Left ventricular false tendon in a patient undergoing mitral valve replacement

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Left ventricular false tendons (LVFTs) are thin, discrete, cord like fibromuscular bands that connect either two walls, both papillary muscles and papillary muscle to the interventricular septum, without connecting to the mitral leaflets.[1]

A 28-year-old female patient presented with a history of breathlessness on exertion and palpitation for 6 years. On examination, there was pansystolic murmur in the precordium. Her electrocardiogram was normal, and chest X-ray showed no cardiomegaly. Transthoracic echocardiography (TTE) revealed bileaflet prolapse with non-coaptation and posteriorly directed eccentric jet. Left atrium was dilated. Both the ventricles were normal in size and function. She was posted for mitral valve replacement. Intraoperative transesophageal echocardiogram (TEE) confirmed preoperative findings. There was a band like structure seen in the midcavity of left ventricle connecting the two walls (Figure: 1, Video: 1). Mitral valve replacement with 27ATS valve and false tendon (FT) excision was done under cardiopulmonary bypass with moderate hypothermic cardioplegic arrest. However, postoperative TTE showed that the midcavity FT was still intact (Figure: 3). Retrospective interpretation of intra operative TEE images showed the presence of two false tendons. Along with first FT in LV mid-cavity (Figure: 1, Video: 1), a second FT was faintly seen below the mitral valve (Figure: 2, Video: 2) coursing towards the left ventricular outlet. Only the second FT was excised by the surgeon. The patient recovered from the surgery uneventfully.

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False tendons are benign anatomic variants present in 40% of people.[2] When LVFTs are oriented perpendicular to the flow of blood, they vibrate like the strings of an Aeolian harp.[3] These vibrations are seen as fine fibrillations on M-mode images[Figure 3]. Patients with LVFTs are said to have violin heart as they frequently have musical systolic murmurs on precordial examination. Echocardiographically LVFTs can be differentiated from LV thrombus, LV hypertrophy, hypertrophic cardiomyopathy and trabeculations by the presence of echo-free space on both sides of the tendons. LVFTs have systolic laxity and become taut during diastole. LVFT may be cut by the surgeon as in our case or may rupture spontaneously or after an myocardial infarction, after percutaneous trans luminal mitral commissurotomy.[4] Ruptured LVFT must be differentiated from ruptured chordae, vegetations or thrombi.[5,6]

The presence of LVFT carries both merits and demerits. Its presence can prevent left ventricular (LV) remodeling. However it may be a causative factor in the development of discrete subaortic stenosis (DSS)

Left ventricular false tendons were first described by Sir Turner,[7] a British anatomist and surgeon who proposed that they retard LV enlargement. In dilated cardiomyopathy or after myocardial infarction, LV dilates due to remodelling. Dilated ventricle increases wall stress and leads to LV failure. Increased wall stress also increases metalloproteinase activity that degrades myocardial extracellular matrix causing further LV dilatation.[8] Transverse midcavity LVFTs absorb the distorting forces

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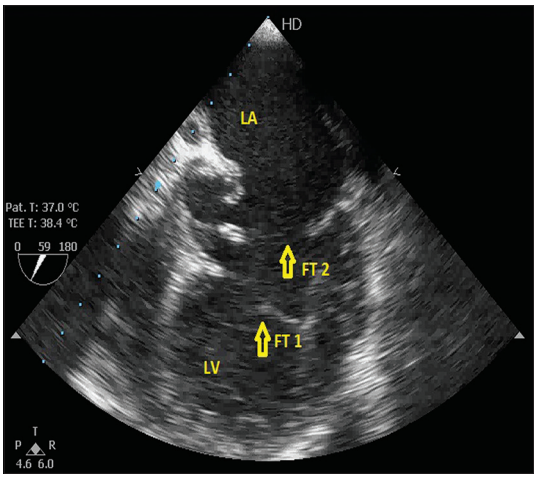

Figure 1: Mid esophageal commisural view with false tendon 1 (FT1) and false tendon (FT2)

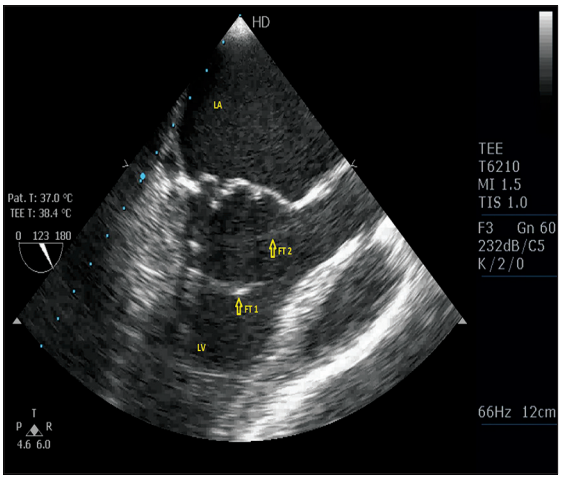

Figure 2: Mid esophageal aortic long axis view with FT1 and FT2

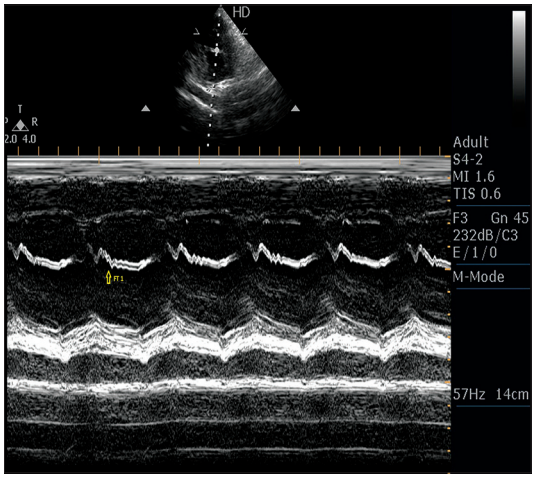

Figure 3: Trans thoracic Mmode showing FT1 with fine diastolic fluttering

resulting in less remodelling, less mitral annular dilatation and LVEDD. In patients with dilated cardiomyopathy, Coapsys device (Myocor, inc) is utilized to reduce the LV wall stress.[9] It is a flexible cord that is attached to the LV walls by epicardial pads and contains a cord that traverses the LV cavity anteroposteriorly. It is considered as a prosthetic false tendon. LVFT produces atrial natriuretic peptide(ANP) similar to left atrium when LV filling pressure are increased which promotes natriuresis, diuresis and vasodilatation resulting in reduced LV wall stress.[10] ANP also retards LV remodelling by inhibiting endothelins, angiotensin-II and aldosterone, all of which promote cardiac fibrosis and remodelling.[11] LVFT also produces ANP.

Dilated and ischemic cardiomyopathy cause functional mitral regurgitation (FMR) by LV remodelling. The papillary muscles are displaced away from the mitral annulus as measured by tenting height and area. Incomplete mitral coaptation occurs due to the traction on the chordae tendinae.[12] Coaptation device or surgical struts help to preserve the subvalvular anatomy and reduce the degree of FMR. In a study conducted by Bhatt *et al*., transversely oriented LVFTs were found to produce less FMR by having less coaptation depth and area.[13]

Left ventricular false tendons may contain Purkinje fibres,[14] which have a gating mechanism that may inhibit or promote re-entry from ventricular tachycardia.[15] In patients with dilated ventricles, conduction of the electrical impulse through LVFTs may produce a more homogenous LV depolarization similar to biventricular pacing and improve LV dP/dt.[16]

The presence of LVFTs in patients with DSS may add to the flow turbulence across the LVOT. Some surgeons excise LVFTs during DSS excision.[17]

In our patient, there were two false tendons, one was traversing LVOT and it was excised by the surgeon. The transverse midcavity tendon was left intact, which may protect the patient from future possibility of FMR. People with LVFTs may be at evolutionary advantage due to reduced LV remodelling after MI and reduced the degree of FMR.

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