Cardiac Computed Tomography and Magnetic Resonance Imaging

The Clinical Use From a Cardiologist's Perspective

Jacob Cross Townsend, MD and David Gregg, IV, MD

Abstract: The introduction and continued evolution of cardiac computed tomography and magnetic resonance imaging have added considerable noninvasive diagnostic insight into a wide range of frequently encountered clinical cardiology scenarios. With an increasing range of imaging modalities, and multiple methods of image acquisition in each, a detailed understanding of the clinical question at hand is often necessary to select the proper study and make optimal use of imaging data. We review common cardiac issues from a clinician's perspective, along with the unique role to be played by computed tomography and magnetic resonance imaging in each condition. This review will hopefully facilitate a strong dialogue between imagers and managing clinicians, creating a shared knowledge of both the capabilities of imaging and the management challenges that treating clinicians face.

Key Words: cardiac computed tomography, cardiac magnetic resonance imaging, coronary artery disease, review, infiltrative cardiomyopath, constrictive pericarditis

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The increasing clinical use of cardiac computed tomography (CT) and magnetic resonance imaging (MRI) over the last decade has created a new partnership between cardiology and radiology. Depending on the institution, studies may be interpreted by radiologists, cardiologists, or jointly. This blended approach to performing and reading cardiac studies helps facilitate optimal interpretation and clinical management, as a complete interpretation of the data usually requires both excellent image evaluation and an understanding of the clinical scenario. Instead of reviewing the details of CT and MRI interpretation, this paper is written with the hope of providing additional clinical insight into the application of cardiac CT and MRI studies. We will begin by looking at the use of CT and MRI in coronary artery disease and will then look at less frequent indications, such as evaluation of pericardial disease, congenital heart disease, and arrhythmia, and will try to place the use and evaluation of these studies in a clinical context.

ISCHEMIC HEART DISEASE

In 2002, chest pain was the second leading cause of emergency department visits, accounting for 5.1% of nearly 110 million evaluations.[1] This was not without reason, as coronary heart disease (CHD) was the cause of 1 in every 5 deaths, making it the overall leading cause of death in the United States among those older than 20 years of age.[2] The lifetime risk of developing CHD after the age of 40 years according to Framingham data is 49% for men and 32% for women.

There are 3 clinical questions that are frequently considered when treating CHD: Is the disease present? Is the disease impairing myocardial perfusion? In patients with known obstructive coronary lesions, is the myocardium supplied by these lesions still viable and a potential target for revascularization? Here both CT and MRI have the unique ability to offer noninvasive answers to these clinical questions.

Detection of CHD

The presence of CHD, irrespective of symptoms, is a marker for future events.[3] Of course, if there was no advantage to intervention, this knowledge would be of little benefit. We now know, however, that with aggressive medical management, including tight control of blood pressure, and perhaps more importantly lipids, patients may actually achieve plaque stabilization, if not regression.[4]

Determining who is at risk for CHD, and would therefore benefit from intensive medical therapy, has proven to be difficult on the basis of clinical risk factors alone. Clinical risk stratification models have largely driven treatment recommendations, especially in patients without known CHD.

The National Cholesterol Education Program-III guidelines use a history of cigarette smoking, hypertension, diabetes, low high-density cholesterol, family history, and age to categorize patients into low-, intermediate-, and high-risk groups.[5] Unfortunately, while these groups do largely correlate with cardiovascular risk, their predictive power is relatively limited in women and younger patients.[3] This clear limitation of clinical risk assessment created the need for additional CHD markers, in which noninvasive imaging can play a major role.

Initial attempts to radiographically determine the presence of CHD were made possible by the frequent finding of calcium deposition in coronary plaques. As early as the 1950s, this allowed for fluoroscopic recognition of coronary calcium and, by inference, CHD.[6] The association between coronary calcium and plaque has become better defined in the following decades, and although not all
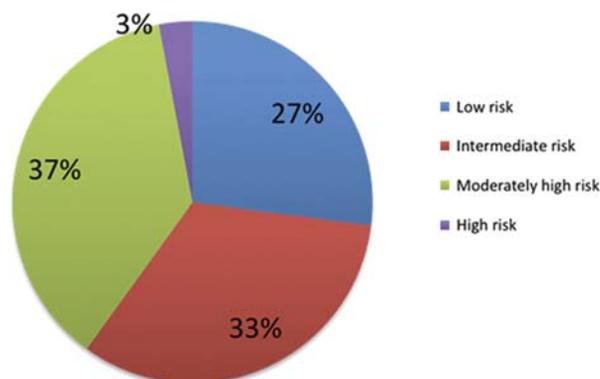
FIGURE 1. The clinically derived risk of CHD in the subset of patients with calcium scores of greater than 400, implying high atherosclerotic plaque burden. Data presented in this pie chart is from Reference 3.

FIGURE 2. A 44-year-old woman presented with chest pain and equivocal electrocardiogram findings. Initial evaluation with a cardiac CT revealed a calcium score of zero with no angiographically apparent stenoses. As her symptoms persisted and clinical suspicion of an acute coronary syndrome remained high, the patient was referred for left heart catheterization, which revealed an acute dissection of the mid-right coronary artery (arrow) extending into the posterior descending artery.

coronary plaques contain calcium, the presence of intra-coronary calcium clearly carries clinical significance.

Although there is some variation by ethnicity and sex, the CT-derived coronary artery calcium score (CAC) shows a linear relationship with the presence of disease. One paper reviewing 16 previous studies found that the sensitivity and specificity of CAC in detecting more than 50% angiographic stenosis were 91% and 49%, respectively.[7] Perhaps even more important, however, is that a lack of CAC correlates with a probability of coronary stenosis of less than 1%.[8]

This informs 3 important clinical situations. First, CAC is quite sensitive to the detection of CHD and augments clinical risk stratification models. As mentioned previously, in patients without known CHD, the decision to treat, and to what target cholesterol level, is largely driven by clinical risk factors. The limitations of this strategy were illustrated by Nasir et al,[3] who reported that 59% of younger patients (men under 55 years and women under 65 years) who were identified as having "severe plaque burden" by a calcium score greater than 400 would not have qualified for therapy under current guidelines (Fig. 1). The clinical significance of this finding was echoed by a separate study by Akosah,[9] in which 75% of asymptomatic young adults presenting with a myocardial infarction would not have been candidates for statin therapy before their event. This suggests that the CAC adds both prognostic insight and potentially the impetus for more aggressive medical therapy.

Second, the knowledge imparted by a lack of CAC is quite robust. The significance of this finding was tested in an emergency department setting in which patients presenting with chest pain and nondiagnostic electrocardiograms underwent CAC scoring. Of 134 patients, 48 had a CAC score of 0, and after excluding 1 patient in whom cocaine use was identified (a cause of non-plaque-related myocardial ischemia), the negative predictive value of the study was 100%. Conversely, the remaining patients had a 30-day event rate of 8%.[10] Other studies have examined the utility of CAC scoring to rule out CHD in other clinical situations, such as determining whether new-onset heart failure is ischemic in origin, and again its usefulness was clearly demonstrated in differentiating ischemic from nonischemic origin with 92% accuracy.[11]
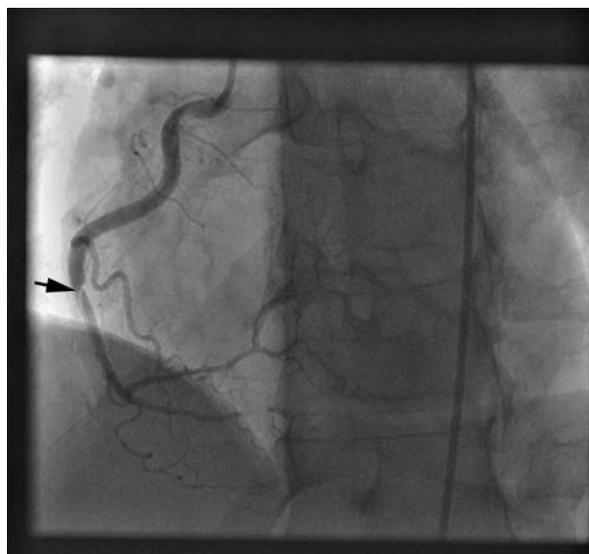
Of course, while these small studies are certainly encouraging, and a recent large review including nearly 85,000 patients found low event rates in both asymptomatic (0.56%) and symptomatic (1.8%) patients without coronary calcium at 51 months, special consideration must be taken in symptomatic patients.[12] This is highlighted by the fact that not all plaque is calcified. A recent retrospective review of patients without baseline coronary calcium found that 16% had obstructive stenosis when angiography was performed for symptoms within the next 18 months.[13] In addition, not all coronary obstruction is plaque related, and a calcium score may be entirely normal in a patient presenting with coronary spasm, cocaine-induced vasoconstriction, or coronary dissection (Fig. 2).

Finally, despite the ability to accurately predict the presence (or absence) of CHD, CAC scoring alone does not adequately predict the presence of significant stenosis, with specificity for detecting significant lesions of only 49%.[7] In addition, it uses the assumption that plaque is calcified, which as already mentioned, may result in an infrequent but important underestimation of CHD. In fact, 1 study demonstrated that the histologic plaque area was 5 times greater than the calcified area demonstrated by CT.[14] These limitations helped push the continued advancement of cardiac CT imaging and the addition of contrast agents to allow for direct coronary visualization.

Direct Coronary Imaging

After confirming the presence of CHD, the next clinical question involves the severity and location of any lesions. Accurate determination of stenosis severity has specific clinical implications, as stenosis greater than 50% may begin to show physiologic significance during hyperemia,[15] and stenoses of 70% are presumed to be flow-limiting. To date, CT has shown the most promise as a

noninvasive coronary imaging modality, and while there are many CT studies comparing stenosis severity with catheterization, 2 more recent examples, in particular, best illustrate the strengths and potential pitfalls of CT technology.

In the ACCURACY trial, 64-slice multidetector CT scanners were used to obtain images with a stenosis that was considered to be significant (>50%).[16] Two hundred and thirty of 245 patients completed the study, and CT was found to have a sensitivity of 95% and a specificity of 83%. Unfortunately, in patients with increased coronary calcium (Agatston score > 400), specificity fell to 53%. A similarly impressive sensitivity of 85% was seen in the CORE 64 study, again using 64-slice multidetector CT scanners for the detection of greater than 50% stenosis.[17] This study was able to achieve remarkable specificity as well (90%), but with the exclusion of more than one-quarter of the study population (291 of 405 completed the study), largely due to high calcium scores.

Clearly, CT has excellent sensitivity to detect obstructive coronary disease, with its major shortcoming being a limited specificity. Whether this limits its clinical application hinges on the patient's presentation. Angina is classified as stable or unstable based on whether the symptoms have been present and unchanged over time, or are new or worsening in severity. Stable angina is mediated by a flow-limiting stenosis but tends to have an insidious course that does not commonly lead to infarction. In contrast, unstable angina reflects a dynamic change in coronary flow with increased concern for infarction. The distinction is imperative, as the treatment, urgency, and outcome of each are quite different.

Of all patients presenting to the emergency department with chest pain, only 25% are determined to have had a coronary event by the time of discharge.[18] Clearly, many chest pain complaints are not cardiac in origin, but as the treatment for an unstable coronary syndrome requires urgent therapy, clinicians have a low threshold for additional evaluation. CT can occupy an important role in this triage. Depending on the prevalence of disease, CT has been shown to have a negative predictive value as high as 99% for the presence of CHD,[16] but it is also important to note that a negative predictive value of only 83% has been reported when the prevalence of disease was higher (nearly 50%).[17] Because an accurate determination of both the presence of disease and lesion severity is important in a patient with unstable symptoms, the use of CT is more frequent in patients felt to be at a lower risk for acute cardiac events. In this population, CT can clearly identify those without disease and allow for safe and rapid triage.[19]

Patients with stable angina do not have the same need for urgent therapy or the same risk for an acute event. Before the publication of the COURAGE trial, the presence of a significant obstruction, especially when accompanied by symptoms, was felt to be cause enough for revascularization.[20] This study showed, however, that in patients with chronic angina and evidence of significant (> 70%) stenosis, there was no mortality difference between those treated medically and those who were revascularized, despite superior anginal control in the revascularization group. It is important to note that this study excluded those with left main coronary disease. With regard to noninvasive imaging, these findings would suggest that the limited ability of CT to discriminate lesion severity may not be clinically relevant, in that as long as high-risk anatomy can be excluded, an initial trial of medical therapy for angina is a safe and reasonable alternative to revascularization regardless of whether a stenosis is 60% or 90%.

More recently, this same idea was validated in the BARI 2D study, in which patients with diabetes were randomized to medical therapy versus revascularization after the presence of significant stenosis was identified by catheterization.[21] Again, there was no difference in mortality between the revascularized group and the medical therapy arm.

The net result of these studies is that in stable patients, even those with a high risk for obstructive coronary disease, CT may have a broadening role, as there is no urgent indication for revascularization. In patients with unstable symptoms, when chest pain is thought to be noncardiac and the risk for obstructive disease is low, CT also does an excellent job of confirming the absence of disease. However, in those with unstable symptoms or a moderate-to-high risk of CHD, CT's limited ability to define the extent of stenosis and the possible need for catheterization in order to revascularize, makes a noninvasive study less appealing.

Although MRI has also demonstrated the ability to visualize coronary gross anatomy, at present not enough data exist to justify its routine use for direct detection of coronary lesions by angiography. Two studies have directly compared CT and MRI using catheterization as the gold standard. The first, which included 52 patients and used 16-slice CT, showed similar sensitivity (92% vs. 88%) and specificity (67% vs. 50%) for the detection of more than 50% stenosis when compared with traditional angiography.[22] The second, again using 16-slice CT in comparison with MRI in 108 patients, found better sensitivity with CT (92% vs. 74%) with similar specificities.[23] One additional caveat to the use of MRI is that the higher degree of operator involvement leads to a significant degree of interoperator variability, making reliability somewhat limited.[24] With lower spatial resolution, ischemic evaluation using MRI generally focuses on impaired perfusion rather than angiography.

Perfusion

The ideal study to assess coronary disease would be one that incorporates detection of disease, location, and severity of coronary stenosis and an assessment of any physiologic impact. In general, there is no indication for revascularization in coronary lesions that do not cause impaired perfusion, but unfortunately most tests at this point measure either perfusion (stress testing) or stenosis (angiography) independently.

The identification and quantification of ischemia is an important clinical tool. Studies using nuclear imaging to determine ischemia have suggested that when less than 10% of the left ventricle is involved, medical therapy is favored over revascularization.[25] In addition, the extent of ischemia is predictive of cardiac death, and perhaps more importantly, the COURAGE nuclear substudy suggested that a reduction in ischemia may be associated with a reduction in cardiac death.[26]

Cardiac MRI (CMRI) is uniquely suited to assess perfusion functionally by looking for stress-induced wall motion abnormalities or by assessing myocardial perfusion. Even without the use of contrast, several studies have shown the utility of CMRI as a stress imaging agent, making use of its excellent tissue discrimination to identify wall motion changes while administering dobutamine.[27,28]
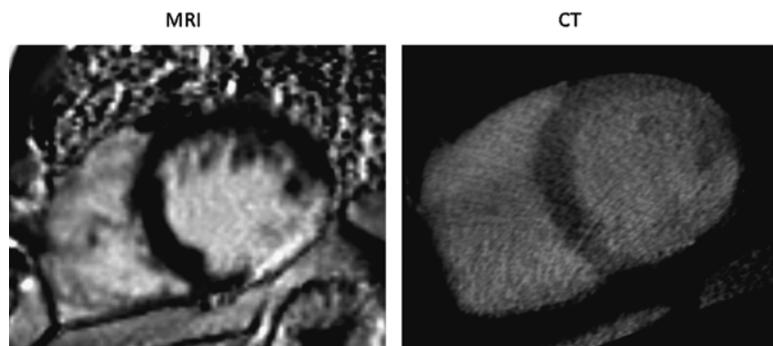
FIGURE 3. Transmural contrast enhancement of the posterior lateral wall as imaged with MRI and CT demonstrating nonviable myocardium.

When compared with stress echo, this technique was found to be both more sensitive (86% vs. 74%) and more specific (86% vs. 74%).[29] More recently, the use of myocardial tagging has been used to further refine the assessment of wall motion changes. When compared with nontagged images, this modality detected more abnormal segments and showed a high correlation with angiographic evidence of high-grade stenosis (62 of 68 patients required revascularization).[30] In addition, a normal tagged MRI scan was associated with a 98% event-free survival rate at 17 months. When gadolinium is used, MRI has the added ability to directly assess for tissue-level hypoperfusion through first-pass imaging. When using adenosine or dipyridamole as the stress agent, first-pass abnormalities identified perfusion defects with a sensitivity of 91% and a specificity of 94%, when compared with positron emission tomography (PET).[31]

Viability

It is estimated that 25% to 40% of patients with ischemic cardiomyopathy will show improvement with revascularization.[32] Unfortunately, many of these same patients, because of their cardiomyopathy, are at a higher procedural risk for revascularization. In light of this increased risk, an accurate discrimination of scar from viable myocardium is of utmost importance (Fig. 3).

CMRI employs several unique characteristics to allow for accurate determination of myocardial viability. Using late gadolinium enhancement (LGE), the late uptake of gadolinium into necrotic or irreversibly damaged myocytes, MRI can pinpoint myocardial scar. Furthermore, its ability to differentiate tissue planes allows for LGE to be identified as transmural or nontransmural. When correlated with the effects of low-dose dobutamine, the regions of transmural LGE show no improvement, whereas nontransmural LGE regions show improved, albeit diminished, contractility.[33] The effects of revascularization have also been shown to be predictable by the degree of LGE, wherein the likelihood of improved contractility is related to the degree of transmural LGE.[34]

Small studies have suggested that CT is also capable of distinguishing infarcted from viable tissue based in the same way as MRI, on delayed enhancement of infarcted tissues.[35,36] Currently, the technique is somewhat limited and often requires additional information, such as wall thickening or motion abnormality, to accurately differentiate scar from ischemic but viable tissue.[37] In an age of increasingly common postsurgical clips and implanted devices, despite its limitations, CT will likely have a role in this arena.

PERICARDIAL DISEASE

Pericardial Effusions

Anything that impedes venous or lymphatic drainage from the heart can lead to fluid accumulating in the pericardial space. Common causes include heart or renal failure, inflammation, infection, and trauma. Small amounts of fluid or slowly accumulating effusions are usually well tolerated, but because the pericardium restricts ventricular filling to a finite space, larger effusions can cause impairment in cardiac filling. As an effusion reaches a critical volume, pressures in all cardiac chambers rise, and ventricular filling becomes closely related to respiratory variation.

In most cases, both the effusion and the accompanying hemodynamic derangements indicative of tamponade are appreciable on echocardiogram; however, there are several specific cases in which CT or MRI offers unique diagnostic capabilities. First, although echocardiography is sensitive for the detection of effusions, it has a limited field of view, making loculated effusions potentially difficult to identify. Second, while echocardiography can establish the presence of pericardial fluid, it has very limited ability to characterize the fluid. With CT attenuation measurements, a simple effusion can often be differentiated from a hemopericardium, purulent pericarditis, or malignancy. MRI shares the ability to differentiate hemorrhagic from nonhemorrhagic pericardial effusions and, with its superior ability to image tissue heterogeneity, also offers detailed imaging of the myocardium for the presence of malignancy to explain the presence of the effusion.[38,39] Finally, both modalities allow for the evaluation of pericardial thickness, which may provide additional insight into the diagnosis.

Constrictive Pericarditis

Constrictive pericarditis often presents clinically with recurrent symptoms of right heart failure, frequently with ascites and lower extremity edema. It is usually accompanied by the appropriate clinical setting (past thoracic surgery, systemic inflammatory disease, prior myocardial infarction, etc), but its diagnosis can pose a challenge as many of the same clinical findings are seen with restrictive cardiomyopathies.
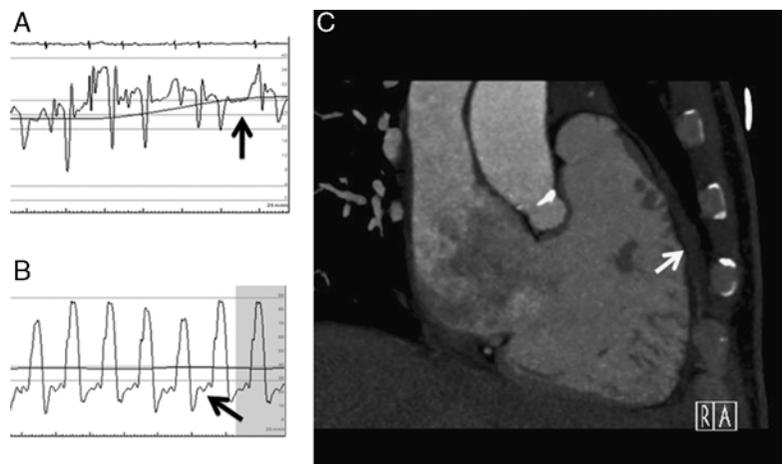
FIGURE 4. A 50-year-old man presented with 6 months of increasing abdominal distention. An echocardiogram demonstrated preserved systolic function with elevated filling pressures. Kussmaul's sign [elevation of the right atrial pressure with inspiration (A)] and "square root sign" [rapid rise in right ventricular pressures during diastole (B)] were found during right heart catheterization, but other support for the diagnosis of constriction was limited. Cardiac CT (C) revealed a thickened (5 mm) and nodular anterior pericardium (arrow), lending further support to the diagnosis and, ultimately, to surgery.

Echocardiography and right heart catheterization are frequently used to document the classic hemodynamic derangements, but these findings are not always present, and even when they are, many can also be seen in restrictive disease.[40] Because the treatment for constrictive pericarditis is surgical, as opposed to the medical therapy indicated for restrictive disease, differentiation between the 2 is imperative. In cases in which the diagnosis is still in question, noninvasive imaging with CT and MRI can help clarify the pathology.

Perhaps the most useful measure to discriminate between constriction and restriction is pericardial thickness. The normal pericardium is less than 2 mm thick, making visualization with echo difficult.[41] In addition, pericardial thickening may be limited to specific regions, making visualization with echocardiography's limited tomographic views even more difficult to appreciate. Although a thickened pericardium is not required for constriction, pericardial thickening has a linear correlation with the risk of constrictive disease (Fig. 4). Indeed, pericardial thickness greater than 4 mm, when associated with appropriate clinical and hemodynamic findings, is highly suggestive of constrictive pericarditis, supporting the diagnosis of construction with a reported accuracy of 93%.[42]

Because both CT and MRI can characterize pericardial thickness, the decision regarding which modality to use may be guided by several other considerations. First, if there is suspicion of calcium deposition in the pericardium (after surgery or radiation), which is independently associated with constrictive disease, the use of CT for its sensitivity in the detection of calcium should be considered. Second, if a small effusion is present, MRI has superior ability to differentiate small effusions from pericardial thickening. Finally, the presence of pericardial thickening requires the setting of appropriate hemodynamic derangements to make the diagnosis. Therefore, with the ability of MRI to provide physiologic data, potentially all of the diagnostic findings can be found in a single study. Because of this unique ability, some have suggested the use of MRI as the initial study for evaluation of constriction.[41]

CONGENITAL

The estimated incidence of complex congenital heart disease is 1.5 in every 1000 live births. With improvements in surgical and medical treatment, this has been estimated to translate into roughly 117,000 adult patients with complex congenital heart disease in the United States alone in the year 2000.[43] Given the wide range of both inborn abnormalities and surgical repairs, this population represents a vast array of anatomic and physiologic variants. Although echocardiography is capable of evaluating some of these variants, CT and MRI have a clear edge in providing a more complete assessment.

Perhaps the most important advantage of CT and MRI over echocardiography is the ability to provide an unrestricted view of the thorax. This allows for an evaluation of any extracardiac anastamosis, mapping of unusual anatomy, and even assessment for commonly associated noncardiac findings such as aortic coarctation or anomalous pulmonary venous return. Furthermore, these unobstructed views allow for an improved assessment of chamber size and function, especially of the right ventricle, which can be hard to accurately assess with echocardiography. In patients with tetralogy of Fallot, this is of particular importance, as serial enlargement of the right ventricle may signal the need for pulmonary valve replacement and suggest an increasing risk for the development of arrhythmias.[44]

In addition to providing anatomic information, MRI has the added capability of quantifying flow and velocity. Thus, not only can regurgitant or stenotic lesions be anatomically identified, but in many cases their severity may also be quantified (Fig. 5).

Although most major congenital abnormalities are identified in the pediatric population, coronary anomalies may present later in life as a cause of exercise-induced syncope, arrhythmias, sudden death, or chest pain. The clinically significant coronary anomalies involve an abnormal origin, with special concern given to arteries coursing between the aorta and the right ventricular outflow tract (Fig. 6). Although CT is superior in providing noninvasive
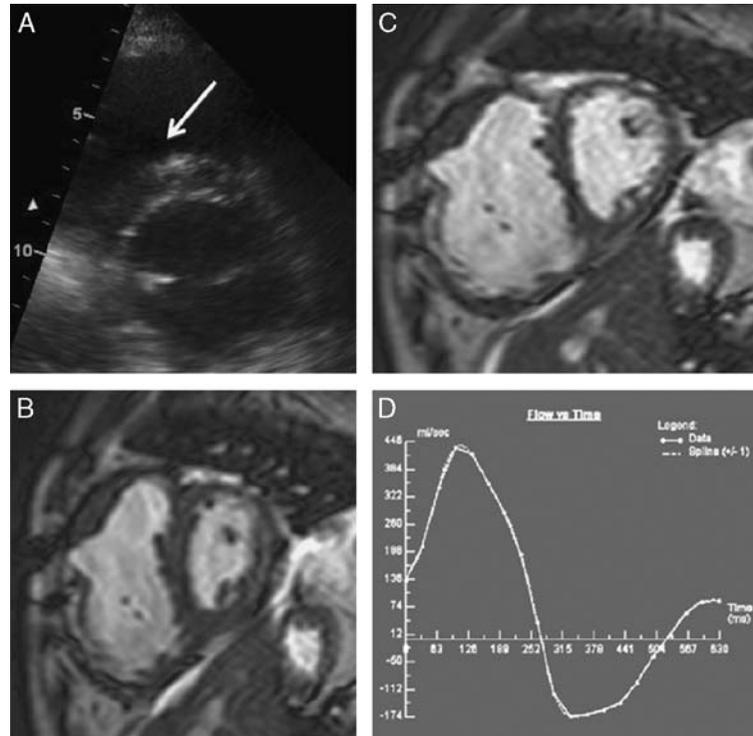
FIGURE 5. A 50-year-old man with a history of tetralogy of Fallot repaired as a teenager. Serial assessment of his right ventricular volume and function was limited with echocardiography [arrow shows the limited view of the right ventricle (A)]. CMRI accurately demonstrated severe right ventricular enlargement [during systole (B) and diastole (C)], suggesting the need for pulmonary valve replacement. Analysis of velocity-encoded imaging of the pulmonary artery allows for the quantification of severe pulmonary regurgitation (D).

FIGURE 6. A 71-year-old woman presented after an abnormal stress test. At cardiac catheterization, the right coronary artery was unable to be cannulated. Cardiac CT demonstrated an anomalous right coronary artery coursing between the aorta and pulmonary artery (arrow). The examination also revealed a markedly enlarged left coronary circulation and multiple coronary cameral fistulas.

coronary angiography, in the case of anomalies wherein only the origin and proximal vessel are of primary importance, both CT and MRI are capable tools.[45]

ARRHYTHMIA

Sudden cardiac arrest is responsible for up to 15% of all deaths in the United States,[46] and is 6 to 10 times more common in patients with clinical heart failure.[47] To compound the issue, the survival rate for out-of-hospital arrest, despite significant advances, was reported to be only 6% in a large population study from the Netherlands.[48] To date, the only effective therapy for sudden cardiac arrest is an implantable defibrillator, but given the cost and invasive procedure required for implantation, accurate identification of those who would benefit is imperative.

Outside the setting of acute ischemia, when the cause of ventricular tachycardia is clear, the evaluation of a ventricular arrhythmia almost always requires cardiac imaging. Some common substrates for a structurally based arrhythmia are easily identifiable with echocardiography, such as hypertrophic cardiomyopathy or severely impaired left ventricular systolic function, but others require a more detailed assessment of the myocardium, for which CT, and especially MRI, can offer added insight.

Involvement of the myocardium with sarcoidosis has been widely reported as a cause of ventricular arrhythmias.[49] It tends to cause patchy ventricular invasion, making diagnosis by echocardiography, or even myocardial

biopsy, difficult. Because of their ability to image the myocardium in its entirety, CT and MRI offer a broader perspective to help make the diagnosis. MRI allows for tissue characterization that can identify even a patchy infiltrative process. CT has a more limited role, but the recognition of myocardial thinning, when paired with a PET scan suggesting metabolically active tissue in the same region, may suggest sarcoid involvement. Subsequent treatment would focus on immunosuppression with steroids, a treatment that has shown promise in quieting the arrhythmia focus.[50]

Arrhythmogenic right ventricular dysplasia (ARVD) is another cause of sudden death that can be quite difficult to diagnose without CT or MRI. ARVD is a genetically determined disease in which right ventricular myocytes are displaced by adipose tissue, leading to right ventricular dysfunction and an arrhythmogenic focus. Up to 40% of these patients may have a normal electrocardiogram at presentation, and while echocardiography classically shows right ventricular dilation, accurate measurements of right ventricular volume and function are often difficult to obtain.[51] MRI is uniquely suited to evaluate for ARVD, as it offers a detailed assessment of right ventricular size and function, but can also illustrate abnormalities in the myocardium. When compared with patients diagnosed with ARVD by clinical criteria, MRI showed 100% sensitivity.[52] The specificity was low at 29%, but the investigators suggested that this may actually have been a limitation of the clinical criteria rather than of MRI, as the specificity when compared with genotype-proven patients was 79%. To date, there are no studies to confirm the high negative predictive value of CMRI for ARVD, but this early study is certainly promising.

MALIGNANCY

Fortunately, primary cardiac and pericardial masses are rare and are frequently benign. Mortality is highly dependent on tumor type, with 3-year survival rates of 90% in myxomas, 70% in nonmyxomatous benign tumors, and 0% in malignant tumors.[39] Far more common are secondary cardiac tumors, with 30% to 50% of melanoma patients having evidence of cardiac metastasis at autopsy. Breast and lung cancer, both more common than melanoma, have cardiac metastasis rates of 10% and 17%, respectively. Clinically, the important distinctions are whether the malignancy is primary or secondary and to what extent it invades the heart and pericardium (Fig. 7).

Frequently, cardiac or pericardial tumors are first noted on echocardiography. However, except in the case of a myxoma, in which a well-visualized thin stalk connecting a mass to the interatrial septum may be diagnostic,[39] most intracardiac tumors cannot be adequately characterized with echo alone. Although many intracardiac tumors ultimately require surgical biopsy for diagnosis, several findings have been reported to correlate with a benign neoplasm. In a study by Hoffman,[53] tumor location in the left ventricle and homogenous tissue composition were both more likely to suggest a benign etiology. There are currently fewer data on the use of CT for intracardiac tumors, but while CT may be less adept in characterizing neoplastic tissue, like MRI it can provide a rapid and accurate assessment of the remainder of the chest for a primary malignant source. In the future, the addition of PET to MRI and CT imaging will add the benefit of metabolic imaging to tissue characterization.

Pericardial neoplastic involvement occurs more frequently, and, similarly, metastatic disease is more common than primary malignancy.[39] Again, lung and breast cancer are the most common primary sources. As discussed earlier, both CT and MRI can accurately image the pericardium and surrounding tissues to yield valuable information.
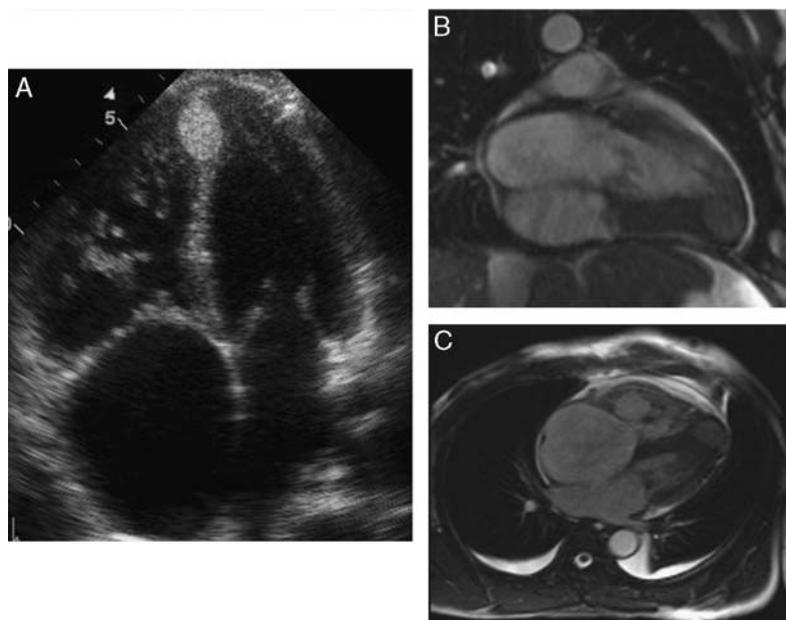

FIGURE 7. A 62-year-old man presented with right-sided heart failure and flushing. An echocardiogram demonstrated dilated right-heart chambers with severe tricuspid regurgitation due to retracted and thickened tricuspid leaflets. The study also revealed an echo-dense mass in the distal septum consistent with cardiac carcinoid (A). CMRI was used to more precisely define the size, number, and location of the intracardiac carcinoid tumors before surgical removal (B, C).

HEART FAILURE

In 2006, the estimated prevalence of heart failure was 5.7 million cases among Americans older than 20 years of age. Although the survival rate after the diagnosis of heart failure has improved over the last 2 decades, the incidence of disease has not changed.[2] The initial study for the majority of these patients is an echocardiogram, which in combination with a detailed history is adequate to diagnose most common causes of left ventricular dysfunction. After accounting for ischemic, valvular, and toxin-mediated cardiomyopathies, there are a cluster of relatively uncommon but important causes of heart failure. These would include infiltrative diseases, acute myocarditis, and structural abnormalities such as noncompaction of the left ventricle.

Cardiac amyloidosis is a relatively rare condition involving infiltration of the extracellular myocardium with fibrillary proteins.[54] This results in ventricular hypertrophy and subsequent restrictive filling. Because cardiac involvement in amyloid is a major factor in prognosis and treatment options, its accurate diagnosis is clinically imperative. Historically, the diagnosis has been made using characteristic but insensitive echocardiographic findings coupled with peripheral or even endomyocardial biopsies. Although still only reported in small numbers, both CT and MRI show promise in offering a noninvasive means of diagnosing cardiac amyloid using late-contrast enhancement[55,56] (Fig. 8). Furthermore, MRI allows for an accurate assessment of the expected restrictive physiology, which, when coupled with subendocardial delayed enhancement, appears to accurately identify cardiac amyloid. One limitation at present, however, is that the use of contrast in these patients is often limited by concurrent amyloid renal disease.

Cardiac sarcoidosis is another of the relatively rare causes of infiltrative cardiomyopathy. It can present with subacute development of heart failure, classically associated with conduction abnormalities or arrhythmias. Like amyloid, there are no specific echocardiographic findings to make the diagnosis, and even endomyocardial biopsy is often of limited utility. With CMRI and CT, the regions of sarcoid involvement produce a scar appearance with late gadolineum enhancement.[57,58] This may be enough to establish the diagnosis in someone without other reasons for scar formation or other evidence of systemic sarcoidosis. When additional confirmation is needed, PET can be used to show that these areas of the "scar" are actually hypermetabolic, consistent with sarcoid. In either case, the ability to identify the exact region of myocardial involvement is clinically important, allowing for reassessment for resolution or improvement with therapy.

Left ventricular noncompaction is another of the rare causes of cardiomyopathy. This genetically derived ventricular malformation results in excessive, deep trabeculations involving the apical portion of the ventricle, along with ventricular systolic dysfunction. Its prevalence has been reported to be up to 3% to 4% among those with heart failure.[59] When the left ventricular cavity can be well seen, echocardiography may be sufficient for the diagnosis; but when the views are limited, or quantification of the extent of trabeculation is unclear, both CT and MRI are more than able to provide the necessary views.

Myocarditis, often secondary to a viral infection, is the last subset of heart failure discussed here. The prevalence is thought to be as high as 10% among patients with unexplained heart failure.[60] The presentation can range from subacute to fulminant heart failure, and while a history of preceding viral symptoms can be suggestive, there are no specific echocardiographic findings to further the diagnosis. MRI is well suited in this condition to evaluate for evidence of myocardial edema.[61] In addition, with contrast imaging, LGE appears in a specific pattern sparing the endocardium, producing a rough inverse of the involvement expected from myocardial infarction.[62] As myocarditis, especially early in the process, can be focal, if endomyocardial biopsy is indicated, MRI can prove useful in identifying targets of active inflammation.[63]

CONCLUSIONS

The last several decades have offered incredible advances in cardiac imaging, allowing for a complete, yet noninvasive, evaluation of cardiac function, hemodynamics, and pathology. As advances continue, even further collaboration between cardiology and radiology will be necessary, and a continued reassessment and understanding of both the clinical thought and technologic acumen required to accurately interpret imaging studies are imperative.
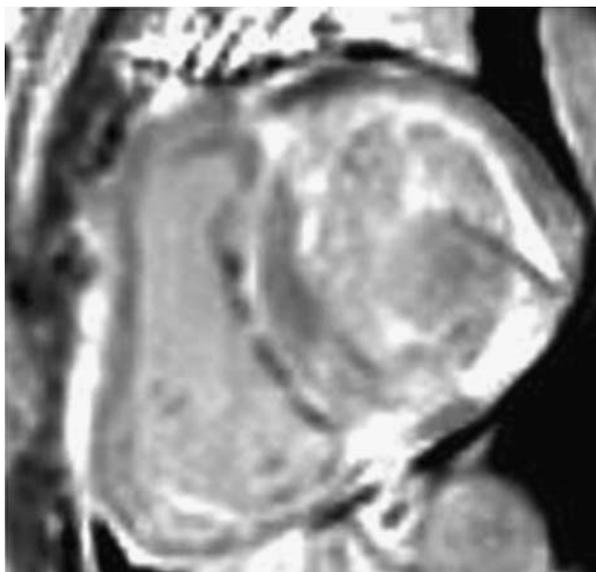

FIGURE 8. A CMRI was ordered to help evaluate infiltrative disease to explain the etiology of progressive heart failure despite aggressive medical management in a 67-year-old woman. The MRI demonstrates diffuse late enhancement in a nonvascular pattern consistent with cardiac amyloidosis. This diagnosis was later confirmed with a cardiac biopsy.

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