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IMAGING (Q TRUONG, SECTION EDITOR)

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Role of Imaging for Suspected Cardiac Thrombus

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Abbreviations *18F-FDG* 18-Fluorodeoxyglucose · *AF* Atrial fibrillation · *CCTA* Cardiac computed tomography · *CT* Computed tomography · *CMR* Cardiac MRI · *CVC* Central venous catheter · *ECG* Electrocardiogram · *LA* Left atrium · *MI* Myocardial infarction · *MR* Magnetic resonance imaging · *NBTE* Nonbacterial thrombotic endocarditis · *NSTEMI* Non ST-elevation myocardial infarction · *PE* Pulmonary embolism · *PET/CT* Positron emission tomography/computed tomography · *RHT* Right heart thrombus · *SSFP* Steady-state free precession · *TEE* Transesophageal echocardiography

Abstract

Purpose of review Cardiac thrombus formation is a frequent complication of a variety of prevalent diseases. Embolism of cardiac thrombus has the potential to result in significant morbidity and mortality from cerebrovascular and peripheral vascular events.

Recent findings Echocardiography is the most commonly used imaging modality for diagnosing intracardiac thrombus. However, technological advances in computed tomography and magnetic resonance imaging have allowed newer noninvasive modalities to evolve into robust tools for the clinical evaluation of patients suspected of disease.

Summary Complimentary use of these imaging techniques is crucial in the diagnosis of cardiac thrombus and initiation of anticoagulation therapy.

Introduction

Cardiac thrombus formation is a frequent complication of a variety of prevalent diseases. Embolism of cardiac thrombus has the potential to result in significant morbidity and mortality from cerebrovascular and peripheral vascular events. Reliable thrombus identification impacts embolic event risk stratification and therapeutic decisions. Many imaging modalities have been explored in the diagnosis of intracardiac thrombus including transesophageal echocardiography (TEE), transthoracic echocardiography (TTE), cardiac computed tomography angiography (CCTA), and cardiovascular magnetic resonance (CMR). The most commonly used method for diagnosing intracardiac thrombus is echocardiography; however, it is limited because intracardiac clots cannot be distinguished from cardiac masses. TEE has high specificity and sensitivity for examining the posterior and inferior heart, and is particularly important for visualizing clots and determining areas of blood stasis in the left atrium. However, TEE is semi-invasive since it involves passing a transducer into the esophagus and gastric fundus. By contrast, TTE is noninvasive and can be used for visualization of clots in the ventricles. However, it provides limited views of the left ventricular apex and distal ascending aorta. CCTA and CMR are well-established alternatives to detecting intracardiac thrombus. These methods require more time and greater resources but may provide improved detection of cardiac thrombi.

Pathophysiology

Intracardiac thrombi occur in association with abnormal blood flow or stasis within the cardiac chambers, subendocardial injury, and/or hypercoagulability—collectively referred to as Virchow's triad [1]. Blood stasis is due to decreased contractility of the heart chambers, as seen in atrial fibrillation, regional wall dyskinesia, or akinesia. Subendocardial injury occurs most commonly after a myocardial infarction leads to mural thrombi formation along the ventricle walls. Hypercoagulability has many causes including advanced age, genetic diseases, inflammatory conditions, and lifestyle conditions such as obesity and immobility. The interaction of factors in the triad increases patient susceptibility to developing intracardiac thrombi.

Imaging modalities

Accurate decision making in the evaluation of cardiac thrombus is critical to direct therapy and, in some cases, guide procedure planning. Echocardiography, computed tomography, and magnetic resonance imaging have emerged as primary complementary imaging modalities in detection and diagnosis (see Table 1).

Table 1. Performance of imaging modalities in the detection of cardiac thrombus

Modality	Percent range				References	Comments
	Sensitivity	Specificity	Positive predictive value	Negative predictive value		
Transthoracic echo (TTE)	35–91	91–100	67–100	94–99	[2–4]	Limited visualization of LAA and atria; better utility in detecting LV/RV thrombus; echo contrast often helpful
Transesophageal echo (TEE)	39–97	96–100	87–93	79–*100	[2]	Historical gold standard; invasive compared with other options, may be time consuming, provided better visualization of atria than TTE, still limited anatomical windows, contraindicated in patients with esophageal pathology, varices, and upper GI bleeding
Computed tomography (CT)	74–100	90–99	87–92	79–100	(58)	Noninvasive compared with TEE, excellent spatial resolution and anatomical coverage and better sensitivity than TTE and high NPV; may need to perform delayed imaging; utilization of ionizing radiation and contrast, may be considered a disadvantage

Table 1. (Continued)

Modality	Percent range Sensitivity	Specificity	Positive predictive value	Negative predictive value	References	Comments
Magnetic resonance imaging (MR)	79–100	99–100	98–100	98–100	[2–4] [2–4] [2–4]	Lack of ionizing radiation, comprehensive anatomical visualization, no limitation to acoustic windows, great for RT heart chambers; LGE provides definitive for evaluation of LV thrombus; more scanner and operator dependent; relative longer scanning time and reduced availability compared with other modalities

Echocardiography

Echocardiography is widely accepted as a primary screening tool in the evaluation of cardiac-related thrombus. Cardiac embolic events account for up to 30% of all ischemic strokes [5]. Left atrial (LA) and, specifically, left atrial appendage (LAA) thrombosis is the most prevalent source of cardio-embolic events and commonly occurs with atrial arrhythmias. Due to the posterior location of the left atrium, the LAA is not well visualized using TTE, and as such, TEE is the imaging modality of choice. The LAA is typically described as having one of four morphologies; chicken wing, cactus, windsock, or cauliflower. The chicken wing morphology is associated with the lowest likelihood of thromboembolic events. [6] Left ventricular (LV) thrombosis is reported to occur in as many as 40% of patients in the immediate post-myocardial infarction period [7], most commonly in anterior infarctions [8]. TTE is the study of choice for evaluation of LV function and the presence of thrombosis, demonstrating excellent sensitivity (95%) and specificity (85–90%) [9]. TTE is superior to TEE in evaluating LV thrombus because of the position of the LV apex in relation to the imaging transducer. Use of contrast-enhancing agents can significantly increase the accuracy of diagnosis in technically limited studies where the apex is not well visualized [10]. Strain imaging may assist in stratifying patients at-risk for LV thrombus post-myocardial infarction, identifying patients with impaired LV contractile function who are at higher risk for thrombus formation [11].

Native cardiac valves can be a source of thrombosis in systemic and pulmonary vascular beds as a result of vegetations, thrombi, and calcific material. In patients in whom there is a suspicion for infectious endocarditis (IE), TTE is appropriate for initial evaluation of valve morphology and hemodynamics. TTE is also useful to assess for progression or resolution of IE or to assess for hemodynamic consequences of IE. TEE is the study of choice, however, when there is a high suspicion for complications such as abscess or fistula formation, or valve perforation.

Foreign intracardiac material such as catheters, pacing wires, or valve prosthesis can be a nidus for infected material or thrombi. Bioprosthetic valves have a lower yearly thrombosis risk (0.5–1%) compared with mechanical prosthetic valves (1–2%). In the identification of prosthetic valve thrombosis, TEE is superior to TTE [12]. Mechanical thrombosis typically occurs during periods of sub-therapeutic anticoagulation. TTE is an appropriate initial screening tool when there is suspicion of valve or catheter infection or thrombosis. Thrombi and vegetations may be difficult to distinguish from one another, depending on chronicity; thus, patient risk factors play a large role in stratifying risk for thrombus formation versus infection.

Computed tomography

Computed tomography (CT) has steadily increased in clinical importance as a noninvasive approach for intracardiac thrombus imaging. CT is not as restricted in capability as TTE imaging regarding patient size, limited acoustic windows, and presence of pulmonary disease. Multidetector CT, usually 64 detectors or more, can obtain thin section, high spatial resolution images of the entire cardiac anatomy. With the addition of electrocardiographic (ECG) gating, artifacts due to cardiac motion are significantly reduced, if not eliminated. Also, the imaging dataset is both volumetric and isotropic, allowing for high-resolution reconstructions in any plane. CT, like echocardiography, involves short scan times and is widely available [13].

Since CT data is acquired using ionizing radiation, radiation dose should always be considered in the study protocol selected. Particularly in ECG-gated CT, radiation dose profiles may exceed exposures of cardiac catheterization, if not optimized. Iodinated contrast is a mainstay of CT protocols and the potential risks of contrast-induced nephropathy and allergic reactions warrant attention. Multi-society guidelines are available for the performance and acquisition of cardiac computed tomographic angiography (CCTA) [14••].

Magnetic resonance imaging

Echocardiography and CT are often sufficient for first-line evaluation of cardiac thrombus. However, CMR can provide additional robust clinical and anatomical information without the use of ionizing radiation. Additionally, CMR provides increased specificity for evaluation of myocardial tissue characteristics and can permit differentiation of cardiac neoplasm from intracardiac thrombi.

Intracardiac thrombi typically have a brighter appearance than tumor or myocardium in inversion-recovery imaging with short inversion times and a darker appearance with long inversion times. Some thrombi show delayed contrast enhancement, which characterizes them as organized clots. However, care must be taken to use thin sections for the detection of small thrombi, and the entire region of interest must be included in the imaging volume. Along with cine and T2-weighted images, the standard perfusion and myocardial delayed enhancement sequences may be helpful. Because thrombi are avascular, they do not show contrast uptake on perfusion sequences and usually appear as dark foci bordering the endocardium. The delayed enhancement sequence is the most sensitive sequence for depicting the thrombus, which usually appears as a dark intracavitary or mural filling defect commonly adherent to hyperenhancing infarcted myocardium [15].

Clinical presentations

Left atrium

Thrombus is the most common intracardiac mass, most frequently involving the LV or LA [16]. The LAA, varying in shape, is a trabecular blind ending structure that acts as a nidus for the majority of thrombi in the LA.

Predisposing factors include mitral valve pathology, left ventricular dysfunction, and arrhythmias, most commonly atrial fibrillation (AF) [17••, 18]. These conditions result in stasis of blood, thus promoting thrombogenesis. The most significant complication from LA thrombus is arterial embolization, particularly to the brain, which leads to stroke.

AF results in decreased contractility leading to remodeling and dilation of the LA and LAA. The average LAA volume is three times larger than normal in patients with AF [19•]. Other prothrombotic changes caused by AF include a smoother endocardial surface and a higher degree of endocardial fibroelastosis [19•]. The risk of systemic embolism in patients with AF not on anticoagulation ranges from < 1%/year to > 20%/year [19•, 20]. AF can be divided into nonvalvular and valvular subtypes [20]. Valvular AF, usually due to stenosis of the native or a mechanical prosthetic mitral valve, is associated with a higher risk of thromboembolism [20]. Up to 80% of patients with mitral stenosis and systemic embolism have concurrent AF [20]. Studies have reported a four- to five-fold increased risk of stroke in the presence of AF and up to 17-fold in the presence of concurrent mitral stenosis [21, 22].

The majority of thrombi due to nonvalvular AF occur in LAA which is more thrombogenic due to trabeculations from pectinate muscles and its blind ending pouch morphology [23]. Some studies report an association between LAA morphology and an increased risk of stroke [6, 19•, 24]. Enlarged and bifid LAA are known structural risk factors for thrombus formation [18].

The incidence of cardiac thrombosis after cardiac transplantation ranges from 2 to 15% which may be underestimated since most patients are asymptomatic and thrombus is detected incidentally [25, 26]. The activation of multiple components of Virchow's triad results in increased prevalence of LA thrombus formation. This phenomenon is enhanced by the classic biatrial anastomosis which results in a greater degree of atrial dysrhythmias and enlargement compared with the preferred bicaval anastomosis [27, 28]. Thrombus formation is noted more frequently along the suture line suggesting that other technical surgical factors are implicated in thrombosis formation. Other alterations in cardiac transplant patients include a hypercoagulable state, reduced endothelial function, and altered platelet function. This results in patients being resistant to conventional antiplatelet therapy [29].

LV thrombus

The majority of left ventricular thrombi (LVT) are apical in location, most commonly associated with myocardial infarction (MI), more specifically ST-elevation myocardial infarction (STEMI) of the anterior myocardial wall [2]. MI leads to activation of multiple components of Virchow's triad, resulting in endothelial injury, increase in procoagulant factors resulting in hypercoagulability, and dyskinesia leading to blood stasis [3].

Despite significant reduction in the incidence of MI since the introduction of aggressive therapy with percutaneous interventions, LV thrombus is reported in 3–4% of all post MI patients and 9% of those with anterior STEMI on TTE [19•, 30•]. Other risk factors for LVT include cardiomyopathy, heart failure, LV aneurysms, and endomyocardial fibrosis [17••]. The risk of LVT is systemic embolization, reported in up to 10% of post MI patients [31]. As with thrombus formation throughout the body, Virchow's triad plays an important role in inciting and propagating LVT [30•].

LV thrombus can occur within 24 h after an acute MI with about 90% of thrombi forming within 2 weeks of the inciting event [1]. More remote thrombus is usually associated with continued or worsening LV dysfunction [3]. The predictors for post MI LVT include reduced ejection fraction, anteriorly located infarct, regional wall-motion abnormalities, and multiple aneurysms [32, 33].

Endomyocardial fibrosis is a restrictive cardiomyopathy endemic to parts of Africa, Latin America, and Asia [34]. Its pathophysiology is not well understood, but is characterized by deposition of fibrous tissue in the endocardium, predominantly in the apices of the right and left ventricles, with progression to obliteration of the apex and deposition of thrombus. The presence of thrombus in these patients is a bad prognostic sign with death occurring from chronic heart failure, arrhythmia, and thromboembolism [34]. Patients with left-sided disease fare worse than those with right-sided disease, which can be asymptomatic for years [34]. Early identification and treatment of LVT in these patients is important to improve outcomes.

TTE is the initial modality used for evaluating LVT [19•]. However, reported sensitivity is as low as 21–53%, although this can be improved with the use of intravenous contrast, which raises sensitivity to approximately 64% [30•].

Delayed enhanced CMR is the gold standard for evaluating LVT, with a sensitivity of up to 88% and specificity of 99–100% verified by surgical findings [2]. Studies have shown an incidence of post MI thrombosis using CMR of 9–15% in all patients and 14–25% in those with anterior MI, which is higher compared with thrombosis detected by TTE [3, 30•]. TTE can only detect around 10% of thrombi less than 1 cm [2]. Screening algorithms that use the extent of wall-motion abnormality on TTE to trigger consideration of further imaging with CMR have been proposed [4•].

Delayed enhancement CMR relies on tissue characterization in detection of LVT rather than anatomic appearance, allowing thrombus to be differentiated from myocardial structures regardless of location or morphology [19•]. It also allows for myocardial characterization and cine imaging. CMR is also more useful in differentiating thrombus from tumor due to improved specificity for evaluation of hemorrhage, calcification, and cystic necrosis which are commonly present in the stroma of tumors [18]. CCTA has been reported to be comparable to CMR in detecting LVT with advantages that include shorter scanning time and widespread availability [17••]. Other reported advantages of CCTA include detection of thrombus-associated calcification and evaluation for ventricular assist device pump thrombosis in patients with end stage heart failure, an increasingly recognized complication [17••, 18]. Disadvantages of CCTA include increased radiation to patients and the need for intravenous iodinated contrast [17••].

RA/RV thrombus

Right heart thrombus (RHT) is rare compared with left heart thrombus. The most common etiology is embolization of clot from the venous system. Other etiologies include stasis from global/regional wall-motion impairment including arrhythmogenic right ventricular cardiomyopathy (or after right ventricular myocardial infarction), arrhythmia, in situ thrombosis (related to foreign bodies such as indwelling vascular catheters, pacemaker leads, prosthetic valve, assist device), or vasculitides (e.g., Behcet's syndrome, coagulopathies, Churg-Strauss syndrome, Loffler's endocarditis) [19•, 35–38].

Central venous catheters (CVCs) are used in up to 8.1% of hospitalized patients [37, 39]. The incidence of CVC-associated right heart thrombi widely varies, from 8 to 66% [37, 40, 41]. Most commonly, CVC-associated thrombi are incidentally detected on echocardiography. It is postulated that thrombus formation is due to mechanical trauma of the catheter repeatedly striking the wall of the vena cava or the right heart, resulting in coagulation cascade [42–44]. CVC-related altered flow dynamics may also contribute. Additional risk factors include large-bore catheter and fibrin sheaths.

RHT can become infected and/or lead to pulmonary embolism. The prevalence of an RHT in the setting of an acute PE is reported to be 4 to 18% [45, 46]. The right heart can fail in the setting of a large thrombus/PE burden, leading to sudden cardiovascular collapse. The overall mortality rate for RHT is reported to be 27.1–44.7% and as high as 100% in undiagnosed patients [46, 47].

TTE is the initial modality of choice for evaluating suspected RHT due to its noninvasive nature, low cost, availability, and portability. Nevertheless, TEE has greater sensitivity, approximately 97%, compared with 50–60% for TTE [48]. With contrast agents, more detailed assessment of suspected RHT can be performed. However, distinguishing between normal myocardium and cardiac mass from thrombus on echocardiography may be difficult.

CCTA is an accurate method for detecting right heart thrombi and is more sensitive in detection than TTE [49]. Despite the advantages of the excellent spatial resolution of CT, diagnostic difficulties may arise from misdiagnosis of normal anatomical variants such as prominent crista terminalis, Chiari network, or the right ventricular moderator band as thrombus.

CMR gradient-echo sequences or steady-state free precession (SSFP) cine images are robust and sensitive in detecting intracardiac thrombi including RHT. Thrombi are isointense to myocardium on T1 and T2 images. They do not enhance on first pass perfusion and late gadolinium enhancement (LGE) sequences. After administration of contrast, the myocardium and cardiac cavity demonstrate high signal intensities relative to thrombi, allowing for the easy detection of intracardiac thrombi. Hence, post-contrast sequences are most useful for diagnosing intracardiac thrombi. In addition, CMR allows for detection of underlying structural heart disease such as MI, arrhythmogenic right ventricular cardiomyopathy, and other cardiomyopathies which may contribute to thrombus formation.

Fluorodeoxyglucose positron emission tomography/computed tomography (18F-FDG PET/CT) may play a critical imaging role in challenging cases of cardiac thrombus where other modalities prove equivocal. As a functional imaging modality, PET/CT may help differentiate intracardiac lesions as non-malignant vs malignant based on their degree of metabolic activity [50–52].

Valve-associated thrombus

Valvular thrombosis

Anatomical and functional abnormalities of native heart valves can predispose patients to developing valvular thrombus due to altered flow dynamics. This risk increases with replacement of the native heart valve with a prosthetic valve. The reported annual incidence of prosthetic valve thrombosis (PVT) varies from 0.1 to 5.7% [53]. It is most often found during work-up of an embolic episode. The patient may also present with heart failure symptoms due to valvular obstruction.

Echocardiography is the primary diagnostic modality used to evaluate PVT because it allows direct dynamic evaluation of the prosthesis. Thrombus typically presents as hypo-to-isoechoic well-circumscribed lesion on the valve. Using color Doppler, echocardiography may show abnormal valve closure, abnormal transvalvular flow (as evidenced by aliasing), and central regurgitation. Echocardiography also permits the evaluation of valvular mobility, effective prosthetic area, and transvalvular gradient measurement [54]. However, evaluation can be limited due to acoustic shadowing caused by the prosthesis or by surgical clips or wires. Small thrombi can be difficult to visualize on TTE. By contrast, TEE allows for a more precise evaluation of the heart valve leaflets and flow dynamics. In challenging cases where TTE is equivocal, invasive TEE is contraindicated, and clinical suspicion remains high, CCTA or CMR can be obtained for further evaluation.

CCTA can aid in the diagnosis of prosthetic valve thrombus. It typically demonstrates a hypodense mass on the associated prosthetic valve leaflet. ECG-gated retrospective exam allows for the visualization of the thrombus throughout systole and diastole including assessment of restricted valvular opening [55, 56]. However, CT artifacts from the mechanical prosthesis can limit the detection of a subtle thrombus.

On CMR, thrombus is T1 and T2 hypointense relative to the heart muscle. It shows no enhancement on first pass perfusion and LGE sequences. SSFP cine images allow for assessment of location of thrombus, valvular morphology, and function throughout the systole and diastole [57].

Limitations of echocardiography and CCTA are that they do not always allow for differentiation between thrombus and valvular vegetations. On the other hand, contrast-enhanced CMR allows for accurate differentiation of thrombus from valvular/nonvalvular intracardiac neoplasms based on lack of enhancement on first pass perfusion and LGE sequences. However, CMR images can suffer from susceptibility artifact from the mechanical prosthesis, limiting evaluation.

Compliance with Ethical Standards

Conflict of Interest

The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

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