Letters to the Editor

IS RECONSTRUCTION OF THE
LEFT ANTERIOR DESCENDING
ARTERY WITH SAPHENOUS
VEIN PATCHING EQUAL TO
ONLAY PATCH
RECONSTRUCTION USING THE
LEFT INTERNAL THORACIC
ARTERY?

To the Editor:

We read with interest the recent report by Myers and associates.1 The authors presented their experience and outcomes of extensive endarterectomy and reconstruction of the left anterior descending artery (LAD) and compared 2 different reconstruction methods: vein patch reconstruction with the left internal thoracic artery (LITA) and LITA onlay patch grafting. The authors showed that the early results were excellent and that the reconstruction method did not have a significant effect on long-term survival in this challenging group of patients. However, we have several concerns about, and comments on, this retrospective review.

First, the actuarial 10-year survival in both groups was less than 50%, which seems lower than expected compared with the overall long-term results of off-pump coronary artery bypass surgery.2 The causes of death were not clearly documented in their report. Because patients who required extensive coronary endarterectomy might have had severe coronary artery disease, it is important to know the early and late causes of deaths. Nevertheless, we also recently published long-term results of long segmental reconstruction of the LAD using the LITA in 112 patients.3 Of these patients, 37 underwent endarterectomy, and their 10-year survival rate was approximately 75%, comparable with the results of the previous report for patients who underwent conventional coronary artery bypass surgery.2 Although we have no data on vein patch reconstruction with the LITA, we believe that onlay patch reconstruction of the LAD using the LITA is quite important to achieve a favorable outcome in the treatment of patients with diffusely diseased coronary arteries. In patients with LITA onlay patch grafts, the wall of the LITA forms a new coronary lumen, and the atheromatous plaques are almost totally excluded from the LAD lumen. Intimal hyperplasia rarely occurs in a LAD reconstructed with the LITA, although vein graft disease has been proved to progress with time.4 An optimal endarterectomy technique and appropriate postoperative anticoagulation strategy are vital to achieve excellent long-term outcomes in these groups of patients. We routinely tack the divided intima of the distal LAD with 8-0 polypropylene sutures. We believe this maneuver, coupled with postoperative anticoagulation, is important to prevent obstruction of distal run-off by residual intima, where thrombosis readily occurs. Our current anticoagulation strategy is as follows. First, low-molecular-weight heparin (5000 U/d) is initiated, after confirmation of heparinization in the intensive care unit. Second, low-dose aspirin, clopidogrel, and warfarin (with a target international normalized ratio of 2.0) are started after the patients have resumed oral intake. Third, low-molecular-weight heparin is discontinued after warfarin is effective. Finally, clopidogrel is continued for 1 month and warfarin for 6 months. We reported immediate postoperative angiographic results with a 97% LITA patency rate (34/35) in patients undergoing long onlay patch reconstruction of the LAD with endarterectomy.1 Their long-term (approximately 10 years) patency rate was 100% (7/7).3 In our recent study, we also reported a 94% immediate postoperative LITA patency in 134 patients using the same technique.5 A similar survival rate between vein patch reconstruction with the LITA and LITA onlay patch grafting does not always mean a similar patency rate. Reconstruction of the LAD with a vein patch can result in a lower patency rate. We all know that postoperative angiography is rarely performed unless patients have significant symptoms. However, it is important to know the fate of these 2 different reconstruction techniques late after surgery. We wonder whether any late angiographic data were available for these 2 groups of patients. We congratulate the authors for their effort in tackling this difficult group of

References


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patients. However, to further improve the late outcome of extensive endarterectomy and reconstruction of the LAD, a precise analysis of the late mortality and late angiographic findings is necessary.

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IS A “NARROW AORTA–MITRAL ANGLE AND ASSOCIATED FACTORS” ASSOCIATED WITH DEVELOPMENT OF SYSTOLIC ANTERIOR MOTION?

To the Editor:

The report by Manabe and colleagues highlights an important factor that might contribute to the likelihood of the occurrence of systolic anterior motion (SAM), which is an undesired complication to be encountered in mitral valve repair procedures.

Many attempts have been made to understand the underlying mechanisms of SAM, which the authors mentioned in their report. We congratulate the authors for their enthusiasm in revealing the mechanisms underlying SAM, and we believe their contributions have great importance.

We wanted to further contribute to this issue, in line with the statements of the authors. As mentioned by the authors, some of the factors that contribute to the development of SAM have been previously defined. These factors can be grouped into major and minor factors. The major group includes excess valvar tissue and an undersized annuloplasty, and the minor group includes a narrow aortic mitral angle, a hyperkinetic small ventricle, a bulging interventricular septum, and an abnormal configuration of the anterior leaflet. We agree with the authors’ statement that a “high ejection fraction and low left ventricular systolic diameter are associated with development of SAM.”

Moreover, we also believe that in cases with a low left ventricular volume, a similar association can be determined. The aorta–mitral angle must be narrow in all cases with a low left ventricular systolic diameter and low left ventricular systolic or diastolic volume. This also results in overestimation of the measured ejection fraction. A narrow aorta–mitral angle facilitates SAM by positioning the anterior leaflet close to outflow tract and displacing the filling chamber of the left ventricle, which becomes a part of the subaortic region. This condition explains why the measured ejection fraction is greater; that is, why the measured left ventricular end systolic residual volume is less. Therefore, a narrow aorta–mitral angle, low left ventricular systolic diameter, low left ventricular systolic volume, and high ejection fraction can be grouped under the same heading “narrow aorta–mitral angle and associated factors.”

We believe that this different viewpoint when exploring the factors related to the development of SAM could help surgeons understand and manage the issue.

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Notice of Correction

In the above-mentioned article, the spelling of Dr Buzzatti’s surname was incorrect. The corrected author list is printed below.

Michele De Bonis, MD, Elisabetta Lapenna, MD, Roberto Lorusso, MD, PhD, Nicola Buzzatti, MD, Sandro Gelsomino, MD, PhD, Maurizio Taramasso, MD, Enrico Vizzardi, MD, and Ottavio Alfieri, MD

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