Case report

Reoperative revascularization of an occluded left subclavian artery and left internal mammary artery ostial stenosis

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Abstract

An ostial stenosis of the left internal mammary artery graft anastomosed to the left anterior descending artery was responsible for unstable angina in a patient with a previous coronary artery bypass graft. A T-shape arterectomy was performed between the left subclavian artery and left internal mammary artery. Successful revascularization of the lesion was achieved with a carotid-to-subclavian bypass and surgical ostial plasty extending to the proximal left internal mammary graft using a Hemashild a graft. This procedure was performed through a transverse supraclavicular incision to avoid potential hazards of a redo median sternotomy.

Keywords: Left internal mammary artery; Ostial stenosis; Subclavian steal syndrome

1. Introduction

The internal mammary artery remains the conduit of choice for coronary artery bypass grafting (CABG) because of enhanced long-term survival with a well documented long-term patency rate [1]. With growing experience and increased number of patients undergoing CABG, the reoperative coronary revascularization is occurring more frequently. The left internal mammary artery (LIMA) is rarely the source of atherosclerotic disease [1], however, myocardial perfusion through this conduit may be compromised by obstructive disease in proximal left subclavian artery (LSA) inducing coronary steal syndrome. Another rare cause of LIMA malperfusion is the presence of the ostial stenosis. We present a case of reoperative revascularization of a de novo LSA occlusion and LIMA graft ostial stenosis.

2. Case report

A 68-year-old woman, who 3 years before was undergone triple CABG (LIMA-normal free flow, to LAD, RIMA to the obtuse marginal artery and a vein graft anastomosed distally to the right coronary artery and proximally to RIMA due to heavily calcified ascending aorta), was recovered at the intensive care unit presenting pulmonary edema, unstable angina and congestive heart failure and left upper limb claudication. The patient was intubated and medical therapy consisting in inotropes, diuretics, heparin and nitroderivates initiated. The echocardiographic examination demonstrated a LVEF = 30% and mild-to-moderate mitral valve regurgitation. Cardiac catheterization was tempting through the femoral approach but without success due to both femoral arteries occlusion. The left radial artery was surgically cannulated and the angiographic examination showed a total occlusion of the proximal LSA and LIMA ostial stenosis (Fig. 1A). The coronary angiography revealed patent venous grafts. The left ventriculography showed inferior acinesia due to preoperative inferior infarction. The subclavian steal syndrome was present, diagnosed by the contrast medium coursing down the LAD during injection and flowed up slowly into the LIMA and faintly opacifying the LSA. The distal portions of the LIMA and LAD remained free of significant atherosclerotic disease. The angio MRI examination demonstrated a total occlusion of the LSA, and a common brachiocephalic trunk from which originated the left and right carotid artery with non-critical stenosis (Fig. 1B,C). The stress-Tl201-scintigraphy demonstrated a reversible ischemic territory in the anterior region. A transesophageal echocardiography demonstrated a heavily calcified ascending and descending aorta.

A transverse left supraclavicular incision was performed. The lateral head of sternocleidomastoideum muscle was

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retracted medially, and the scalenus anterior was divided at its origin. The LSA was exposed after carefully noting the course of phrenic nerve. The proximal LIMA was isolated and mobilized. The left common carotid artery was soft and without significant atherosclerotic involvement. Heparin sodium (150 U/kg) was administered to maintain an activated clotting time in the range of 300–360 s. A test occlusion of the LIMA resulted in no hemodynamic adverse effect. A T-arterectomy between the LSA and LIMA was performed. A longitudinal arterectomy of the LSA at LIMA origin was made initially. Another longitudinal LIMA arterotomy was performed at its origin extended until joining the LSA arterectomy and to the proximal LIMA. Then, the carotid-to-LSA bypass was performed using a 10-mm Hemashield CSG graft. The graft was remodelled for performing the anastomosis to LSA and simultaneously the LIMA ostial plasty (Fig. 2). Postoperatively, the patient remained under treatment with enoxaparin and aspirin. At 3 months after surgery, the Doppler echocardiography demonstrated a good flow at the LIMA main stem (Fig. 2B). She remained free of anginal symptoms at 6-month follow-up.

3. Comment

One of the reasons of the IMA grafts’ long term patency is the low occurrence of atherosclerotic disease which usually develops either at the distal anastomosis or at the body of the vessel. Angioplasty has been performed successfully to treat lesions at these locations [2]. The IMA’s ostial stenosis is a rare phenomenon and its management remains to be a challenging issue. Different authors have documented the successful use of angioplasty and stenting for RIMA [3] and LIMA [4] ostial lesions, although the restenosis incidence remains to be high requiring adjunctive dilation procedures [5,6]. In the present case, because of the total proximal occlusion of the LSA, we opted for surgical revascularization. The presence of proximal LSA occlusion, induced the subclavian steal phenomenon, although the reversed LIMA flow indicating such phenomenon was particularly slow, probably due to the presence of ostial stenosis, which reduces the flow through the LIMA ostium, so, preventing partially a greater reversed flow from LIMA to LSA.

The patient was critically evaluated for LIMA and LSA surgical revascularization through a transverse left supraclavicular approach. The presence of severe aortic calcification contraindicated the application of different surgical strategies such as LIMA graft’s reattachment to aorta or an aorta-to-LIMA, proximal LSA-to-LIMA and aorta-to-LSA bypass grafting. The employed approach offered the theoretical advantages of avoiding the resternotomy, pleural cavity opening, and provides a direct access to the site of LIMA graft and LSA stenosis. Such a technique can be applied safely in patients with porcelain aorta as a ‘non-touch’ approach.

Different authors have reported, that the ostial LIMA stenosis may be managed with a vein jump graft from the subclavian artery [7] or common carotid artery [8] to the LIMA graft. We preferred the LIMA surgical ostial plasty extended proximally to the arterial conduit employing the same prosthetic carotid-to-LSA graft. Such a technique offers the same advantages of the arterial revascularization.
and also employs less time than constructing a different graft to LIMA. Especially in critical situations such as in our case, such a technique should be taken in consideration. Concomitantly, the carotid-to-LSA bypass grafting was performed as the treatment of choice for the management of subclavian steal syndrome due to proximal LSA stenosis [9].

We believe that the LIMA ostial plasty in concomitance to carotid-to-LSA bypass grafting is a procedure that can be performed safely through a transverse supraclavicular approach. Potential hazards of a redo median sternotomy can be avoided. This approach should be reserved only for patients with the unique problem of LIMA-to-LAD stenosis or with proximal LSA stenosis causing subclavian steal syndrome, without other myocardial territory that would benefit from surgical revascularization.

References