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Review Article

Contemporary Imaging in Chronic Pulmonary Thromboembolic Disease

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Abstract

Pulmonary hypertension is the end result of rarefaction of functioning blood vessels within the lung. This can be caused by a number of pathologies, but in the case of chronic thromboembolic pulmonary hypertension, occlusion of the blood vessel is usually initiated by acute thrombus, which, over time changes to chronic fibrous plaque which has a limited response to medical therapy. The most effective treatment options involve mechanical solutions in the form of surgery or balloon pulmonary angioplasty. Treatments are most effective when introduced early in the disease process. In order that patients may benefit from these treatments, an effective diagnostic pathway is essential. Echocardiogram remains the most effective screening tool in the diagnosis of pulmonary hypertension. Right heart catheterization is the gold standard tool for the assessment of pulmonary hemodynamics. Cardiac magnetic resonance (CMR) imaging is an evolving tool that can be used to evaluate the right ventricular function. It is the gold standard in evaluating right ventricular function and has lower interobserver variability than echocardiography. Despite being a more expensive and less available tool, CMR has the ability to accurately assess blood flow within the pulmonary vasculature, which can enable early detection of disease and response to therapy. Cardiac magnetic resonance imaging is now recognized as an essential component of the imaging armamentarium to assess pulmonary vascular disease.

Key words: Conventional medical therapy, pulmonary hypertension, right ventricle

Introduction

Pulmonary hypertension refers to a constellation of conditions defined by an elevated pulmonary arterial pressure. It is usually a progressive disease, associated with a poor prognosis at 5 years and a high associated symptom burden.[1] The etiologies of pulmonary hypertension have been divided into 5 groups as defined by the 2018 National Institute for Health and Care Excellence (NICE) classification. The disorder may be idiopathic or complicate a number of medical conditions, including connective tissue disease and the majority of cardiovascular and respiratory diseases. It may also be the result of occlusive pulmonary emboli, in the form of chronic thromboembolic pulmonary hypertension (CTEPH), classed as Group IV in the 2018 NICE classification of pulmonary hypertension.[2] This is defined by a mean pulmonary arterial pressure obtained at right heart catheterization (RHC) above 25 mmHg and a pulmonary capillary wedge pressure of <15 mmg along with at least one persistent perfusion defect seen on imaging (V/Q scanning or pulmonary angiography) after 3 months of anticoagulation. This often occurs as a complication following acute pulmonary embolus and is said to occur in 2–4% of cases.[3]

There are a number of treatment options which are available for patients with CTEPH. This includes surgical clearance (pulmonary thromboendarterectomy), percutaneous procedures (balloon pulmonary angioplasty [BPA]), and medical therapy. The treatment offered depends on the anatomical location of the thromboembolic disease and whether it is proximal pulmonary artery (PA) (surgically operable) or distal disease (BPA and medical therapies available).

In order for patients to benefit from these well-established treatments, an appropriate diagnosis is required.

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Echocardiography [Figure 1] remains the main screening investigation for pulmonary hypertension, mainly because it is inexpensive, widely available, and convenient to perform. The presence of a tricuspid regurgitation jet is used to estimate right ventricular systolic pressure but can over or underestimate pressure and is not present in up to 30% of patients.[4] The gold standard diagnostic investigation remains RHC,[5] in conjunction with a number of imaging modalities, such as computer tomography pulmonary angiography (CTPA), cardiac magnetic resonance (CMR) imaging and magnetic resonance pulmonary angiography (MRPA). RHC is an invasive investigation, which carries some associated risks and discomfort for patients but allows direct measurement of the pressure within the main PA, in addition to pulmonary vascular resistance and cardiac output. Ideally there would be an accurate non-invasive investigation, which would reliably provide the diagnosis without exposing patients to such risk.

The use of CMR has been increasing in pulmonary arterial hypertension in recent years due to increased availability, improved acquisition times and ease of use.[6] CMR has several advantages over echocardiography, including improved assessment of left and right ventricular mass, volume and ejection fraction, and is the gold standard for structural and functional assessment of the right ventricle (RV).[7]

Pathophysiology of Chronic Thromboembolic Disease (CTED)

Present evidence suggests that the development of CTEPH follows an acute thromboembolic event and in the majority of cases of CTEPH a history of acute VTE can be elicited.[8] The presence of acute thrombus induces an inflammatory response within the vessel lumen which organizes the thrombus, before spontaneously lysing it using endogenous fibrinolytic system. Over time, there is usually complete resolution of the thrombus,

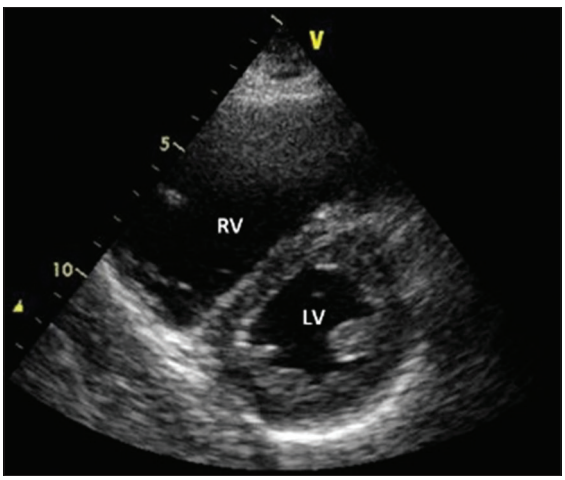

Figure 1: Echocardiography image of parasternal short axis demonstrating a dilated right ventricle and compression of the interventricular septum toward the left ventricle

restoring normal blood flow. However, in some cases, the clot is not completely lysed and instead, the thrombus material changes to a fibrous plaque, containing macrophages and lymphocytes,[9,10] leading to permanent pulmonary vascular occlusion. This vascular occlusion leads to increased pulmonary vascular resistance and increased pulmonary arterial pressure leading to the condition of CTEPH. CTEPH can also cause remodeling of the small distal pulmonary arterioles, in addition to chronic stenosis of larger more central vessels. Clinically, it leads to impaired exercise capacity and can proceed to right heart failure and premature death.[11] The management of CTEPH is well validated:[12] there is a potential cure for CTEPH in the form of surgery. Pulmonary endarterectomy has been shown to cure, or at least substantially reduce the pressure within the pulmonary vasculature and RV in those with central disease.[12] For those with more distal disease, or those with persisting pulmonary vascular obstruction following surgery, there is an option for BPA, which has been shown to improve pulmonary vascular hemodynamics and functional state.[13]

CTED is a term currently employed for those patients in whom following acute pulmonary embolus there remains chronic vascular obstruction and exercise intolerance, without evidence of pulmonary hypertension at rest. It has been shown that these patients can have a functional limitation when tested with cardiopulmonary exercise testing[11,14,15] and rarely successful treatment with PEA has been performed.[16] It should be recognized that CTED represents a spectrum of conditions characterized by as few as a single residual obstruction versus, on the other extreme, enough disease to lead to elevated pulmonary vascular resistance and the development of pulmonary hypertension. Differentiating between these conditions in patients is important since it dictates the individuals' treatment strategies.

Magnetic resonance imaging (MRI) is now central to the investigation of patients with thrombotic pulmonary vascular disease. This manuscript will cover some of the advances in imaging techniques that help in the evaluation of patients with thrombotic pulmonary vascular disease.

Physics of MRI

MRI was discovered by Paul Lauterbur and Peter Mansfield in the 1940s. It uses the natural magnetic properties of the body to produce images. The human body is composed mainly of water, which is principally made from the hydrogen ion (H_2O). Under normal circumstances, H_2O have two poles, north and south, and spin on their axes, which are randomly aligned. When exposed to a magnetic field, such as in an MRI scanner, the protons axes then align. The uniform position of the ions then creates a magnetic vector along the axis of the MRI scanner. The vector is created by an electromagnetic wave and represents the instantaneous magnetic field strength and direction at any point in which the wave is propagating.[17] The magnet in the MRI scanner can act upon the positively charged H_2O, with radiofrequency pulses,

causing the ions to spin. The direction of the pulses can change the direction of the spins, allowing the build-up of layers of detail. The frequency of atomic nuclei rotation is dependent on the strength of the magnetic field to which they are exposed.[17] This can be changed using a series of electric coils and can be done for different parts of the body with different sections of the body resonating as different frequencies are applied.

As the radiofrequency is turned off, the vector returns to its resting state with proton axes randomly aligned, this in itself causes a radio wave to be emitted. This is the signal used to create the MRI images. There are coils used around the region of the body that is being imaged to optimize the detection of that emitted signal. These signals are plotted and with multiple signals, images are developed.[18] Multiple radio wave pulses can be used to highlight particular tissues or abnormalities. Tissue differentiation is seen as different tissues, such as water and fat, have different relaxation times once the radio waves are turned off. This relaxation time can be measured. T1 relaxation is the time taken for the vector to return to its normal resting state and T2 is the time taken for the axial spin to return to baseline.[19]

Clinical Role of CMR Imaging

Despite echocardiography being a valuable first line investigation, there are some weaknesses and a more comprehensive evaluation is merited when there is diagnostic uncertainty. CMR is the gold standard imaging modality for assessing the right ventricular structure such as size and function. As pulmonary hypertension progresses and the PA is unable to accommodate the full cardiac output, the RV begins to maladapt to the pressure changes with progressive dilatation. CMR can characterize these morphological and functional changes over time. As pressure increases, the interventricular septum shifts towards the left ventricle (LV) during late systole and, in severe cases, the septum bows towards the LV as RV and PA pressures exceed systemic pressure.[20] There are a number of specific imaging techniques which can be used in MRI that can characterize the RV with increasing accuracy and utility.

Cine MRI images are obtained by repeatedly imaging an area of interest over a period of time. In CMR, this is achieved by acquisition of images at multiple time points during the cardiac cycle, synchronized with the electrocardiogram. These images can be arranged such that the blood flow within the heart can be seen during a cardiac cycle. From this, both RV and LV end-diastolic and end-systolic volumes can be measured and hence ejection fraction and stroke volume can then be calculated[7] along with accurate determination of ventricular dimensions and muscle masses [Figure 2]. In pulmonary hypertension, these are both seen to increase. Factors such including RV: LV ratio >1 and RV wall thickness >4 mm are suggestive of the presence of PH.[21] A further advantage of the high-level tissue characterization and multi-planar images that can be obtained from CMR is the ability to assess for and quantify intracardiac shunting. In addition, a hot topic in contemporary pulmonary vascular research is the determination of risk for the individual patient using risk scores. CMR is proving to be increasingly useful in risk stratifying the likelihood of clinical deterioration. Higher RV volume and reduced RV ejection fraction are predictive of worse outcomes.[22]

Phase contrast imaging [Figure 3] is an MRI technique that can be used to visualize moving fluid. Proton spins moving in the same direction as the magnetic field gradient develop a phase shift that is proportional to the spin velocities. In a pulse sequence, bipolar gradients are used to encode spin velocities. Moving spins in fluid will experience a different magnitude of the second gradients compared to the first because of the spatial position. This information can be used to determine the velocity of the fluid. This technique can be applied to the PA to assess cardiac output.[23] Blood velocity can be calculated, which has been shown to strongly correlate with PA pressures and pulmonary vascular resistance.

However, despite the many strengths of cardiac MRI in the evaluation of the RV, there is as yet no reliable way of using it to measure the PA pressure. Therefore, the "gold standard" investigation for pulmonary arterial pressure remains RHC. This is the most accurate way of measuring pressure and can estimate RV function by the assessment of the right atrial pressure and right ventricular end diastolic pressure. These represent a measure of preload, PA pressure, pulmonary vascular resistance and stroke volume, to measure contractility.

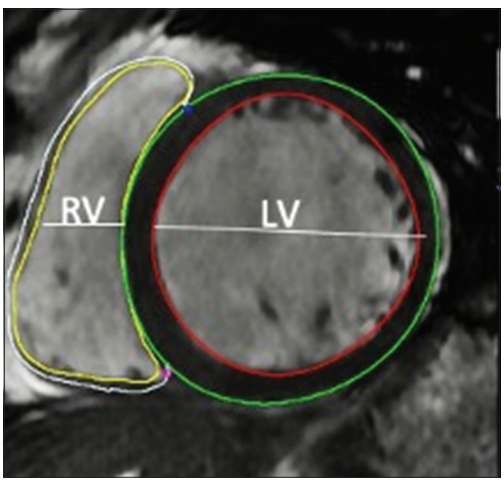

Figure 2: Short axis CMR images of the right ventricle and left ventricle in a healthy individual

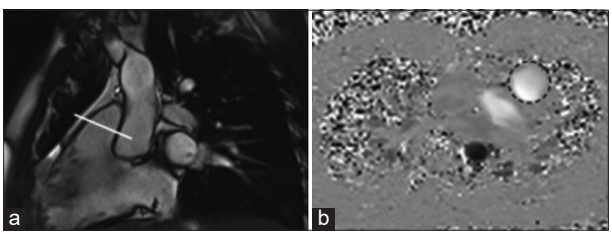

Figure 3: Steady State Free Precession cine sequence used to identify the location of the pulmonary valve (a) and phase imaging (b) of the blood flow across the valve

Flow Mapping

Phase contrast measurements of blood flow through the great vessels can be used to calculate the cardiac output, shunt fractions and regurgitant flow over the valves. It is a phase-sensitive method that can calculate velocity of blood flow from the detected signals in vessel lumen. Flow is measured by obtaining thin, cross-sectional images of the vessels that are sensitized to through-plane velocity during a single cardiac cycle, usually 3 mm or less. An oblique slice is obtained to intersect the vessel [Figure 3]. The lumen is covered by a set of pixels by drawing a region of interest (ROI) around the blood pool. If a ROI is drawn around each slice in the cardiac cycle, the cardiac output can be calculated. The flow through each pixel is calculated and multiplied by the number of pixels covering the area of the vessel.[24] This gives an accurate estimate of cardiac output and can be therefore used to determine the functionality of the RV.

Flow velocity can also be measured. This is done by applying a flow-encoding gradient along the direction of the imaging pulse sequence after the excitation. This generates a flow curve and can be used to determine if there is significant valve regurgitation and allows quantification of intracardiac shunts. In addition, vessel compliance can be evaluated using this technique. Measuring the change in vessel area in cross section during the cardiac cycle can be used to calculate the distensibility, which is reduced when there is high pressure in the vessel.[24] These measures again have been used to evaluate the function of the RV and has prognostic use with regards to pulmonary hypertension.

Although phase contrast measurements are the most accurate way of measuring the cardiac outputs on CMR, there are some issues that may introduce inaccuracies to the measurements. Regions of interest around the vessel lumen need to be accurately drawn, otherwise signals will only partially be from flowing magnetization and tissue will be used as part of the flow calculation. This can lead to underestimation of the blood flow. Changes in flow velocity using a 2-dimensional acquisition can lead to ghosting artifacts. This can occur during respiration. As such, there can be replication of blood vessels along the direction of the phase-encoded signal. Using signal averaging during non-breath-hold can reduce the risk of these flow variables.[25]

Tissue Tracking

Regional wall abnormalities can impact upon ventricular function and influence clinical outcomes. Regional ventricular function can be measured using both CMR and echocardiography. However, the latter can be complicated by poor image quality using subcostal views. CMR provides higher quality images and is able to provide biventricular imaging. This imaging can be done using a number of techniques, including myocardial tagging, phase contrast velocity imaging, displacement encoding, strain encoding and feature tracking (CMR-FT). The exact methodology employed is dependent on the software package used for analysis.[26]

CMR-FT is a useful technique because, unlike the other techniques, it can be applied to the short axis stack acquired during standard CMR protocols, whereas other techniques tend to require specific tissue tracking image acquisition in addition to the standard protocol. Tracking methods identify a small window on one image and search for a comparable image on the subsequent frame. The displacement detected on serial images represents the local tissue displacement. In cardiac tissue tracking, the window is required to be at least 8 × 8 pixels. Much larger images leads to degradation of the quality and any smaller may mean that the tissue displacement is beyond the limits of some of the images, and therefore does not detect the tissue movement. The resolution of the images is important. If this is too low, the larger displacement leads to larger search areas and images become less comparable. If too high, frame to frame displacements are too small, such that the pixels are difficult to identify. The sequences detect movement inward and outward, during systole and diastole. It is easier to track tissues moving apart, and, as such, the sequence begins near end-systole and tracks the tissue during diastole. Initially, tracking was designed for 2-dimensional images, but has now been developed for 3-dimensional volumetric regions, such that radial, longitudinal and circumferential tracking of the RV wall can be performed.[26]

Dimensional Flow

This is an exciting novel measurement technique which is in its infancy in its application to pulmonary vascular medicine. Blood flow in the cardiac chambers and great blood vessels is multidimensional and multidirectional. Whilst CMR can measure cardiac chamber volumes, and therefore calculate cardiac output using the difference between volumes measured at end diastole and end-systole, there are some inaccuracies in using this method, with the inclusion of papillary muscles and trabeculations, leading to interobserver variability. A more accurate and comprehensive way of measuring intracardiac flow and cardiac output is using velocity encoded phase contrast imaging and is now recognized to be the gold standard for these measurements.[27] Using these methods, a shunt fraction can be calculated in the presence of intracardiac abnormalities. Furthermore, it allows the estimation of peak blood flow velocity and the effects of turbulent blood flow on the vessel wall. The measurement encodes flow in all three special directions for the duration of the cardiac cycle. This had previously been performed using 2D cine CMR but 4D flow has been shown to have improved reproducibility compared with 2D.[28]

4D CMR includes phase-contrast MR with blood flow encoded in all spatial dimensions in addition to the dimension of time. It allows for the visualization of the multidirectional blood flow throughout the pulmonary circulation [Figure 4]. It has been shown that the 4D flow appearances are altered in those with pulmonary hypertension and it has been used to assess for hemodynamic changes.[29] Abnormal flow patterns,

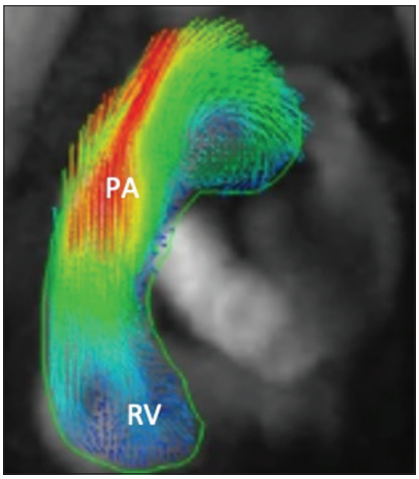

Figure 4: Flow mapping of blood travelling from right ventricle to pulmonary artery. Color mapping represents the flow velocity ranging from highest in red (55 cm/s) to the slowest in blue (0 cm/s)

mainly in the form of vortex formation, are associated with raised intrapulmonary pressures. The persistence time of the vortex has been demonstrated to correlate with mean PA pressure, making it an attractive tool not only in the diagnosis of pulmonary hypertension, but in monitoring response to treatment.

Wall sheer stress can also be evaluated using 4D flow. This is the frictional force along the inner wall of the artery, caused by viscous drag. At a pathophysiological level, a reduction in wall sheer stress in the systemic circulation is thought to lead to endothelial cell dysfunction because higher wall sheer stress increases endothelial nitric oxide release.[30] In pulmonary hypertension, wall sheer stress has been demonstrated to be reduced. As such, it can be said that reduction in sheer stress does lead to reduced endothelial cell health, possibly leading to further vascular remodeling. Wall sheer stress assessment remains a technique used only in research studies, but there is an increasing body of evidence to support its use and it may become a clinical parameter in due course.[30] Pulmonary vascular resistance and clinical indices are currently the main methods by which response to therapy is measured. However, having another objective measurement would lead to a more holistic evaluation of an individual's response to therapy.

MRPA

MRPA has the benefit of being able to identify the presence of acute or chronic pulmonary embolus, without using ionizing radiation. Performed using gadolinium contrast, MRPA has sensitivity of 78% and specificity of 99%[31] for the presence of acute pulmonary thromboembolism, which makes it an attractive option for routine diagnostic use. However, acquiring technically optimal images is more challenging than other modalities used to image the pulmonary vasculature. Longer breath-hold times and acquisition times limits the routine use of MRPA. In the PIOPED III study, up to 25% of studies were shown to be technically inadequate[31] due to poor arterial opacification, breathing artefact and wrap-around artefact. As such, although pulmonary embolus can be diagnosed using MRPA, the absence of it does not exclude its presence to the same degree as CTPA.

Given that widespread use of MRPA appears not to be possible, it is important to ensure the correct population is chosen for its use. Examples for the use of MRPA include: (1) those with a low to intermediate probability for the presence of pulmonary thromboembolism; (2) patients who are intolerant of iodine-based contrast agents, and; (3) females of childbearing age who are at higher risk of the effects of ionizing radiation. Using MRPA in those who are claustrophobic, who are unable to hold their breath and have a high probability of venous thromboembolism the diagnostic yield is likely to be low and require a second imaging modality.[32]

In current UK practice, MRPA is not a part of the routine diagnostic pathway for the diagnosis of acute pulmonary embolism. Its role lies mainly in the multi-modality evaluation of those with CTED and pulmonary hypertension and assessing the potential to benefit from surgical intervention. Defects seen in CTEPH on MRPA are similar to that seen on CTPA, such as intraluminal bands, narrowing or obstructed vessels, central thrombus and bronchial arterial hypertrophy. MRPA is used as a "road map" for surgeons performing pulmonary endarterectomy.[33] It has the advantage over conventional angiography of being non-invasive, as well as allowing evaluation of the RV in the same investigation. Whilst this has not replaced RHC, it has the ability to evaluate patients in response to therapy, both medical and surgical.[34]

Whilst MRPA has limitations for routine use for the diagnosis of acute and chronic pulmonary thromboembolism, this is an evolving technology. With its lack of exposure to ionizing radiation and strong sensitivity and specificity, there are strengths that make it an attractive option for common usage if the evolution of technology can reduce costs improve image acquisition.

Conclusions

CTED is a complex disease which represents a significant burden for patients following acute VTE. The recognition of this disease is important as it can be associated with significant morbidity and mortality for patients and if detected early enough there are potential curative options available for management. Imaging plays a critical role in this process in determining the diagnosis, risk stratification and management strategies. Cardiac MRI is now recognized as an essential component of the imaging armamentarium to assess pulmonary vascular disease. There are a number of emerging MRI-facilitated techniques for determining key functional and structural parameters of the RV which can guide the clinician in making the best decisions for the care of their patients. This includes MR-pulmonary angiography which can identify key anatomical occlusions in the pulmonary vascular tree.

It is clear that over the next 5 years that cardiac MRI will become central to the diagnosis and management of patients with CTED.

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