

**IMAGES IN EMERGENCY MEDICINE**

Cardiovascular

# Bedside cardiac ultrasound evaluation of a young male with chest pain

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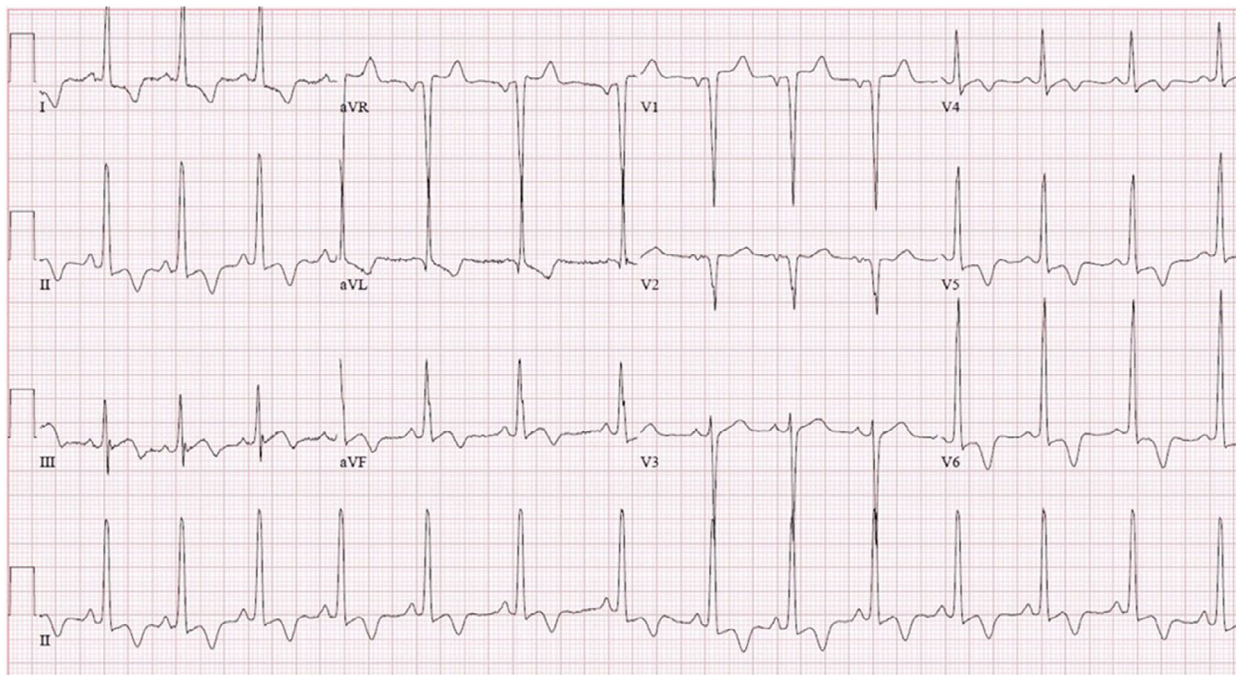
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## 1 | CASE PRESENTATION

A 26-year-old man without a past medical history presented to the emergency department (ED) for evaluation of a brief self-resolving

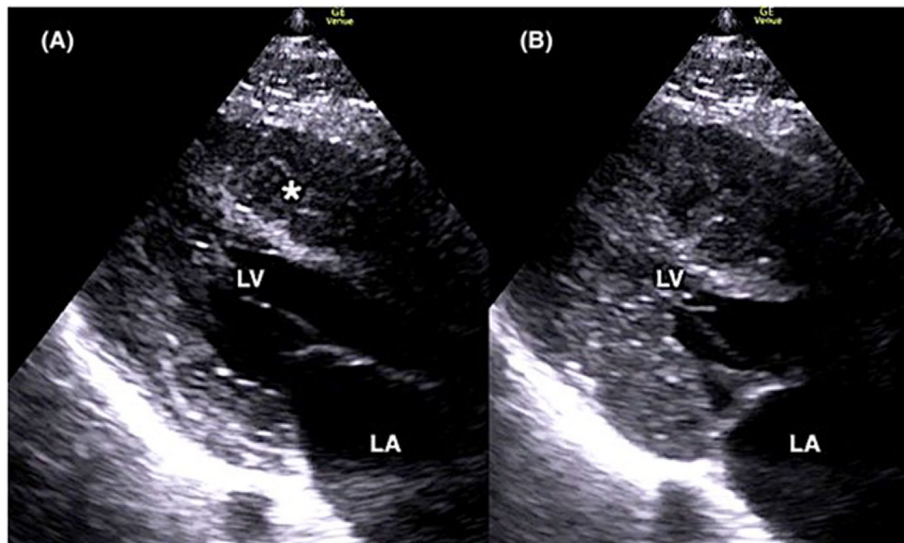
episode of chest pain the day prior described as substernal, non-radiating, non-exertional, and pressure-like. He denied any associated palpitations, dyspnea, or syncope. There was no known family history of sudden cardiac death. Vital signs and physical examination



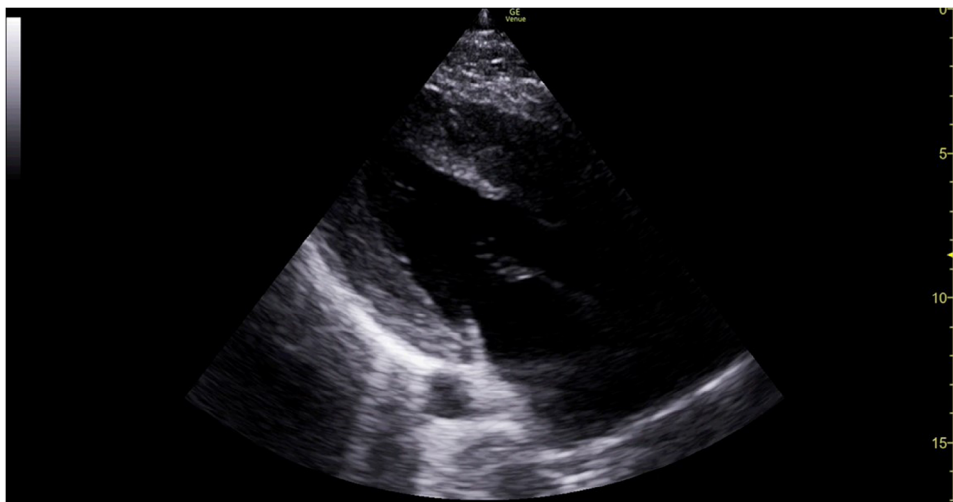
**FIGURE 1** ECG demonstrating normal sinus rhythm, LVH, and T-wave inversions of inferior-lateral leads. ECG, electrocardiogram, LVH, left ventricular hypertrophy, TWI, T-wave inversions.

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**FIGURE 2** Bedside transthoracic echocardiography demonstrating asymmetric left ventricular wall hypertrophy (asterisk) in the parasternal long-axis window during diastole (A) and mid-cavity obliteration during systole (B). LA, left atrium; LV, left ventricle.



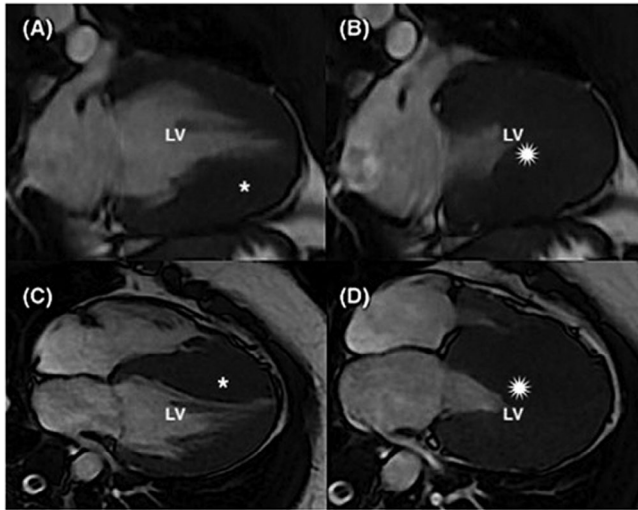
**VIDEO 1** Bedside transthoracic echocardiography demonstrating asymmetric left ventricular wall hypertrophy in the parasternal long-axis window during diastole and mid-cavity obliteration during systole.

were unremarkable. An electrocardiogram (ECG) demonstrated normal sinus rhythm with notable left ventricular (LV) hypertrophy (LVH) and T-wave inversions in the inferior and lateral leads (Figure 1). Focused cardiac ultrasound (FOCUS) demonstrated a preserved LV ejection fraction without pericardial effusion or signs of right ventricular strain. FOCUS was notable for asymmetric LVH with maximal wall thickness in end diastole of 24 mm and dynamic mid-cavity obliteration (Figure 2, Video 1). Ultimately, the patient was admitted for further cardiology evaluation and cardiac magnetic resonance (CMR) imaging (Figure 3), which confirmed apical hypertrophic cardiomyopathy (ApHCM).

## 2 | DIAGNOSIS

### 2.1 | Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a genetic cardiovascular disorder with multiple variants of LVH that may clinically manifest as arrhythmias, heart failure, and/or sudden cardiac death.<sup>1</sup> This case describes the rare HCM subtype, ApHCM, which accounts for about only 8% all HCM cases and carries annual mortality rates of 0.5%–4%.<sup>1</sup> ApHCM can be undetected by echocardiography alone in 40% of cases; therefore, additional imaging modalities, such as angiography



**FIGURE 3** Cardiovascular magnetic resonance (CMR) demonstrating asymmetric LV hypertrophy (asterisks) of hypertrophic cardiomyopathy in 2-chamber Long axis during diastole (A) and systole (B). CMR of hypertrophic cardiomyopathy in 4-chamber long axis during diastole (C) and systole (D). Left ventricular mid-cavity obliteration (stars) demonstrated during systole (B, D). LV, left ventricle.

and CMR, are often warranted.<sup>1-3</sup> The echocardiographic diagnostic criteria for ApHCM that emergency physicians (EPs) should be familiar with include the following: asymmetric LVH with LV wall thickness  $\geq 15$  mm during diastole and a ratio of maximal apical to posterior wall thickness  $\geq 1.5$ .<sup>3</sup>

FOCUS is a critical bedside tool for EPs in the evaluation of potential life-threatening cardiovascular emergencies.<sup>4</sup> Although it has been previously demonstrated that ultrasound-trained EPs can distinguish more advanced echocardiographic pathologies, timely and accurate diagnosis of HCM remains challenging.<sup>5</sup> FOCUS was instrumental to our case in providing early identification of underlying

HCM. Notably, echocardiographic screening for HCM in youth athletes by novice sonographers has provided some encouraging evidence to support further point-of-care ultrasound use.<sup>6</sup> This report highlights that expanding the scope of EP-performed FOCUS to include targeted echocardiographic screening of ED patients for HCM may have significant clinical impacts.

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