Occupational thorax disease: a pictorial review

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Learning objectives

- To describe pathophysiological mechanisms in expositional diseases of the thorax, particularly pneumoconiosis
- To illustrate the most frequent imaging findings in pneumoconiosis and some industrial exposure diseases.
- Show the importance of knowing the patient's exposure history
Background

Exposure changes of the thorax derived from organic or inorganic elements conditions the development of changes in the architecture of the pulmonary parenchyma, with the presence of local or generalized inflammatory phenomena from which fibrotic changes occur, with deterioration of respiratory function.

Pneumoconiosis is a group of diseases of very high prevalence in the world; in particular, developing countries is a source of public health concern for widespread exposure and irregular prevention measures. Radiological manifestations are broad and varied, and it is essential to have access to the patient's exposure data.

The objective of this work is to illustrate the different characteristic imaging features described in the literature on pneumoconiosis, such as silicosis, asbestos disease and less common ones such as exposure to talcum powder, iron dust or heavy metals.
Findings and procedure details

**Pneumoconiosis** can be classified into two large groups according to whether they are **fibrotic** or **non-fibrotic**. (Fig 1)

**Silicosis** is the disease derived from exposure to silica (SiO$_2$) material found mainly in the glass industry, underground and open-pit mining, and in sand quarries. In the world around 2.3 billion people are exposed to this material.

The development of the disease occurs after there is access of the particle to the small airway, phagocytosis and accumulation of charged macrophages in the pulmonary interstitium, with the onset of inflammation and parenchymal destruction and stimulus for fibrous degeneration (Fig 2).

Three forms of clinical-imaging presentation are described, each with variable exposure times and clinical manifestations that are subject to them (Fig 3).

- **Silicoproteinosis**: type of manifestation with short exposure times (weeks - months), presents accelerated respiratory deterioration, even respiratory failure, and imaging findings similar to alveolar proteinosis and ARDS. Areas of confluent alveolar occupation are observed in the imaging studies. It is not uncommon to observe peripheral areas in ground-glass due to the partial occupation of the alveoli of surfactant protein and eosinophilic exudate (Fig 4).

- **Accelerated silicosis**: mean exposure time between 5 to 10 years, with clinical manifestations one year after exposure, may evolve towards respiratory failure (Fig 6, 7).

- **Chronic silicosis**: exposure times greater than 10 years are initially asymptomatic, however with the development of the disease will appear dyspnea, pulmonary hypertension, emphysema and fibrosis (Fig 9). There is a 3-fold increased risk of over-aggregated TB infection (Silicotuberculosis).

In accelerated and chronic silicosis, there may be a spectrum of manifestations that are subclassified in **simple** when the presence of nodules between 1 - 10 mm is identified, which are distributed diffusely in the upper and posterior areas of both lungs; Up to 20% of them are calcified (Fig 5). The second group **complicated** silicosis also known as **progressive massive fibrosis**, which is characterized by the confluence of silicotic nodules with parenchymal retraction and mass formation that appear predominantly in upper
lobes, with irregular edges (in "angel wings"), which can have punctate calcifications, and that associate peripheral emphysema, in addition to signs of loss of lung volume. In both cases reactive hilar lymphadenopathy manifested as calcified lymph nodes in "egg shell" can be observed (Fig 8).

Special mention of the **coalworker’s pneumoconiosis**, recognized disease of physiopathology similar to silicosis, even coexisting both, in patients exposed in coal mines; it is characterized by observing the presence of multiple small nodules, distributed in both lung parenchyma. The confluence of the nodules can be observed with the presence of opacities like-mass. There is an atypical presentation showing the presence of basal reticular opacities with traction bronchiectasis, and sometimes micronodules, findings similar to asbestosis (Fig 10).

In the **disease due to exposure to Asbestos**, two types of pathogenic fiber, the amphiboles and the serpentines, are recognized, being the first of the most carcinogenic potential. They are found in cement industry compounds, vehicle brakes and construction materials in developing countries. Around 8-9 million people are exposed in the United States. In Latin America there are no precise records of its epidemiology but it could exceed this figure.

Once the asbestos fibers are deposited, they interact with type I pneumocytes, there is translocation towards the interstitium, which triggers an inflammatory response that leads to alveolitis and fibrotic changes of the parenchyma. The average exposure time is around 10 - 20 years. It is recognized that the affectation of the pleura is due to the directed lymphatic drainage or penetration of the fibers towards this (Fig 11).

The disease by exposure to asbestos has a spectrum of manifestations depending on the anatomical commitment, although it is not uncommon to find coexistence of both; In the pleura **benign pleural disease** and **mesothelioma** can be recognized. In the pulmonary parenchyma **Asbestosis**.

**Benign pleural disease** manifests as generally **pleural plaques**, circumscribed focal areas of pleural thickening, usually bilateral, although it may be asymmetric. They appear in posterior and lateral regions of both hemithorax, even adjacent to the diaphragmatic dome. Up to 15% are calcified. Other forms of manifestation are diffuse pleural thickening and pleural effusion (Fig 14).
Pleural thickening is observed in up to 22% of patients exposed to asbestos, is characterized by compromising up to a quarter of the chest wall and is predominantly smooth. The pleural effusion in turn affects not more than 3% of exposed patients, being the most common manifestation in the first 20 years of exposure, recurrent and sometimes bilateral.

If there is primary malignant pleural involvement secondary to asbestos exposure, it is known as mesothelioma; manifest as nodular pleural thickenings with extension towards the fissures; it can coexist with unilateral pleural effusion (90%) and even loss of lung parenchyma volume (40%) (Fig 15, 16).

The compromise of the lung parenchyma or Asbestosis is defined as interstitial fibrosis of the pulmonary parenchyma with evidence of the presence of asbestos fibers in the pathological study or expositional antecedent; No findings are found on chest radiographs until at least 15 years after exposure, even with latency periods reaching 40 years. The characteristic finding is the presence of fibrotic changes in the lung parenchyma that is observed as reticular or reticulonodular opacities on chest radiography, or changes of fibrosis on high resolution chest tomography; The coexistence of pleural manifestations such as those described inclines towards the diagnosis of asbestosis. Depending on the severity of the disease, it is possible to find architectural distortion, traction bronchiectasis, subpleural lines, and honeycomb (Fig 12, 13).

Other findings in the pulmonary parenchyma secondary to exposure to asbestos include round atelectasis, the presence of opacity like-mass with pleural folding and vascular structures and bronchial tubes converging towards it. It is associated with pleural disease and should be differentiated from malignant lesions, with an increased incidence in exposed to asbestos.

Other types of pneumoconiosis are more rare and their diagnostic imaging approach will depend on epidemiological data such as work area and exposure to organic or inorganic dusts. Some examples to show: talcosis secondary to exposure to talcum powder in ceramic industry (Fig 17), siderosis in patients exposed to iron dust in factories of the steel industry (Fig 18). Various heavy metals found in work environments such as tungsten, carbon, cobalt, titanium, tantalum and nickel may be associated with changes in the pulmonary architecture by chronic exposure (Fig 19).
Fig. 1: Pneumoconiosis classification. Fibrotic vs. Non-fibrotic

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Fig. 2: Silicosis pathophysiology.

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Fig. 3: Silicosis classification.

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Fig. 4: Chest x-ray shows poorly defined alveolar opacities in both parenchyma, more confluent on the left side. Thoracic tomography, peribroncovascular alveolar occupation areas, air bronchogram, and peripheral ground-glass opacity zones. Pathology sample with occupation of alveoli by surfactant protein.

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**Fig. 5:** Chest X-ray shows poorly defined nodular opacities in both lung parenchyma, thickening of the right paratracheal stripe probably mediastinal lymph nodes. Thoracic tomography in mediastinum and lung reconstruction demonstrates right and subcarinal hilar calcified adenomegalies; multiple micronodules disseminated in both parenchymas, not confluent.

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Fig. 6: Chest X-ray shows signs of volume loss, poorly defined reticulonodular opacities, and opacities like-mass in both upper lobes with retraction of pulmonary hila. There are no pleural effusions. Thoracic tomography, multiple confluent micronodules with round calcifications and areas of adjacent emphysema. Pleuroparenchymal bands without pleural thickening.

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Fig. 7: Thoracic tomography, multiple punctate nodules, some with calcium attenuation, confluent, slight signs of parenchymal retraction. Histopathological cut under polarized light shows crystals of silica.

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**Fig. 8:** Chest x-ray and coronal reconstruction of thorax tomography show reticulonodular opacities and dense confluent opacities in both pulmonary apices (progressive massive fibrosis), with retraction of pulmonary hilums and signs of volume loss. In axial reconstruction, multiple nodules with calcium attenuation and reactive hilar lymphadenopathy (lymph nodes in "egg shell")

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Fig. 9: Confluent opacities like-mass in both lung parenchyma with signs of volume loss (progressive massive fibrosis). Histopathological section of silicotic nodule.

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**Fig. 10:** Chest x-ray, ill-defined reticular opacities in both parenchyma. Thoracic tomography, multiple small nodules disseminated, not confluent, not calcified.

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Fig. 11: Asbestos exposure disease pathophysiology

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Fig. 12: Distortion of the architecture of both lung parenchyma and loss of volume, fine intralobular reticulation and areas of bilateral posterior smooth pleural thickening.

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Fig. 13: Loss of volume and opacities of alveolar occupation poorly defined in left upper lobe, there are no pleural effusions. Thoracic tomography shows distortion of parenchymal architecture with fine intralobular reticulum, traction bronchiolectasis and subpleural bands. Greater commitment of the left parenchyma. Poorly defined areas of ground-glass opacity. Histopathological section showing asbestos fibers.

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**Fig. 14:** Pleural plaques some of which are calcified. Diffuse pleural thickening.

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Fig. 15: Irregular pleural thickening in the left hemithorax, with enhancement after administration of iodinated contrast. Pattern of nodular extension towards the fissures. Discrete pulmonary architecture distortion in left pulmonary parenchyma.

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Fig. 16: Irregular pleural thickening of the base of the right hemitorax, with extension towards fissures. There are no pleural effusions.

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Fig. 17: Poorly defined micronodules, diffusely distributed in both lung parenchyma.

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Fig. 18: Reticulonodular opacities, without clear signs of volume loss, there is no pleural effusion. Tomography with multiple non-confluent nodules in both parenchyma. There is no mediastinal adenomegaly. Histopathological prussian blue stain demonstrating iron deposit.

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**Fig. 19:** Chest x-ray shows thick reticular opacities in the middle lobe and lingula. There are no pleural effusions. Thoracic tomography in lung window reconstruction, architectural distortion, with traction bronchiectasis and consolidation in the middle lobe.

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Conclusion

Pneumoconiosis is a group of pathologies derived from exposure to materials that condition changes in lung architecture and deterioration in respiratory function. There are findings indicative of the type of etiology responsible for lung involvement and it is the radiologist's responsibility to know and describe them.
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References