Perfusion Scintigraphy in Diagnosis and Management of Thromboembolic Pulmonary Hypertension

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Abbreviations: CTEPH = chronic thromboembolic pulmonary hypertension, MAA = macroaggregates of human serum albumin, PAH = pulmonary arterial hypertension, PE = pulmonary embolism, V/Q = ventilation-perfusion

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

■ Describe the role of lung scintigraphy in the examination of patients with pulmonary hypertension.

■ Identify perfusion abnormalities in CTEPH and the expected changes after pulmonary thromboendarterectomy and pulmonary balloon angioplasty.

■ Recognize the various scintigraphic findings of PAH, interstitial lung disease, and/or right-to-left shunts.

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Chronic thromboembolic pulmonary hypertension (CTEPH) is a life-threatening complication of acute pulmonary embolism (PE). Because the treatment of CTEPH is markedly different from that of other types of pulmonary hypertension, lung ventilation-perfusion (V/Q) scintigraphy is recommended for the workup of patients with unexplained pulmonary hypertension. Lung V/Q scintigraphy is superior to CT pulmonary angiography for detecting CTEPH. Perfusion defect findings of CTEPH can be different from those of acute PE. Familiarity with the patterns of perfusion defects seen during the initial workup of CTEPH and the expected posttreatment changes seen at follow-up imaging is essential for accurate interpretation of V/Q scintigraphy findings.

Introduction

Chronic thromboembolic pulmonary hypertension (CTEPH) is a complication of acute pulmonary embolism (PE). Chronic thromboembolism is a relatively common cause of pulmonary hypertension (1). Owing to mechanisms that are not fully understood, the thrombotic material residing in the pulmonary arteries after acute PE may organize into chronic scars that can occlude the pulmonary arteries and increase pulmonary vascular resistance (2,3). It is important to distinguish CTEPH from pulmonary hypertension due to other causes (Table) (4) because there are distinctive treatments for CTEPH, which include potentially curative surgical resection (5).
Although the frequency of CTEPH after acute PE is somewhat controversial, it occurs in 0.4%–3.8% of patients in most series (6–15). There are no effective screening procedures to detect CTEPH routinely after PE (16), so most cases are diagnosed during the investigation of symptoms related to pulmonary hypertension. In fact, only about half of patients who are diagnosed with CTEPH have had clinically recognized PE (17,18). A delayed diagnosis of CTEPH could result in a progressive increase in pulmonary vascular resistance (due to upstream extension of the intravascular scars and downstream arteriopathy of small muscular arteries and arterioles) and eventually right-sided heart failure (19). These conditions increase the morbidity and potential complications associated with CTEPH.

**Diagnosing CTEPH**

In patients with pulmonary hypertension, CTEPH is diagnosed on the basis of lung scintigraphy findings and/or specific evidence of arterial defects at CT, MRI, or conventional pulmonary angiography (16). A subset of patients with symptomatic disease, referred to as chronic thromboembolic disease, may have normal pulmonary hemodynamic parameters at rest. In these patients, poorly perfused alveoli constitute physiologic dead space that results in dyspnea, hypoxia, and/or poor exercise capability (20). Imaging can help to confirm the clinical suspicion and guide appropriate clinical management.

An algorithm for diagnosing CTEPH is shown in Figure 1 (16,21). V/Q lung scintigraphy is recommended for the workup of all patients with unexplained pulmonary hypertension (16) and is the procedure of choice for evaluation of CTEPH (22). Lung scintigraphy is superior to CT pulmonary angiography for the diagnosis of CTEPH because of its higher sensitivity (23). However, advances in CT technology have helped to improve its sensitivity (24).

In patients referred specifically for evaluation of CTEPH, the interpretation of V/Q scan findings is usually straightforward, with reported sensitivities of 90%–100% and specificities reaching 94%–100% (16). Most patients with CTEPH have very abnormal V/Q scan findings and often have multiple moderate and large perfusion defects, without matching ventilation defects, in their lungs (23,25). A large defect generally involves more than 75% of the expected volume of a bronchopulmonary segment;
Figure 2. Temporal evolution of perfusion abnormalities in a 35-year-old man who developed CTEPH. He initially presented with acute-onset chest pain and shortness of breath. (a) Axial (top) and coronal (bottom) contrast material–enhanced CT images obtained at the time of the acute presentation show filling defects (arrows) in the bilateral lobar and segmental pulmonary arteries, consistent with acute PE. (b) Technetium 99m (\(^{99m}\text{Tc}\)) macroaggregated human albumin (MAA) perfusion scans obtained 3 days later show multiple peripheral wedge-shaped defects throughout the lungs (anterior segment of left upper lobe and anteromedial basal segment of left lower lobe, left posterior-oblique view), consistent with moderate and large segmental defects. \(^{99m}\text{Tc}\)-MAA scans obtained 3.5 months and 1 year later show persistent but smaller and less conspicuous defects, which are the most noticeable in the anteromedial basal segment on the left posterior-oblique view. In Figure 2 and subsequent figures, \(\text{ANT} = \) anterior, \(\text{EQUIL} = \) equilibrium phase, \(\text{LAO} = \) left anterior-oblique, \(\text{LLAT} = \) left lateral, \(\text{LPO} = \) left posterior-oblique, \(\text{POST} = \) posterior, \(\text{Q} = \) perfusion, \(\text{RAO} = \) right anterior-oblique, \(\text{RLAT} = \) right lateral, \(\text{RPO} = \) right posterior-oblique.

Figure 3. Temporal evolution of perfusion abnormalities in a 74-year-old woman with PE, who initially presented with shortness of breath. \(^{99m}\text{Tc}\)-MAA perfusion scans obtained the day of initial presentation (top row) and 2 months, 3.5 months, and 4 years later show the resolution or near resolution of several defects (eg, in anterior segment of left upper lobe and superior segment of left lower lobe) (white arrows). Other defects (eg, in inferior lingula) (red arrows) persisted.

A moderate defect, 25%–75% of the expected segment volume; and a small defect, less than 25% of the expected segment volume (26).

Compared with acute PEIs, perfusion defects evolve over time and generally decrease in size (Figs 2, 3). Consequently, to improve sensitivity, albeit at the cost of reduced specificity, a scan with findings indicating an intermediate probability of CTEPH could be considered a CTEPH-positive image (25). A perfusion defect involving more than 50% of the bronchopulmonary segment volume without a matched ventilation defect could be sufficient for the diagnosis of pulmonary thromboembolism (27–29). The extent of perfusion defects does not correlate well with the severity of CTEPH (30), and the conspicuity of perfusion defects can decrease over time despite the progression of hemodynamic abnormalities (31).
Normal scintigraphic findings exclude the presence of surgically accessible chronic thromboembolic disease (Fig 4) (25). Small subsegmental or nonsegmental defects can be seen in idiopathic PAH (23,32,33). The presence of a perfusion defect involving less than 50% of a bronchopulmonary segment should not be considered evidence of thromboembolic disease (28).

Patients with pulmonary hypertension and V/Q scan findings suggestive of CTEPH should be referred to a specialist center for further evaluation, such as right-sided heart catheterization for assessment of pulmonary artery pressure, capillary wedge pressure, and vascular resistance (21). Other causes of pulmonary hypertension should be considered if the pulmonary capillary wedge pressure is elevated (>15 mmHg) or the pulmonary vascular resistance is low (≤2 Wood units). Hemodynamic parameters are important for prognostication and risk assessment before pulmonary endarterectomy is performed.

Catheter-based selective pulmonary angiography is the reference-standard modality for diagnosing CTEPH (34). In some instances, high-quality CT or MR angiography has been used to confirm the diagnosis (35), although false-negative findings have been reported in up to 25% of cases (24). CT pulmonary angiography can complement lung scintigraphy and catheter-based pulmonary angiography in the assessment for operability and provide additional information about the lung parenchyma, mediastinal structures, and bronchial arteries.

Lung Scintigraphy Technique

Ideally, to help evaluate any coexisting underlying heart or lung disease, a chest radiograph should be obtained before lung scintigraphy is performed. In a patient with normal chest radiograph findings, perfusion-only imaging might be sufficient to diagnose or rule out CTEPH (36). However, even in cases with normal chest radiograph findings, ventilation scanning can help define the lung borders and facilitate the recognition of peripheral perfusion defects (37). $^{133}$Xe ventilation imaging yields physiologic information about ventilation and is more sensitive for the detection of obstructive airway disease than aerosol ventilation imaging. Ventilation defects are best evaluated during the wash-in phase because the collateral ventilation of radioactive gas through the canals of Lambert and pores of Kohn may reduce the conspicuity of defects during the equilibrium phase. Washout-phase images can show areas of air trapping in the lungs.

The choice of radiotracer for ventilation scanning is controversial. In our practice, $^{133}$Xe ventilation imaging is performed before perfusion imaging in patients who are referred for evaluation of pulmonary hypertension. The main advantage of $^{133}$Xe ventilation imaging in the evaluation of CTEPH is that it does not interfere with subsequently performed $^{99m}$Tc-MAA perfusion imaging or cause artifacts due to central airway deposition. The absence of artifacts allows one to accurately evaluate the extent and severity of perfusion defects.
Figure 5. CTEPH in a 59-year-old man. (a) V/Q scans obtained at an outside institution, with $^{99m}$Tc-pentetic acid used to perform ventilation (VENT) imaging before the perfusion (PERF) portion of the study, show multiple perfusion defects (bottom row), including a large confluent mismatched defect involving the basal segments of the right lower lobe (rectangles). There is some activity along the diaphragm, mimicking the “stripe” sign (activity at periphery of perfusion defect), in the right posterior-oblique view. (b) The severity of the perfusion defects (ie, absent perfusion in the right lung base) (rectangles) is better appreciated on the repeat V/Q scans obtained with $^{99m}$Tc-MAA used for perfusion imaging, which was performed immediately after $^{133}$Xe ventilation scanning (images not shown).

If $^{99m}$Tc-labeled aerosolized radiotracers are used for ventilation scanning, residual radiotracer activity (ie, “shine-through”) could mask or obscure perfusion defects and result in an underestimation of the degree of perfusion abnormality (Fig 5). To minimize shine-through activity, the perfusion images should have, at least, three times higher prescribed activity (counts per second) than the initially obtained ventilation scan.

False-positive mismatch defects may occur more frequently with $^{133}$Xe scanning than with aerosolized $^{99m}$Tc scanning owing to image quality degradation and higher soft-tissue attenuation (37). The anatomic correlation between ventilation defects and perfusion defects is more accurately assessed with $^{99m}$Tc-labeled aerosolized radiotracers, the use of which enables imaging in planes similar to those in which perfusion imaging is performed. In contrast, $^{133}$Xe ventilation imaging is limited to one to two views. In Europe, where the availability of $^{133}$Xe is limited, with use of krypton 81m ($^{81m}$Kr), the advantages of using a gas are combined with high image quality and capability for imaging in multiple projections or with SPECT, without interfering with perfusion imaging.

Ideally, perfusion imaging should be performed with use of freshly prepared $^{99m}$Tc-MAA. The number of particles administered may be reduced to 100,000–200,000, and the administered dose can be reduced to 40 MBq (1 mCi) in young adults or pregnant patients without significantly degrading image quality (26,37). Similarly, reducing the number of particles should be considered in patients with severe pulmonary hypertension and patients with significant right-to-left shunts (26).

Using SPECT or SPECT/CT, as compared with planar imaging, increases sensitivity and specificity (Fig 6) (38,39). Investigators in a meta-analysis (40) found SPECT to be comparable to CT pulmonary angiography in the diagnosis of acute PE, but with a lower radiation dose (~2 mSv per correct diagnosis vs 5 mSv with CT pulmonary angiography). In the setting of CTEPH, SPECT may help improve the diagnostic accuracy of perfusion imaging, and when combined with CT pulmonary angiography, it might aid in the assessment for operability (41). However, in the setting of SPECT, as compared with planar imaging, the probability of a false-positive study in patients with group 1 pulmonary hypertension (ie, PAH) can be high (33). However, this has not been our experience (38,39).
Figure 6. Imaging findings in a 72-year-old woman with a history of PE. (a) $^{99m}$Tc-MAA perfusion (PERF) images show multiple segmental defects in the lungs. (b–d) Axial low-milliamperage CT (b), SPECT (c), and fused SPECT/CT (d) images show a defect (circle in c and d) in the medial-basal segment of the lower lung lobes that was not well evaluated on planar images.

Posttreatment Changes

Pulmonary Endarterectomy

Pulmonary endarterectomy is the definitive treatment for CTEPH. This procedure is associated with improved hemodynamic parameters and good outcomes with respect to functional status, quality of life, and survival (42,43).

Reperfusion edema may occur in lung regions of newly performed endarterectomy (44). Although severe reperfusion edema can cause respiratory failure, milder cases usually resolve over time (45). Hypoventilation, subsegmental atelectasis, and pleural effusions commonly occur during the perioperative period and affect perfusion images (Fig 7). Hyperperfusion may be seen at lung scintigraphy, even after resolution of the edema seen at radiography. However, over time, the pulmonary vasculature adapts to the normalized pressures and the hyperemia resolves (Fig 8).

On postoperative perfusion scans, lungs that were previously normally perfused commonly appear to be photopenic relative to the reperfused segments (46). In CTEPH, increased blood flow and wall shear stress over time cause adaptive changes and increased pulmonary vasculature resistance in segments that were not affected by PE. Decreased pulmonary artery pressure and
Figure 7. $^{133}$Xe ventilation (top row) and $^{99m}$Tc-MAA perfusion (middle and bottom rows) scintigraphic findings in a 26-year-old man 10 days after bilateral pulmonary thromboendarterectomy. Ventilation scan obtained during the wash-in phase best depicts the relative hypoventilation in the left lung. Air trapping in the lung bases is seen during the washout phases. A hot spot in the lingula (arrow, middle row) seen on the perfusion images is consistent with a $^{99m}$Tc-MAA clump.

Figure 8. $^{99m}$Tc-MAA pulmonary perfusion scan findings in a 69-year-old man after bilateral pulmonary thromboendarterectomy for CTEPH. (a) Perfusion scans obtained initially, 1.5 months before the surgery, show multiple segmental defects (eg, in lateral-basal right lung lobe) (white arrows), with sparing of the superior right lung lobe (red arrows in a–c). (b) Perfusion scans obtained 6 days after the surgery show improved perfusion and hyperemia in regions of the previous perfusion defects (white arrows). The superior segment appears to be photopenic compared with the rest of the lung. (c) On the perfusion scans obtained 6 months after the surgery, the pulmonary perfusion (white arrows) appears to be more homogeneous, with persistent marked improvement compared with the perfusion seen on the initially obtained scans (a).
preferential blood flow due to low resistance in lung regions where endarterectomy was performed cause a redistribution of blood flow away from the rest of the pulmonary artery tree—that is, a vascular steal (Fig 9) (47). This can lead to the appearance of a high-probability–type V/Q scan pattern after surgery.

These pseudodefects are an expected finding. In the absence of clinical or radiographic evidence of an actual acute PE, further workup is not warranted. The extent and severity of these pseudodefects decrease within 9–12 months in most patients (48).

In the postoperative period, acute PE can occur but is rare because patients typically receive anticoagulant medication. However, the complete absence of perfusion in previously perfused lobar or segmental regions should raise suspicion for acute PE (Fig 10). At scintigraphy, this can have an appearance similar to that of a pseudodefect, and semiquantitative SPECT/CT may be useful in this scenario.

Pulmonary Balloon Angioplasty
Pulmonary endarterectomy is a complex surgical procedure and can be associated with significant mortality in patients of advanced age who have other comorbidities (18). Pulmonary balloon angioplasty (PBA) is an alternative interventional procedure for patients who are not surgical candidates, and, like pulmonary endarterectomy, can facilitate improved outcomes compared with those associated with medication therapy (49). A balloon catheter is used to dilate stenotic pulmonary arteries and improve perfusion to lung segments downstream from partially occluded arteries. PBA cannot be used to remove the intravascular scars characteristic of CTEPH and might not be feasible when segmental arteries are completely occluded at the orifice (50). Angioplasty forces the intravascular scars to the side and subjects the vessel lumen to arterial pressures that may further expand it over time.

The role of lung scintigraphy in evaluating response to PBA is not established (51).
Figure 10. PE after pulmonary endarterectomy in a 38-year-old woman with a history of CTEPH. (a–c) $^{99m}$Tc-MAA perfusion scans were obtained before (a) and 12 days (b) and 1.5 months (c) after the surgery. (d, e) Axial (d) and coronal (e) contrast-enhanced reconstructed CT images obtained at the time of the second V/Q scanning examination (b) show a massive new embolus (arrow) involving the right upper lobe pulmonary artery.

Figure 11. $^{99m}$Tc-MAA perfusion scintigraphy findings in a 41-year-old woman with CTEPH before (top row) and after (bottom row) two sessions of pulmonary balloon angioplasty of the right and left lower lobe segments. Improved perfusion (arrows) is most conspicuous in the posterior-basal segment of the right lower lobe.

Perfusion changes seen on planar images are often subtle after PBA (Fig 11). Knowledge of the vessels that have been surgically managed can help in identifying small improvements in perfusion defects. When multiple interventional sessions are needed, a preprocedural baseline perfusion scan should be obtained close to the time of the intervention to help evaluate subsequent improvement. Pretreatment imaging also helps to identify target vascular territories and assess the severity.
Figure 12. 99mTc-MAA perfusion scintigraphy abnormalities in pulmonary hypertension. (a) Low-probability scintigraphic scans were obtained in a patient with left-sided congestive heart failure and incidental right diaphragmatic eventration. (b) Scintigraphic scans obtained in two patients (top and bottom rows) demonstrate variability in the severity and extent of CTEPH-related perfusion defects. (c) Scintigraphic scans obtained in a different patient, who has mediastinal fibrosis, shows multiple large perfusion defects.

Lung Scintigraphy Findings of Pulmonary Hypertension of Other Causes

Familiarity with the spectrum of perfusion abnormalities that can be seen at initial workup is essential for correct and accurate interpretation of lung scintigraphy findings (Fig 12). Patients with idiopathic PAH can have normal perfusion scans or scans that show nonsegmental patchy defects with a “moth-eaten” appearance (Fig 13), which corresponds to pruning of the pulmonary vessels. Reverse mismatch defects (ie, larger or more severe defects seen at ventilation imaging, as compared with perfusion imaging) also have been reported (53). Patients with global perfusion abnormalities appear to have a worse prognosis than do patients with normal scans or only focal defects (33).

Large mismatched segmental defects may be observed, albeit infrequently, in pulmonary hypertension of nonthromboembolic causes. For example, in idiopathic pulmonary fibrosis, areas of honeycombing and traction bronchiectasis may have normal ventilation but absent perfusion, consistent with large mismatched defects (54) (Fig 14). In these patients, correlation of scintigraphic findings with radiographic or CT findings is essential to exclude superimposed PE in preserved lung parenchyma. Nonthrombotic lesions involving pulmonary arteries (ie, angiosarcoma, invasion by mediastinal or lung malignancy, compression by adjacent mass, vasculitis, or fibrosing mediastinitis) also can cause large mismatched perfusion defects.
defects (Fig 15). In the setting of a unilateral large perfusion defect involving nearly the entire lung, additional imaging may be warranted to exclude these causes (Fig 16).

Venous obstruction can result in regional perfusion abnormalities in the lungs. Large mismatched perfusion defects with normal pulmonary angiogram findings can be seen in pulmonary veno-occlusive disease (PVOD) (55). However, the majority of patients with PVOD have normal perfusion or small mismatched defects (56). Lung scintigraphy can be used to compare activity in the lung quadrants drained by individual pulmonary veins to diagnose pulmonary vein stenosis (Fig 17) (57).

Pulmonary capillary hemangiomatosis is an uncommon cause of pulmonary hypertension. Lung perfusion scintigraphy may depict small subsegmental and nonsegmental defects similar to idiopathic pulmonary hypertension (58). Increased (augmented) basal activity has been reported (Fig 18) (59,60). As in cases of PVOD,
in the setting of pulmonary capillary hemangiomatosis, lung scintigraphy is not helpful for the diagnosis but it may guide subsequent treatment involving lung transplantation (61).

Pulmonary hypertension due to lung disease (group 3) (Fig 19) is diagnosed on the basis of a pattern of findings such as clinical signs and symptoms, radiographic abnormalities, pulmonary function test defects, and right-sided heart catheterization. Mild pulmonary hypertension is common in severe chronic obstructive pulmonary disease. If pulmonary hypertension is severe but in the absence of concomitant left-sided heart disease, CTEPH may be considered (16). In some patients, aerosol or $^{81m}$Kr ventilation imaging in multiple projections or SPECT might be necessary for accurate assessment of the degree of ventilation and perfusion mismatch.

**Right-to-Left Shunt**

Extrapulmonary activity at $^{99m}$Tc-MAA perfusion scanning should raise suspicion for a right-to-left shunt (Fig 20). However, activity in the thyroid gland, gastric mucosa, or urinary collecting system can be seen owing to the inadvertent presence of free $^{99m}$Tc-pertechnetate at scintigraphy, particularly if imaging is delayed (62). Gallbladder activity and biliary excretion of radiotracer into the bowel can also be seen in the absence of a right-to-left shunt (63). In patients with superior vena cava obstruction (Fig 20b), retrograde flow through the chest wall, mediastinal, and paravertebral collateral veins could result in focal activity along the venous collaterals, hepatic parenchyma, and even vertebral body bone marrow (64). Evaluation of brain activity is the most accurate method for detection of right-to-left shunts (26).

Imaging of the head or whole body should be considered if extrapulmonary activity is identified on lung images. At whole-body scintigraphy, an extrapulmonary uptake ratio of greater than 0.1 (ie, $>10\%$) is consistent with an abnormal shunt (65) and should alert the clinician to a possible intracardiac shunt.

Congenital intracardiac shunts can complicate the management of or cause pulmonary hypertension. The development of pulmonary hypertension can result in reversal of the direction of flow through the shunt and hypoxemia. In patients with CTEPH, congenital intracardiac shunts can result in significant hypoxemia at rest.

In CTEPH, a right-to-left shunt can lead to an increased risk of paradoxical embolism and neurologic complications. In patients who undergo surgery for CTEPH, cardiac defects could be repaired simultaneously. Even if the cardiac defect remains, decreased pulmonary arterial pressures should result in a reduced magnitude of the right-to-left shunt. There is a theoretical possibility that in patients with intrapulmonary vascular...
malformations (such as hereditary hemorrhagic telangiectasia [66] and hepatopulmonary syndrome), increased flow through pulmonary arteries may result in an increased right-to-left shunt after surgery.

**Conclusion**

Lung scintigraphy is recommended for the workup of patients with unexplained pulmonary hypertension. $^{133}$Xe ventilation scanning can be performed in conjunction with $^{99m}$Tc-MAA perfusion scintigraphy without affecting the quality of the perfusion images. The presence of a perfusion defect (or defects) that involves greater than 50% of the bronchopulmonary segment volume could support the diagnosis of CTEPH, and further workup for CTEPH or referral to a specialist center is recommended. Normal perfusion scan results rule out CTEPH.

Atypical distribution such as unilateral involvement may be seen with other causes of CTEPH such as fibrosing mediastinitis and pulmonary vein stenosis. Areas of hyperperfusion and relative perfusion defects are commonly seen after pulmonary thromboendarterectomy and gradually decrease or resolve over time.
Figure 17. Left pulmonary vein stenosis after stent placement in the inferior vein, and asymmetrically decreased perfusion to the left lung in a 60-year-old man. ⁹⁹ᵐTc-MAA perfusion scans (left, middle and bottom rows) show diffusely decreased activity in the left lung compared with perfusion activity in the right lung. ¹³³Xe ventilation scans are shown in the top left row. Posterior three-dimensional volume-rendered CT image (top right) shows the stent. Axial CT image (bottom right) shows narrowing of the left superior pulmonary vein (red arrow).

Figure 18. Capillary hemangiomatosis in a 48-year-old woman after right lung transplantation. Left: The native left lung has markedly decreased radioactive activity at ¹³³Xe ventilation scanning (top row) and nearly absent perfusion (PERF). These findings correspond to the confluent alveolar opacities seen on the corresponding frontal radiograph (top right) and axial CT image (bottom right). ⁹⁹ᵐTc-MAA perfusion scans (left, middle and bottom rows) show heterogeneous activity, with increased perfusion in the lung base (arrows). Right: Interlobular septal thickening and ground-glass opacities in the right lung indicate recurrent capillary hemangiomatosis after transplantation. 1MIN and 2MIN = 1 and 2 minutes, respectively, after radionuclide injection; WOUT = washout.

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References
Figure 19. Cystic fibrosis in a 25-year-old woman. Left: $^{133}$Xe ventilation (top row) and $^{99m}$Tc-MAA perfusion (middle and bottom rows) scans show matched defects in the posterior view. Right: Axial CT images obtained at the level of the aortic arch (top) and below the carina (bottom) show bronchial wall thickening, bronchiectasis, centrilobular opacities, and cystic change in bilateral lungs.


Figure 20. Extrapulmonary activity at V/Q scanning, with $^{99m}$Tc–pentetic acid ventilation (V) imaging performed first. (a) Ventilation and perfusion scans obtained in a 61-year-old patient show left ventricular myocardial activity (red arrows) due to an intra-atrial right-to-left shunt and deposition of $^{99m}$Tc-MAA particles in the myocardial capillaries via the coronary arteries. Hot spots represent deposition of aerosolized radiotracer in the trachea (white arrows) and focal $^{99m}$Tc-MAA activity at the tip of a central venous catheter in the superior vena cava (black arrows). Subsequent echocardiography performed with an agitated saline–contrast material bubble examination had early positive findings, confirming an intra-atrial shunt. (b, c) $^{99m}$Tc-MAA perfusion images (b) were obtained in a 42-year-old woman with a history of deep vein thrombosis, superior vena cava syndrome, and nontherapeutic anticoagulation, who was referred for evaluation of shortness of breath. $^{99m}$Tc-MAA was injected into an upper extremity vein before perfusion images were acquired. Axial contrast-enhanced CT images (c) were obtained at the level of the main pulmonary artery (top) and liver (bottom), with contrast material injected into a right arm vein. Intense radioactivity in the liver, particularly in the quadrate lobe (arrows in b), corresponds to the large collateral veins along the liver capsule (arrows in c, bottom image) at axial contrast-enhanced CT, which also revealed retrograde opacification of the chest wall and azygos veins (arrows in c, top image).