EDITORIAL

What's So False about a False Chord?

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I sat down to read a list of echo studies that had begun to pile up … as they tend to do. The first study was requested in a patient who had suffered a stroke. This study was essentially normal with normal heart size and left ventricular (LV) systolic function. As part of my routine approach to evaluating and reporting echo studies performed in patients with acute strokes, I added a series of pertinent negatives, including that there was no valve disease, the left atrial volume index was normal, the bubble study was negative (at rest and Valsalva), and the rhythm was regular sinus. I carefully evaluated the LV apex to confirm that there was no LV thrombus, and it was during this careful apical review that I bore witness to a thin, filamentous structure stretching across the distal third of the left ventricle (LV). This anatomic structure was well defined and not an artifact. It connected the apical anterolateral wall segment with the midinferoseptal wall segment. It was taut in diastole and lax in systole. It did not connect to the mitral valve, nor did it involve the mitral subvalvular apparatus. It did not interfere with the laminar color-flow Doppler pattern. I concluded that this was an aberrant chordae tendineae (aka a false chord) that I was seeing.

The next echo was requested in a patient with fever, bacteremia, and suspected endocarditis. Again, no findings of valvular heart disease or suspected endocarditis, but there was a clearly demonstrated false chord stretching across the midanterior to the midinferior wall segments (Figure 1), which, of course, I mentioned in my report.

The third echo of the day was requested for a loud systolic murmur in a patient being assessed preoperatively. This patient had a small LV with LV hypertrophy and mildly reduced LV systolic function. The murmur was from aortic valve sclerosis. There again, seen as I carefully reviewed the apical views, was that recurring normal variant—a *false chord*. In fact, this patient actually had two diverging chords stretching from the apical lateral wall to the mid LV septum. Or maybe *from* the septum *to* the lateral wall, who really knows?

All totaled, after my first 10 studies of the day, I had reported the finding of 7 false chords (actually 8 chords in 7 patients). This 70% prevalence started me thinking about what is *false* about something I was seeing so commonly? Of course, I've been known to ponder excessively over questions such as this. For example, "Where is the *paradox* in *pulsus paradoxus*?" and "Why is the most *typical* chest pain presentation considered *atypical*?" But I will save those questions for a future editorial.

In my opinion, the best review of these structures was published over a decade ago in *JASE*.[1] I recommend you read that review to learn more about their proposed embryologic origin and the variation in phenotypic appearance and check out some of the gross pathologic examples. Citing multiple sources, an incidence of ~50% can be found. In an autopsy report of nearly 500 human hearts, false chords were found in 55%. In addition, there is a slightly increased likelihood of finding these in men, where the incidence exceeds 60%.[2]

So now when you ask yourself what's false about false chords, it becomes clearer that the untrue (aka false) portion of that nomenclature is the "chord" component. The term "chord" is actually a misnomer and should probably be "cord." In music, a chord is typically defined as three or more notes sounded together, whereas cord is conventionally defined as a thin, flexible string (e.g., umbilical cord, spinal cord, etc.).[3] Since chordae (plural form of *chord*) are anatomic tendineae (plural form of tendon) and a tendon is commonly defined as a flexible but inelastic cord of strong fibrous collagen tissue, the term *false chord* begins to make sense.[4] Chordae tendineae are attached to the mitral (or tricuspid) valve, but these "false" *chords* do not involve the valve apparatus or interact in any way with valve function. They may be single or multiple and have many variations in exact location. Importantly, they are very common. In fact, based on my review, it would be more unique *not* to see a false chord since they occur in >50% of all autopsy findings. Referring to these as "normal variants" or "normal anatomic variants" is a correct statement, but given the complexity of echocardiography, we should weigh the value of extraneous reporting against succinct, clinically meaningful reporting.

Although beyond the scope of this editorial, some have questioned the clinical impact of these commonly found structures. In a review on *false tendons*, the authors reported (retrospectively) that these structures were associated with LV hypertrophy, LV dilation, and arrhythmias.[5] Their proposed mechanism for these structures being pathologic rather than benign was based on their suspicion that these structures cause an alteration of blood flow. From my experience, this is not very likely. Also, in that study, the authors reported finding false tendons in 111 patients over a 2-year period during which they performed ~20,000 exams. This was in 2014 using a nearly 10-year-old ultrasound platform, and it demonstrates how infrequent this finding was only one decade earlier.

I realize that the reason I started this editorial was the frequency with which I was seeing these normal variants. With limited research, however, I was able to confirm that my frequency was not excessive but was previously demonstrated in autopsy studies. So why did this seem to me to be at an increased frequency? I assume it may have something to do with the improved image quality of today's ultrasound systems. I suggest that as our imaging capabilities continue to improve, our reporting should also evolve to address these structures we are now seeing with increased frequency. It is important that our interpretations match the ability we now possess to detect subtle anatomic variations that were previously hidden. Certain normal anatomic variations may mimic diseases if not carefully considered. Some examples may include normal LV trabeculation (mistakenly reported as noncompaction cardiomyopathy), normal mitral valve anatomy (erroneously reported as mitral valve prolapse), and even aortic valve imperfections from nodules of Arantius (incorrectly reported as endocarditis). Misinterpreting these normal structures (previously unseen with older ultrasound technology) as pathology has the consequential risk of unnecessary downstream testing with associated potential complications.

In this issue of *CASE*, there are many patient case examples provided to help guide you in your daily echo lab clinical practice.

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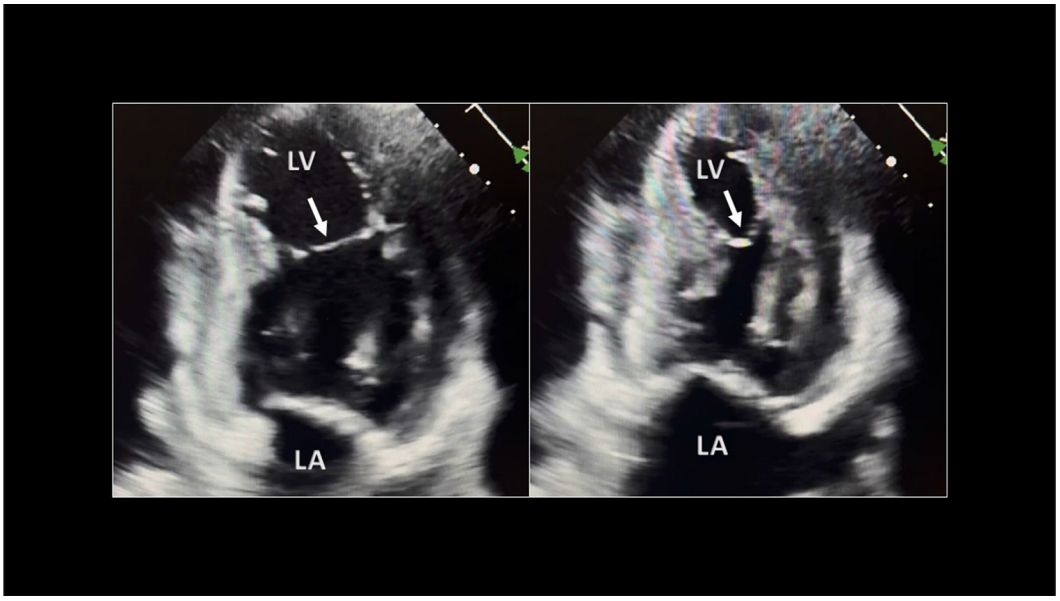

Figure 1 Two-dimensional transthoracic echocardiography, apical 2-chamber view, diastolic (*left*) and systolic (*right*) frame, demonstrates normal LV size and function with a visible, echo-bright linear false chord (*arrow*) connecting the anterior to the inferior midwall segment. The chord is taut in diastole and lax (bowed) in systole. *LA*, Left atrium.

Hackett *et al.* demonstrate the expertise required to interpret fetal echocardiography and report how easy it is to misinterpret a fetal mass. Their report adds another possible but rare cause for the discovery of an atrial mass on fetal echocardiography. In another congenital heart case, Akingbade *et al.* present a very cleverly titled report that beautifully demonstrates the possibilities you may find in patients who present with dilated right hearts. Their figures and videos are well worth your time to review. Dow *et al.* report on the important strengths and weaknesses of multimodality imaging for the investigation of rare pericardial tumors. Their report includes transthoracic echocardiography, transesophageal echocardiography, invasive coronary angiography, cardiac magnetic resonance imaging, cardiac computed tomography, positron emission tomography-computed tomography, and both gross and histologic pathology. Mendez-Ruiz *et al.* report a pregnant patient found to have an embolized atrial septal defect closure device that had been placed years earlier. The device was in the pulmonary artery, and they managed the patient conservatively through pregnancy. Ghantous *et al.* report on 3 patients with impressively large right heart masses that resolved with treatment. Their serial imaging is simply eye-opening and offers hope to many of us who would not have believed complete cardiac resolution was even a possibility after seeing the initial echocardiograms. In a very similar report, using serial echo and positron emission tomography-computed tomography, Frederiksen *et al.* demonstrate complete resolution of a large mass located in the LV outflow tract that represented cardiac metastases from melanoma. These serial imaging studies provide visual evidence of the value of the radio-targeted molecular and immune therapies available today. Lastly, in a letter to the editor, Vainrib *et al.* offer additional three-dimensional echocardiography insights to help us when we are investigating the left atrial appendage.

So, the next time you see a heart tugging on its strings, it's #JADEL (just another day in the echo lab)! False chords are "real," and they are common. But they are not chordae tendineae and have no connection to the atrioventricular valves. And if you happen to live in Ohio, the Ohio Craft Brewers Association established the False Chord Brewing company in 2020. And bringing this all back to echocardiography, or at least closer to the heart, the official State of Ohio website is called "The Heart of It All" and includes a summary of the False Chord microbrewery (https://ohio.org/things-to-do/destinations/false-chord-brewing).

Remember, every echocardiogram you see today has a teaching point, and every teaching point is a potential new CASE report.

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