Differential Diagnosis in diffuse pulmonary calcifications.

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Learning objectives

To be aware of different causes of diffuse pulmonary calcifications.

Background

Diffuse pulmonary calcifications occur in a wide variety of disorders. Most often these are caused by previous infectious processes. Other known causes are metabolic disorders, occupational disorders or interstitial lung diseases (1). Specific symptoms are often lacking, but calcification may be a marker of disease severity and its chronicity. Pathophysiologic states predisposing to pulmonary calcification and ossification include hypercalcemia, a local alkaline environment, and previous lung injury. Factors such as enhanced alkaline phosphatase activity, active angiogenesis, and mitogenic effects of growth factors may also contribute (2).

Diffuse pulmonary calcifications are divided into 2 types, based on their underlying mechanism. The first type is the dystrophic calcification, caused by deposits of calcium salts into abnormal tissue (altered by necrosis or scarring). The second type is metastatic calcification, which is less common and occurs in the presence of serum mineral ion imbalance.

Metastatic pulmonary calcification is further subdivided into benign and malignant causes.
Two physiologic mechanisms are required to generate metastatic pulmonary calcifications: the release of excess calcium salts from bone and the transport of these calcium salts through the circulation.

In contrast to metastatic calcification, dystrophic pulmonary calcification occurs in sites of previous cellular and tissue injury. Calcium is an important ion for cellular integrity, and an increase in intracellular calcium may contribute to the injury (1).

Findings and procedure details

Imaging

The chest radiograph is useful for the detection of pleural calcification, hilar-mediastinal lymph node calcification or calcified lung nodules. However, diffuse calcification is often mistaken for another process such as pulmonary edema or intrapulmonary hemorrhage, as it appears as a non-specific infiltrate.
High-resolution CT (HRCT) is the modality of choice to detect and evaluate these calcifications (2).

**Cases**

We present one case of metastatic pulmonary calcification and five cases of dystrophic pulmonary calcification.

*Metastatic pulmonary calcification*

Metastatic pulmonary calcification (MPC) occurs in disease states with concurrent derangement of calcium and phosphorus metabolism. It is most commonly seen in chronic renal failure. Metastatic pulmonary calcification is characterized by diffuse calcium deposition in the lung. Pathologically, it is an interstitial process with calcium deposited in the alveolar septa and bronchial walls and, to a lesser extent, in bronchioles and pulmonary arterioles. Calcium deposition can lead to interstitial fibrosis in severe cases and can result in respiratory insufficiency.

Three patterns have been described in metastatic pulmonary calcification on HRCT. The first pattern is multiple diffuse calcified nodules that are either distributed throughout the whole lung or show a predilection for the apices or the bases (Fig. 1 on page 5). The second pattern is diffuse or patchy areas of ground-glass opacity or consolidation. Finally, MPC may appear as a confluent high attenuation parenchymal consolidation in a predominantly lobar distribution, mimicking lobar pneumonia. Several authors have also described calcification in the bronchial walls, myocardium and within the vessels of the chest wall (3,4).

*Varicella pneumonia*

Varicella (chickenpox) is a highly contagious viral illness transmitted by respiratory droplets. The incidence of primary varicella pneumonia increases significantly with age and peaks in the 3rd-5th decade. Histologically, the varicella pneumonia consists of an interstitial inflammatory infiltrate with an intra-alveolar proteinaceous exudate, edema and hemorrhage. Late development of tiny, widespread, micronodular calcifications through both lungs is an uncommon sequela of varicella pneumonia (Fig. 2 on page 6, Fig. 3 on page 7). There are no associated calcified lymph nodes, as opposed to calcifications secondary to tuberculosis or histoplasmosis (5).

*Silicosis*
Silicosis is caused by inhalation of free silica particles, usually during occupational exposure such as mining, sandblasting, and masonry.

HRCT findings of silicosis and coal workers' pneumoconiosis include diffuse and randomly distributed, small, well-defined nodules that are most prominent in the middle and upper lung zones. The nodules usually measure less than 5 mm in diameter and may calcify. Complicated silicosis is characterized by development of large masses, produced by coalescence of these small nodules (Fig. 4 on page 8).

Lymph node involvement with or without calcification is prominent in silicosis. So-called "eggshell calcification" occurs in 3-6% of miners with silicosis (Fig. 5 on page 9). For diagnosis, peripheral solid or broken calcification up to 2 mm thick must be present in two or more nodes >1 cm in diameter. At least one ring shadow must be complete. "Eggshell calcification" has also been described in sarcoidosis, Hodgkin's disease after radiation, blastomycosis, histoplasmosis, scleroderma, and amyloidosis (1,5,6).

**Amyloidosis**

Amyloidosis is a systemic disease caused by extracellular accumulation of amyloid. It can be idiopathic (primary form) or associated with various inflammatory, hereditary, or neoplastic pathogeneses (secondary or reactive form). Pulmonary amyloidosis may be part of a widespread process that involves many organs, or it may be localized to the airways and lung parenchyma.

Primary pulmonary amyloidosis is a localized form of amyloidosis that is confined to the lung parenchyma. Unlike systemic amyloidosis, localized pulmonary amyloidosis usually follows a benign course. It can occur in three forms: diffuse interstitial deposits, single or multiple pulmonary nodules, or, most commonly, submucosal tracheobronchial deposits (Fig. 6 on page 10, Fig. 7 on page 11). Nodular pulmonary amyloidosis is characterized by multiple round or oval, sharply defined areas of increased opacity of variable size and number. Approximately 50% of the nodules calcify or ossify (1,5).

**Disseminated pulmonary ossification**

Disseminated pulmonary ossification is a chronic process characterized by progressive, metaplastic ossification. It affects the pulmonary interstitium or alveolar spaces and is associated with chronic, diffuse pulmonary or systemic pathology. There are 2 forms of pulmonary ossification: dendriform pulmonary ossification (DPO) and nodular pulmonary ossification (NPO). Of the 2, DPO is the less common, and it can be seen in patients with chronic pulmonary disease and usual interstitial pneumonitis. Dendriform is the preferred term because of the delicate, dendritic, branching appearance of the metaplastic
(heterotopic) mature bone and striking radiographic appearance that distinguishes it from other forms of pulmonary ossification. Dendriform pulmonary ossification preferentially affects the alveolar interstitium, expanding the alveolar septa rather than the alveolar spaces (Fig. 8 on page 12, Fig. 9 on page 13). In contrast, NPO affects the alveolar spaces, preferentially the lower lobes, and is encountered in the clinical setting of passive congestion, such as in mitral valve stenosis (1,7).

**Pulmonary alveolar microlithiasis**

Pulmonary alveolar microlithiasis (PAM) is a rare disease, characterized by presence of diffuse innumerable, minute calculi called microlithiasis in the alveoli of the lungs. The mutation in SLC34A2 gene that encodes a sodium-phosphate co-transporter in alveolar type II cells resulting in the accumulation and forming of microlithiasis rich in calcium phosphate (due to impaired clearance) are considered to be the cause of the disease. A hallmark of PAM is a striking dissociation between the radiologic findings and the only mild clinical signs and symptoms.

CT demonstrates diffuse, ground-glass increased attenuation throughout the lungs. The calcifications are more dense and numerous along the bronchovascular bundles and in the subpleural regions (Fig. 10 on page 14). With progression of the disease, interstitial fibrosis and subpleural cysts may be found. Differentiation from other forms of pulmonary calcifications is based on the characteristic radiographic features and the paucity of clinical symptoms relative to the massive pulmonary involvement (5,8).

**Images for this section:**
Fig. 1: Metastatic pulmonary calcification. Axial CT scan image chest shows centrilobular ground-glass nodules and several small calcified nodules. The patient was known with chronic renal impairment and secondary hyperparathyroidism.
Fig. 2: Healed varicella pneumonia. Coronal MIP reconstruction image shows numerous bilateral small nodules. Nodules are smoothly marginated and sharply defined.
Fig. 3: Healed varicella pneumonia. Axial image of the same patient demonstrates no associated parenchymal or interstitial abnormalities.
Fig. 4: Silicosis. Axial CT scan image obtained using bone window settings shows a conglomerate mass of fibrosis containing multiple calcified small nodules.
**Fig. 5:** Silicosis. Axial MIP reconstruction image of the same patient using bone windows setting demonstrates mediastinal egg-shell calcifications, typical for silicosis.
Fig. 6: Amyloidosis. Coronal MIP reconstruction image depicts multiple small nodular opacities scattered through lungs, containing several calcified foci, and confluent subpleural opacities.
Fig. 7: Amyloidosis. Axial high-resolution image in lung window setting of the same patient demonstrates thickening of several bronchi, suggestive for tracheobronchial involvement.
**Fig. 8:** Disseminated pulmonary ossification. Coronal MIP reconstruction depicts diffuse subpleural calcifications, representing dendritic calcification.
Fig. 9: Disseminated pulmonary ossification. Axial image with lung window setting of the same patient shows subpleural honeycombing, characteristic for fibrosis.
Fig. 10: Pulmonary alveolar microlithiasis. Coronal MIP reconstruction image depicts a diffuse bilateral calcified fine nodular pattern, predominantly along the bronchovascular bundles.
Conclusion

We presented in short the most common causes of diffuse pulmonary calcification. This pattern can result from a variety of different conditions. These conditions all have a different presentation. A diagnostic approach is based on the patient's history and associated intrathoracic findings. A better understanding of these can lead to a more confident and specific diagnosis without the need for a open lung biopsy.

Personal information

References


