Thickened myocardium with a two layered structure consisting of a thin measurement of N/C ratio at end
Bhatia et al. Isolated non ratio between the non attack
Magnetic Resonance imaging:
• Trabeculation
Table 1: Diagnostic criteria for left ventricular non
Cardiac MRI:
Cardiac MRI (CMR) corroborated the echocardiography findings with a ratio of noncompacted myocardium to compacted myocardium of greater than 2.3. There were no mural thrombi by CMR. Her findings were consistent with left ventricular non-compaction (LVNC).

Case Outcome:
This patient’s heart failure was treated medically and her dyspnea improved with following diuresis. She started oral anticoagulation for secondary prevention of likely cardioembolic disease without further neurologic symptoms.

Discussion:
Left ventricular non-compaction is an increasingly recognized cardiomyopathy, characterized by deep ventricular recesses and trabeculations, which are appreciated on echocardiography by a classic "sponge-like" appearance.

Normally, the myocardium undergoes a process of "compaction" of the initial loose myofibrillar networks during the first trimester of pregnancy. Disruption of this compaction process can lead to congenital appearance of LV non-compaction.

While its true prevalence remains unknown, in patients referred for echocardiography, 0.01-0.3% patients meet criteria for diagnosis. There is controversy as to whether LVNC can be acquired, as evidenced by the development of characteristic findings on serial echocardiograms in case reports. Several authors conclude that LVNC is a phenotypic expression of varying processes, congenital or acquired, rather than a distinct cardiomyopathy.

Most patients who are incidentally identified with LVNC remain asymptomatic. Symptomatic patients typically present with heart failure symptoms and have an average LV EF of 36%. Thromboembolic events occur in 8% patients, more frequently in those with systolic dysfunction. The mortality rate from sudden cardiac death in patients with LVNC is 8-9%. Treatment options vary on an individual basis and usually consist of medical management for heart failure with consideration of anticoagulation if there is a history of thromboembolic disease.

Conclusion:
Although LVNC is a rare clinical entity, the incidence is expected to rise with growing recognition given the increasing ubiquity and sensitivity of cardiovascular imaging. For general providers, LVNC is a consideration for patients presenting with new onset heart failure, dysrhythmia or thromboembolic disease.

References:

Presentation:
A 52 year old woman presented with fatigue and shortness of breath. She had a progressive dyspnea on exertion and a 20 pound weight gain. Two months previously, she had experienced a transient ischemic attack (TIA) with acute onset of left sided hemiparesis and paresthesias; her symptoms completely resolved within hours. Evaluation of her TIA symptoms at that time included a non-contrast head CT and carotid ultrasound which were both normal.

Exam:
On physical exam, she had faint bilateral crackles, an elevated JVP to 14cm above the right atrium and an S3 gallop. Her neurologic exam was normal.

EKG & Labs:
An EKG demonstrated normal sinus rhythm with left axis deviation and lateral T-wave flattening. Her labs were notably for mildly elevated pro-NPBNP (282 pg/mL). Troponins were trended and remained normal.

ITTE:
Transthoracic echocardiography demonstrated asymmetric LV wall thickness and abnormal endocardial trabeculations with a spongiform appearance in the lateral and inferior segments. The LV ejection fraction was 25-30%.

Cardiac MRI:
Cardiac MRI (CMR) corroborated the echocardiography findings with a ratio of noncompacted myocardium to compacted myocardium of greater than 2.3. There were no mural thrombi by CMR. Her findings were consistent with left ventricular non-compaction (LVNC).