

Consolidation With Diffuse or Focal High Attenuation Computed Tomography Findings

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Abstract: This pictorial essay aims to present various lesions that could present as consolidations with diffuse or focal high attenuation on computed tomography, helping to make the diagnosis more confident and specific. The radiologic literature has limited information about such findings and the role of computed tomography in the differential diagnosis. The following diseases are presented: metastatic pulmonary calcification, pulmonary alveolar microlithiasis, amiodarone lung, talcosis, iodinated oil embolism, tuberculosis, silicoproteinosis, and amyloidosis. In conclusion, air-space consolidations can be seen in a wide variety of diseases affecting the lungs. The identification of the different patterns of consolidation with focal high attenuation narrows the differential diagnosis. We present a diagnostic approach based on appearance and distribution of these lesions.

Key Words: consolidation, high-resolution CT, high attenuation
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The computed tomography (CT), and especially high-resolution CT (HRCT), when compared with plain films has higher sensitivity to detect mild differences in lung density, morphology, and distribution of the lesions, and associated abnormalities. Such characteristics make possible the HRCT to depict several diagnoses of pulmonary diseases.

Diffuse hyperdense pulmonary abnormalities could be divided into 4 patterns: (a) diffuse small hyperdense nodules; (b) multiple calcified large nodules or masses; (c) hyperdense linear or reticular pattern; and (d) hyperdense consolidations.¹

Air-space consolidations seen in chest radiographs and CT are usually nonspecific, making the differential diagnosis difficult when based on such aspects. Clinical and laboratorial data could help narrowing diagnostic possibilities.

Measurement of the attenuation at noncontrast-enhanced CT inside the areas of consolidation can give the clue for the specific diagnosis. Attenuation higher than muscle seen as dense diffuse pulmonary opacities are due to calcium deposits in metastatic pulmonary calcification (MPC) and pulmonary alveolar microlithiasis (PAM), talc powder deposition in talcosis, and iodine accumulation in amiodarone lung toxicity. Deposition of iodinated oil embolism can occur also after transcatheter oil embolization or after lymphangiography. Tuberculosis, amyloidosis, and silicoproteinosis may have focal calcifications, usually seen as small punctate calcified foci inside the areas of consolidation.

This pictorial essay has the aim to present various lesions that could present as consolidations with diffuse or focal high attenuation on CT, helping to make the diagnosis more confident and specific. The radiologic literature has limited information about such findings and the role of CT in the differential diagnosis.¹

MPC

MPC is consequence of calcium deposition in normal pulmonary parenchyma.² This condition can occur in a variety of disorders: primary and secondary hyperparathyroidism, chronic renal failure, intravenous calcium therapy, and massive osteolysis due to metastases or multiple myeloma.¹ MPC usually presents as an asymptomatic condition. A fulminant course evolving to respiratory failure and early death is seen in some cases.^{2–4}

HRCT findings are characterized by centrilobular fluffy ground-glass nodular opacities, which contain foci of calcification, seen mainly in the upper pulmonary zones. The calcifications can be punctate within the nodular opacities, ringlike, or diffuse (involving the entire nodule). These lesions may mimic air-space nodules, and rarely present as lung consolidations (Fig. 1). Most of these conditions can have high attenuation without consolidation. Other common feature seen in this condition is calcified vessels of the chest wall.^{1,2} Soft tissue window settings are useful to demonstrate extensive calcification.²

Histologic analysis in MPC is characterized by calcium deposition in alveolar septa, small pulmonary vessels, and bronchial walls.^{3,4} More severe interstitial calcification can result in dense consolidation. Calcium

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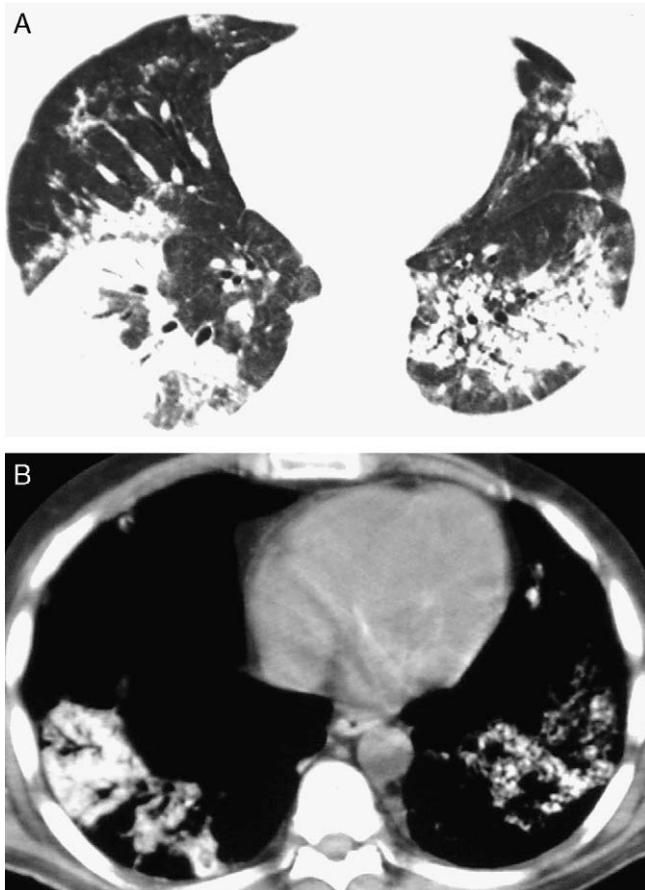


FIGURE 1. A 49-year-old man with multiple myeloma and metastatic pulmonary calcification. A, Computed tomography through the lung bases shows bilateral areas of consolidation, and a few small nodular opacities. B, Mediastinal window demonstrates extensive calcifications within areas of air-space consolidation.

deposits may induce a lung reaction with alveolar organizing exudates evolving to fibrosis. These alveolar infiltrates may calcify.²

PAM

PAM is a rare chronic disease characterized by widespread calcific intra-alveolar concretions within alveolar spaces.^{4,5} The etiology and pathogenesis of microcalcific nodules formation are still unknown, probably related to an recessive autosomal heritage. Clinical symptoms are usually absent, and, when present, being characterized as dyspnea on exertion. Patients with PAM usually demonstrate extensive pulmonary abnormalities, with mild clinical manifestation. Therefore, radiographic diagnosis goes before any clinical complaints. Occasionally, progressive deterioration of pulmonary function may occur. Death is generally due to respiratory failure and “cor pulmonale.”^{4,5}

Characteristic HRCT findings consist of multiple bilateral calcified micronodules measuring less than 1 mm in diameter, which tend to confluence.^{1,4} The lesions have

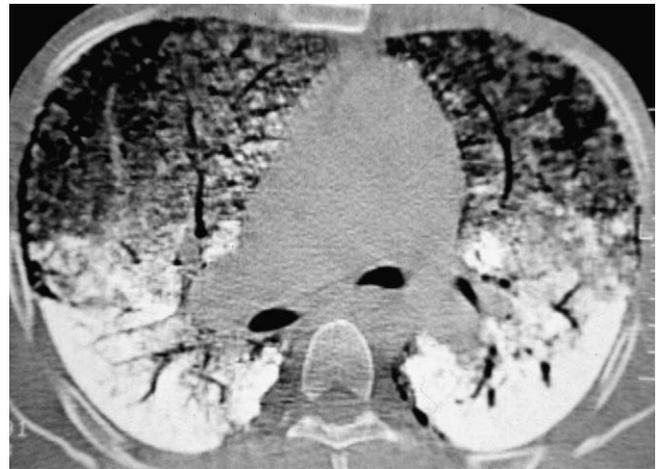


FIGURE 2. A 21-year-old male patient with pulmonary alveolar microlithiasis. High-resolution computed tomography with mediastinal window shows hyperdense consolidations in the lower portions of the lungs, and ill-defined confluent nodules in the upper lung areas.

predominance to cardiac borders and posterior pulmonary zones.^{4,5} Micronodules show often peripheral lobular distribution, resulting in a pattern resembling interlobular septa and subpleural calcification.^{3,5} Another feature seen on HRCT scans includes a very low attenuation line alongside the pleura, called “black pleural line,” and, probably due to subpleural cysts or a thin dark fat layer below the ribs.⁴ Apical “bullae” may also be seen in the lungs. Confluent nodules eventually present as air-space consolidations in patients with long-standing disease¹ (Fig. 2).

Intra-alveolar accumulation of spherical microliths is seen at histopathology. In the early stages of the disease, the alveolar walls are normal; eventually interstitial fibrosis develops. Blebs and bullae are often present, particularly in the lung apices.^{1,4}

AMIODARONE LUNG

Amiodarone-induced pulmonary toxicity is a serious adverse effect seen in patients receiving large doses of amiodarone to prevent cardiac arrhythmias. The treatment with this drug results in deposition of iodine in the lung parenchyma, a constituent of the amiodarone molecule. Clinical complaints are insidious, consisting of nonproductive cough, dyspnea, and occasionally fever. The diagnosis is one of exclusion because the signs and symptoms are not specific, and there is no laboratory test allowing the diagnosis.^{1,6}

The most common CT findings include septal thickening, interstitial fibrosis, and consolidations. These opacities usually are peripheral in location. High iodine content makes possible the detection of amiodarone deposits in the lung by CT as a high-attenuation focal or multiple parenchymal opacities^{1,4,6} (Fig. 3). The association of dense lung air-space consolidations with high density of the liver and spleen is characteristic of

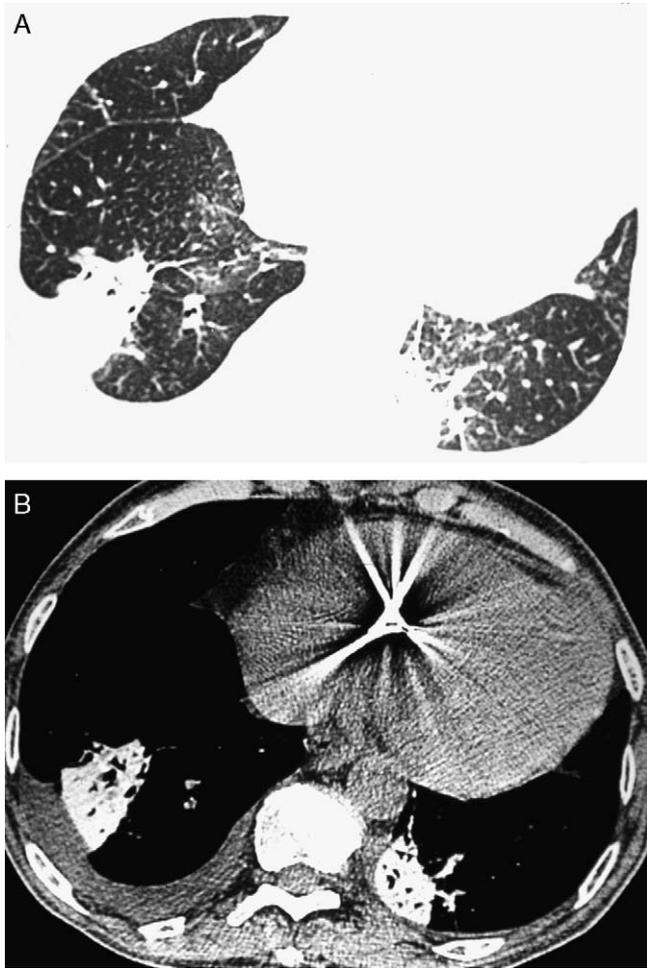


FIGURE 3. A 63-year-old female patient with amiodarone lung. High-resolution computed tomography with parenchymal (A) and mediastinal (B) windows demonstrate peripheral consolidations with air bronchogram, increase of heart volume, and right pleural effusion. In B, the high-density consolidations are shown.

amiodarone impregnation. However, it is not known whether this change in lung density indicates toxicity or the normal accumulation of amiodarone in lung tissue resulting from its therapeutic effects.⁶

Pathologic examination of the lung in amiodarone toxicity typically reveals chronic inflammation and fibrosis of the alveolar septa, and hyperplasia of type II pneumocytes. In addition, accumulation of intra-alveolar macrophages, which contain vacuolated cytoplasm with iodine inclusions, is also seen.⁴

TALCOSIS

Talc pneumoconiosis has been described in workers exposed to talc during extraction of magnesium silicate from mines, grinding, packing, and transportation of the product. Another form of talcosis is caused by the endovenous administration of talc seen in drug abusers. Clinical manifestations of talcosis consist of dry cough

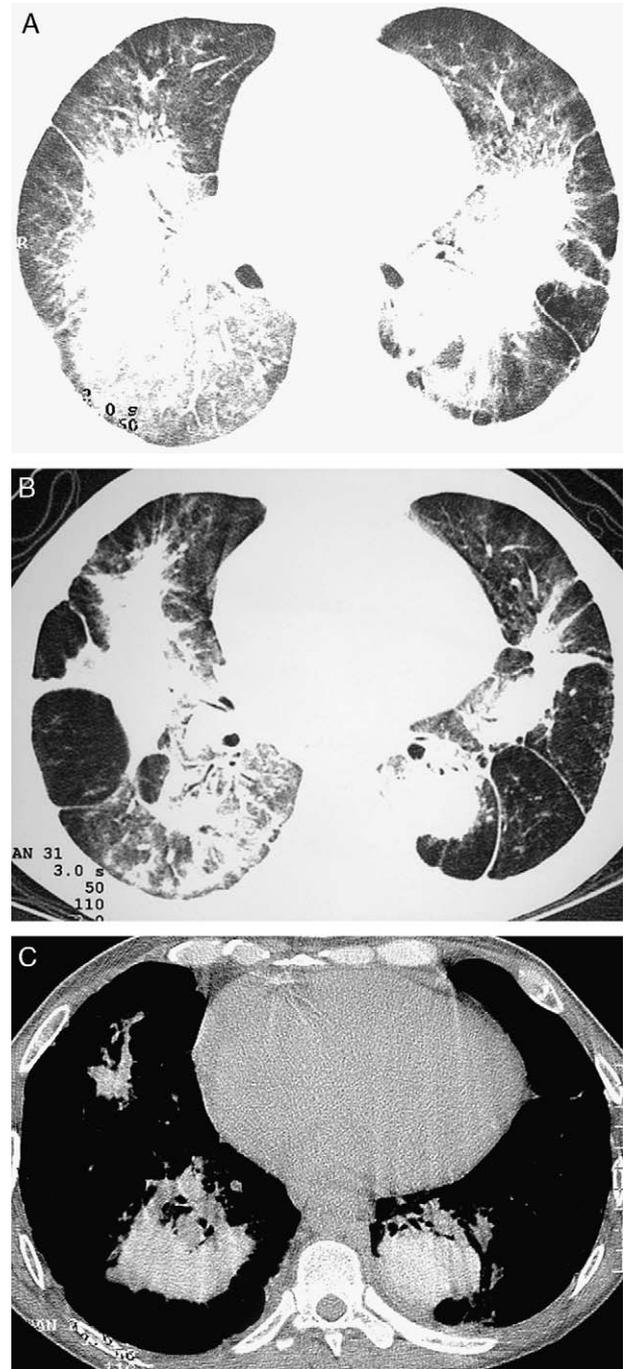


FIGURE 4. A 41-year-old male patient with talcosis. A, High-resolution computed tomography at the level of the main bronchi demonstrates bilateral conglomerate masses. Linear opacities, architectural distortion adjacent to the conglomerate masses, and evidence of emphysema are also seen. B, High-resolution computed tomography at the level of the lower lobes shows air-space consolidations and large areas of panlobular emphysema. C, Mediastinal window demonstrates high attenuation within the conglomerate masses, suggesting talc deposition.



FIGURE 5. A 46-year-old woman with iodinated oil embolism. Nonenhanced computed tomography scan at the level of the lung bases after chemoembolization in an asymptomatic patient with hepatocellular carcinoma shows bilateral hyperattenuating ethiodized oil (Lipiodol) in the collapsed lower lobes. Note significant bilateral pleural effusion.

and chronic dyspnea. Late complications include pulmonary arterial hypertension and cor pulmonale.^{1,4}

Earlier tomographic manifestations consist of a diffuse micronodular pattern with well-defined nodules, or diffuse ground-glass opacities. As the disease progresses the nodules can become confluent, resulting in hyperdense consolidations or confluent perihilar masses (Fig. 4). These lesions are similar to those seen in progressive massive fibrosis caused by silicosis. The dense opacities result from talc deposition within the pulmonary arterioles, capillaries, and interstitium. Panlobular emphysema involving predominately the lower lobes was seen almost exclusively in patients with talcosis secondary to endovenous injection of Ritalin.^{1,4,7}

Pathologically, in the early stages of the disease, talcosis consists of multiple small granulomas composed of multinucleated cells containing birefringent crystals, which are identified in the alveolar septa and alveolar air spaces. In long-standing disease the nodules tend to confluence, producing large foci of consolidation associated with progressive fibrosis, resembling the progressive massive fibrosis seen in other pneumoconiosis. Panacinar emphysema, sometimes with bulla formation, is often evident. Foreign material is readily identifiable within the giant cells and is particularly well seen by polarization microscopy.^{4,7}

IODINATED OIL EMBOLISM

Iatrogenic causes of iodinated oil embolism occur either after lymphangiography or after transcatheter oil chemoembolization. Chemoembolization of the liver for unresectable malignancy using ethiodized oil is being used with increasing frequency. Small and usually invisible intratumoral arteriovenous shunts allow chemoembolization material to pass into the hepatic veins and thence

into the lungs. Although ethiodized oil may cause pulmonary inflammatory changes, most patients with iodinated oil embolism are asymptomatic. Rarely, cough, dyspnea, and hypotension occur. Extrahepatic chemoembolization material is commonly seen in other organs, but usually do not cause problems.^{1,8}

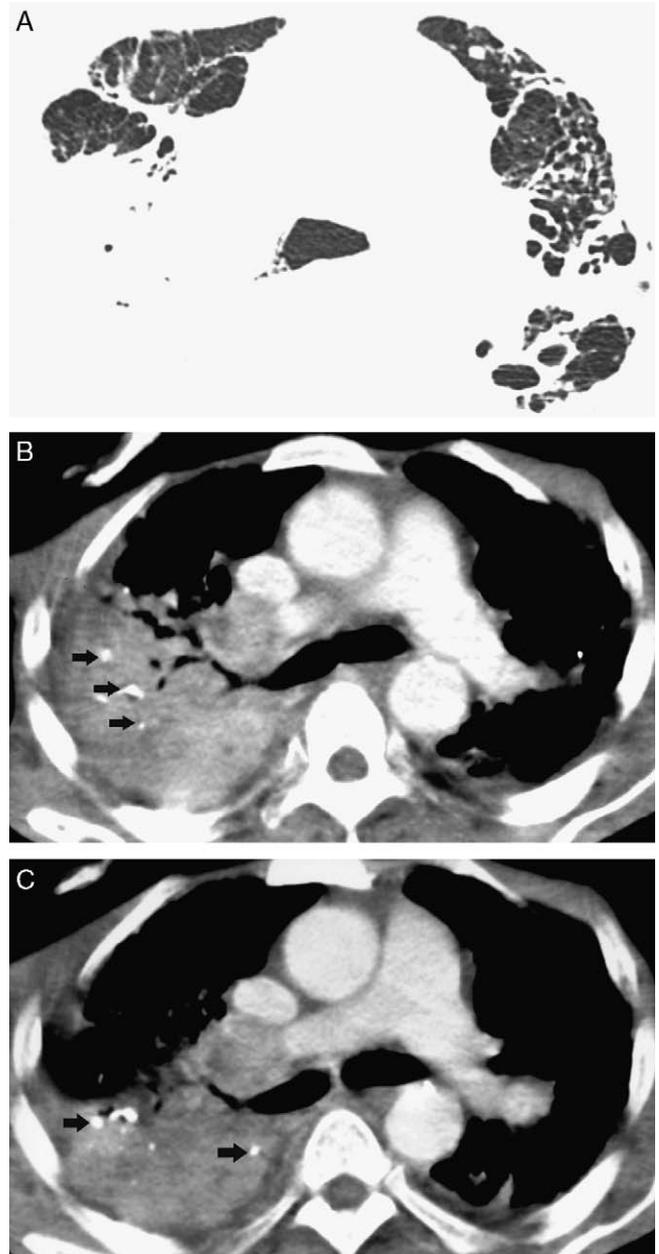


FIGURE 6. A 22-year-old woman with tuberculosis. High-resolution computed tomography shows in A, extensive consolidations in both lungs, with architectural distortion and cavitary lesions on the left. In B and C, contrast-enhanced mediastinal window images in the upper regions of the chest. Punctate calcifications are seen within the consolidation areas located in the right upper lobe (arrows).

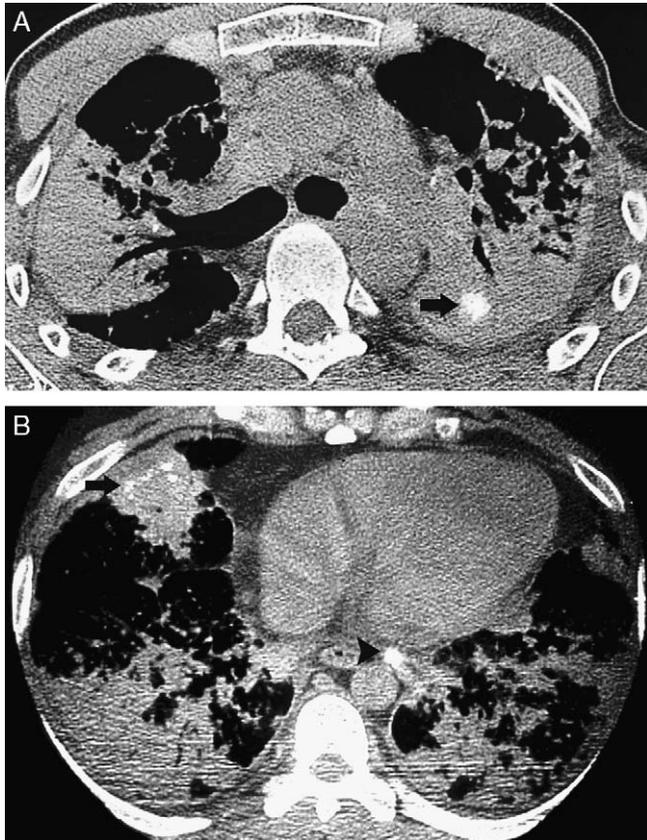


FIGURE 7. A 33-year-old male sandblaster with silicoproteinosis (A and B). Computed tomography with mediastinal window demonstrates extensive consolidation with air bronchogram and calcifications (arrows), and calcified mediastinal lymph nodes (arrow head).

CT findings consist of multifocal patchy areas of ground-glass attenuation and high-attenuation areas of consolidation and collapse (Fig. 5).

TUBERCULOSIS

Pulmonary tuberculosis is a chronic recurrent infection caused by *Mycobacterium tuberculosis*. Symptoms are most commonly nonspecific, and include fatigue, weakness, anorexia, weight loss, and mild fever. Unproductive or mildly productive cough is usual, occasionally associated with hemoptysis.⁴

The patients with tuberculosis may present on CT scans focal areas of air-space consolidation, which could cavitate. Ill-defined centrilobular branching nodules may be seen, assuming the so called tree-in-bud pattern.⁴ Dystrophic calcifications are frequently seen in chest tuberculosis, being related to pulmonary granulomas, mediastinal lymph nodes, and irregular fibrotic lung lesions. Eventually, early inflammation with caseous formation presents higher phosphatase activity inside the necrotic foci, which invariably calcify⁹ (Fig. 6).

At histology, the initial reaction presents as an alveolar exudate composed by edema, fibrin, and polymorphonuclear leukocytes. Later, the mononuclear cells replace the former histologic pattern, presenting predominantly as necrotizing and non-necrotizing granulomas. These lesions may become confluent, resulting in the formation of necrotic areas containing the causal agents

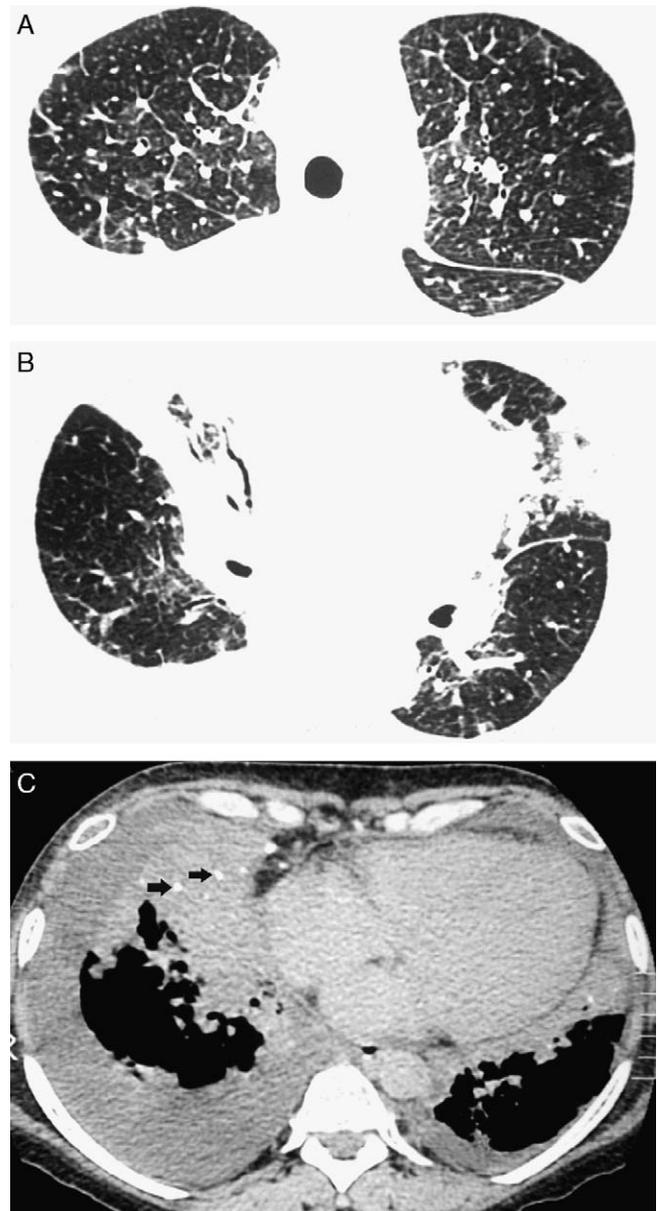


FIGURE 8. A 50-year-old woman with diffuse alveolar septal amyloidosis. A, High-resolution scan (parenchymal window) through upper lung zone shows interlobular septal thickening and small nodules. B, A computed tomography scan through lower zones shows paramediastinal consolidations with air bronchograms. C, A soft-tissue window shows that the consolidations contain punctate calcifications (arrows). Note bilateral pleural effusion.

TABLE 1. Consolidations With Diffuse or Focal High Attenuation

Disease	Clinical Findings	Associated Computed Tomography Findings
Metastatic pulmonary calcification	Conditions associated with hypercalcemia (primary and secondary hyperparathyroidism, chronic renal failure, osseous metastases, or multiple myeloma) Patients generally asymptomatic	Centrilobular fluffy ground glass nodular opacities, that contain foci of calcification
Pulmonary alveolar microlithiasis	Clinical-radiologic dissociation Pulmonary alveolar microlithiasis frequently have very extensive pulmonary abnormalities, which are usually not associated to symptoms or physical signs	Calcified vessels in the chest wall Multiple bilateral calcified micronodules Interlobular septal and subpleural calcification
Amiodarone-induced pulmonary toxicity	Patients receiving large doses of amiodarone to prevent cardiac arrhythmias	High density of the liver and/or spleen
Talcosis	Workers exposed to talc during extraction, grinding, packing, and transportation of the product Endovenous administration of talc seen in drug addicts (particularly Ritalin)	Conglomerate masses Panacinar emphysema
Iodinated-oil embolism	Previous history of lymphangiography or hepatocellular cell carcinoma chemoembolization	Deposition of iodinated oil in other localizations: pleural cavity or abdominal organs
Tuberculosis	History of previous tuberculosis or pulmonary infection symptoms Symptoms are most commonly nonspecific, and include fatigue, weakness, anorexia, weight loss, and mild fever	Ill-defined centrilobular branching nodules, assuming the tree-in-bud pattern Cavitation, thin-walled or thick-walled
Silicoproteinosis	Workers exposed to dust with high concentration of silica, especially sandblasters	Fibrotic areas Ill-defined ground-glass centrilobular nodules Lymph node calcifications
Amyloidosis	Progressive dyspnea	Nodular interlobular septal thickening

Criteria for differential diagnosis.

inside them. Larger portions of the pulmonary parenchyma are progressively affected as the disease evolves, being characterized by inflammatory and necrotic lesions. Caseous, necrotic, and fibrotic areas may present further dystrophic calcification.⁴

SILICOPROTEINOSIS

Silicoproteinosis is related to heavy exposure to silica dust, which evolves as a rapidly progressive disease. This abnormality results in air spaces filled by proteinaceous material similar to that seen in idiopathic alveolar proteinosis. The disease develops over a period of a few months to 1 year, in individuals exposed to dust with high concentration of silica, especially sandblasters. These patients had rapidly progressive shortness of breath and presents high rate of mortality in the first year after the onset of symptoms.¹⁰

HRCT of the lung shows ill-defined centrilobular nodules, bilateral ground-glass opacities, and bilateral areas of air-space consolidation. Nodular calcifications are common findings in this subject, usually presenting a punctate pattern within the areas of consolidation¹⁰ (Fig. 7).

The classic pathologic finding is deposition of periodic acid-Schiff–positive proteinaceous material within the air spaces. In contrast to chronic exposure to

silica-containing dust, silicoproteinosis presents minimal collagen deposition and fibrosis.^{4,10}

AMYLOIDOSIS

Pulmonary amyloidosis is a rare disease presenting 3 forms: submucosal deposits in the airways (tracheo-bronchial form), parenchymal nodules (nodular parenchymal form), or diffuse interstitial damage (diffuse parenchymal or alveolar septal form).^{1,3} Diffuse parenchymal amyloidosis is the least common form of this disease, but the most clinically significant, assuming higher association to systemic amyloidosis than to localized one.^{3,4} Patients with this form are prone to die of respiratory failure, and most common symptoms are related to progressive dyspnea.¹¹

HRCT abnormalities consist of abnormal reticular opacities, interlobular septal thickening, multiple small nodules, and air-space consolidation.^{1,3,4} The diffuse parenchymal pattern is mostly nodular, although confluent consolidations may be seen. Basal and peripheral distribution is the dominant aspect.⁴ Lymph node enlargement, and unilateral or bilateral pleural effusions are associated findings.⁴ Nodules and areas of consolidations could show calcifications, some of them with punctate aspect^{3,11} (Fig. 8).

Histologically, amyloid is a proteinaceous material collecting alongside the pulmonary interstitium, the

media of small blood vessels, and the endothelial and epithelial basal membranes, usually assuming a uniform linear or micronodular appearance.¹¹ Foci of calcification inside consolidations and small amyloid nodules can be found in pathologic specimens. Eventually, osseous metaplasia are seen in the calcified areas.^{3,4}

CONCLUSIONS

Air-space consolidations can be seen in a wide variety of diseases affecting the lungs. The identification of consolidation with diffuse or focal high attenuation narrows the differential diagnosis. The most common causes of diffuse hyperdense consolidations are MPC, PAM, amiodarone lung toxicity, talcosis, and deposition of iodinated oil material. Consolidations with punctate calcifications suggest as differential diagnosis tuberculosis, silicoproteinosis, and parenchymal amyloidosis. The association of consolidation with high attenuation, clinical presentation, and additional CT findings could narrow even more the differential diagnosis (Table 1) avoiding, in some cases, invasive procedures, such as pulmonary biopsy.

Consolidations with diffuse or focal high attenuation can result from a variety of different conditions. We have presented a diagnostic approach based on appearance and distribution of these lesions. Despite the limitations, we believe that the proposed diagnostic approach can be helpful in the differential diagnosis of

the various conditions that result in consolidations with high attenuation of the pulmonary parenchyma.

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ERRATA

In the August 2008 issue of *Journal of Thoracic Imaging*, two authors' names were misspelled in the article by Gilkeson et al. The corrected author list follows:

Robert C. Gilkeson, MD, Eric M. Nyberg, MD, Peter B. Sachs, MD, Amanda M. Wiant, MD, Kenneth G. Zahka, MD, and Ernest S. Siwik, MD

1. Gilkeson RC, Nyberg EM, Sachs PB, et al. Systemic to pulmonary venous shunt: imaging findings and clinical presentations. *J Thorac Imaging*. 2008;23:170–177.

The institutions listed for the authors of two of the Award-Winning Abstracts published in the August 2008 issue of *Journal of Thoracic Imaging* were incorrect. The authors of “Radiologic-pathologic correlation of complications post-lung transplantation” are from the University of Texas Health Science Center at San Antonio, while the authors of “The diaphragm at MDCT: covering the thoracic outlet” are from the University of Cincinnati.

The Society of Thoracic Radiology apologizes for the error.

1. Oral Scientific & Educational Abstract Winners. Annual Meeting and Post-graduate Course, Fort Myers, Florida, March 2–5, 2008. *J Thorac Imaging*. 2008;23:227–231.