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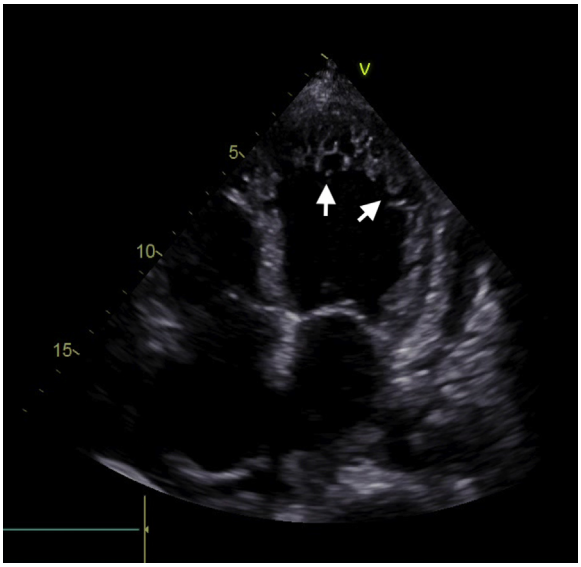

Figure 1. Apical 4-chamber view of the left ventricle, demonstrating prominent trabeculation at the apical and lateral walls (arrows).

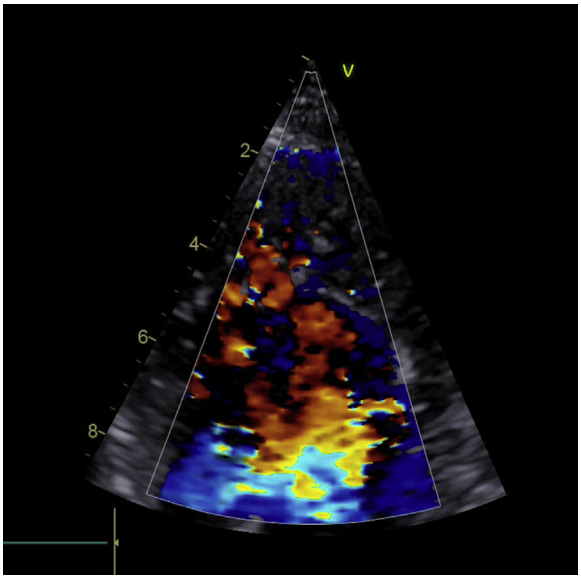

Figure 2. Blood flow in and out of the trabeculae, visible on color flow imaging.

[Ann Emerg Med. 2019;74:186.]

An 87-year-old woman presented with dyspnea, orthopnea, and ankle edema for 3 weeks. Medical history included diabetes mellitus, hypertension, and ischemic stroke. Blood pressure was 110/70 mm Hg and pulse rate was 86 beats/min. Jugular venous pressure was elevated. ECG showed sinus rhythm and new-onset left bundle branch block. Chest radiograph result was consistent with pulmonary congestion and right-sided pleural effusion. Troponin T level was 54 ng/L (reference <14 ng/L), similar to her baseline level. Bedside echocardiography was performed (Figures 1 and 2, Video E1, available online at http://www.annemergmed.com).

For the diagnosis and teaching points, see page 259.
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(continued from p. 186)

DIAGNOSIS:

Left ventricular noncompaction. Figure 1 shows an apical 4-chamber view of the left ventricle, demonstrating prominent trabeculation at the apical and lateral walls and severely depressed systolic function (Video E1, available online at http://www.annemergmed.com). Blood flow in between trabeculae is visible on color flow imaging (Figure 2). The ratio of compacted to noncompacted myocardium measured 1:3, consistent with left ventricle noncompaction, a rare cardiomyopathy that is being increasingly recognized.[1] Mutations in sarcomere genes underlie approximately 30% of cases.[2] It is believed to arise from failure of myocardial sinusoids in the developing embryo to compact, which normally progresses from base to apex. Consequently, noncompaction preferentially affects apical, inferior, and lateral walls. Hypertrabeculation of the left ventricle serves as a nidus for thrombus formation that merits prophylactic anticoagulation. The left ventricle ejection fraction is the major determinant of sudden cardiac death risk.[3] Treatment is according to guidelines for heart failure; disease-specific therapy is unavailable. Despite variable penetrance, family screening is advisable.

The patient improved with diuretics, bisoprolol, lisinopril, and spironolactone. She was anticoagulated but declined implantable cardioverter defibrillator. Family screening result with echocardiography was negative.

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