3 Imaging of Deep Vein Thrombosis and Pulmonary Emboli

Olga R. Brook

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3.1 Ultrasound

Ultrasound is the imaging modality of choice for diagnosis of deep vein thrombosis (DVT) in the extremities[1] since studies in the early 1980s first showed its value.[2,3] Ultrasound is cost-effective, relatively accurate, portable, and does not require radiation exposure and therefore can be utilized successfully in the variety of health care settings.

3.1.1 Accuracy of Doppler Ultrasound for Diagnosis of DVT

The accuracy of DVT diagnosis with ultrasound varies depending on the presence of symptoms (symptomatic [pain, swelling, and cramping] vs. asymptomatic) and suspected anatomical location of DVT (proximal lower extremity, distal lower extremity, or upper extremity). Asymptomatic DVT is diagnosed when ultrasound is performed for risk stratification in patients with pulmonary emboli (PE) or in screening patients with cancer or immobile patients. Approximately one-third of patients with DVT do not have any symptoms.[4]

Lower extremity: A review of studies and systematic review by Segal et al showed that for *symptomatic* patients, sensitivity of Doppler ultrasound for *proximal lower extremity* thrombosis ranges from 89 to 96% with specificity of 94 to 99%.[5] In different studies, the accuracy of *symptomatic distal lower extremity* DVT ranges from 75 to 93% in sensitivity and from 73 to 99% in specificity.[6,7,8] Sensitivity of detecting DVT in *asymptomatic* patients is significantly lower, ranging from 47 to 62%; however, specificity remains high at 94 to 97%.[8,9]

Upper extremity: Di Nisio et al conducted a systematic review of 11 studies involving symptomatic patients and found that despite the fact that overall most studies suffer from poor methodology, it appears that sensitivities of compression ultrasound with and without Doppler or Doppler ultrasound by itself or contrast venography are similar, ranging from 85 to 97% with specificity ranging from 87 to 96%, with lower numbers for contrast venography.[10] The authors suggest that the addition of Doppler did not seem to make a significant difference, while the best accuracy is provided by compression ultrasound alone. However, given the overall low quality of available literature, it is prudent to perform compression ultrasound with Doppler to rule out upper extremity DVT (▶ Table 3.1).

3.1.2 Indication

DVT in lower extremities has an estimated incidence of 5/10,000 patients per year in the general population with incidence increasing with age and comorbidities.[11] PE have been diagnosed in up to 60% of patients with DVT, especially as it progresses more centrally from the calf veins. Death

Table 3.1 Summary of accuracy of ultrasound for detection of deep vein thrombosis (DVT)

	Lower extremity	
	Proximal	**Distal**
Symptomatic	Sensitivity: 89–96% Specificity: 94–96%	Sensitivity: 75–93% Specificity: 73–99%
Asymptomatic	Sensitivity: 47–62% Specificity: 94–97%	
	Upper extremity (symptomatic only)	
Compression ultrasound	Sensitivity: 97% Specificity: 96%	
Doppler ultrasound	Sensitivity: 84% Specificity: 94%	
Compression + Doppler	Sensitivity: 91% Specificity: 93%	
Contrast venography	Sensitivity: 84% Specificity: 87%	

from all causes occurs in approximately 6% of DVT cases and 12% of PE cases within 1 month of diagnosis.[12] Several algorithms have been developed to assess the pretest probability of DVT to accurately detect patients at higher risk of DVT and PE, while reducing unnecessary imaging studies. The Wells criteria, based on clinical signs and symptoms that are most commonly used in clinical practice,[13] classified patients into risk categories for thromboembolic disease as low, intermediate, or high. Patients with low or intermediate risk should be further evaluated with serum D-dimer, while patients classified as high risk should be evaluated with CT pulmonary angiogram to rule out PE. More recently, Le Gal et al simplified risk groups to "DVT likely" and "DVT unlikely."[14] Because the prevalence of DVT in the "DVT unlikely" or low-risk groups is not zero, a follow-up D-dimer test is recommended for further decision-making.[5] If the D-dimer test is negative, a DVT can be safely excluded. If a D-dimer test is positive, patients should undergo ultrasound evaluation of the symptomatic extremity. In a patient with intermediate or high risk or "DVT likely," D-dimer evaluation is unnecessary and ultrasound evaluation will be required to rule out DVT.

3.1.3 Technique

Collaborative practice guidelines for peripheral venous ultrasound examinations were developed by the American College of Radiology, American Institute of Ultrasound in Medicine, Society for Pediatric Radiology, and Society of Radiologists in Ultrasound to set standards for evaluation of venous thromboembolism (VTE) in both the lower and upper extremities.[15] Evaluation of the lower extremity generally begins with optimal grayscale ultrasound technique in both the transverse and sagittal planes, beginning with the common femoral and femoral veins moving down to the popliteal vein, distal to the tibioperoneal trunk. Similarly, evaluation of the upper extremity should include the internal jugular, subclavian, axillary, brachial, cephalic, and basilic veins in both the transverse and sagittal planes. In both examinations, compression by the ultrasound transducer is applied every 2 cm or less in the transverse plane to completely compress the normal vein lumen to eliminate a possibility of acute isoechoic thrombus or lack of thrombus visualization due to poor technique or technical factors. Areas of clinical concern, such as focal pain, require special attention with focal imaging in the transverse and sagittal planes. Doppler waveforms should be obtained in the bilateral common femoral veins and bilateral subclavian veins, to evaluate for asymmetry or loss of phasicity during the respiratory cycle that will indicate distal (pelvic) thrombus or compression of the pelvic veins. Doppler waveforms should be obtained in the sagittal plane. At the authors' institution, in lower extremity evaluation, compression ultrasound is extended to the posterior tibial veins and peroneal veins. Additionally, augmentation is utilized at our institution at the common femoral, femoral, and popliteal veins when obtaining sagittal Doppler waveforms. Augmentation is a sonographic technique when manual compression or a "squeeze" is briefly applied below the level being imaged (i.e., if imaging the upper calf, transient compression of the lower calf). A brief increase in venous inflow should be visualized on Doppler ultrasound. Lack of increase in venous inflow indicates obstruction to the flow (thrombus or mass) between the area of the "squeeze" and imaging site. Augmentation should be avoided if thrombus is visualized, as it may dislodge the thrombus and cause a pulmonary embolus. Finally, if DVT is identified on a unilateral study, in the authors' institution, the contralateral leg needs are assessed to determine the burden of the disease.

3.1.4 Imaging Features

The imaging features of acute DVT include presence of echogenic clot within a distended and noncompressible vein, without or limited color flow on Doppler imaging. Imaging features that can be seen in chronic DVT include thickened vessel walls, atretic or diminutive venous segments, and the presence of collateral vessels. Chronic clot will become more echogenic over time and can calcify.[16] The clot can have varying degrees of echogenicity, up to being completely anechoic in hyperacute presentation.[16] Therefore, lack of vein compression by the ultrasound transducer is the most definitive test for the detection of DVT. Pelvic DVT can be detected by indirect sonographic signs, such as lack of normal respiratory variation with respiration and Valsalva maneuvers with Doppler tracing over common femoral vein (▶ Fig. 3.1). Further evaluation with computed tomography (CT) or magnetic resonance (MR) venography can be useful in this scenario.[17] Indirect signs of DVT in the calf veins

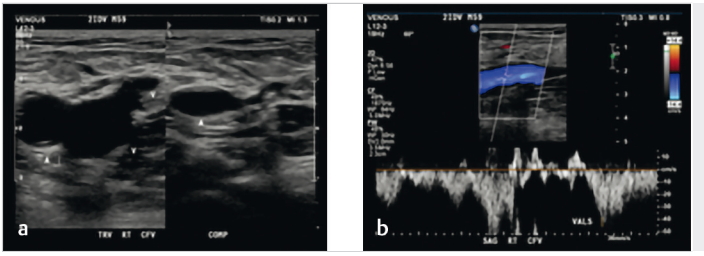

Fig. 3.1 **(a)** Normal transverse images of the right common femoral vein (CFV) with and without compression. **(b)** Normal color Doppler and waveform in the right CFV showing normal respiratory variation and Valsalva.

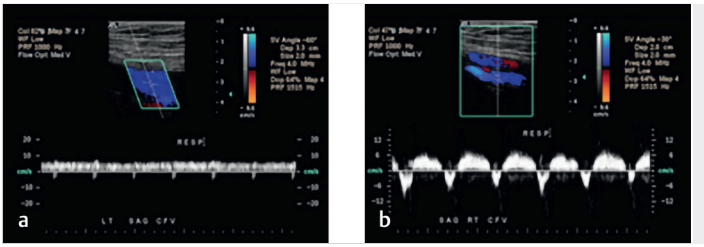

Fig. 3.2 **(a)** Abnormal respiration variability of the left common femoral vein (CFV) raising suspicion for distal (central) pathology. Confirmed left deep venous thrombosis in a case of May–Thurner syndrome. **(b)** The same patient with normal right CFV respiration variability.

are lack of augmentation (increase in flow) with calf squeeze in the deep thigh veins as seen on Doppler ultrasound[16] (▶ Fig. 3.2).

3.1.5 Pitfalls

False-positive ultrasound diagnosis of DVT can be caused by pelvic masses compressing vasculature, such as adenopathy, malignancy, abscesses, and hematomas, as well as diffuse soft-tissue edema. At times, an arterial abnormality can be confused for venous one.[16]

Important pitfall for false-negative results is aberrant anatomy, such as duplicated femoral and popliteal veins. In these cases, all veins should be evaluated for DVT.[17] *Patient factors*, such as morbid obesity, extreme edema, postoperative bandaging, and lack of cooperation with the sonographer can also limit usefulness of the study.

Technical expertise of the sonographer as well as use of the established protocol is of vast importance in reducing variability and thus improving accuracy of the examination.

3.2 Computed Tomography Pulmonary Angiography

Use of computed tomography pulmonary angiography (CTPA) for diagnosis of PE has increased significantly since 1992, when it was shown to be as effective as conventional angiography[18] and more accurate than ventilation perfusion (V/Q) scan. Multidetector CTPA has been shown to have a sensitivity of 83% and specificity of 96%, as identified in the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) II study.[19]

3.2.1 Indication

Evaluation of a patient's pretest probability of pulmonary embolism can be performed using several decision-making tools, including Wells' criteria, Geneva criteria, and pulmonary embolism rule-out criteria (PERC).[20,21,22] Using these tools, low- and intermediate-risk patients undergo high-sensitivity D-dimer testing. A negative D-dimer effectively excludes deep venous thrombosis.[23] In intermediate-risk patients with a positive D-dimer or those with a high pretest probability of pulmonary embolism, multidetector CTPA is indicated.[24,25]

3.2.2 Technique

The CTPA technique has continued to evolve over time with the improving technology of CT scanners, although the main premise behind the examination has not significantly changed. Axial CT imaging is obtained from the lung apices to the diaphragm following administration of intravenous (IV) contrast, with the scan timed for opacification of the pulmonary arteries. Optimally, CTPA is obtained using 16- or 64-channel multidetector CT scanner to allow for single breath hold imaging to

BMI	kVp	IV contrast (cc)	LA threshold (HU)
<25	80	80	170
25-33	100	100	135
>35	120	120	100

Fig. 3.3 kVp, contrast dose, and scan triggering threshold based on the patient's body mass index (BMI).

reduce motion artifacts. In our institution, kVp, volume of contrast, and HU threshold for scan initiation are chosen based on the patient's body mass index (BMI; ▶ Fig. 3.3) in order to improve vessel opacification while reducing radiation exposure and contrast load. A bolus tracking technique is recommended for optimal timing of the scan, usually focused on the main pulmonary artery. At the authors' institution, the left atrium is used to track the bolus with the scan obtained during shallow inspiration in order to perform "a double rule-out" to evaluate for both PE and aortic dissection, as symptoms between these conditions can sometimes overlap. Scan slices are acquired in thin 0.5-mm sections, with 2.5- and 1.25-mm axial reformats produced for review. In addition, 5-mm sagittal and coronal and 15-mm maximal intensity projections (MIPs) of left and right oblique images are obtained to demonstrate pulmonary vasculature. Our protocol allows for opacification of both pulmonary arterial and central arterial circulation in order to rule out both PE and aortic dissection. This "double rule-out" technique requires larger volume contrast than solely targeting pulmonary circulation, which can be performed with as little as 40 mL of IV contrast with concentration of 350 to 370 mgI/mL (milligrams of iodine/milliliter) and utilization of low kVp in patients with small body habitus.

3.2.3 Imaging Features

Diagnosis of pulmonary embolism on CTPA is made by identification of filling defects in the pulmonary arteries. The filling defect may cause a complete occlusion of the vessel with nonenhancing vessel, a partial central filling defect, or a peripheral filling defect (▶ Fig. 3.4).[26] A complete filling defect may also result in vessel enlargement compared to adjacent or contralateral, nonoccluded pulmonary arteries. In the case of a partial filling defect, the "polo mint" sign may be seen on transverse slices through a vessel with central hypoattenuation surrounded by hyperattenuating contrast material (▶ Fig. 3.5). Longitudinal slices through the vessel will demonstrate a "tram track" sign (▶ Fig. 3.6).

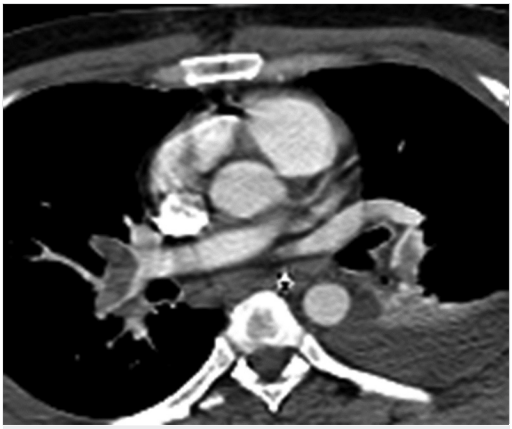

Fig. 3.4 Filling defect in the distal right main and left lower lobar pulmonary arteries.

Pulmonary infarcts may result from pulmonary arterial occlusion. These appear on CTPA as peripheral nonenhancing wedge-shaped opacities (▶ Fig. 3.7). Supporting features include internal lucency (air) within the wedge-shaped opacity, consistent with cavitation within the infarct, in

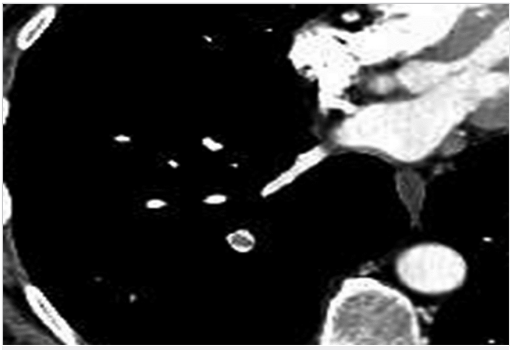

Fig. 3.5 "Polo mint" sign of a partial filling defect.

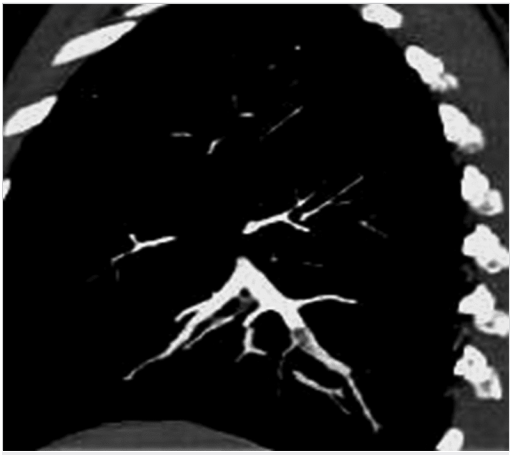

Fig. 3.6 "Tram track" appearance of a filling defect on longitudinal views of the pulmonary arteries.

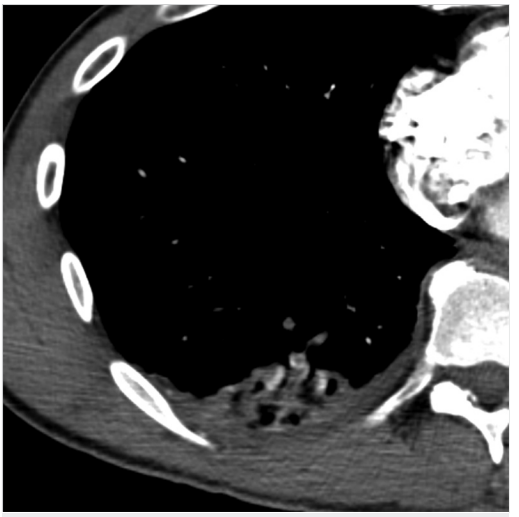

Fig. 3.7 Wedge-shaped opacity with central lucency suggestive of pulmonary infarct.

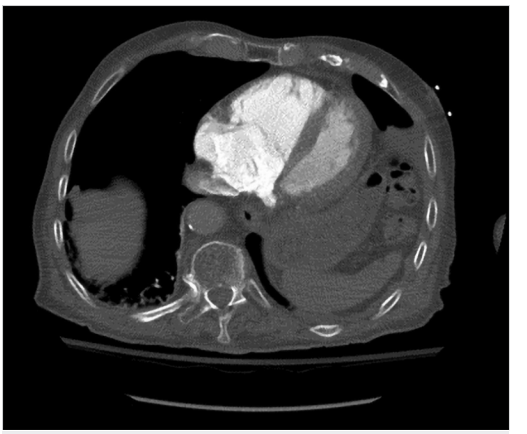

Fig. 3.8 Right ventricular strain pattern. The right ventricle is dilated with respect to the left and the interventricular septum is deviated to the left.

cases of old pulmonary infarcts. In the absence of a filling defect within the pulmonary arterial system, these findings are not specific for pulmonary embolism and may reflect other causes of pulmonary consolidation. In this case, additional evaluation with V/Q scanning or repeat CTPA may be indicated.

Pulmonary embolism can lead to right ventricular (RV) dysfunction and pulmonary arterial hypertension. Recognition of RV dysfunction is vital as it is a predictor of morbidity and mortality. A ratio of RV to left ventricular diameter in short axis greater than 1, deviation of the interventricular septum, increased azygos vein diameter, increased SVC diameter, and increased pulmonary artery diameter are all signs of RV dysfunction (▶ Fig. 3.8). Clot burden does not appear to be predictive of patients' mortality.[27,28]

3.2.4 Pitfalls

Misdiagnosis of pulmonary embolism or missed pulmonary embolism can result from a multitude of technical and interpretive errors. The most common technical error is respiratory motion during image acquisition, accounting for 42% of misdiagnosed PE[29]

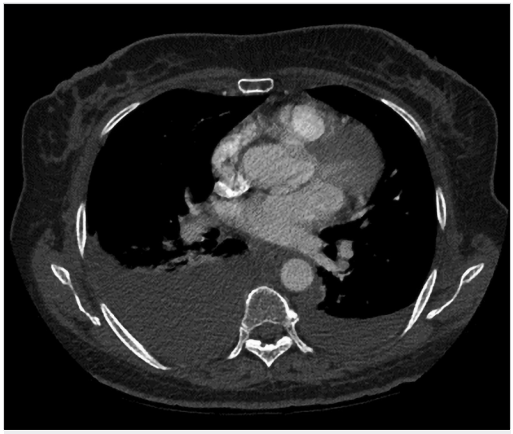

Fig. 3.9 Motion artifact in the left lower lobe causes an artifactual filling defect.

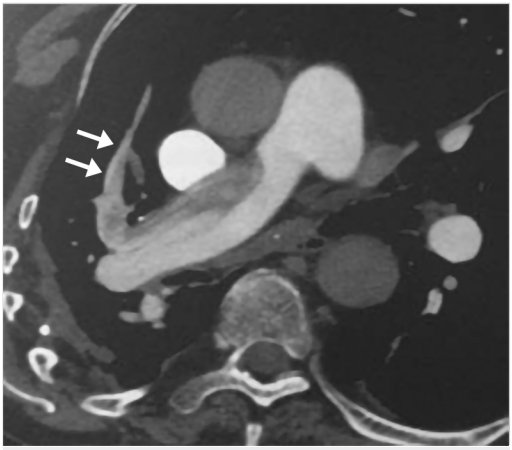

Fig. 3.10 Deep inspiration before contrast injection leads to differential opacification of pulmonary arteries. (arrows depict pseudofilling defect in the pulmonary artery, due to influx of unopacified blood).

(▶ Fig. 3.9). Cardiac motion can also result in erroneous filling defects. Streak artifact results from tubes, lines, or even the patient, if the patient's arms are at their side. Consequently, it is imperative that all extraneous metals be removed from the patient when possible and that the arms be placed above the patient's head. Deep inspiration prior to scanning results in decreased intrathoracic pressure, which draws unopacified blood into the right heart. This can result in mixing of contrast, which may appear as filling defect (▶ Fig. 3.10). This can be prevented by instructing the patient to take a shallow inspiration prior to scanning. Failure to trigger image acquisition at the appropriate time can result in reduced opacification of pulmonary arteries. This can lead to apparent filling defects, particularly in segmental and subsegmental vessels.

3.2.5 Special Considerations

Iodinated contrast agents pose their own potential risks, most importantly allergic reactions and contrast-induced nephropathy (CIN). In patients with a history of allergic reaction to iodinated contrast agents, it is imperative to determine the severity of their reaction. In the case of mild and moderate reactions, the patient can be premedicated with steroids prior to contrast administration. The American College of Radiology contrast manual provides specific guidelines on nonemergent and emergent premedication that include 50 mg of oral prednisone at 13, 7, and 1 hour prior to contrast administration or IV corticosteroid administration 4 to 5 hours prior to contrast administration[30] with a dose of 50-mg diphenhydramine 1 hour prior to contrast administration. Of note, administration of steroids < 2 hours prior to contrast administration has not been shown to be efficacious.

Postcontrast acute kidney injury (PC-AKI) and CIN: PC-AKI refers to any acute kidney injury that occurs in conjunction with IV iodinated contrast administration and includes CIN. CIN is a type of acute tubular necrosis (ATN) defined by an increase in serum creatinine by > 0.3 mg/dL or by 50%, or urine output ≤ 0.5 mg/kg/h for at least 6 hours within 48 hours following contrast administration. While the precise mechanism of CIN is not known, research indicates that it may be related to vasoconstriction and direct toxicity to the tubules. The predominant risk factor for CIN is underlying severe renal insufficiency. There are multiple strategies to reduce the risk of CIN. Volume expansion using 0.9% saline is the main strategy in high-risk patients and is the only strategy that is currently supported by literature. N-acetylcysteine (NAc) has not been shown to reduce the risk of CIN and is not routinely administered prior to contrast administration. Finally, use of iso-osmolar contrast agents such as iodixanol has not been shown to decrease CIN versus low osmolar contrast media (LOCM).[30]

Pregnancy is associated with an increase in the risk of venous thrombosis, which is related to venous stasis and a hypercoagulable state associated

with pregnancy. Clinical diagnosis of pulmonary embolism in pregnant patients is hindered by physiologic changes of pregnancy, including tachycardia, tachypnea, and lower extremity edema. Consequently, clinical decision tools are difficult to apply. D-dimer becomes elevated, which can lead to false-positive results. The American Thoracic Society has developed guidelines for imaging of pregnant patients based on symptoms and clinical suspicion.[31] CTPA is indicated if a patient has an abnormal chest radiograph or if a V/Q scan is nondiagnostic. Fetal doses of less than 50 mGy are thought to be inconsequential, although no safe fetal radiation dose has been established. Fetal radiation exposures in CTPA and V/Q scanning are similar. However, maternal exposure is greater in CTPA than in V/Q scan. Fetal radiation exposure during CTPA occurs via direct and scattered radiation. Dose reduction and shielding techniques should be implemented to reduce the effective dose to the mother and the fetus. A reduced-dose protocol includes decreasing the scanning range to include from the aortic arch to the diaphragm and reducing kVp and mA to 100.[32] Ingestion of oral barium attenuates the radiation dose to the fetus.[33] Lead shielding has also been shown to reduce fetal radiation dose.[34]

Radiation exposure is a consideration for every patient undergoing CTPA. The average dose of CTPA using a standard protocol is about 5 mSv. Maximum tube voltage can be reduced to 100 kVp without loss of diagnostic accuracy, which decreases the effective dose by 44%.[32]

CT venography (CTV) of the pelvis can be performed to diagnose DVT of the pelvic veins. However, diagnostic yield of adding either CTV or ultrasound for diagnosis of VTE is relatively low.[35] Therefore, CTV of the pelvis should be performed only in selected cases, as it carries significant radiation exposure. Patients who benefit the most from this study include those in the ICU, whose CTPA quality is more likely to be suboptimal. Patients who have undergone recent pelvic surgery may also benefit, as it provides direct visualization of the deep pelvic vessels. Finally, patients with lower extremity casts cannot undergo sonographic evaluation and therefore may require CTV when they present with the appropriate pretest probability.

3.3 Nuclear Medicine

Current utilization of V/Q scans is limited to patients with renal disease, contrast allergies, or morbidly obese patients,[19,36] as CTPA has been shown to be an accurate and robust technique in PE evaluation.

Although this section is dedicated to V/Q scanning, newer methods of utilizing radiopharmaceuticals have been developed and/or retooled for evaluating DVT. Radiolabeled peptides were developed in recent years, specifically for the detection of DVT with ^{99m}Tc–apcitide gaining the most notoriety demonstrating high sensitivity and specificity in acute DVT; however, it failed to prove PE in 83% of patients with known embolus.[37] Similarly, FDG positron emission tomography (PET)/CT has gained recent success for detection of DVT, although further evaluation of this technique is needed.[38]

3.3.1 Accuracy of Nuclear Medicine Studies for VTE Diagnosis

High-probability V/Q scan can detect the presence of PE with a sensitivity of 77.4% and a specificity of 97.7%.[19] Adding single-photon emission computed tomography (SPECT) increases test sensitivity by improving upon segmental defect detection by 13% and subsegmental defect detection by 80%.[39]

3.3.2 Indication

Similar to CTPA, the patient's pretest probability of PE is calculated by the clinical team using decision-making tools including Wells' criteria, Geneva criteria, and PERC score.[20,21,22] A review by Metter et al summarized nuclear medicine evaluation of PE, including indications for V/Q scanning.[40] If there is a high clinical and biochemical concerns for PE (using decision-making tools and D-dimer), a chest radiograph should be obtained first. If the chest radiograph is normal or near normal, a V/Q scan is indicated with CTPA as a reasonable alternative. In patients in whom CTPA is contraindicated due to renal insufficiency, contrast allergy or morbid obesity V/Q scan can be performed instead. In patients with significantly abnormal chest radiographs or those who are clinically unstable, CTPA is the preferred alternative.

3.3.3 Technique

Evaluation of PE with V/Q scan is a multistep process. A chest X-ray should be obtained prior to the V/Q scan to evaluate the anatomy and allow for comparison of findings per PIOPED II and Prospective Investigative Study of Acute Pulmonary Embolism

Diagnosis (PISAPED) recommendations. For the ventilation scan, the radiopharmaceutical most often used is ^{99m}Tc–diethylenetriamine pentaacetic acid (DTPA),[39] although ^{133}Xe is used in some academic centers because of its ability to assess all phases of ventilation and ability to provide physiological information, particularly in obstructive airway disease.[39,41] The aerosol is delivered through a nebulizer and mouthpiece with the nose occluded as the patient is breathing and multiple images are taken, in the supine position for ^{99m}Tc and upright for ^{133}Xe. A minimum of six planar images are captured with the gamma camera of both lungs from the posterior, anterior, left posterior oblique (LPO), right posterior oblique (RPO), left anterior oblique (LAO), and right anterior oblique (RAO) positions for comparison to perfusion imaging taken from the same positions.

The perfusion scan utilizes ^{99m}Tc-macroaggregated albumin (MAA). If ^{99m}Tc DTPA is used, the ventilation scan is performed first with a dose that is considerably lower than the ^{99m}Tc-MAA to ensure that an adequate perfusion phase scan is performed. ^{133}Xe ventilation scan is frequently performed first; however, at some institutions, the perfusion is performed first allowing for the scenario of a normal perfusion scan with a normal chest X-ray, which then could predicate the omission of the ventilation scan.[39] Disadvantages of the "perfusion-first" method are that if the perfusion scan is nondiagnostic the background activity would contribute to the subsequent ventilation scan, further complicating the study. For the perfusion scan, the patient is placed in the supine position during normal respiration. The ^{99m}Tc-MAA dose is injected intravenously and images are taken in six planes as discussed previously.

The addition of SPECT imaging has emerged over recent years to aid in the detection of pulmonary embolus. Limited to ^{99m}Tc DTPA, this allows the overlapping lung parenchyma to be better assessed in comparison to planar imaging, especially at the subsegmental levels using a gamma camera that captures multiple 2D images over multiple planes and processes them into a 3D rendering. To build upon the V/Q SPECT, low-dose CT has been introduced, allowing for mapping of the perfusion defect to CT imaging and increasing specificity further.

3.3.4 Imaging Features

Study results are reported in terms of pulmonary embolism probability. Ventilation and perfusion imaging are obtained with a preceding chest radiograph although through the PIOPED findings by Sostman et al, ventilation scans are superfluous in most patients when evaluating for PE with a normal chest radiograph.[41] The result of this is that both ventilation and perfusion scans are obtained with the ventilation portion utilized for equivocal cases where body habitus or chronic lung disease may provide information on the ventilation portion.

In a study with high probability of PE, or "PE present," two or more segments of perfusion scan–chest radiograph mismatch (two or more perfusion defects) can be identified (▶ Fig. 3.11). In a study with very low probability of PE, or "PE absent," there can be up to three small perfusion defects or one perfusion defect smaller than a matched radiographic lesion, as well as a single mid- or upper zone segment perfusion scan–chest radiograph mismatch (▶ Fig. 3.12). Multiple radiographic findings not overtly apparent on the accompanying perfusion scan can equal low probability as well including prominent hilum, cardiomegaly, elevated hemidiaphragm, linear atelectasis, and pleural effusions, in at least one-third of a pleural cavity.[41] In all other instances, the study is termed "nondiagnostic" or "intermediate probability."

3.3.5 Pitfalls

During perfusion imaging, blood clotting can occur in the syringe during ^{99m}Tc-MAA injection causing

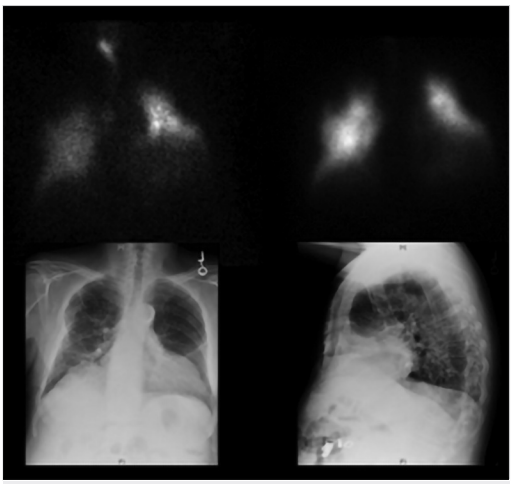

Fig. 3.11 An 86-year-old man with a history of chronic lung disease (CLD) matched small segmental defects in the lung apices corresponding to a large apical bullous seen on the chest radiograph = low likelihood ratio for recent pulmonary emboli.

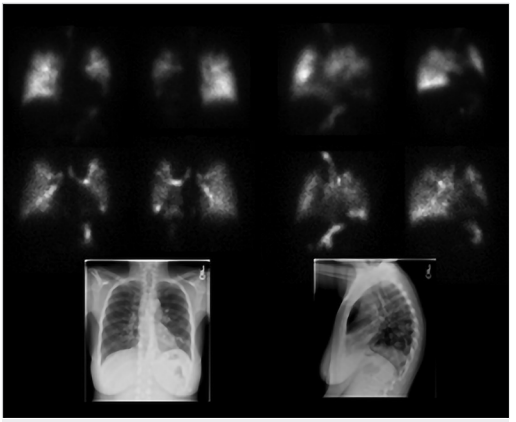

Fig. 3.12 A 54-year-old woman with sarcoidosis with normal chest X-ray and LLL-matched perfusion defects concerning with high probability pulmonary emboli (PE) in left lower lobe.

hotspots on perfusion imaging. Abnormal comparability can be seen between ventilation and perfusion scans secondary to positioning differences and changes in the respiration cycle, given the scans are obtained at two separate time points. Uptake in the thyroid and kidneys may be visualized in cases of right to left shunts.[39] Finally, because the high sensitivity and specificity of the modified PIOPED II criteria rely upon a lack of significant chest X-ray findings, lung pathology such as pneumonia, atelectasis, or even diffuse lung disease may obscure results of the V/Q scan and render a study nondiagnostic.

3.3.6 Special Considerations

Chronic cardiopulmonary concerns can prove problematic in the V/Q scan interpretation. Some of them can be mitigated. For example, in the case of patients with acute obstructive lung disease (chronic obstructive pulmonary disease [COPD] exacerbation), bronchodilator therapy may improve the accuracy of the V/Q scan via improvements in ventilation. Similarly, patients with acute heart failure exacerbation should be medically optimized prior to V/Q scanning to mitigate nondiagnostic imaging resulting from sequelae of heart failure (pulmonary edema, pleural effusions, etc.).[39]

V/Q SPECT shown to improve detection of pulmnary emboli over planar imaging.[43] in sensitivity, and, with the addition of CT, increased specificity in initial studies.[43] Despite this, no large prospective study has been performed to assess this methodology compared to planar imaging to establish a standard in the same manner as PIOPED II and PISAPED[44]; thus, the use of SPECT varies by institution.

3.4 Conventional Pulmonary Angiography

Conventional angiographic techniques were initially developed in the early 1960s using IV contrast as opposed to selective catheterization due to concern for dislodging emboli. Selective angiography was later shown to be safe and effective and able to detect peripheral emboli. The advent of digital subtraction angiography led to improved procedure time and reduced volume of contrast. For many years, angiography was the gold standard for the diagnosis of pulmonary embolism. With the advent of multidetector CTPA, which is noninvasive with high sensitivity and specificity, conventional angiography has fallen out of favor as a diagnostic technique. It is now reserved for interventions such as catheter-directed thrombectomy and thrombolysis.

3.4.1 Indication

Given the sensitivity and specificity of CTPA without the need for an invasive procedure, the indications for conventional pulmonary angiography are primarily to facilitate intervention. These indications are described in that dedicated section. Diagnostic pulmonary angiography is still used in preoperative planning in the case of chronic pulmonary embolism. It is also used during planning for intervention of pulmonary arteriovenous malformations and fistulas.

3.4.2 Technique

Pulmonary angiography is performed in a similar fashion to many minimally invasive central venous procedures. Briefly, central venous access is obtained through either the internal jugular or common femoral veins. A 6- to 7-Fr sheath can be used, depending on catheter selection. Pressures should be measured in the right atrium, right ventricle, main pulmonary artery, and pulmonary capillary wedge pressure. After accessing the pulmonary

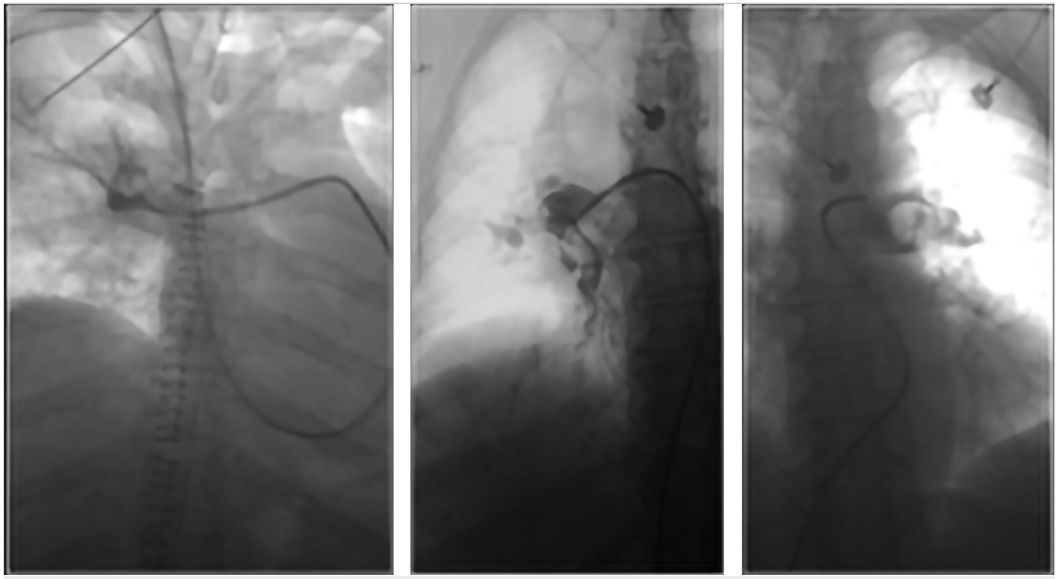

Fig. 3.13 Central main right pulmonary artery and segmental clot demonstrating pulmonary emboli.

artery, angiographic images may be obtained. Injection rates vary based on the vessel being interrogated. A rate of 25 to 30 mL/s for 2 seconds should be used for the main pulmonary artery, 15 to 20 mL/s for 2 seconds for the left and right pulmonary arteries, and 5 to 10 mL/s for 2 seconds for lobar and segmental branches. Extreme care should be taken in performing large-volume power injections in patients with acutely elevated pulmonary pressures, as the increased afterload of the contrast can precipitate acute cardiovascular collapse. Anteroposterior images of each lung and oblique images of the lower lobes should be obtained, with additional images taken at the radiologist's discretion. The frame rate for acquisition of images can also be increased, particularly in patients who are unable to suspend their respiration while imaging.

3.4.3 Imaging Features

As described by Dalen et al in 1971, angiographic abnormalities in pulmonary embolism include intraluminal filling defects, cutoffs of arteries, areas of oligemia, and asymmetric contrast flow[45] (▶ Fig. 3.13).

3.4.4 Pitfalls

Accuracy of pulmonary angiogram is shown to be high in proximal pulmonary artery branches but diminishes in higher-order pulmonary artery branches where branches overlap and can make visualization of filling defects difficult to assess.[46] As in CTPA, respiratory motion can also reduce the sensitivity of this technique.

3.4.5 Special Consideration

Given the rise of CTPA and V/Q scans for PE and ultrasound for DVT, angiography is rarely performed for diagnosis and generally reserved for the cases which intervention will be performed (i.e., thrombectomy or thrombolysis).

References

[1] Heng Tan C, Bedi D, Vikram R. Sonography of thrombosis of the deep veins of the extremities: clinical perspectives and imaging review. J Clin Ultrasound. 2012; 40(1):31–43
[2] Raghavendra BN, Rosen RJ, Lam S, Riles T, Horii SC. Deep venous thrombosis: detection by high-resolution real-time ultrasonography. Radiology. 1984; 152(3):789–793
[3] Talbot S. Use of real-time imaging in identifying deep venous obstruction: a preliminary report. Bruit. 1982; 6:41–42

[4] Beyer J, Schellong S. Deep vein thrombosis: current diagnostic strategy. Eur J Intern Med. 2005; 16(4):238–246

[5] Segal JB, Eng J, Tamariz LJ, Bass EB. Review of the evidence on diagnosis of deep venous thrombosis and pulmonary embolism. Ann Fam Med. 2007; 5(1):63–73

[6] Gottlieb RH, Widjaja J, Tian L, Rubens DJ, Voci SL. Calf sonography for detecting deep venous thrombosis in symptomatic patients: experience and review of the literature. J Clin Ultrasound. 1999; 27(8):415–420

[7] Cogo A, Lensing AW, Wells P, Prandoni P, Büller HR. Noninvasive objective tests for the diagnosis of clinically suspected deep-vein thrombosis. Haemostasis. 1995; 25(1–2):27–39

[8] Wells PS, Lensing AW, Davidson BL, Prins MH, Hirsh J. Accuracy of ultrasound for the diagnosis of deep venous thrombosis in asymptomatic patients after orthopedic surgery. A meta-analysis. Ann Intern Med. 1995; 122(1):47–53

[9] Kearon C, Julian JA, Newman TE, Ginsberg JS. Noninvasive diagnosis of deep venous thrombosis. McMaster Diagnostic Imaging Practice Guidelines Initiative. Ann Intern Med. 1998; 128(8):663–677

[10] Di Nisio M, Van Sluis GL, Bossuyt PMM, Büller HR, Porreca E, Rutjes AW. Accuracy of diagnostic tests for clinically suspected upper extremity deep vein thrombosis: a systematic review. J Thromb Haemost. 2010; 8(4):684–692

[11] Fowkes FJ, Price JF, Fowkes FG. Incidence of diagnosed deep vein thrombosis in the general population: systematic review. Eur J Vasc Endovasc Surg. 2003; 25(1):1–5

[12] White RH. The epidemiology of venous thromboembolism. Circulation. 2003; 107(23) Suppl 1:I4–I8

[13] Wells PS. Integrated strategies for the diagnosis of venous thromboembolism. J Thromb Haemost. 2007; 5 Suppl 1: 41–50

[14] Le Gal G, Carrier M, Rodger M. Clinical decision rules in venous thromboembolism. Best Pract Res Clin Haematol. 2012; 25(3):303–317

[15] Guideline developed in collaboration with the American College of Radiology, Society of Pediatric Radiology, Society of Radiologists in Ultrasound. AIUM practice guideline for the performance of peripheral venous ultrasound examinations. J Ultrasound Med. 2015; 34(8):1–9

[16] Hamper UM, DeJong MR, Scoutt LM. Ultrasound evaluation of the lower extremity veins. Radiol Clin North Am. 2007; 45 (3):525–547, ix

[17] Hanley M, Donahue J, Rybicki FJ, et al. ACR Appropriateness Criteria: Suspected Lower Extremity Deep Venous Thrombosis [online publication]. Reston, VA: American College of Radiology; 2013

[18] Remy-Jardin M, Remy J, Wattinne L, Giraud F. Central pulmonary thromboembolism: diagnosis with spiral volumetric CT with the single-breath-hold technique: comparison with pulmonary angiography. Radiology. 1992; 185(2):381–387

[19] Sostman HD, Stein PD, Gottschalk A, Matta F, Hull R, Goodman L. Acute pulmonary embolism: sensitivity and specificity of ventilation-perfusion scintigraphy in PIOPED II study. Radiology. 2008; 246(3):941–946

[20] Kline JA, Mitchell AM, Kabrhel C, Richman PB, Courtney DM. Clinical criteria to prevent unnecessary diagnostic testing in emergency department patients with suspected pulmonary embolism. J Thromb Haemost. 2004; 2(8):1247–1255

[21] Le Gal G, Righini M, Roy PM, et al. Prediction of pulmonary embolism in the emergency department: the revised Geneva score. Ann Intern Med. 2006; 144(3):165–171

[22] Wells PS, Anderson DR, Rodger M, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. Ann Intern Med. 2001; 135(2): 98–107

[23] Wells PS, Anderson DR, Rodger M, et al. Evaluation of D-dimer in the diagnosis of suspected deep-vein thrombosis. N Engl J Med. 2003; 349(13):1227–1235

[24] Bettmann MA, Baginski SG, White RD, et al. ACR Appropriateness Criteria® acute chest pain: suspected pulmonary embolism. J Thorac Imaging. 2012; 27(2):W28–W31

[25] Stein PD, Woodard PK, Weg JG, et al. PIOPED II Investigators. Diagnostic pathways in acute pulmonary embolism: recommendations of the PIOPED II Investigators. Radiology. 2007; 242(1):15–21

[26] Wittram C, Maher MM, Yoo AJ, Kalra MK, Shepard JA, McLoud TC. CT angiography of pulmonary embolism: diagnostic criteria and causes of misdiagnosis. Radiographics. 2004; 24(5): 1219–1238

[27] van der Meer RW, Pattynama PMT, van Strijen MJ, et al. Right ventricular dysfunction and pulmonary obstruction index at helical CT: prediction of clinical outcome during 3-month follow-up in patients with acute pulmonary embolism. Radiology. 2005; 235(3):798–803

[28] Ghaye B, Ghuysen A, Willems V, et al. Severe pulmonary embolism:pulmonary artery clot load scores and cardiovascular parameters as predictors of mortality. Radiology. 2006; 239 (3):884–891

[29] Hutchinson BD, Navin P, Marom EM, Truong MT, Bruzzi JF. Overdiagnosis of pulmonary embolism by pulmonary CT angiography. AJR Am J Roentgenol. 2015; 205(2):271–277

[30] ACR Committee on Drugs and Contrast Media. ACR Manual on Contrast Media. Version 10.3. Reston, VA: American College of Radiology; 2017

[31] Leung AN, Bull TM, Jaeschke R, et al. ATS/STR Committee on Pulmonary Embolism in Pregnancy. An official American Thoracic Society/Society of Thoracic Radiology clinical practice guideline: evaluation of suspected pulmonary embolism in pregnancy. Am J Respir Crit Care Med. 2011; 184(10): 1200–1208

[32] Heyer CM, Mohr PS, Lemburg SP, Peters SA, Nicolas V. Image quality and radiation exposure at pulmonary CT angiography with 100- or 120-kVp protocol: prospective randomized study. Radiology. 2007; 245(2):577–583

[33] Yousefzadeh DK, Ward MB, Reft C. Internal barium shielding to minimize fetal irradiation in spiral chest CT: a phantom simulation experiment. Radiology. 2006; 239(3):751–758

[34] Chatterson LC, Leswick DA, Fladeland DA, Hunt MM, Webster ST. Lead versus bismuth-antimony shield for fetal dose reduction at different gestational ages at CT pulmonary angiography. Radiology. 2011; 260(2):560–567

[35] Goodman LR, Sostman HD, Stein PD, Woodard PK. CT venography: a necessary adjunct to CT pulmonary angiography or a waste of time, money, and radiation? Radiology. 2009; 250 (2):327–330

[36] Yazdani M, Lau CT, Lempel JK, et al. Historical evolution of imaging techniques for the evaluation of pulmonary embolism. Radiographics. 2015; 35(4):1245–1262

[37] Dunzinger A, Hafner F, Schaffler G, Piswanger-Soelkner JC, Brodmann M, Lipp RW. 99mTc-apcitide scintigraphy in patients with clinically suspected deep venous thrombosis and

pulmonary embolism. Eur J Nucl Med Mol Imaging. 2008; 35 (11):2082–2087

[38] Hess S, Madsen PH, Iversen ED, Frifelt JJ, Høilund-Carlsen PF, Alavi A. Efficacy of FDG PET/CT imaging for venous thromboembolic disorders: preliminary results from a prospective, observational pilot study. Clin Nucl Med. 2015; 40(1):e23–e26

[39] Parker JA, Coleman RE, Grady E, et al. Society of Nuclear Medicine. SNM practice guideline for lung scintigraphy 4.0. J Nucl Med Technol. 2012; 40(1):57–65

[40] Metter D, Tulchinsky M, Freeman LM. Current status of ventilation-perfusion scintigraphy for suspected pulmonary embolism. AJR Am J Roentgenol. 2017; 208(3):489–494

[41] Mettler FA, Guiberteau MJ. Respiratory system. In: Essentials of Nuclear Medicine Imaging. 6th ed. Philadelphia, PA: Elsevier/Saunders; 2012:197–219

[42] Stein PD, Sostman HD, Matta F. Critical review of SPECT imaging in pulmonary embolism. Clin Transl Imaging. 2014; 2: 379–390

[43] Reinartz P, Wildberger JE, Schaefer W, Nowak B, Mahnken AH, Buell U. Tomographic imaging in the diagnosis of pulmonary embolism: a comparison between V/Q lung scintigraphy in SPECT technique and multislice spiral CT. J Nucl Med. 2004; 45 (9):1501–1508

[44] Sostman HD, Miniati M, Gottschalk A, Matta F, Stein PD, Pistolesi M. Sensitivity and specificity of perfusion scintigraphy combined with chest radiography for acute pulmonary embolism in PIOPED II. J Nucl Med. 2008; 49(11): 1741–1748

[45] Dalen JE, Brooks HL, Johnson LW, Meister SG, Szucs MM, Jr, Dexter L. Pulmonary angiography in acute pulmonary embolism: indications, techniques, and results in 367 patients. Am Heart J. 1971; 81(2):175–185

[46] Stein PD, Athanasoulis C, Alavi A, et al. Complications and validity of pulmonary angiography in acute pulmonary embolism. Circulation. 1992; 85(2):462–468