

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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with rapid ventricular rate. The patient had received a permanent DDD pacemaker in our department 2 months earlier because of nodal rhythm. Coronary angiogram was normal. Her electrocardiogram showed intermittent sinus rhythm and some pacing beats during follow up. Cardioversion was contemplated for atrial flutter which was probably recent-onset. A 2-D echocardiogram was normal except for slightly dilated left atrium. A transesophageal echocardiography (TEE), showed that LAA was free of thrombus. Imaging of the LAA in multiple planes demonstrated a thin, linear, mobile membrane traversing the body of the LAA (Fig 1). Color Doppler did not show flow acceleration across this membrane (Fig. 2). Pulsed-wave Doppler confirmed low flow velocities across the membrane (Fig 3), indicating no obstruction.

Discussion

Further developments in imaging techniques and the use of biplane and multiplane TEE have allowed visualization of the LAA, which

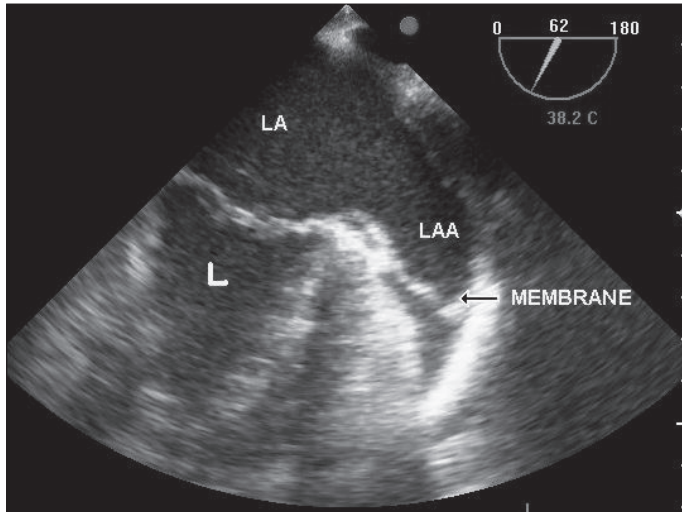


Figure 1. Transesophageal (2-chamber view) images of membrane of left atrial appendage

L - left ventricle, LA - left atrium, LAA - left atrial appendage

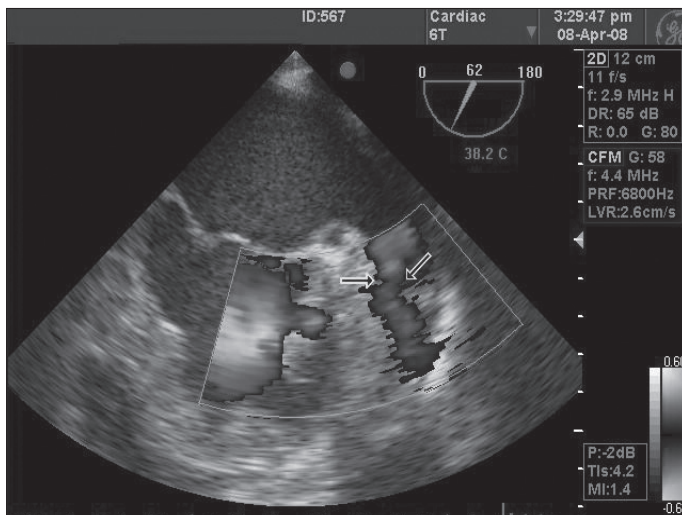


Figure 2. Color flow Doppler image of absence of flow acceleration across the membranes

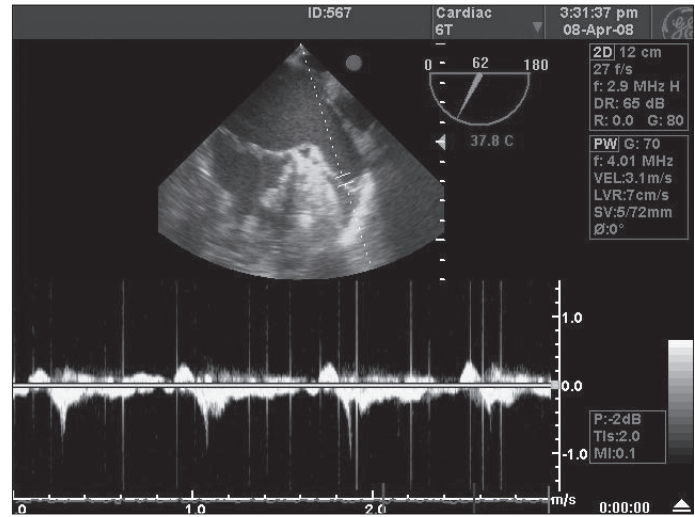


Figure 3. Pulsed-wave Doppler image of low flow velocities across the membrane

previously was difficult to demonstrate by other imaging methods. Accuracy of LAA thrombus detection with TEE is important in the pre-cardioversion evaluation of patients with atrial fibrillation and flutter (1).

Several studies have emphasized that LAAs vary in volume and shape, and are often composed of multiple lobes (3, 4). To our knowledge, very few cases of a membrane involving the LAA have been described (2, 5-8). In two reports (5, 6), obstructing membranes at the opening of the LAA, causing functional stenosis have been described and in four reports nonobstructive membranes located in the body of the LAA have previously been presented (2, 7). Similar to previous findings, we presented a case that has a thin mobile membrane-like structure across the LAA cavity. It does not cause an obstruction in the LAA cavity as demonstrated by normal flow velocities on pulsed-wave Doppler of the LAA and by a lack of turbulence with color flow Doppler. As emphasized by Coughlan et al. (5) the origin of membranes in LAA is not clear. The most likely explanation for the origin of these membranes would appear to be a congenital anatomic variation. Previous reports after incomplete surgical ligation or recanalization of the LAA have emphasized the potential for stagnant blood flow within the LAA and possible thrombus risk with systemic embolization (9, 10).

The differential diagnosis of linear structures which appear in LAA may also include prominent pectinate muscles, side lobe artifacts and partial resorption of prior LAA thrombi. Most LAAs (97%) had pectinate muscles ≥ 1 mm in width. Small pectinate muscles (< 1 mm (3%)) were noted only in the first and last decades (1). In our case there were no imaging characteristics of prominent pectinate muscles. Correale et al. (2) presented a case of membrane-like structure which seems to be the roof of LAA cavity and the echo-free space below might be localized pericardial fluid within the pericardial sinus. They suggest that this situation should be taken into account in differential diagnosis of linear structures appearing within the LAA. In our case there was no pericardial effusion. Limitation of our case includes the lack of surgical confirmation and the lack of detailed pathologic analysis of the excised membrane. In our case, there was no thrombus in LAA, and the patient was cardioverted without any complications and she was discharged in sinus rhythm and in good condition.

Conclusion

We described, in one case, pre-cardioversion TEE findings of a thin, linear, mobile, and nonobstructive membrane within the cavity of the LAA. The clinical implications and origins of these kinds of membranes are not clear; however, they may represent an anatomic variant. The echocardiographer should pay attention to the LAA during examination.

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Double etiology of recurrent thrombophlebitis: Behçet's disease and inferior vena cava agenesis

Tekrarlayan tromboflebit çift etiolojisi: Behçet hastalığı ve vena kava inferior agenizisi

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Introduction

Behçet's disease (BD), a systemic vasculitis with unknown origin, mostly involves vascular lesion (1). Thrombosis was a commonly feature of the disease which it may affect large vessels, such as vena cava (2). There are different considerations about the pathogenesis of the vascular complications and the tendency for thrombosis in BD. However, less knowledge considering vascular malformations was clarified. Absence of the inferior vena cava (IVC) which is an uncommon vascular anomaly is exceptionally associated to BD. In this field, we report a case.

Case report

A 36-year-old man was admitted because of the fifth episode of left leg thrombophlebitis. He had suffered from recurrent erythema nodosum, oral and scrotal ulcers for five years. Since one year, when he developed a bilateral pan uveitis, he had diagnosed as Behçet's

disease and treated by colchicine, platelet suppressive agent and over dose of corticosteroid. He fulfilled all criteria of International Study Group of Behçet's disease (3) and he had positive HLA B 51. Despite regular intake of his treatment, he had noticed engorgement of his left leg and he was hospitalised for further exploration. On admission, the classical signs of poor venous drainage were present. There were several oral and scrotal aphthous showing a BD flare.

Ultrasonography examination showed acute deep venous thrombosis extending from the popliteal to the distal external iliac vein. Thrombophilia testing was in normal value; it included antithrombin III, protein C, protein S, homocysteine, activated protein C resistance test, and presence of antiphospholipid antibodies. Importance of collateral superficial vein of chest, abdomen and lower limb led to practice thoraco-abdominal angiographic tomography to search vena cava obstruction. It showed absence of retro-hepatic portion of inferior vena cava (Fig. 1) and developed collateral veins. No visceral malformations were detected. Heart sonographic exploration was normal. Treatment was consisting in low molecular weight heparin for a week associated with adjusting dose of acenocoumarol. No place for surgical treatment

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