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CAUGHT IN THE WEB; A CASE REPORT OF LEFT VENTRICLE FALSE TENDON (LVFT) IN A 15 YEAR OLD BOY

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Abstract: **One of the (unintended) outcomes of echocardiography is the increasing recognition of small or thin intracardiac structures previously seen only on autopsy. Examples of this are the nodules of Arantius, Lambl's excrescences, eustachian valve remnants, the crista terminalis, and the discrete fibromuscular structures known as left ventricular false tendons (LVFTs). Left ventricle false tendon (LVFT) is a rare structural anomaly of the heart, characterized by fibrous strands within the left ventricular cavity. It is usually considered a benign finding; however, it can occasionally be associated with clinical symptoms. We present a case of LVFT in a 15-year-old boy, highlighting the importance of thorough evaluation in young individuals with atypical cardiac presentations.**

Key Words: **Left ventricle false tendons (LVFT), arrhythmias, sudden cardiac death (SCD), Congenital Heart Disease (CHD), benign early repolarisation (BER).**

1. CASE SUMMARY

Presenting illness and physical examination

A 15 year old boy from Nakuru County, Rift Valley province Kenya, presented to us for cardiology review from a peripheral facility with history of sporadic palpitations, exercise intolerance and back pain for 2 years. He reported that he was not able to participate in competitive sporting activities at the same level with his peers due to easy fatigability and vague non specific lower back pain. He did not experience chest pain, syncope or pre syncope symptoms. There was no history of shortness of breath at rest or on ordinary activity. His family history was unremarkable for any significant acquired/congenital heart disease or sudden cardiac death. His current medications included monthly Benzathine Penicillin prescribed in a peripheral facility ostensibly due to a positive anti-streptolysin O qualitative test.

Clinically, he was a pleasant adolescent in no obvious distress. He was not pale and all other clinical parameters were normal. His pulse rate was 72b/min and blood pressure was 107/68 mmhg. He was afebrile with a temperature of 36.9 degrees Celsius. His cardiovascular examination was unremarkable with regular S1 and S2 heart sounds and no murmur. There was no obvious chest dysmorphology. Peripheral vascular examination was normal. The rest of the systemic examination was also normal except for mild thoracolumbar paraspinal tenderness with no apparent spinal dysraphism.

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Diagnostic workup

His basic work up at the referring facility included a complete blood count which was unremarkable and a positive qualitative anti streptolysin O titre. A plain thoracolumbar radiograph and MRI had also been performed at the referring facility and showed mild loss of normal lumbar lordosis due to muscle spasms. A resting 12 lead ECG and bedside point-of-care cardiac ultrasound was requested at our facility. The ECG showed normal sinus rhythm with elevated J point in V2 and V3 suggestive of benign early repolarisation (BER). Cardiac ultrasound showed a thin fibrous strand extending transversely from the posteromedial papillary muscle to the basal interventricular septum. Cardiac systolic function was preserved in both ventricles. Situs was solitus with levocardia position, concordant arterioventricular, ventriculoatrial and atriovenous connections. There were no echocardiographic features of rheumatic fever or its long term consequence, rheumatic heart disease.

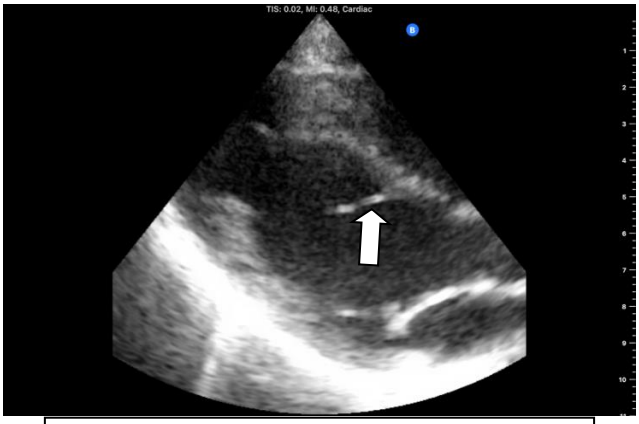

Fig 1; Parasternal long axis view of LVFT.

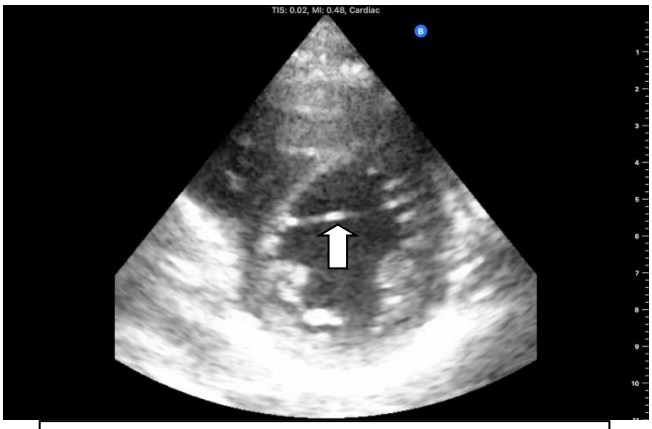

Fig 2; View of the LVFT from Parasternal short axis

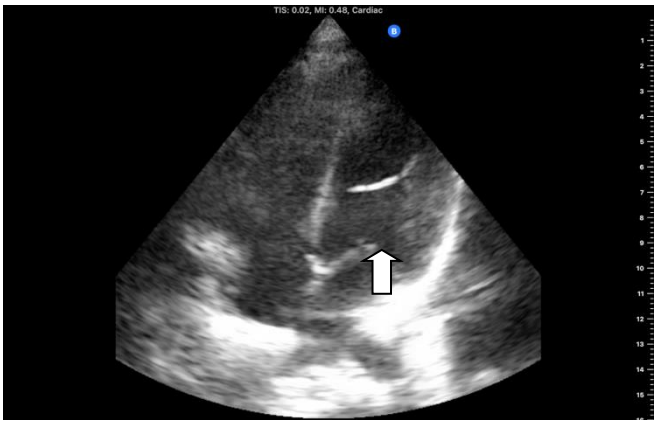

Fig 3; Modified apical 4 chamber view of the LVFT

Fig 4; resting 12 lead ECG showing a normal sinus rhythm and elevated J point in V2, V3 suggestive of benign early reporalisation (BER)

Management and follow-up

The patient was diagnosed with left ventricular false tendon (LVFT) with benign early repolarisation (BER) and mechanical lower back pain (LBP). Advanced cardiac diagnostics such as cardiac MRI (CMR), exercise stress testing (EST), cardiopulmonary exercise testing (CPET) and ambulatory cardiac monitoring were deemed unnecessary in the patient's current clinical context and were therefore not requested. Mechanical lower back pain was managed conservatively with rest and oral paracetamol only when necessary. No sporting restrictions were imposed on the patient. Within one month of follow up, the patient reported improved exercise tolerance as the back pain resolved and he did not report any palpitations, pre-syncope or syncope symptoms.

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2. DISCUSSION

Left ventricular false tendons (LVFTs) are fibromuscular structures, connecting the left ventricular free wall or papillary muscle and the ventricular septum. These structures span the left ventricular cavity to connect distant sites; on an endocardium, these structures appear beside the chordae tendineae (Velthius S, 2021). In general, LVFTs contain conductive tissue; an exception is the less common, thinner, oyster-white LVFT, which consists entirely of dense connective tissue. A few myocardial cells are diffusely distributed within LVFTs (Lotkowski D, 1997). Turner first described LVFTs by autopsy, but the role of LVFTs was not investigated due to the lack of noninvasive equipment. The application of ultrasound diagnostic instruments in medicine has made it possible to detect LVFTs noninvasively. Although LVFTs can be readily identified with routine two-dimensional echocardiography, the echocardiographic detection rates vary widely from 0.5–70% due to several factors, including operator skill, the equipment used, and knowledge of these phenomena. Thus, there is a need for a large-scale investigation of the prevalence of LVFTs (Liu Y, 2015).

Although first described more than 100 years ago, the pathophysiological significance of these structures remains unclear. There is some discussion about safety issues during intense exercise in athletes with LVFTs, as these bands have been associated with ventricular arrhythmias and abnormal cardiac remodelling. However, presence of LVFTs appears to be much more common than previously noted as imaging techniques have improved and the association between LVFTs and abnormal remodelling could very well be explained by better visibility in a dilated left ventricular lumen. While LVFTs may result in electrocardiographic abnormalities and could form a substrate for ventricular arrhythmias, it should be considered as a normal anatomic variant. Persons with LVFTs do not appear to have increased risk for ventricular arrhythmias or sudden cardiac death. An LVFT from the basal IVS to the apical segment of the left ventricular free wall may be a normal anatomical structure in the left ventricular cavity. On the contrary, transverse false tendons in the left ventricular cavity as is the case with our patient, may be associated with BER (Liu Y, 2015). Salazar reported a child with considerable electrocardiographic repolarization who showed an elevated ST segment with a negative T wave in the anterolateral and inferior leads. The patient was diagnosed with an anomalous LVFT that was 6 mm in diameter (Salazar, 1997).

LVFTs could also complicate with endocarditis as described in a case report of an 80-year old male with extended-spectrum beta-lactamase *Escherichia coli* bacteremia who presented with a partially ruptured false tendon mimicking mural vegetation (Sah A, 2020). Additionally, caution should be taken in patient undergoing cardiac surgery for structural heart disease to avoid resection of LVFTs which may contain conductive tissue. In patients undergoing trans-catheter mitral valve interventions, presence of LVFTs may complicate delivery of devices or instruments (Darwish, 2023).

Some studies have suggested that false tendons reduce the severity of functional mitral regurgitation by stabilizing the position of the papillary muscles as the left ventricle enlarges. It has also been suggested that they retard LV remodeling by tethering the walls to which they are attached, but there are few data to substantiate this and therefore large scale studies are necessary to explore the pathophysiological and clinical significance of these structures (Silbiger, 2013).

In our patient the LVFT wass an incidental finding with only minor non specific clinical symptoms which may or may not be related to it. However due diligence was done to reasonably evaluate him for more significant potential cardiovascular findings.

3. CONCLUSION

Left ventricular false tendons (LVFTs) are intriguing fibromuscular structures that have garnered attention in the realm of cardiology for over a century. Although their presence was first documented through autopsies, advancements in noninvasive diagnostic tools, particularly ultrasound, have made it feasible to detect LVFTs in living patients. Nevertheless, the understanding of the pathophysiological significance of LVFTs remains a subject of ongoing exploration. The variability in detection rates of LVFTs in echocardiography (ranging from 0.5% to 70%) highlights the significance of operator skill, equipment quality, and awareness of these structures in influencing their identification. Current evidence suggests that LVFTs, although capable of causing electrocardiographic anomalies and potentially serving as substrates for ventricular arrhythmias, should be regarded as normal anatomic variants with no proven link to increased risk of sudden cardiac death. A more comprehensive understanding of these structures and their potential clinical implications is vital for enhancing

patient care and guiding future research in the field. Our patient will be monitored routinely in our outpatient clinic and further work up shall be conducted should the need to do so arise.

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INFORMED CONSENT

Written informed consent was obtained from the patient to publish this case report, including the images.

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