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Case Report

Pathology Section

Calcified False Tendon Linked to Sudden Cardiac Death in a Young Adult: An Autopsy-based Case Report

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ABSTRACT

Left Ventricular False Tendons (LVFTs) are cord-like structures that traverse the cavity of the left ventricle, connecting the left ventricular free wall or papillary muscle and the ventricular septum without attachment to the mitral valve leaflets. They are incidentally found in autopsy heart specimens. False tendons are generally benign anatomic variants and can be associated with functional murmurs and electrocardiographic abnormalities. False tendons can also be a cause of ventricular arrhythmias. Two-dimensional echocardiography serves as a useful imaging modality in detecting false tendons. They should be differentiated from other entities such as thickened ventricular trabeculations, an accessory anterior mitral leaflet, thrombus, vegetation, and ventricular masses or tumours. Most LVFTs are transverse and are located in the apex. They are found to arise from the inner trabeculated layer of the myocardium. The exact prevalence of LVFTs remains unclear. Present report is a case of postmortem findings in the heart of a 34-year-old male who experienced a sudden collapse while walking after lunch. On gross examination, the left ventricular wall and the lower part of the interventricular septum showed focal grey-white areas. A well-defined, grey-white, hard polypoidal lesion was noted in the left ventricle near the apex, attached to the papillary muscles. The left anterior descending artery showed early atherosclerotic changes. LVFT was diagnosed, which could have been the reason for the arrhythmia. Hence, the evaluation of cardiac murmurs and arrhythmias by two-dimensional echocardiography and cardiac Magnetic Resonance Imaging (MRI) serves as helpful parameters in detecting LVFTs.

Keywords: Arrhythmia, Calcification, Cardiac hypertrophy, Echocardiography, Left ventricular false tendon, Murmur

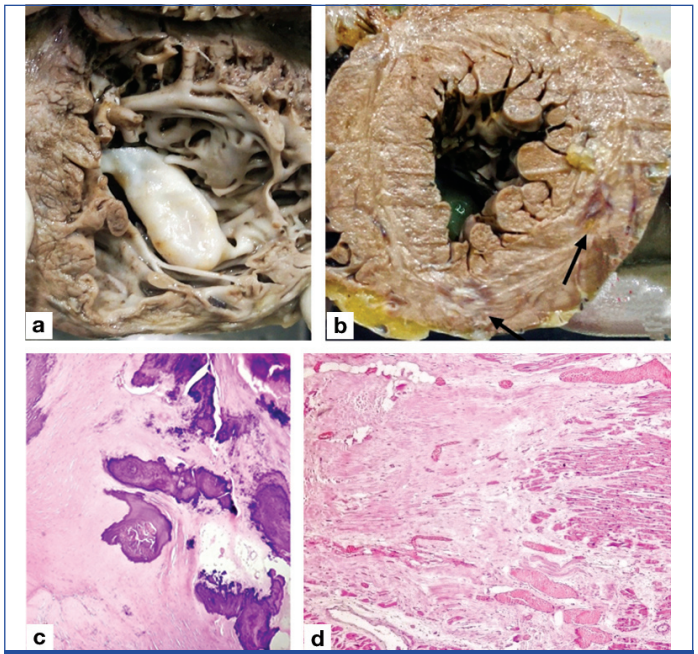
CASE REPORT

A 34-year-old male was brought to the emergency department by his relatives with a history of a sudden collapse while walking after lunch. On examination, his vital signs were absent, and he was declared dead. Past medical history revealed that he was a chronic smoker and alcoholic, with no other co-morbidities. After obtaining informed consent from the deceased relative, an autopsy was conducted by the forensic team, and the entire heart with a portion of the aorta was sent to our histopathology lab.

Gross examination of the heart: The heart weighed 326 grams, measuring 15 cm (apex to base)×14.5 cm (mediolateral)×7 cm (anteroposterior), and had a smooth pericardial surface. The heart was opened up and dissected using the short-axis method. A well-defined, grey-white, hard polypoidal band-like structure (possibly a false tendon, thickened papillary muscle, or trabeculae) was noted in the left ventricle near the apex, attached to the papillary muscles, measuring 2 cm×0.7 cm×0.5 cm [Table/Fig-1a]. Upon slicing the lesion, a gritty sensation was felt. The cut surface appeared grey-white and calcified. The left ventricular wall and the lower part of the interventricular septum showed focal grey-white areas (suspicious old healed infarcts) [Table/Fig-1b]. The left anterior descending artery and left circumflex artery showed atherosclerotic changes.

Histopathological examination: Sections from the white polypoidal lesion within the left ventricle revealed extensive areas of fibrosis with central dystrophic calcification [Table/Fig-1c]. Sections from the anterior and lateral walls of the left ventricle and interventricular septum showed areas of fibrosis (indicating old healed infarcts) [Table/Fig-1d].

The final diagnosis of left ventricular false tendon with calcification, along with early coronary artery disease, was made.



[Table/Fig-1]: a) Well-defined, grey white, hard polypoidal lesion (band like structure); b) Focal grey white areas (suspicious old healed infarcts); c) Extensive areas of fibrosis with central dystrophic calcification in sections from white polypoidal lesion (x400); d) Areas of fibrosis (old healed infarcts) (H&E x100).

DISCUSSION

LVFTs are fibrous or fibromuscular bands of varying length, number, and thickness that traverse the cavity of the left ventricle without attaching to the mitral valve leaflets [1,2]. LVFTs were first described by Sir William Turner, a British anatomist and surgeon, in 1893 [3,4]. They are also referred to as anomalous left ventricular cords, aberrant tendons, bands, moderator bands, and pseudotendons [5,6]. These bands can be attached to the septum, papillary muscles, or the free wall of the ventricle, but not to the mitral valve, hence the term “False Tendon” [5].

False tendons are commonly detected by two-dimensional echocardiography (2-D echocardiography) [1-3,5-12] and have

also been found as incidental findings in autopsies [2]. 2-D echocardiography not only provides clear visualisation of LVFTs but also provides dimensions of these structures. They appear as linear echoes running from the left ventricular free wall or from the papillary muscle of the mitral valve to the interventricular septum in the four-chamber approach with long-axis, upwards-downwards tilting of the ultrasonic beam [1].

Embryologically, the heart is composed of two myocardial layers: an outer condensed layer and an inner, less compact layer with trabeculations [11]. LVFTs arise from the inner trabeculated myocardial layer [11,12]. They are more common in males and occur with equal frequency in both normal hearts and those with congenital malformations. They are usually located in the apex, mid, or basal third segment along the axis of the left ventricle. Based on their location, LVFTs can be classified as transverse (localised to one zone), diagonal (extending across two zones), or longitudinal (extending across all three zones) [5,9]. Most LVFTs are transverse and located in the apex, as was the finding in the present case.

LVFTs are considered normal anatomical structures and are generally considered benign anatomic variants [5,6]. Their thickness ranges upto about 3 mm [9]. They can be associated with precordial murmurs, Electrocardiogram (ECG) repolarisation abnormalities like giant T-wave inversion, pre-excitation ventricular premature beats and/or arrhythmias, mitral regurgitation, systolic and diastolic dysfunction, and a dilated left ventricle [2,5,9,10,12]. LV false tendons have also been implicated in helping membrane formation in Discrete Subaortic Stenosis (DSS) [11].

A study by Plácido R et al., in elderly individuals showed that calcium deposits in the heart are more common and are found in association with coronary artery disease, dilated cardiomyopathy, aorto-mitral valvular disease, and renal disease [8]. LVFTs can be associated with a functional murmur and may show significant electrocardiogram abnormalities in patients with congenital heart disease [2].

The prevalence of LVFTs remains unclear, as different identification methods have shown a wide range of prevalence, ranging from 0.4-68% [1,6,7,12]. Recent advancements in echocardiographic technologies and cardiac MRI have facilitated the detection of LVFTs [5].

A community-based, prospective screening study by Kenchaiah S et al., on 3931 eligible individuals between 1987 and 1990 demonstrated the presence of LVFTs in 101 patients [9]. Hall ME et al., conducted a study in the general population, which showed the presence of LVFTs in 15 out of 100 matched controls [10]. Luetmer PH et al., studied the incidence and distribution of LVFTs in 483 human autopsy heart specimens, where 55% of hearts showed the presence of LVFTs [6].

Histopathological features of LVFTs include fibrous tissues, myocardial fibres, elastic and connective tissue fibres, conductive tissue, blood vessels, and calcifications in the elderly. The presence of conductive tissue in the bands serves as a source of ventricular arrhythmias [2,3,10].

Mimickers of LVFTs include thickened ventricular trabeculations, discrete subaortic membrane, intraventricular masses or tumours, an accessory anterior mitral leaflet, thrombus, vegetation, and a flail mitral valve with ruptured chordae tendineae [3,7].

LVFTs are rare findings that can present with sudden cardiac death due to arrhythmias, particularly in the younger population. Therefore, awareness of LVFTs is necessary for treating physicians to evaluate such presentations with advanced echocardiography and cardiac MRI to prevent sudden cardiac death.

CONCLUSION(S)

LVFTs are anatomical variants of the normal human left ventricle and can be mistaken for a papillary muscle, thrombus, trabeculation, vegetation, flail aortic valve, or parachute accessory anterior mitral leaflet. Hence, a careful understanding of their morphology and existence may be helpful in correlating them with cardiac murmurs and arrhythmias. In the present case, the probable cause of death could be due to arrhythmia produced by the LVFT and early coronary artery disease.

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