Current Problems in Diagnostic Radiology 48 (2019) 387–392

Current Problems in Diagnostic Radiology

journal homepage: www.cpdrjournal.com

Original Articles

Beyond Pulmonary Embolism; Nonthrombotic Pulmonary Embolism as Diagnostic Challenges

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A B S T R A C T

Nonthrombotic pulmonary embolism (NTPE) is less well understood and is encountered less frequently than pulmonary embolism from venous thrombosis. NTPE results from embolization of nonthrombotic material to the pulmonary vasculature originating from many different cell types as well as nonbiologic or foreign materials. For many radiologists NTPE is a challenging diagnosis, presenting nonspecific or unusual imaging findings in the setting of few or unusual clinical signs. The aim of this paper is to review the pathophysiology of diverse causes of NTPE, which should aid radiologists to better understand and, more importantly, diagnose these infrequent events.

Introduction

Venous thromboembolism (VTE) typically aris3ing from deep venous thromboses in the legs can lead to subsequent pulmonary embolism (PE), treatment of which depends on accurate diagnosis by radiologic methods. This is a commonly encountered problem for the radiologist as the incidence of PE in the United States is approximately 1 per 1000 patients annually. As approximately 6%-10% of these cases are fatal,[1,2] VTE/PE as a differential diagnosis looms large in the minds of many practitioners. However, nonthrombotic pulmonary embolism (NTPE) is less well appreciated as it is encountered much less frequently. NTPE is defined as the embolization of nonthrombotic material to the pulmonary vasculature. Nonthrombotic material can arise from various cell types (adipocytes, hematopoietic, amniotic, trophoblastic, or tumor), bacteria, fungi, or, as well, from foreign material or gas[3] (Fig 1).

As with VTE/PE, NTPE can be a formidable clinical diagnostic challenge, as the condition often presents with nonspecific or unusual clinical signs. Typically, signs and symptoms are of low specificity (eg, difficult breathing, chest pain on inspiration, palpitations, low blood oxygen saturation, cyanosis, tachypnea, and tachycardia). Despite the rarity of NTPE, the clinical history should suggest that the patient belongs to one of the categories described below, which will provide clues as to the nature of potential nonthrombotic emboli. On the positive side, there are certain imaging findings which are specific to NTPE.[3,4]

This article will review these imaging findings and provide illustrative examples so that these rare, but serious conditions can be promptly diagnosed by the radiologist to allow rapid treatment of a potentially fatal condition (Fig 2A-C).

Biologic Materials

Septic Pulmonary Embolism

A septic pulmonary embolism (SPE) is characterized by thrombotic material from an infected intravascular vegetation that is dislodged and eventually reaches the pulmonary arteries.[5] Microorganisms responsible for these vegetations can arise from an iatrogenic cause, such as an infected intravascular catheter or device, be seeded from a soft-tissue infection, or most classically arise from infective endocarditis. Up to 75% of cases of SPE occur in patients with right-sided infective endocarditis resulting from IV drug abuse.[6] Clinical presentation can vary from febrile illness with cough and shortness of breath to sepsis.

For diagnosis, CT is the preferred imaging modality along with an echocardiogram and blood cultures for confirmation of bloodstream and right-sided heart infection.[7] Common imaging findings on CT include wedge-shaped opacities secondary to pulmonary infarction, and multiple peripheral nodules with a predilection for lower lobe distribution. Variable cavitation of the nodules may occur depending on the chronicity of the disease course. In some cases, it is possible to visualize the blood vessel leading to the nodule (the “feeding vessel sign”) indicating the hematogenous origin of the nodules.[8,9] A central nodule with surrounding ground glass opacity (the “Halo sign”), which arises from surrounding parenchymal infarction and

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https://doi.org/10.1067/j.cpradiol.2018.07.007
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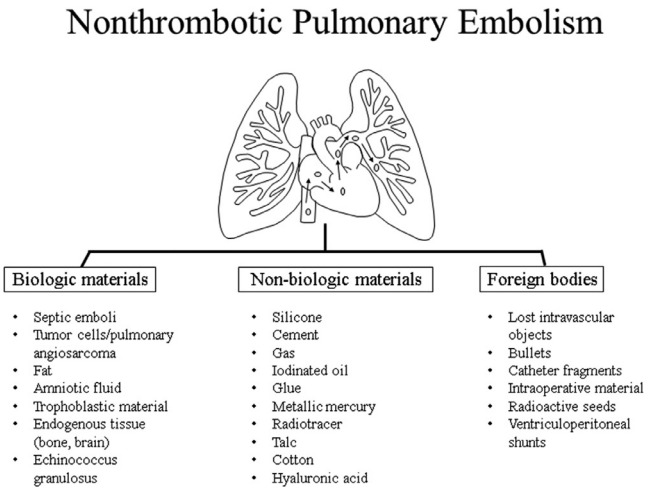

FIG 1. Nonthrombotic pulmonary embolism (NTPE) is defined as embolization to the pulmonary circulation caused by a wide range of substances of endogenous and exogenous biological and nonbiological origin and foreign bodies.

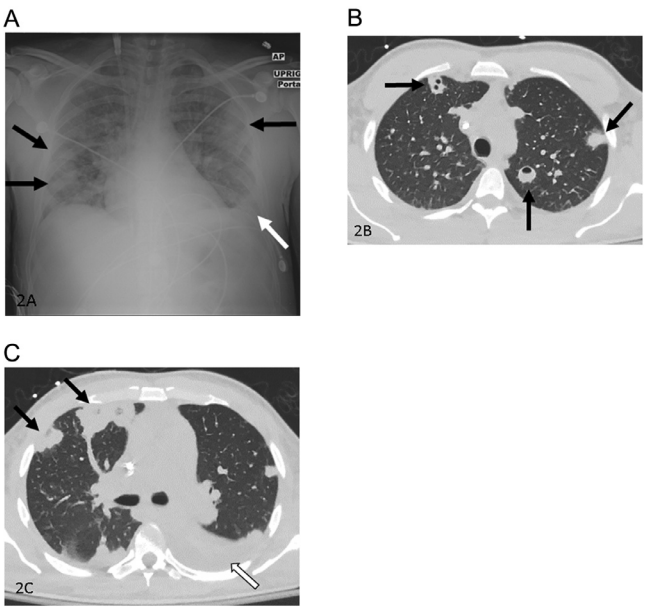

FIG 2. Septic embolism: 29-year-old male with a history of IV drug abuse and infective endocarditis involving the tricuspid valve presented with shortness of breath. (A) Chest radiograph shows scattered ill-defined nodular opacities and consolidative lesions throughout both lungs (black arrows) as well as a pleural effusion (white arrow). (B, C) A non-contrast CT examination shows multiple bilateral pulmonary nodules (black arrows), some of which are centrally necrotic and cavitary. Bilateral pleural effusions are seen (white arrow).

inflammation, is another nonspecific sign commonly seen in SPE.[10] Though isolated imaging findings are nonspecific, when occurring together in the appropriate clinical setting (eg, IVDA) a diagnosis of SPE can be made with more confidence (Fig 3A-C).

Tumor Embolism

Tumor embolism occurs when malignant cells within the blood stream become lodged in the lumen of the pulmonary vasculature. The true incidence of this complication is difficult to assess, but has been reported at 2.4%-26% based upon postmortem examinations.[11,12] At postmortem, the most common solid tumors are breast, gastric, renal cell, prostate, choriocarcinoma,

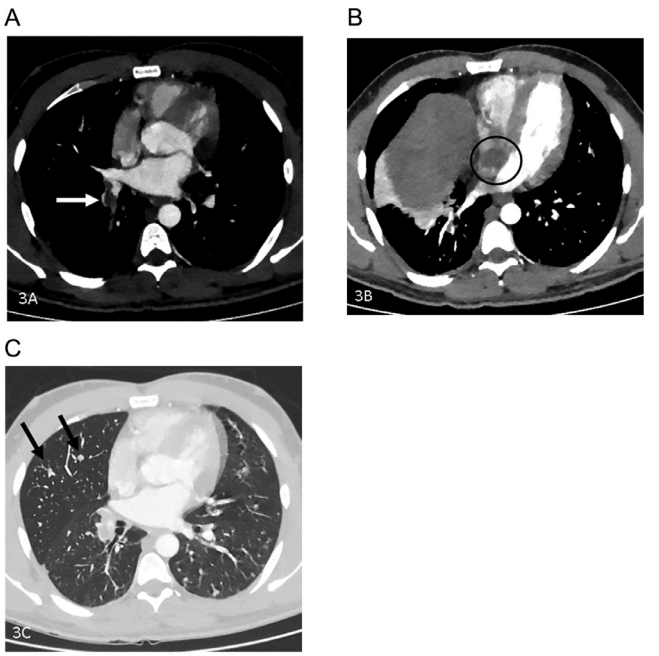

FIG 3. Tumor embolism: 27-year-old female with a history of infiltrative hepatocellular carcinoma. (A) An enhanced CT demonstrates a large hypodense filling defect in the right lower lobe pulmonary artery extending into segmental branches (white arrow). (B) Additional thrombus was detected at the right atrium (black circle). (C) CT with lung window demonstrates intravascular metastasis in the right middle lobe (black arrows).

and hepatocellular malignancies.[13] Tumor embolism has been classified into 3 categories. Type I is the "classic," or "true" type originating from a distant primary tumor via hematologic seeding. There is no invasion into vessel walls.[14,12] Type II tumor embolism results from a tumor growing into the pulmonary arteries (eg, renal cell carcinoma). Type III tumor embolism occurs when local disease, either primary lung cancer or metastatic tumor, infiltrates, and obstructs pulmonary vasculature.[4]

Imaging findings of tumor embolism are often difficult to distinguish from thromboembolism. The intra-arterial filling defects seen with macroembolism are identical to thromboembolism.[11] Interestingly, macroembolisms usually cause slow progressive dyspnea and rarely result in sudden death.[15] The classic CT finding of microembolism is a "tree-in-bud" appearance, consisting of small centrilobular nodules of soft tissue attenuation connected to multiple branching linear structures originating from a single stalk. Imaging signs specific for tumor embolism include dilated and beaded peripheral arteries, IV contrast enhancement of intravascular filling defects, and F18-fluorodeoxyglucose (FDG) avidity on PET/CT.[11] As expected, tumor emboli are resistant to recannulation and fibrinolytic therapy (Fig 4A-D).

Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is a leading cause of maternal mortality in the developed world, accounting for approximately 10% of all maternal deaths in the United States.[16] Despite this high percentage, AFE remains a rare occurrence with an incidence of approximately 7.7/100,000 births.[17] Patients typically present with acute respiratory distress, mental status changes, central nervous system irritability, and a cutaneous rash.[18] Historically, AFE was thought to occur after amniotic fluid entered maternal circulation during normal labor via uterine vein tears or during cesarean section.[19] However, mechanical obstruction of pulmonary blood flow is not always evident in AFE, and the constellation of symptoms is broader than other forms of

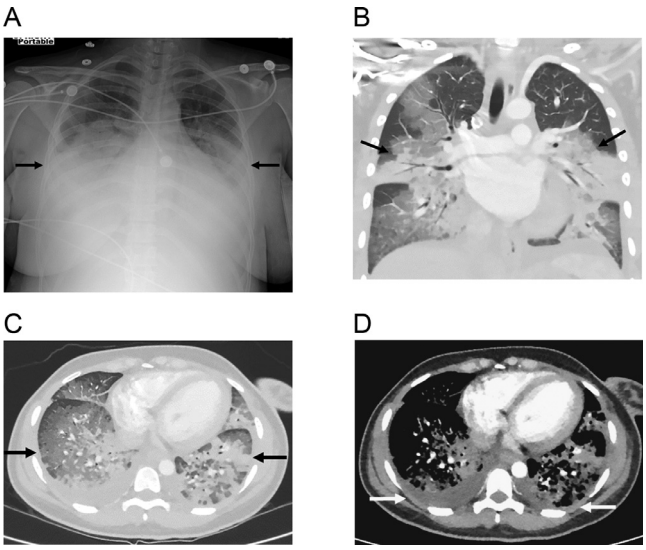

FIG 4. Amniotic fluid embolism: 23-year-old female with hemoptysis, tachycardia and chest pain 24 hours after cesarean section. Chest radiography (A) demonstrates diffuse airspace opacities in the bilateral mid and lower lung fields (black arrows). Coronal and axial CT images (B, C) demonstrate patchy and confluent ground glass and consolidative opacities in the mid and lower lung fields (black arrows). (D) Axial CT with soft tissue window shows small to moderate bilateral pleural effusions (white arrows).

pulmonary embolism.[20,21] Consequently, some now hypothesize that the disease has an immunologic origin.[22,23]

Due to the acuity of the condition at the time of presentation, imaging is not recommended until the patient is stable. Diagnosis is based on clinical findings, and is one of exclusion. Chest radiography findings include diffuse bilateral homogenous opacities suggestive of pulmonary edema. On CT, diffuse bilateral ground glass opacities have been reported (Fig 5A).[24]

Fat Embolism

Fat embolism occurs when fat enters the systemic circulation following bone fracture or surgical manipulation. Following simple fractures of long bones, for example, femur or tibia, fat embolism occurs in approximately 1%-3% of patients. This incidence increases up to 20% in patients with severe trauma.[25] The traditional diagnostic triad includes acute respiratory distress, mental status changes and a cutaneous petechial rash, reported in approximately 85% of patients.[18] However, these signs may also be present in amniotic fluid embolism.

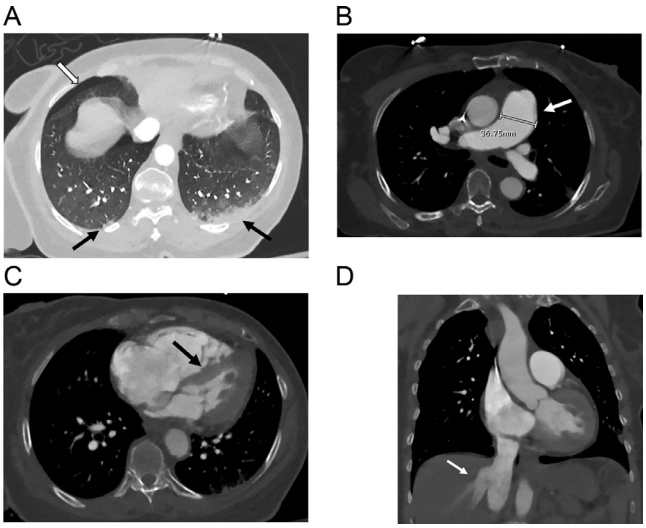

FIG 5. Fat embolism: 87-year-old female with shortness of breath 2 days after right hip arthroplasty. CT Pulmonary angiography with lung window (A) reveals airspace opacities dependently in the lower lobes (black arrows). There is a small right pneumothorax (white arrow). CT Pulmonary angiography (B, C, D) demonstrates evidence of right heart strain including mild enlargement of the main pulmonary artery (white arrow), right heart enlargement with interventricular septal flattening (black arrow), and reflux of contrast into the IVC and hepatic veins (white arrow). No filling defects are identified in the pulmonary arteries.

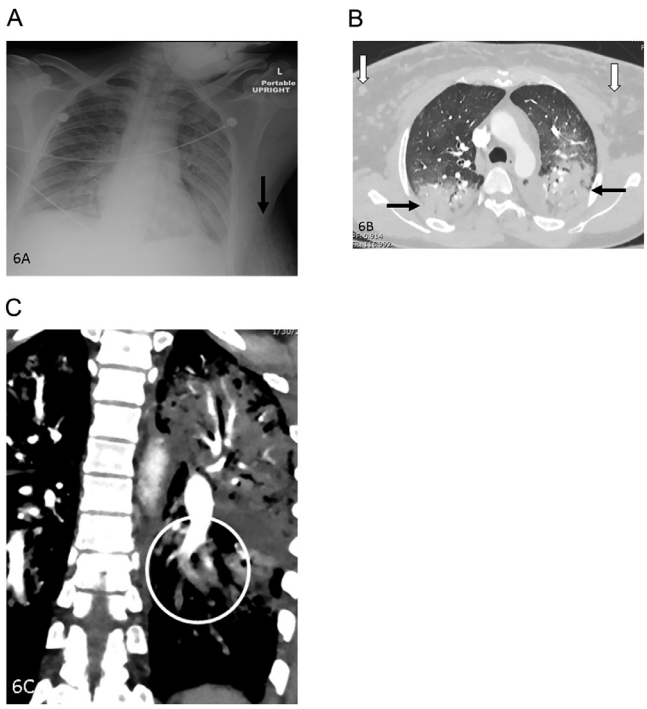

FIG 6. Silicone embolism: 36-year-old transgender female with progressing respiratory distress that began immediately after cosmetic silicone injections to the buttocks. Frontal radiograph (A) shows diffuse hazy opacities throughout the lungs, most pronounced in the lower lobes. There are multiple nodular densities in the left breast from previous silicone injection (black arrow). Axial image of CT pulmonary angiography with lung window (B) shows diffuse bilateral airspace opacities in a dependent distribution (black arrows) consistent with silicone pneumonitis and ARDS. Nodular soft tissue densities in the breast (white arrows) are sequelae of prior silicone injection. Coronal image (C) shows diffuse dependent intraluminal filling defects in multiple bilateral subsegmental and distal pulmonary arteries (circle).

Imaging findings of fat embolism can be confused with pulmonary contusion if the time of injury is not taken into consideration. Typically, pulmonary contusions produce immediate changes whereas those related to fat embolism develop over 1-2 days after fracture or trauma.[26] CT findings include bilateral, patchy opacities in peripheral and dependent areas or manifest with no zonal predominance. No definitive intravascular filling defects are seen, and there may be signs of right heart strain on the basis of acute pulmonary hypertension (Fig 6A-C).[27]

Nonbiologic Materials

Silicone Embolism

Polydimethylsiloxane (liquid silicone) is increasingly used for legal as well as illicit cosmetic augmentation. A significant increase in the number of published case reports of silicone embolism has occurred over the past 2 decades.[28] Common sites for injection include breasts, hips, and buttocks.[29] It is believed that the injected silicone enters the venous system, and eventually reaches the pulmonary vasculature causing mechanical obstruction.[4] The clinical presentation is similar to fat embolism with acute dyspnea, petechial rash, altered mental status, and hypoxemia.[29]

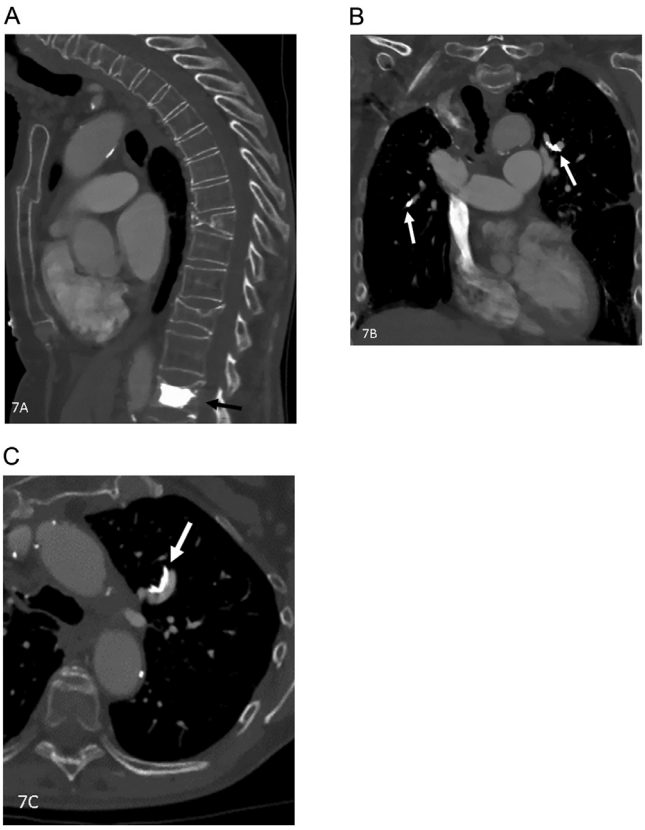

FIG 7. Cement embolism: 80-year-old female with chest pain and a history of vertebroplasty. Sagittal CT image with bone window (A) shows diffuse osteopenia with multilevel compression fractures of the thoracolumbar spine as well as vertebroplasty cement in the L1 vertebral body (black arrow). Coronal and axial images of CT pulmonary angiography (B, C) depict high density material in the bilateral upper lobe pulmonary arteries consistent with migration and embolization of vertebroplasty cement (white arrows).

Imaging demonstrates diffuse, dependent, bilateral ground-glass opacities with a lower lobe predominance. The appearance of the lungs will be consistent with noncardiogenic pulmonary edema. Dependent intraluminal filling defects in pulmonary arteries will also be present (Fig 7A-C).[11,30,29]

Cement Embolism

Cement embolism is always iatrogenic, occurring after cement injection into an intraosseous space. Vertebroplasty and kyphoplasty are the most common procedures associated with this complication ranging in incidence from 3.5% to 23%.[31,32] The higher incidence of cement embolism in these procedures is thought to result from 2 factors. Firstly, the cement used has less viscosity than that used in other orthopedic procedures, and secondly, the embolism is aided by the absence of any physiological valves in the paravertebral venous plexus.[4] Most commonly, no clinical symptoms are present and emboli are identified on follow-up imaging.[32]

Rarely emboli are identified on intraoperative fluoroscopy.[33] Chest radiography may reveal small, high-density linear opacities, that may branch, seen near the spine or in either central or peripheral lungs. On CT, multiple tubular areas with high attenuation, outlining the pulmonary arteries, can be seen (Fig 8A-D).[34]

FDG Embolism

Positron emission tomography/computed tomography (PET/CT) is now the standard of care in staging, restaging, and monitoring of treatment response in many patients with cancer. Though uncommon, FDG

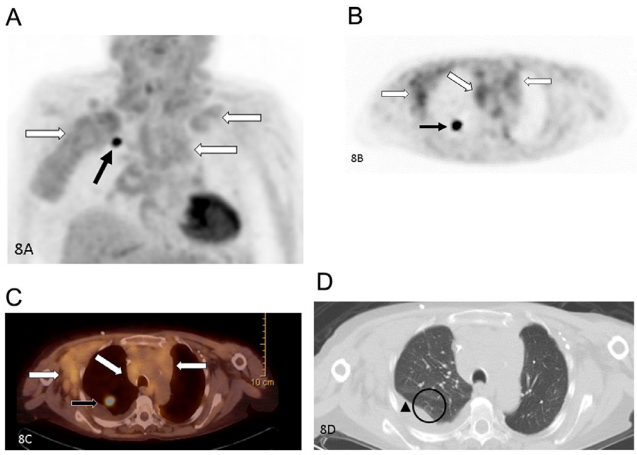

FIG 8. FDG Embolism: 77-year-old female under surveillance for a history of chronic lymphocytic leukemia and remote breast cancer. Maximum intensity projection image (A), axial PET image (B) and fused axial PET/CT image (C) show an area of markedly increased FDG uptake in the right upper lobe (black arrows). Axial CT image (D) lacks a corresponding structural abnormality, therefore, the diagnosis of FDG embolus was made. Incidental note of minimal to mild FDG uptake in the bilateral axillary and mediastinal lymphadenopathy (white arrows) consistent with known CLL. Right pleural effusion is seen (arrow head).

embolism is important to recognize as a false positive result appearing to represent hypermetabolic viable tumor.[35] In the literature reviewed, all instances of FDG microembolism resolved spontaneously.[35-37]

Focal and intense FDG uptake without a detectable corresponding lesion on accompanying CT imaging is highly suggestive of FDG microembolus. One way to avoid microembolus is to use a preplaced IV cannula when injecting FDG.

Y-90 Radioembolization and Radiation Pneumonitis

A now established technique for treatment of hepatocellular carcinoma or metastatic liver disease is the intravascular administration of yttrium-90 (Y-90) microspheres. The hypervascularity of these tumors make them amenable to radioembolization. However, if an arteriovenous shunt large enough to allow passage of microspheres is present, Y-90 may pass into the pulmonary circulation and cause radiation pneumonitis.[38,39] To mitigate this problem, patients now undergo a pretreatment study with Tc-99m to evaluate the degree of arteriovenous shunting present.[39] Clinically, patients with pneumonitis present with nonproductive cough, dyspnea, fever, bronchoalveolar lymphocytosis, and eosinophilia.[40]

On imaging, radiation pneumonitis demonstrates ill-defined, patchy ground glass opacities with perihilar and hilar sparing. There is often an asymmetrical distribution with greater right-lung involvement.

Talc (Hydrous Magnesium Silicate) Embolism

Fillers such as talc, cellulose, or corn starch, are added to oral medications containing small quantities of active drug in order to provide bulk. This is problematic when these medications are dissolved in water to facilitate IV injection.[41] Upon injection insoluble particles from filler materials can result in pulmonary embolization as well as producing an inflammatory response. The inflammatory response can cause a giant cell reaction eventually leading to granulomatosis.[41]

Most patients diagnosed with talc granulomatosis are IV drug abusers who present with a history of slowly progressive dyspnea.[4,41] Chest radiography findings range from early disease with widespread small nodules to late-stage with large areas of opacity indicative of massive fibrosis.[42] Pulmonary hypertension may also be present.[43] As with chest radiography, CT demonstrates a progression of findings. Initially, small (1-mm) nodular and ground-glass opacities may be

seen diffusely or in a well-defined centrilobular distribution.[44] Subsequent granulomatous reaction within the vessel walls results in the classic "tree-in-bud" appearance.[41] As the disease progresses, nodules can become confluent resulting in heterogeneous conglomerate masses with dense opacities representing talc deposition.[45]

Air Embolism

Venous air embolism can occur following both diagnostic and therapeutic procedures, surgery, and trauma. Interestingly, it has been reported that up to 23% of patients who undergo contrast-enhanced CT have small quantities of air identifiable within their central venous systems.[11] The amount and rate of entry of air determines the degree of symptomatology, with an estimated minimum lethal dose in humans of 300-500 mL.[46] Air embolism may also occur following rapid ascent while scuba diving.[47]

Patients present with sudden onset dyspnea, hypotension, chest pain, altered mental status, or convulsions.[46] On radiography it is possible to see areas of hyperlucency in the heart, main pulmonary artery, or hepatic veins. Findings also include focal pulmonary oligemia, and pulmonary edema.[48] CT may demonstrate small amounts of air in the right side of the heart, main pulmonary artery, or other systemic veins (Figs 9A-C and 10).[49]

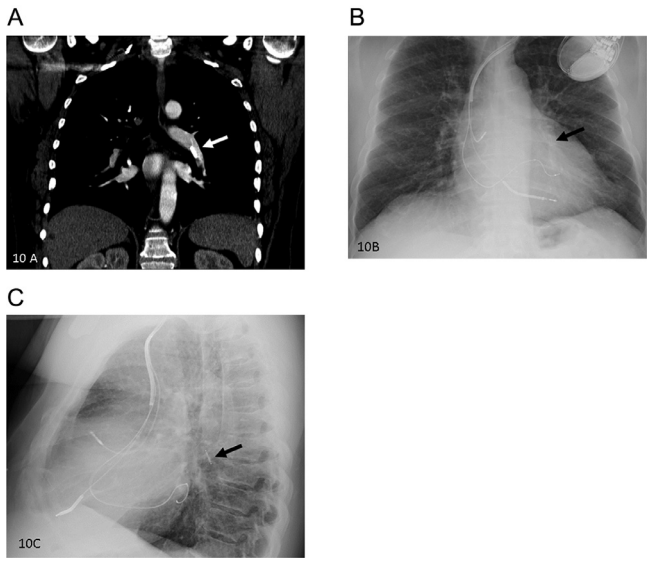

FIG 10. CardioMEM device: 57-year-old female with a history of Lupus and pulmonary hypertension. (A) Coronal image of CT shows an oval metallic density in the left interlobar pulmonary artery (white arrow). (B, C) Frontal and lateral radiographs show the metallic density overlying the region of the left pulmonary artery (black arrows).

Foreign Bodies

Foreign body pulmonary embolism can be iatrogenic in the case of intravascular devices, or traumatic, as in the case of bullet fragment embolization. The number of intravascular procedures is rapidly increasing, and so too the number of associated complications.[50] Embolization can result from dislodged fragments from coils, stents, guide wires, or catheters to name a few. Embolization not only may lead to pulmonary vascular occlusion, but also cardiac arrhythmias if devices become lodged in the right side of the heart.[50] Catheter embolization most often results from shearing of the distal tip of the catheter during improper removal via the introducing needle.[51] Regardless of the nature of the intravascular foreign body, the recommended treatment is attempted endovascular retrieval. If unsuccessful, or too large for endovascular removal, vascular surgery may be indicated.[50]

The best imaging modality to locate an intravascular object is CT or angiography. The imaging of a hyperdense object visible in the pulmonary vasculature or right heart confirms embolization. With the multiplicity of devices, it is recommended that the radiologist refers to an image of the deployed device to aid in search for embolic fragments.

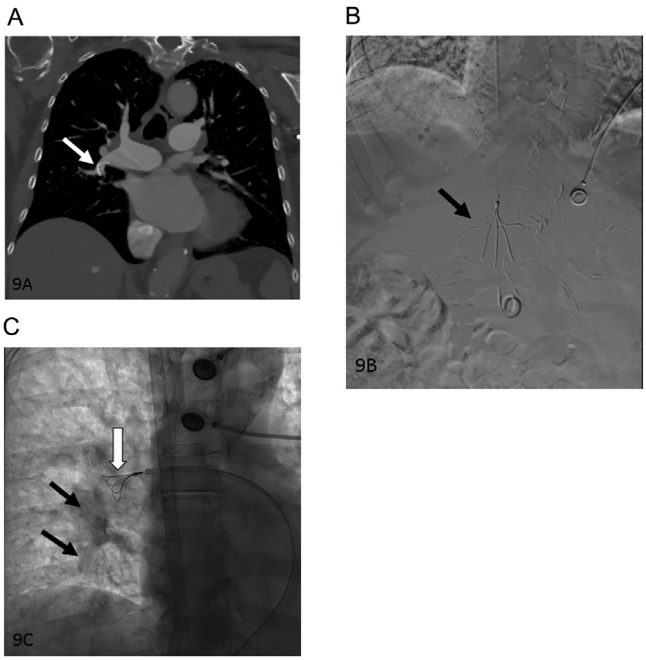

FIG 9. Migrated IVC filter fragments in a 68-year-old male. Coronal CT pulmonary angiography (A) shows a curvilinear metallic density in the right lower lobar pulmonary artery (white arrow). Frontal digital subtraction image (B) shows a slight migration of an infrarenal IVC filter (black arrow). Conventional agiographic image (C) shows migrated fragments in the right lower lobe pulmonary arteries (black arrows) and approaching retrieval device (white arrow).

Radioactive Seed Embolization

Brachytherapy seeds, such as those used in prostate cancer therapy, have been known to embolize from their implantation sites. In the case of the prostate, migration to the pulmonary vasculature is possible through the adjacent periglandular and hemorrhoidal venous plexuses.[52] Several studies have reported serious adverse events attributed to seed embolization in this patient population, which include pneumonitis, small-cell lung cancer, and acute myocardial infarction.[53-55]

The characteristic shape of the brachytherapy seeds is seen on chest radiography as small distinct linear radiopacities. On CT seeds can appear as punctate hyperdense attenuations with possible streak artifact resulting from their titanium or silver casings.[56]

Of note is the CardioMEMS (Atlanta, GA) device, which is an implantable pulmonary artery monitoring device used in patients with heart failure (Figs 9A-C). As the device is deployed within the pulmonary arteries by design, it can easily mimic a foreign body embolus. On imaging a tubular metallic density will be visible in the pulmonary arteries, with the radiopaque central sensor component also visible.

Conclusion

NTPE is defined as the embolization of nonthrombotic material to the pulmonary vasculature, resulting from a wide range of biologic and nonbiologic materials which include intravascular foreign bodies. The lack of specificity of clinical symptoms and imaging signs

associated with NTPE present a diagnostic challenge to the radiologist. Definitive clues may be present in imaging, which when combined with appropriate clinical setting, allows the radiologists to determine the correct diagnosis promptly and thus guide appropriate patient management.

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