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Computed tomography and cardiac magnetic resonance imaging in pulmonary hypertension

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Abstract Recent advances in imaging technology have allowed for better temporal and spatial resolution in cardiovascular imaging. The idea of a "one-stop shop" for anatomical and functional cardiopulmonary and vascular assessment in patients with pulmonary hypertension is very appealing since diagnostic, prognostic, and therapeutic response can be measured. Modalities, such as computed tomography (CT) and cardiac magnetic resonance (CMR), are better suited to image the right heart and associated structures in multiple projections allowing for three-dimensional data sets and image reconstruction. This review will focus on the use of CT and CMR in the assessment of the right ventricle and pulmonary structures as they relate to pulmonary vascular disease. (Prog Cardiovasc Dis 2012;55:161-171)

Keywords: Hypertension; Pulmonary; Computed tomography; Magnetic resonance imaging; Right ventricle

Pulmonary circulation under normal physiologic conditions is a low-resistance, high-capacitance system allowing the right ventricle (RV) to maintain its stroke volume performing one sixth of the work of the left ventricle.[1] With increasing afterload and diminished vascular elasticity due to pulmonary hypertension (PH), the coupling of the RV–pulmonary circuit is disrupted leading to RV hypertrophy, eventual dilatation and impaired contractility.[2] Since RV failure is the most common cause of death in PH,[3] identifying early signs of right heart disease is paramount to treatment.

While two-dimensional transthoracic echocardiography is the most readily available technology for initial screening and assessment of PH, it is limited by acoustic windows, the irregular geometry of the RV and the inability to quantify indexes of RV function. Modalities such as computed tomography (CT) and cardiac magnetic resonance (CMR), are better suited to image the right heart and associated structures in multiple projections allowing for three-dimensional data sets and image reconstruction.[4,5] Our discussion will focus on the use of CT and CMR in the assessment of the RV and pulmonary structures as they relate to pulmonary vascular disease.

Computed tomography

CT of the chest is an important part of the evaluation for patients with PH.[6,7] The current generation of 64-slice (and higher) multidetector CT (MDCT) scanners allow for better spatial (versus CMR) and temporal (versus older scanners) resolution, shorter scanning times and breath-holds, and electrocardiogram (ECG)-gated acquisition for detailed cardiac structural analysis.[8] The use of intravenous iodinated contrast further permits assessment of ventricular volumes, ejection fraction, and vascular structures. The specific protocols for image acquisition are beyond

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Abbreviations and Acronyms

AA = ascending aorta

ce-MRA = contrast-enhanced magnetic resonance angiography

CMR = cardiac magnetic resonance

CT = computed tomography

CTE = chronic thromboembolic

DCE = delayed contrast enhancement

ECG = electrocardiogram

IVRT = isovolumic relaxation time

LV = left ventricle

MDCT = multidetector computed tomography

NPV = negative predictive value

PA = pulmonary artery

PH = pulmonary hypertension

PPV = positive predictive value

PVR = pulmonary vascular resistance

RHC = right heart catheterization

RV = right ventricle

RVEF = right ventricular ejection fraction

TR = tricuspid regurgitation

VMI = ventricular mass index

WHO = World Health Organization

the scope of this review, but are well described elsewhere.[9,10] Thus, the systematic evaluation of cardiac, pulmonary, and vascular structures with image reconstruction makes MDCT a favorable single-test modality.

Cardiovascular assessment

The use of retrospective ECG gating has allowed for more accurate quantification of ventricular volumes and ejection fraction that correlate well with CMR with minimal intra- and inter-observer variability.[11-13] Bi- and multi-phasic contrast injection protocols can be used to optimally visualize right and left heart structures, including the coronary arteries if needed.[14,15] Although retrospective ECG gating typically requires a higher radiation dose, recent technical developments allow for large reductions in radiation dose without compromising diagnostic accuracy.[16]

There are multiple studies using CT of the thorax to look for useful measures to diagnose PH.[4] Chan et al demonstrated that several parameters were predictive of PH of varying etiologies independent of age, sex, pulmonary capillary wedge pressure, and body size (body surface area, thoracic diameter, and ascending aorta [AA] diameter).[17] Specifically, dilatation of the main pulmonary artery (PA) ≥ 29 mm had a sensitivity and specificity for the detection of PH of 77.4% and 89.6%, respectively. However correlation of main PA diameter with mean PA pressure by right heart catheterization (RHC) varies widely and may depend on the severity of disease.[17,18] The main PA to AA diameter ratio >1.0 was 86.8% sensitive and 79.2% specific for a diagnosis of PH.[17] Additionally, the PA/AA diameter ratio correlated strongly with RHC-derived mean PA pressure ($R^2=0.45$, $p<0.001$), which was enhanced when combined with echocardiography-derived RV systolic pressure ($R^2=0.55$, $p<0.001$), giving a 96% specificity and 59% sensitivity to detect PH.[19] In patients with connective tissue disease, however, the PA/AA diameter ratio showed only modest correlation with hemodynamic quantification of PH.[20] Others have found that CT-measured PA volumetric analysis normalized to body surface area correlates highly with mean PA pressure ($r=0.89$, $p<0.05$), although this was a small sample size of patients with sleep apnea.[21] In patients being evaluated for lung transplantation, the strongest correlation with mean PA pressure was seen by combining the cross-sectional areas of the main and left main PA indexed to body surface area ($r=0.81$, $p=0.0001$).[22]

ECG-gated MDCT measures of RV and left ventricular (LV) wall thickness and dimensions have also been studied. RV hypertrophy suggests that exposure to chronic pressure overload and septal bowing into the LV during systole (pressure overload) or diastole (volume overload) are important markers of RV strain (Fig 1).[10] Regions of the RV demonstrated changes in structure and function in patients with PH, most notably at the infundibulum, with compensatory regional wall hypertrophy regardless of right-sided filling pressures.[23] Simon et al described that wall stress of the infundibulum is no different between compensated and decompensated patients with PH, but those with decompensated disease demonstrated increased infundibular end-systolic wall thickness.[23] Others have shown that increased RV free wall, RV/LV free wall ratio, and RV/LV lumen ratio are also predictive of PH.[17] The

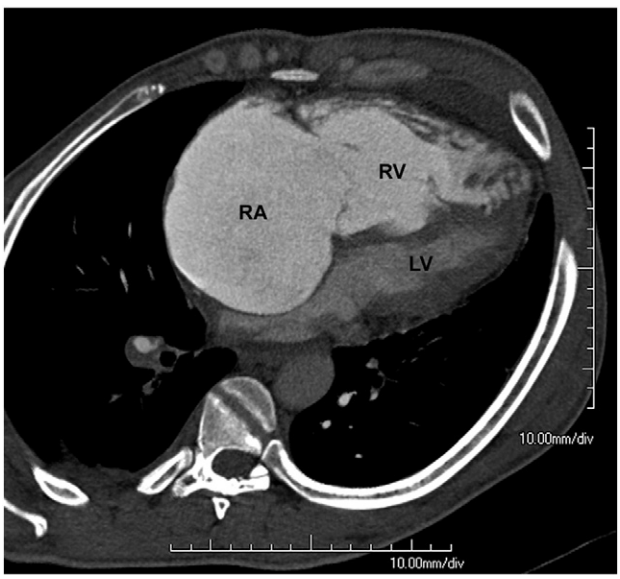

Fig 1. Axial CT scan image of a patient with pulmonary hypertension. This is a four-chamber view in diastole. Note the massive RA, RV hypertrophy, RV enlargement, and septal bowing into the LV cavity causing reduced LV volume. RA, right atrium; RV, right ventricle; LV, left ventricle.