

Eosinophilic periarteritis of the left internal thoracic artery graft

Sol internal torasik arter greftinde saptanan eozinofilik periarterit bulgusu

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Introduction

The use of left internal thoracic artery (LITA) as a bypass conduit is associated with the highest long term patency rate and greater life expectancy with respect to saphenous vein (SV) and radial artery grafts after coronary artery bypass grafting (CABG) (1). In this report, we presented a patient reoperated due to the failure of the LITA graft to the left anterior descending (LAD) artery and the pathologic examination of the LITA graft revealed eosinophilic periarteritis.

Case Report

The patient was 60 year old woman who had undergone triple vessel CABG (LITA to left anterior descending artery (LAD)) (90% proximal stenosis), a SV graft to right coronary artery (75 % proximal stenosis) and circumflex artery (80% proximal stenosis) five years ago at our clinic. She was re-admitted due to angina on exertion and signs of ischemia on the anterolateral and posterior walls three years after first operation. Her past medical history was significant for hypertension. She had no history of asthma or any other allergic diseases. Her physical examination and chest X-ray were normal. Acute phase reactants, routine biochemistry, complete blood count and viral serology were all within normal range. Peripheral blood smear revealed 68% of neutrophils, 27% of lymphocytes, 3% of monocytes, 1% of basophils and 1% of eosinophils. Coronary angiography demonstrated diffuse narrowing of the distal half of the LITA graft like a string sign (Fig. 1) and 80% proximal stenosis of the right SV graft. The SV graft to the left circumflex artery was patent. Left ventriculography was normal with an ejection fraction of 70%. Two-vessel re-CABG was performed by using a SV graft to the mid-LAD and a SV graft the distal right coronary artery (RCA). Her postoperative course was uneventful.

Histopathologic examination of the LITA graft (Fig. 2) revealed diffuse inflammatory infiltrate with a good number of eosinophils and some scattered lymphocytes and plasma cells throughout the adventitial and periadventitial layers. Upper half of the LITA graft revealed normal histology except for mild to moderate fibrous intimal hyperplasia (FIH). Pathology of the SV graft was consistent with vein graft atherosclerosis.

With the suspicion of a systemic vasculitic disease, autoimmune serology including serum rheumatoid factor, anti-nuclear antibody and

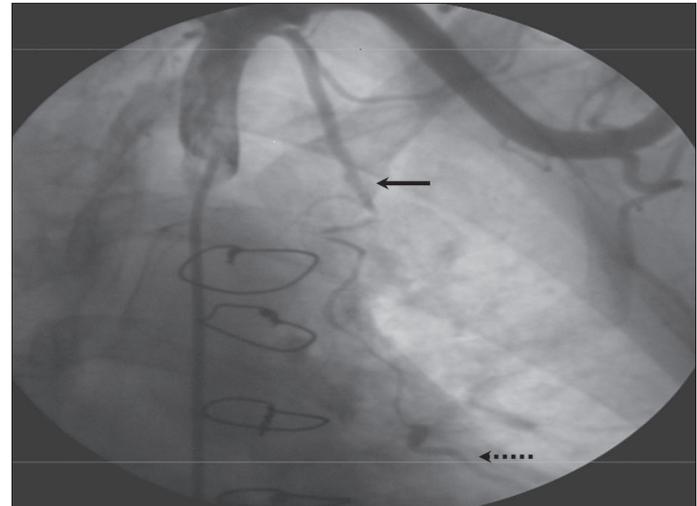


Figure 1. Diffuse narrowing of the distal half of the LITA graft on angiogram (right anterior oblique view, black arrow and dashed arrow show LITA and LAD artery, respectively)

LAD - left anterior descending artery, LITA- left internal thoracic artery

anti-neutrophil cytoplasmic antibodies (ANCA) was studied and all were negative. Patient refused any further diagnostic investigation.

Discussion

Due to the intrinsic structural properties, LITA graft is known to be resistant to atherosclerosis. Significant atherosclerosis narrowing has been shown to involve less than 5% of LITA grafts in the long term (1). Fibrous intimal hyperplasia, around the anastomoses, as an adaptive mechanism to normal conditions of flow or wall tension, develops in all grafts and was proposed to be an important factor in the failure of LITA grafts in the long term (1). Aside from FIH and atherosclerosis, there is only one previous report of systemic arterial disease affecting the LITA in the literature. Fishbein et al (2), reported chronic arteritis and cystic medial necrosis pattern in the LITA grafts of four patients undergoing CABG.

To our knowledge, this is the first report demonstrating the eosinophilic periarteritis in the LITA graft after CABG. Eosinophilic

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Figure 2. A. Photomicrography of the distal LITA (anastomotic region) demonstrating the mild to moderate FIH (black bar), which was defined as 2 to 2.5 times the normal intimal fibrous thickening causing less than 50% luminal narrowing. Arrows indicate the internal elastic laminae (Trichrome staining, original magnification, X40). B. Photomicrography of the LITA wall demonstrating a diffuse inflammatory infiltrate from the media to the periadventitial tissue. There were well preserved endothelial cells. The internal elastic membrane, elastic and elastomuscular tissue in the media appeared to be preserved. Neither granuloma nor fibrinoid necrosis was detected. Also, there were no atherosclerotic alterations (hematoxylin and eosin, original magnification, X100). C. Photomicrography demonstrating the adventitial inflammation with an eosinophilic predominance (hematoxylin and eosin, original magnification, X200)

LITA- left internal thoracic artery

vasculitis is usually seen with some systemic vasculitic diseases such as Churg-Strauss syndrome or Wegener's granulomatosis. In this case the clinical and histopathological findings did not fulfill the criteria of eosinophilic vasculitis in which there should be damage to the elastic fibers or fibrinoid necrosis concomitant to eosinophilic cellular infiltration.

The entity of eosinophilic periarteritis as an isolated finding was previously described in coronary arteries. Kajihara et al (3) and Taire et

al. (4) reported two sudden cardiac death cases and eosinophilic periarteritis in the LAD and RCA was the only pathologic finding without any coronary obstruction or atherosclerosis in the autopsy of these cases. They speculated that coronary vasospasm induced by the adventitial inflammation could be a cause for death in these patients. It is proved that long term stimulation of the adventitia with some inflammatory cytokines such as platelet activating factor and leukotriene-C4 results in vasospasm and neointimal proliferation in coronary arteries (5).

Conclusion

Overall, eosinophilic inflammation in the adventitia of the LITA graft may be an important factor in some patients for the failure of LITA graft by causing a chronic vasospasm or accelerating underlying FIH.

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Nonobstructive membrane of the left atrial appendage

Sol atriyum apendiksinde nonobstrüktif membran

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Introduction

The left atrial appendage (LAA) is a small, muscular extension of the left atrium. It is located anterolaterally and lies in the left atrioventricular sulcus, superior to the proximal portion of the left circumflex artery (1).

The membranes of the LAA cavity are very rare. The origin of membranes involving the LAA, and their clinical significance is not clear (2). To our knowledge, only six cases of LAA membrane have been

described to date. In this report, we describe a case with nonobstructive membrane within the body of LAA and discuss the transesophageal images mimicking a membrane in the body of LAA.

Case Report

A 69-year-old female presented with fatigue and worsening palpitations at rest. A 12-lead electrocardiogram showed atrial flutter

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