Anastomotic stenosis after minimally invasive direct coronary artery bypass

In the February issue of the journal (page 59) Morin and colleagues reported on a 52-year-old man who underwent minimally invasive direct coronary artery bypass grafting and 4 days postoperatively was found at angiography to have 80% stenosis at the distal end of the anastomosis between the left internal mammary artery and the left anterior descending (LAD) coronary artery. The patient was discharged home. Four months later there was no residual stenosis.[1]

The ultimate technical goal of coronary artery surgery is to achieve a conduit-to-coronary-artery anastomosis that has no residual stenosis. Even though the authors estimated the postoperative stenosis at 80%, the angiographic image depicted in their Fig. 1 appears to be much more severe. Although difficult to ascertain with a single view, the stenosis is probably greater than 95%. Because the patient's preoperative LAD stenosis was reported as 90% the operation at that time provided no benefit or improvement.

The authors could not diagnose this stenosis as spasm because it did not respond to intracoronary nitroglycerin administration. In their Fig. 1 there appears to be coronary poststenotic dilatation that may represent a response to nitroglycerin by the native LAD coronary artery. Could the administration of intracoronary calcium channel blockers have improved the situation?

Because there were no symptoms, the patient was discharged home under the assumption that "collateral flow was demonstrated by the lack of hemodynamic compromise during occlusion of the LAD coronary artery while performing the anastomosis."

Why was the patient discharged without further evidence of the absence of ischemia (i.e., viability scanning)? The authors recognized that limited postoperative activity minimizes the development of symptoms. After discharge, resumption of activity could have precipitated a significant ischemic event should this stenosis have been permanent. It may otherwise be argued that either the operation was not necessary or the patient remained at risk for myocardial infarction.

Anastomotic stenosis can be caused intraoperatively by an improperly constructed anastomosis, by probing of the native coronary artery, by the use of coronary occluders or intraluminal shunts and by the use of tourniquets or vascular clamps as in this case. Thus, postoperative images of stenoses always carry the risk of permanence. Once a significant stenosis has been demonstrated we do not feel confident discharging a patient under the assumption that it would cause no harm. Furthermore, from the legal standpoint it could prove indefensible. This report is valuable because it alerts treating physicians to this possibility although it does not provide a therapeutic answer.

Further research is needed to clarify the etiology and pathophysiology of post-bypass stenosis, its incidence and, more importantly, how to determine which patients will or will not require further intervention.

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