Coronary Artery or Myocyte
Wherein Lies the Diagnosis?

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Concision in style, precision in thought, decision in life.
—Victor Hugo, 1907*

Patients who have left ventricular (LV) contractile dysfunction with dilatation of one or both ventricles receive the broad diagnosis of dilated cardiomyopathy (DCM), though further refinement is invariably required to guide appropriate therapy. Etiologic considerations span metabolic, toxic, infiltrative, structural, and congenital disorders, many of which may be apparent on initial history; hypertension and atherosclerotic coronary artery disease (CAD) are often the lead suspects (Table). Even in the absence of a defined genetic syndrome, DCM often occurs within a milieu of genetic susceptibility, underscoring the need to elicit a careful family history.

Accurate diagnosis in the initial workup of DCM dictates distinct treatment pathways and attendant variability in prognosis. Excluding CAD in the patient with newly diagnosed DCM typically includes one or more diagnostic procedures. The ECG has proven utility amidst an array of imaging options for the patient with DCM, even if the merit of Q waves in defining infarcted myocardium has waned. Recognition of epsilon waves should promote consideration of arrhythmogenic right ventricular cardiomyopathy, atrioventricular block with the appropriate family history should trigger suspicion of lamin A/C cardiomyopathy, and low QRS voltage despite thickened myocardium should raise one’s suspicion of amyloidosis.

If bedside assessment does not point to an obvious etiology, the initial workup of newly diagnosed DCM typically includes one or more diagnostic procedures. The ECG has proven utility amidst an array of imaging options for the patient with DCM, even if the merit of Q waves in defining infarcted myocardium has waned. Recognition of epsilon waves should promote consideration of arrhythmogenic right ventricular cardiomyopathy, atrioventricular block with the appropriate family history should trigger suspicion of lamin A/C cardiomyopathy, and low QRS voltage despite thickened myocardium should raise one’s suspicion of amyloidosis.

The varied approaches taken to exclude coronary artery disease in DCM reflect a spectrum of diagnostic methodologies. Current heart failure guidelines for the diagnosis of heart failure in adults continue to advocate invasive coronary angiography, with some authors even cautioning against underuse of invasive angiography in the initial evaluation of heart failure. Catheter angiography is recommended particularly for DCM patients with chest pain or angina in whom revascularization would not be contraindicated. Such patients might be considered to have a higher pretest likelihood of CAD, not necessarily the ideal candidates for CT coronary angiography. Cardiac catheterization also provides invasive pressure measurements, which may be preferred to define hemodynamic status over Doppler-based estimates in patients with decompensated heart failure. Coronary angiography, particularly with CT, may also be suitable in the patient with established nonischemic cardiomyopathy who has an unexpected decline in function that may be due to interval development of CAD.

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Nuclear scintigraphy might hold appeal in distinguishing ischemic from nonischemic cardiomyopathy by defining the extent of myocardial ischemia, though limited specificity compared with invasive angiography reminds us that microvascular ischemia may be present in DCM even without epicardial coronary stenosis. Recent advances in targeted tracer design hold promise in their ability to image molecular processes in heart failure that have diagnostic and prognostic value. Transthoracic echocardiography is widely used and advocated for the initial evaluation of the patient with heart failure to define cardiac size and function as well as to detect valvular pathologies. In nondecompensated DCM, Doppler-derived E/E' is a widely accepted surrogate for LV end-diastolic pressure. Dobutamine stress echocardiography, specifically failure to augment long-axis deformation and velocities by M-mode and tissue Doppler, respectively, performs surprisingly well in distinguishing ischemic from nonischemic cardiomyopathy. This approach has mechanistic appeal with increasing appreciation of a subendocardial population of longitudinally oriented myofibers, rendering them especially sensitive to epicardial coronary disease–induced ischemia.

If CAD produces infarct scar and subsequent dilatation, then late gadolinium enhancement by cardiac magnetic resonance (LGE-CMR) would seem to be the logical choice for etiologic discrimination. McCrohon et al showed the limitations of coronary angiography in defining CAD as the etiology of DCM in patients who had previously sustained myocardial infarction with culprit vessel recanalization. In such instances, coronary angiography finds only a nonobstructive lesion, whereas the signature of prior infarct scar is readily apparent by LGE-CMR. In this and subsequent confirmatory studies, 9% to 13% of patients with DCM were found to be incorrectly classified as having nonischemic cardiomyopathy by coronary angiography and correctly classified as having ischemic cardiomyopathy by LGE myocardial imaging.

Beyond diagnostic value in excluding CAD and defining other etiologies of cardiomyopathy, myocardial imaging provides prognostic value in evaluating the patient with DCM; the presence of midwall fibrosis portends a poor response to medical therapy, lower likelihood of functional recovery, and greater mortality rates. These factors could affect decisions regarding aggressiveness of treatment and timing of referral for cardiac transplantation or ventricular assist device placement. Although the decision to resynchronize the LV whose ejection fraction falls below 35% may be influenced by echocardiography-based dysynchrony measures, it may be abandoned once transmural inferolateral wall scar is demonstrated by LGE-CMR—a finding that predicted complete absence of response to cardiac resynchronization regardless of dys synchrony in one study.

Despite published evidence favoring one or more approaches, diagnostic styles vary among centers providing cardiovascular care, reflecting differences in regional expertise and availability as well as patient-specific clinical questions. Thus, if one’s primary concern is whether or not a patient with DCM has obstructive epicardial CAD, particularly if the pretest likelihood is low, obtaining a concise view of the coronaries noninvasively with CT may suffice. However, for diagnostic precision and prognosis-guided decision for improved patient outcomes, the myocyte deserves our attention as well.

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### References


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