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ECG-Gated CT Pulmonary Angiography: Anatomy, Imaging Techniques and Severity Assessment

Reham Ramadan Ali , Huda Ahmed Mohamed Ismael , Elsayed Hamed Zidan , Rabab Mohamed Abd El Hay

Department of Radiodiagnosis, Zagazig University, Zagazig city, Sharkia governorate, Egypt

***Corresponding author: Huda Ahmed Mohamed Ismael**

Abstract

Background: Pulmonary embolism (PE) is a potentially life-threatening cardiovascular condition that requires rapid and accurate diagnosis. Computed tomography pulmonary angiography (CTPA) is the principal imaging modality for diagnosing PE and evaluating its cardiovascular consequences. However, conventional non-ECG-gated CTPA may be affected by cardiac motion artifacts, potentially limiting the assessment of right ventricular morphology and function. ECG-gated CTPA allows image acquisition synchronized with the cardiac cycle, potentially improving the visualization of cardiac structures and the assessment of CT-derived parameters associated with PE severity.

Aim: This review article aims to discuss the anatomical and pathophysiological basis of pulmonary embolism, the role of CTPA in diagnosis and severity assessment, and the potential benefits and limitations of ECG-gated CTPA compared with conventional non-ECG-gated imaging, with particular emphasis on right ventricular strain and pulmonary vascular changes.

Conclusion: CTPA plays an important role in the diagnosis and assessment of pulmonary embolism by providing information about pulmonary arterial obstruction and associated cardiovascular abnormalities. ECG-gated CTPA may improve the evaluation of right ventricular morphology and other CT-derived parameters by reducing cardiac motion artifacts. However, its potential advantages should be considered alongside technical limitations, radiation exposure, and clinical applicability. The selection of an appropriate imaging technique should therefore be guided by the diagnostic requirements and clinical condition of each patient.

Keywords: Pulmonary embolism; ECG-gated CTPA; CT pulmonary angiography; Right ventricular strain; Pulmonary hypertension; Imaging techniques.

Introduction

Acute pulmonary embolism (PE) is a potentially life-threatening cardiovascular condition characterized by the obstruction of the pulmonary arterial circulation by thromboemboli, most commonly originating from deep venous thrombosis of the lower extremities or pelvic veins. The clinical consequences of PE vary considerably, ranging from limited pulmonary vascular obstruction with minimal physiological disturbance to extensive obstruction associated with acute right ventricular (RV) dysfunction, systemic hypotension, obstructive shock, and death. The severity of the condition depends on several interrelated factors, including the anatomical extent of vascular obstruction, pulmonary vasoconstriction, pre-existing cardiopulmonary disease, and the ability of the right ventricle to accommodate the sudden increase in afterload [1,2].

Following embolic occlusion, the pulmonary circulation experiences an increase in vascular resistance caused by both mechanical obstruction and additional vasoconstrictive responses. The resulting elevation in pulmonary arterial pressure increases the workload imposed on the right ventricle. Because the right ventricle is normally adapted to a low-pressure circulation, it is relatively sensitive to an abrupt increase in afterload.

Consequently, extensive acute PE may impair RV contractility, reduce forward blood flow through the pulmonary circulation, and ultimately compromise left ventricular filling and systemic cardiac output [1,2].

Importantly, the degree of anatomical obstruction does not invariably correspond to the clinical severity of PE. Pulmonary vascular resistance is influenced not only by the volume and distribution of thromboembolic material but also by vasoconstriction, hypoxemia, and the patient's underlying cardiopulmonary reserve. Therefore, a comprehensive assessment of PE severity requires integration of clinical findings, cardiovascular function, laboratory markers, and imaging parameters rather than reliance on clot burden alone [2,3].

Pathophysiology and Severity of Acute Pulmonary Embolism

The severity of acute PE is assessed primarily according to its early mortality risk and hemodynamic consequences. Contemporary clinical risk-stratification approaches integrate hemodynamic status, clinical prediction scores, imaging evidence of RV dysfunction, and biomarkers of myocardial injury or stress. This approach distinguishes patients who are hemodynamically unstable from those who remain stable but may have evidence of right-sided cardiac strain [3].

High-risk PE is characterized by hemodynamic instability, including cardiac arrest, obstructive shock, or persistent hypotension attributable to PE. In these patients, acute obstruction of the pulmonary vascular bed can cause severe RV failure and an inability to maintain adequate systemic perfusion. In contrast, patients without hemodynamic instability may fall into intermediate-risk or low-risk categories depending on their clinical risk scores, RV function, and laboratory findings. Intermediate-risk patients may demonstrate RV dysfunction or myocardial injury despite preserved systemic blood pressure, whereas low-risk patients generally lack evidence of significant hemodynamic compromise or clinically important RV dysfunction [3].

Right ventricular strain is one of the principal pathophysiological consequences of clinically significant acute PE. Embolic obstruction increases pulmonary vascular resistance, producing an abrupt rise in RV afterload. Unlike the left ventricle, which is adapted to generate high pressures against the systemic circulation, the right ventricle is less capable of sustaining a sudden and substantial increase in pressure. The resulting pressure overload may impair RV contraction and cause acute ventricular dilatation [1].

Initially, the right ventricle attempts to compensate for the increased afterload by increasing myocardial contractile effort. However, when the obstruction and associated vasoconstriction exceed its compensatory capacity, RV emptying becomes impaired and end-systolic and end-diastolic volumes increase. This results in RV enlargement and increased wall tension. As the right ventricle dilates, its geometry changes, and the interventricular septum may flatten or bow toward the left ventricle. These changes reflect the interaction between the two ventricles within the relatively constrained pericardial space and may further impair left ventricular filling [1,2].

RV dilatation and dysfunction have important consequences for systemic circulation. As the right ventricle becomes less effective at ejecting blood into the pulmonary circulation, pulmonary venous return to the left atrium decreases. At the same time, RV enlargement and interventricular septal displacement may restrict left ventricular diastolic filling. The resulting reduction in left ventricular preload decreases stroke volume and cardiac output.

In severe cases, this sequence produces systemic hypotension and inadequate coronary perfusion. Reduced coronary perfusion, combined with increased RV wall stress and myocardial oxygen demand, can further impair RV contractility and establish a cycle of worsening ventricular dysfunction. This process may culminate in obstructive shock, cardiac arrest, or death if the acute hemodynamic disturbance is not reversed [1,3].

CTPA provides several anatomical indicators of RV pressure overload and dysfunction. The RV/LV diameter ratio is widely used because it reflects the relative enlargement of the right ventricle compared with the left ventricle. An increased ratio, particularly a value greater than 1, suggests RV enlargement and may indicate

increased risk in the appropriate clinical context. Nevertheless, this finding should not be interpreted in isolation, as its significance depends on the clinical presentation and other indicators of RV dysfunction [2].

Additional CT findings include interventricular septal flattening or leftward bowing, enlargement of the right-sided cardiac chambers, and reflux of contrast medium into the inferior vena cava or hepatic veins. Septal flattening or bowing reflects altered ventricular pressure relationships and may accompany significant RV pressure overload. The presence of these findings can strengthen the imaging evidence of right-sided cardiac strain, although their absence does not exclude clinically important PE.

Pulmonary artery enlargement may occur in association with increased pulmonary arterial pressure and altered pulmonary vascular hemodynamics. In acute PE, thromboembolic obstruction reduces the effective cross-sectional area of the pulmonary vascular bed. The resulting increase in pulmonary vascular resistance, together with vasoconstriction in unobstructed vascular segments, increases the pressure against which the right ventricle must eject blood. Enlargement of the central pulmonary arteries may therefore accompany the hemodynamic effects of PE, particularly when the pressure burden is substantial.

On CTPA, the diameter of the main pulmonary artery can be measured as an anatomical parameter, and its relationship to the ascending aortic diameter can be expressed as the pulmonary artery-to-aorta (PA/Ao) ratio. These measurements may provide supplementary information about pulmonary vascular enlargement and the cardiovascular consequences of PE. However, pulmonary artery diameter is influenced by factors other than acute embolic obstruction, including pre-existing pulmonary hypertension, chronic cardiopulmonary disease, and individual anatomical variation. Consequently, an enlarged main pulmonary artery should not be regarded as a specific or independent indicator of acute PE severity [2,4].

A further distinction is required between acute pulmonary artery enlargement and chronic pulmonary arterial remodelling. Acute PE may increase pulmonary arterial pressure, but established enlargement may also reflect longstanding pulmonary hypertension or chronic thromboembolic disease. Persistent thromboembolic obstruction and vascular remodelling can lead to chronic thromboembolic pulmonary hypertension (CTEPH), in which elevated pulmonary vascular resistance imposes a sustained pressure load on the right ventricle. Thus, pulmonary artery diameter should be interpreted in conjunction with RV dimensions, septal configuration, the patient's clinical history, and other available imaging findings rather than being used as a stand-alone marker of acute severity [2,4].

Pulmonary infarction is a potential parenchymal complication of acute PE, particularly when emboli obstruct distal pulmonary arteries. It develops when regional pulmonary perfusion becomes sufficiently compromised to produce ischemic injury, hemorrhage, and ultimately necrosis of lung tissue. Although the lungs receive blood from both the pulmonary and bronchial circulations, this dual blood supply does not completely prevent infarction in all circumstances. Following pulmonary arterial occlusion, bronchial circulation may contribute to alveolar hemorrhage; if the injury and hemorrhage are not adequately resolved, tissue necrosis and subsequent fibrotic scarring may occur [5].

Pulmonary infarction may be associated with pleuritic chest pain, hemoptysis, and peripheral pulmonary opacities on imaging. It can also be accompanied by pleural inflammation or effusion. However, not all patients with PE develop infarction, and the presence of an embolus does not automatically indicate irreversible parenchymal injury. The development of infarction depends on the location and extent of vascular occlusion, the adequacy of collateral perfusion, and the condition of the underlying lung tissue [5].

Other pulmonary consequences may include ventilation–perfusion mismatch and hypoxemia. Occlusion of pulmonary arterial branches reduces perfusion to ventilated lung regions, while associated physiological disturbances may impair overall gas exchange. Hypoxemia can contribute to pulmonary vasoconstriction and may further increase the pressure load on the right ventricle. The relative contributions of these mechanisms vary among patients and depend on the extent of embolic disease and the underlying cardiopulmonary condition [2].

Although many patients recover following appropriate treatment of acute PE, some experience persistent symptoms or develop long-term complications. Post-pulmonary embolism impairment may manifest as persistent dyspnea, reduced exercise tolerance, or limitations in physical functioning after the acute event. Such symptoms may have multiple contributing causes and should not automatically be attributed to persistent pulmonary vascular obstruction.

One of the most important chronic complications is chronic thromboembolic pulmonary hypertension (CTEPH). This condition develops when organized thromboembolic material and associated pulmonary vascular changes produce persistent obstruction and increased pulmonary vascular resistance. The sustained pressure load can lead to chronic RV remodelling and dysfunction, eventually causing progressive right-sided heart failure if left untreated [2,4].

CT-based findings have been investigated for their potential to identify patients at increased risk of developing CTEPH after acute PE. These include the degree and occlusive nature of thromboembolic obstruction, RV enlargement, pulmonary infarction, and mosaic attenuation of the lung parenchyma. Lorenz et al. (2020) reported associations between selected initial CT findings and subsequent CTEPH, although the predictive performance of individual findings was not sufficient to establish them as definitive predictors. Accordingly, these findings should be interpreted as potential indicators of increased risk rather than proof that a patient will develop chronic thromboembolic disease.

Diagnosis of Pulmonary Embolism

The identification of pulmonary embolism (PE) is often challenging because the condition presents with nonspecific symptoms and requires definitive verification [6]. To achieve an accurate diagnosis, healthcare professionals typically combine clinical assessment, laboratory investigations and imaging modalities [7,8]. Each of these diagnostic approaches contributes unique and complementary information that guides the confirmation or exclusion of PE [6]. Clinical assessment is the first step in evaluating suspected cases. Recognizing risk factors such as recent surgery, prolonged immobilization or obesity plays a crucial role in raising clinical suspicion [9].

In addition to risk stratification, healthcare providers carefully evaluate presenting symptoms. Common manifestations include sudden onset of dyspnea, hemoptysis, and pleuritic chest pain, which, although not specific to PE, are strongly suggestive in the appropriate clinical context. Laboratory testing serves as an important adjunct in the diagnostic process. The D-dimer test is widely employed, as a negative result can reliably exclude pulmonary embolism in patients with low to moderate clinical probability However, elevated levels are nonspecific and must be interpreted alongside clinical findings and further imaging[10].

Imaging modalities remain central to the definitive diagnosis of PE. Multidetector computed tomography (CT) is currently regarded as the preferred imaging method for confirming or excluding pulmonary embolism in most patients [6].

In individuals with contraindications to contrast media, such as renal impairment or allergies, ventilation–perfusion scintigraphy provides a valuable alternative [6].

Imaging plays a central role in the diagnosis of acute pulmonary embolism (PE), allowing direct visualization of pulmonary arterial thrombi, assessment of pulmonary perfusion, and identification of associated cardiopulmonary abnormalities. The choice of imaging modality depends on the patient's clinical probability of PE, hemodynamic stability, contraindications to contrast media, pregnancy status, radiation considerations, and the availability of appropriate equipment and expertise [11,12].

Computed tomography pulmonary angiography (CTPA) is the principal imaging modality for confirming acute PE in most adults because it combines high diagnostic accuracy with rapid acquisition and widespread availability. Alternative techniques, including ventilation–perfusion (V/Q) scintigraphy, lower-extremity venous ultrasonography, echocardiography, and magnetic resonance imaging (MRI), have complementary roles in

selected clinical circumstances. Conventional pulmonary angiography is now used predominantly when catheter-based intervention is planned rather than as a routine diagnostic examination [11,12].

CTPA is a contrast-enhanced multidetector CT examination designed to opacify the pulmonary arterial circulation and demonstrate intraluminal filling defects caused by thromboemboli. It is considered the principal imaging reference standard for diagnosing acute PE because it provides direct anatomical visualization of the pulmonary arteries and permits assessment of the distribution and extent of embolic disease. In addition, CTPA may reveal alternative explanations for acute chest pain or dyspnea, including pneumonia, pleural effusion, pneumothorax, and aortic pathology [11,12].

CTPA should be interpreted within an appropriate diagnostic algorithm rather than used indiscriminately in every patient with suspected PE. Clinical pretest probability assessment and D-dimer testing can help determine whether imaging is necessary. In patients with a low or intermediate clinical probability, a negative appropriately interpreted D-dimer result may exclude PE without further imaging. Conversely, patients with a high clinical probability generally require definitive imaging, provided that it can be performed safely and without delaying necessary emergency management [3].

The principal direct sign of acute PE on CTPA is an intraluminal filling defect within a contrast-opacified pulmonary artery. Depending on its orientation and distribution, the thrombus may appear as a central defect surrounded by contrast material, sometimes described as a polo-mint sign on cross-sectional images or a railway-track sign when viewed along the longitudinal axis of the vessel. An acute thrombus may also appear as a partial filling defect that forms an acute angle with the vessel wall or as complete occlusion of an affected arterial branch [11,12].

The anatomical distribution of emboli should be documented systematically. Thrombi may involve the main pulmonary arteries, the right or left pulmonary artery, lobar arteries, segmental branches, or subsegmental branches. A saddle embolus is located across the bifurcation of the main pulmonary artery and extends into the right and left pulmonary arteries. Although the location and extent of thrombus are important descriptive findings, the presence of a large or centrally located embolus does not, by itself, establish the patient's overall clinical risk category [12].

The distinction between acute and chronic thromboembolic disease is also important. Acute thrombi commonly appear as central or eccentric filling defects that may form acute angles with the vessel wall. Chronic thromboembolic disease may instead demonstrate eccentric wall-adherent defects, webs or bands, vessel narrowing, recanalization, and other features of longstanding vascular obstruction. These findings should be considered when interpreting CTPA in patients with persistent symptoms or a history of previous PE [12].

Beyond direct visualization of thrombi, CTPA provides information about the physiological consequences of PE. Relevant findings include right ventricular (RV) enlargement, an increased RV-to-left ventricular (RV/LV) diameter ratio, interventricular septal flattening or leftward bowing, and reflux of contrast material into the inferior vena cava or hepatic veins. These features may indicate right-sided cardiac pressure overload or dysfunction and can contribute to prognostic assessment when interpreted alongside clinical findings [11,3].

Pulmonary parenchymal and pleural abnormalities may also be identified. Pulmonary infarction can appear as a peripheral, pleural-based, wedge-shaped opacity, sometimes with a reverse-halo appearance. Other findings include small pleural effusions, atelectatic changes, and areas of reduced perfusion. Although these abnormalities may support the overall interpretation, they are not specific for PE and must be assessed in the context of the direct pulmonary arterial findings and the clinical presentation [11,12].

Advantages and limitations of CTPA

The main advantages of CTPA include rapid acquisition, broad availability, direct visualization of pulmonary arterial thrombi, assessment of embolic distribution, and the ability to identify alternative thoracic diagnoses. It can also provide useful anatomical information about the right heart and central pulmonary arteries, making it valuable for both diagnosis and initial assessment of disease severity [11].

However, CTPA involves ionizing radiation and iodinated contrast administration. Its diagnostic performance may be reduced by inadequate contrast enhancement, respiratory or cardiac motion, and other technical artifacts. Interpretation of very small peripheral filling defects can be challenging, particularly when image quality is suboptimal. A negative or indeterminate study should therefore be interpreted in conjunction with clinical probability and the technical adequacy of the examination [12].

CT Pulmonary Angiography and ECG Gating

In general, CT displays excellent spatial resolution, wide field of view, multi-planar reformat and allows direct assessment of the pulmonary parenchyma, pulmonary vasculature, and cardiac chambers [13].

CTPA is a non-invasive technique that assesses the pulmonary arteries and veins using X-rays and computer technology. It is commonly used in practice for diagnosis and follow-up of pulmonary embolism and pulmonary hypertension [13] and [14].

The aim of CTPA is to provide detailed anatomy of the main pulmonary artery, its segmental and sub-segmental branches through injecting intravenous contrast agent. Once the right side of the heart and the pulmonary arteries opacify while the aorta is not, the study is considered optimal. Adequate timing is very crucial in CTPA as any time delay allows the pulmonary veins to opacify along with the pulmonary arteries, making their differentiation difficult [15] and [16].

ECG-gated CTPA enables accurate measurement of RV/LV diameter ratio, interventricular septal bowing, and pulmonary artery dimensions, all of which are key prognostic markers in acute PE and predictors of adverse outcomes [17,18].

ECG-gated Computed Tomography Pulmonary Angiography (CTPA) offers significant advantages over conventional non-gated CTPA in predicting the development of chronic thromboembolic pulmonary hypertension (CTEPH), primarily due to its superior ability to assess cardiac function and hemodynamic impact. By synchronizing image acquisition with the cardiac cycle, ECG-gated CTPA minimizes motion artifacts and enables accurate quantification of right ventricular (RV) parameters, including the RV/left ventricular (LV) diameter ratio, interventricular septal deviation, and right atrial enlargement. These parameters are well-established indicators of right ventricular strain, which is a critical determinant in the progression from acute pulmonary embolism to CTEPH. In contrast, non-gated CTPA is limited by cardiac motion artifacts and provides only indirect or less reliable assessment of ventricular function, reducing its prognostic value. Furthermore, ECG-gated imaging allows multiphase reconstruction and dynamic evaluation of ventricular performance, enhancing detection of subtle functional abnormalities and early pulmonary vascular remodeling. Consequently, ECG-gated CTPA provides more robust risk stratification and improves prediction of long-term complications, including persistent pulmonary hypertension and right heart failure [17,19,18].

Limitations and Considerations

Despite its advantages, ECG-gated CTPA has limitations, including increased radiation exposure (especially with retrospective gating), susceptibility to arrhythmia-related artifacts, and higher technical complexity. However, newer technologies such as iterative reconstruction algorithms and dose modulation techniques have significantly reduced radiation dose, making ECG-gated protocols more feasible in clinical practice [19].

Non-ECG gated CTPA: is best acquired in a helical fashion in a caudo-cranial direction from diaphragm to apices in held inspiration to minimize diaphragm related artefact. Approximately 80 ml of contrast medium is injected at 4-5 mL/s with scan triggering 5 seconds after peak main pulmonary artery (MPA) enhancement. Image reconstructions with 1-2 mm axial sections and 1 mm overlap is suggested [20].

Electrocardiogram (ECG)-gated Computed Tomography Pulmonary Angiography (CTPA) is an advanced imaging technique that synchronizes CT image acquisition with the cardiac cycle to minimize motion artifacts and improve visualization of cardiopulmonary structures. Unlike conventional CTPA, ECG-gated acquisition enables more accurate assessment of right ventricular (RV) size, function, and interventricular septal position, which are critical prognostic indicators in acute pulmonary embolism (PE) and predictors of chronic thromboembolic pulmonary hypertension (CTEPH) [17,19].

Dual energy CTPA: is a scan technique very similar to a standard ungated CTPA but with the addition of a saline flush to minimize superior vena cava related artefact. Simultaneous data acquisitions at both 140 and 80 kV (with dual source technology or via rapid kV switching with single source scanners) is used to generate an iodine perfusion map of the lung parenchyma via a subtraction technique [20].

CT Findings Related to Pulmonary Embolism Severity

Signs related to pulmonary hypertension discussed in this chapter are: RV dilatation, increased thickness of the RV free wall, increased RV outflow tract thickness, right ventricular to left ventricular (RV/LV) ratiom Flattening or reverse bowing of the septumm reflux of intravenous contrast from the RA into the IVC and hepatic veins, The diameter of the main pulmonary artery, the ratio between the PA and the ascending aorta, Increased right pulmonary artery diameter and left pulmonary artery diameter distal to the bifurcation, Ratio of segmental PA to accompanying bronchus, coronary sinus dilatation, decreased left atrial sizeو pulmonary artery distensibility (PAD), mosaic perfusion [15]

Among the most significant radiological predictors described in the literature are right ventricular (RV) changes. Under normal conditions, the right ventricle is a thin-walled chamber with a smaller diameter than the left ventricle. However, elevated pulmonary arterial pressure leads to progressive structural remodeling. [21].

RV dilatation is one of the earliest and most clinically significant findings, reflecting increased afterload and ventricular strain. Studies have shown that enlargement of the RV on CT strongly correlates with disease severity and poor clinical outcomes [21]. RV size is determined by measuring the maximal distance between the ventricular free wall and the interventricular septum, perpendicular to the long axis of the heart at the mid ventricular level. It can be considered enlarged if the transverse diameter exceeds 57 mm when measured on axial slices (Figure 1) [20].

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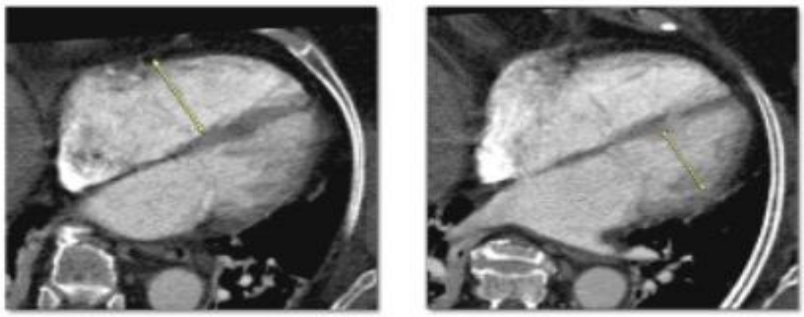


Fig. (1): Axial contrast enhanced CT chest shows measurements of maximal right ventricular and left ventricular diameters [21].

In chronic cases such as CTEPH, increased thickness of the RV free wall is frequently observed, indicating chronic pressure overload and compensatory hypertrophy. In general, the normal thickness of the RV free wall is <4 mm. The RV free-wall thickness is measured at the mid-ventricle level [21]. RV hypertrophy is not an immediate process, and it is a sign of chronic pressure overload that can be encountered in pulmonary hypertension (Figure 2) [22]

Similarly, increased RV outflow tract thickness has been reported as an additional sign of long-standing right ventricular adaptation to elevated pulmonary pressures The musculature of the right ventricular outflow tract is compacted and subjectively easier to measure than the right ventricular free wall. It can be measured anteriorly 1 cm below the pulmonary arterial valve[23].

Particularly important and widely validated prognostic marker is the right ventricular to left ventricular (RV/LV) ratio. This parameter is routinely measured on CTPA and reflects the balance between right and left ventricular dimensions. Cardiac chamber dimensions are typically measured on the short-axis plane in CTPA, allowing calculation of the right ventricular to left ventricular (RV/LV) diameter ratio [24]. An RV/LV ratio greater than 1 is widely accepted as an indicator of right ventricular dysfunction, reflecting increased afterload caused by acute pulmonary embolism. Numerous studies have confirmed its prognostic value, demonstrating strong associations with short-term mortality and adverse clinical outcomes [25,26,27]. Elevated cardiac biomarkers, such as troponin, have also been observed in patients with RV/LV ratios exceeding 1 [28]. Furthermore, an increased RV/LV ratio has been linked to a higher likelihood of intensive care unit admission [27].

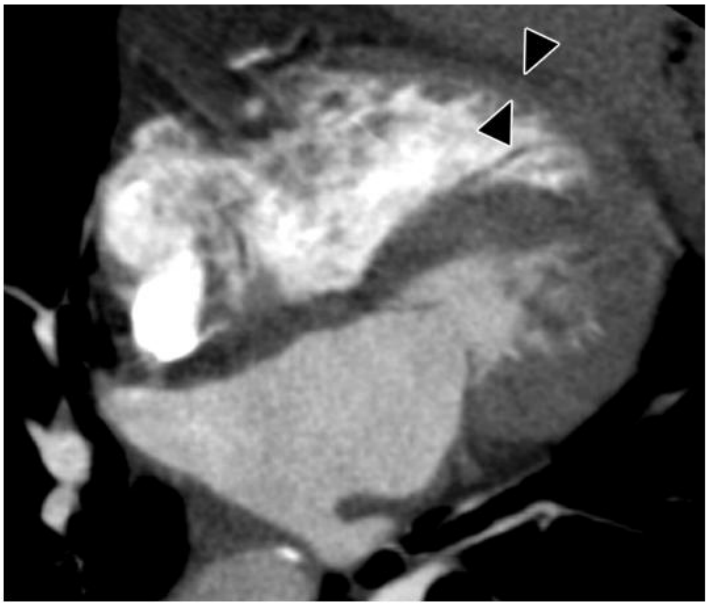


Fig. (2): Contrast-enhanced ECG-gated CT scan shows right ventricle (RV) free wall hypertrophy measured at 7 mm (arrowheads) and mild RV enlargement [22].

Right ventricular strain or dysfunction: There are several chest CT signs for determining the severity of RV dysfunction. Beside changes in the measurements of RV, there are changes in the interventricular septum and reflux of contrast material into the inferior vena cava (IVC) [29].

Other important prognostic signs include Interventricular Septal Displacement Under normal conditions, the interventricular septum bows toward the right ventricle; however, increased right-sided pressures in acute pulmonary embolism may cause leftward displacement. Although septal bowing demonstrates high specificity for right ventricular dysfunction, its sensitivity remains limited [30]. Evidence suggests that abnormal septal

morphology is more frequently observed in high-risk patients [31]. Moreover, a meta-analysis reported significant associations between septal deviation and increased mortality and adverse clinical outcomes [32].

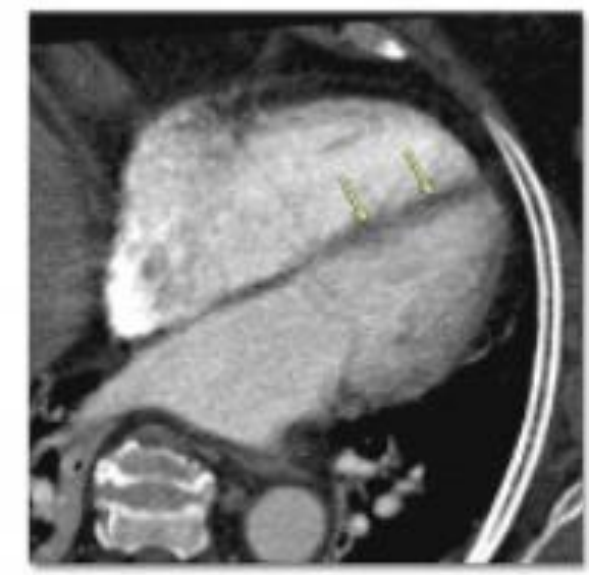


Fig. (3): a 4-chamber view reconstruction of a chest computed tomography scan shows septal flattening (arrows) [29].

Because of right ventricular dilatation, reflux of intravenous contrast from the RA into the IVC and hepatic veins occurs in PH. The severity of reflux of contrast medium into the IVC or hepatic veins is graded into grade 1= no reflux; 2= trace of reflux into IVC only; 3= reflux into IVC but not hepatic veins; 4= reflux into IVC and proximal hepatic veins; 5= reflux into IVC and hepatic veins down to the mid-portion of the liver; 6= reflux into IVC with opacification of distal hepatic veins (Figure 4) [33], [30,28].

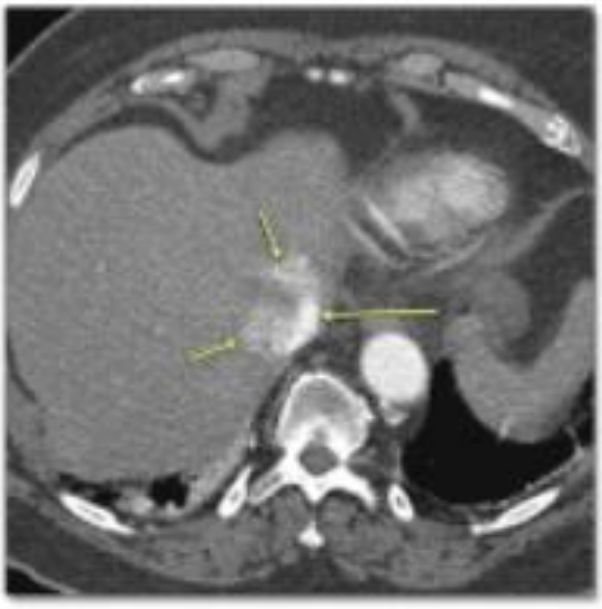


Fig. (4): Axial upper abdomen enhanced CT shows grade 4 reflux of contrast medium into the inferior vena cava (long arrow) and proximal hepatic veins (short arrows) [29].

The diameter of the main pulmonary artery (PA), as well as the ratio between the PA and the ascending aorta, are considered important indicators of pulmonary hypertension. Studies have shown that increased pulmonary and aortic diameters are significantly associated with mortality in patients with pulmonary embolism

[28]. Additionally, enlarged PA diameter has been correlated with adverse clinical outcomes, with reported median values of approximately 29.9 m in high-risk patients [34].

The diameter of the pulmonary artery directly reflects vascular dilation and the thickness of blood vessel walls, making the measurement of MPA diameter an effective indicator of pulmonary artery pressure. PH is suspected when MPA diameter ≥29 mm [35]. The maximum width of the main pulmonary artery (MPA) is measured on axial mediastinal CT images acquired during inspiration, at the level of its bifurcation orthogonal to its long axis (Figure 5) [36].

Factors like age, body surface area, and vascular compliance can influence pulmonary artery dilation. Therefore, relying solely on MPA diameter may not accurately reflect pulmonary artery pressure variations among individuals [35]. Therefore, the ascending aorta is also measured at the same axial level as MPA diameter measurement and compared to it (Figure 5) [37].

Ratio of the MPA to the AO > 1 has shown to correlate well with systolic pulmonary artery pressure (sPAP) rather than using MPA diameter alone as variables affecting MPA diameter will influence the ascending aorta equally [37].

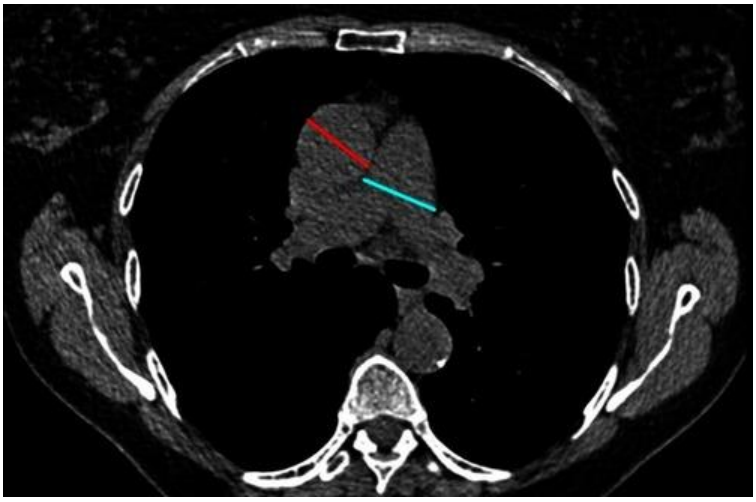


Fig. (5): CT image (mediastinal window) showing measurement of main pulmonary artery (blue line) and aortic diameter (red line) [36].

Peripheral pulmonary vascular signs have also been described extensively. Increased right pulmonary artery diameter and left pulmonary artery diameter distal to the bifurcation are recognized markers of pulmonary hypertension, Both the right pulmonary artery (RPA) and left pulmonary artery (LPA) can be measured 1.5 cm distally from the bifurcation of the MPA RPA and LPA diameters of 16 mm and 21 mm, respectively raise the likelihood of presence of PH [38].

Other important signs include Ratio of segmental PA to accompanying bronchus.Normally, the outer diameter of the bronchus is equal to that of the adjacent pulmonary artery. Increased segmental artery–to-bronchus ratio greater than 1:1 in three or more lobes contributes to the specificity of diagnosing PH[39].

Other important prognostic signs include coronary sinus dilatation Dilatation of the coronary sinus, particularly measurements exceeding 9 mm, has been identified as an independent predictor of 30-day mortality in patients with acute pulmonary embolism [25]. Unlike the RV/LV ratio, this parameter is less affected by cardiac motion or variations in the cardiac cycle, making it a reliable supplementary prognostic marker.

Recent studies have also highlighted the significance of left atrial size as a prognostic marker Acute increase in pulmonary vascular resistance can impair right ventricular function, subsequently reduce pulmonary venous return and left atrial (LA) filling. As a result, decreased LA size has emerged as a prognostic marker in

pulmonary embolism [40]. Studies have demonstrated that smaller LA dimensions and volumes are associated with worse outcomes [41]. For instance, LA volumes below 62 mL have been linked to significantly higher mortality rates compared to larger volumes [42].

From a functional perspective, pulmonary artery distensibility (PAD) has emerged as an important imaging biomarker. Reduced PAD indicates increased vascular stiffness and is strongly associated with pulmonary hypertension and chronic thromboembolic disease The use of ECG-gated CTPA has enhanced the assessment of PAD by enabling dynamic measurement of arterial changes throughout the cardiac cycle [43]. PAD can be calculated using the formula:

Coronary artery calcifications are a well-established marker of coronary artery disease and are frequently detected in patients undergoing CTPA for suspected pulmonary embolism [44]. Emerging evidence suggests their prognostic significance in acute pulmonary embolism, with studies demonstrating associations between calcification severity and both short- and long-term mortality [45]. In a large cohort study, patients with pulmonary embolism and coexisting cardiovascular disease exhibited significantly higher long-term mortality rates [46].

Intravascular webs are considered highly characteristic of chronic thromboembolic obstruction. These web-like filling defects represent residual organized thrombotic material adherent to the vessel wall after incomplete thrombus resolution. Similarly, tapering or abrupt narrowing of pulmonary arteries suggests fibrotic remodeling and chronic vessel obstruction rather than acute embolic occlusion.

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