptomatic, as a "new case." Because of our academic interest in establishing his present clinical picture, we gathered

Fig. 3 Chest radiographs (October 2015) show nothing unusual in the **A**) anteroposterior or **B**) lateral view.

Fig. 4 Stress echocardiogram with dobutamine (October 2015) shows no myocardial ischemia or ventricular dysfunction.

Fig. 5 Transthoracic echocardiogram with contrast (October 2015) confirms good left ventricular function in **A**) diastole and **B**) systole.

diastolic diameter of the left ventricle (LV) was 59 mm, and the LVEF was 0.41; the akinetic apex remained compatible with the nonviable segments; the remaining apical segments and anterior septum, which had been hypokinetic upon baseline examination, had evolved to normal at peak stress, indicating that they were viable and nonischemic; and the LV at rest showed hyperkine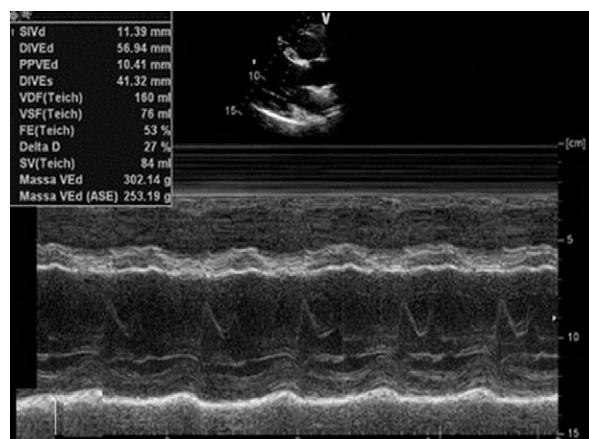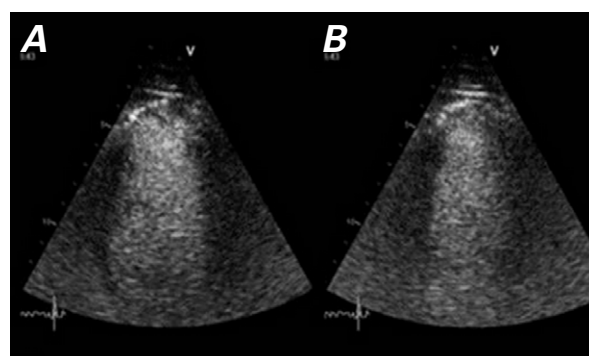sia. Contrast echocardiograms confirmed good LV function (Fig. 5). Despite the ECG evidence of RV overload, a transthoracic echocardiogram showed RV function within normal limits. In the absence of tricuspid valve regurgitation, pulmonary artery pressure could not be estimated.

Although this case report would be much more compelling if it provided angiographic documentation of both the original lesion and the LITA–LAD anastomosis at the 12-year follow-up examination, we did not feel comfortable performing contrast coronary angiography on an asymptomatic patient. A 64-slice multidetector CT scan did show good patency of the LITA anastomosis (Fig. 6). However, the focal point of the CT angiogram was the LITA graft. In reviewing the angiogram, we could say, with certainty, only that the anastomosis showed good patency. In an attempt to reconstruct the CT angiographic slices, we observed (inconclusively) only an artifact in the traumatized region.

Discussion

Blunt thoracic trauma most often affects the LAD (76%), followed by the RCA (12%) and the LCx (6%).[2]
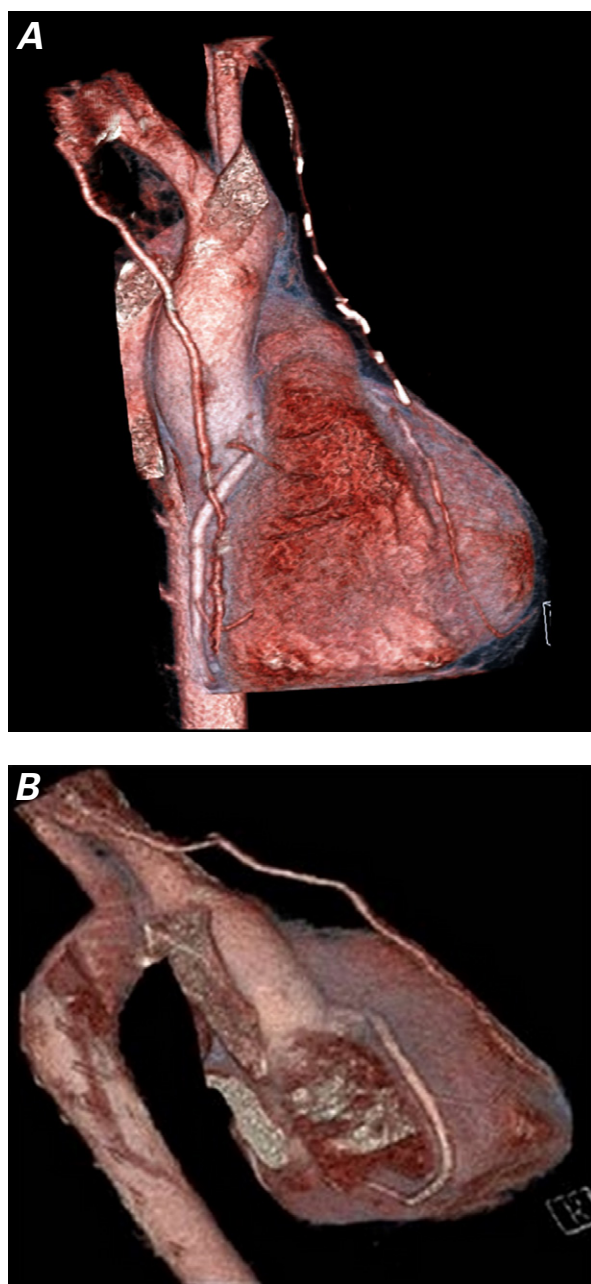
Fig. 6 *A 64-slice multidetector computed tomogram shows good patency of the left internal thoracic artery (LITA) in situ anastomosis with **A**) LITA clips visible and **B**) LITA clips suppressed in reconstructed view to show good LITA flow.*

The higher incidence of LAD involvement could arise from its proximity to the chest wall. The RCA might be most vulnerable to injury when blunt chest trauma occurs during ventricular systole, a time when this vessel lies in its most anterior position.[2]

Coronary artery dissection as a consequence of blunt chest trauma is extremely rare. Such dissection has been reported to occur secondary to percutaneous coronary intervention, coronary angiography, cardiac surgery, and atherosclerosis. Unusual causes are spontaneous occurrence during the peripartum period, congenital disorders of the connective tissue (such as those seen in Marfan and Ehlers-Danlos syndromes), and cardiac sarcoidosis. The mechanism leading to a coronary dissection after blunt chest trauma is most likely a shearing force that produces a small intimal tear, thereby enabling blood to enter the vascular wall and form a blood-filled space.[3] In consideration of information reported by the patient, there probably was no dissection, and the coronary lesion was self-limiting.

In searching the MEDLINE database for "blunt chest trauma" and "myocardial ischemia," Christensen and colleagues[4] found 77 descriptions of AMI after blunt chest trauma, but only one in which angina pectoris was reported. According to these authors, the age distribution was atypical in comparison with that of AMI in general: 82% of the patients with AMI after blunt chest trauma were younger than 45 years of age, and only 2.5% were older than 60 years. Trauma most frequently resulted from traffic accidents, and the LAD was the vessel most often affected. Thirty-one of the 77 patients had occlusion but no atherosclerosis, which strongly suggested a causal relationship between the trauma and occlusion. Our patient had epidemiologic characteristics in accordance with these data (age, traffic accident, and nonatherosclerotic LAD trauma).

Treatment options include medical therapy with anticoagulative and antiplatelet agents, angioplasty (with or without stenting),[5] and coronary artery bypass grafting (CABG) with or without CPB.[6] Thrombolytic therapy should be avoided because of its potentially catastrophic consequences (risk of bleeding from the associated injuries), including extension of the coronary dissection due to hemorrhage into the false lumen.[7] In searching the English-language medical literature for "traumatic coronary artery dissection," Lobay and McGougan[8] found 22 case reports involving 24 patients over a 10-year interval (1998–2007). Eight patients were treated with CABG, 7 were treated with stent placement, and 9 were treated conservatively.[8] This literature review shows that CABG, angiography with stent placement, and conservative management can all be considered viable treatment options for this condition.

We chose conservative treatment for our patient, because he appeared to have no symptoms that called for intervention and because he had already been treated at the community hospital. Almost one year later, clinical and laboratory findings indicated the need for intervention. Even then, we did not opt for coronary angioplasty, because the lesion involved the ostium of an important first diagonal LAD branch and there was increasing evidence of the LITA's advantages over myocardial revascularization in treating atherosclerosis. As of May 2017, the patient was asymptomatic but had a few chronic cardiac sequelae: advanced right bundle branch block, with RV overload; scintigraphic discovery of mild-to-

moderate ischemia of the LAD territory; and discrete depression of LV global systolic function (LVEF, 0.42). However, there was no evidence of myocardial ischemia upon dobutamine stress echocardiography, and 64-slice multidetector CT revealed no restrictive arterial stenosis. Conduction disturbances after blunt chest trauma are probably rare and, in the present case at least, transient. Their pathophysiologic mechanisms are not yet well understood, and they deserve special attention.[9]

Reports on long-term follow-up of coronary blunt trauma are scarce. Kahn and Buda[10] reported the case of a patient who had undergone clinical management for over 2 years, with stable symptoms and good ventricular performance, despite cardiac-chamber enlargement. Yang and colleagues[11] clinically treated a patient with blunt trauma for 7 years, during which he experienced markedly reduced LV function and enlargement of the LV cavity in the presence of angiographically normal coronary arteries. On the basis of this case, the authors concluded that although early revascularization might help to prevent cardiac remodeling, more data are needed to compare the long-term outcomes of different interventions in large studies. Our present report of a 14-year-old case outcome is favorable to surgical revascularization with use of the LITA, but our data appear to stand alone in the literature.

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