

# PATHOLOGY

☒ Sheet

☐ Slide

☐ Handout

Number

6

Subject

**Pneumoconiosis**

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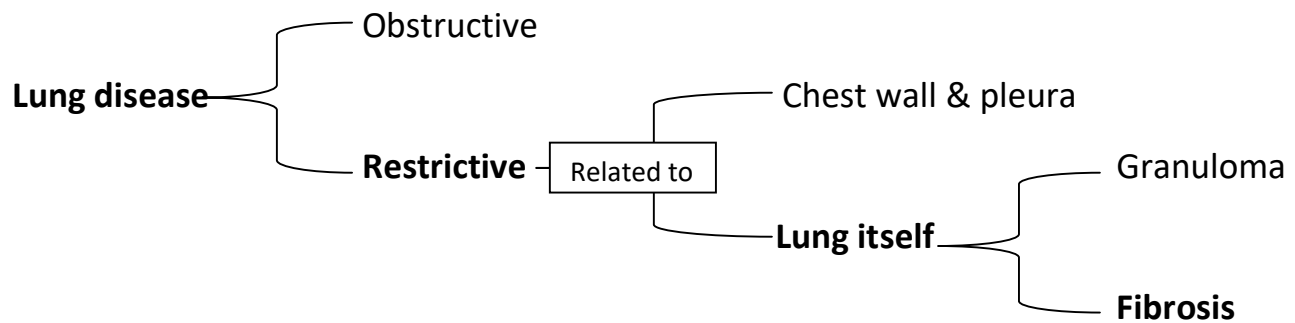
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# Pneumoconiosis

## Introduction



Lung fibrosis can be due to inhalation of certain mineral dust ( such as coal dust , silica & asbestos ) , as they accumulate in the lung & cause lung fibrosis

The accumulation of this mineral dust usually happens in the **occupational sitting**

NOT any dust that we inhale will cause fibrosis in the lung , as there are certain factors that determine if this dust will cause fibrosis in the lung or not

\*\*\* Factors that affect the reaction of the lung to mineral dust:

### 1. Physical factors

**Size :**

>>> certain size to be lodged inside the alveoli

if it is very small ( tiny ) >> soluble >> so enter & leave the alveoli >> No problem

if it **between ( 1-5 )  $\mu\text{m}$**  >> particles can accumulate in the interstitium >> very dangerous size

if it is larger >> sweep by mucus >> go out with cough >> No problem

**Shape :**

As it makes the particle adhesive or not ( we can sweep certain particles while other particles may be stiff & hard to be swept by cilia so they go down to alveoli)

**Solubility :**

If it is not soluble then it might cause problems

**Purity :**

Pure form of silica are more dangerous than silica within mixture of particles

**2. Chemical factors****Reactivity** (Inert vs reactive) :

Coal is inert while silica & asbestos are more reactive so they cause more damage

**3. Host factors**

Certain polymorphism that has no relation with particles

Additional 3 factors :

- 1. Duration:** as 1 year exposure differ from 5 years exposure
- 2. Concentration:** so they happen in occupational siting
- 3. Efficacy of the machinery that removes the particles** as smoking will affect it & so affect the reaction of the lung to the mineral dust!!  
(You remember immotile cilia / Kartagener syndrome)

## Pathogenesis

Now , how these particles enter the lungs & cause fibrosis !!

Simply :

Inflammation → Damage → Repair → Fibrosis

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Particles taken by macrophages >>> active macrophages >>> this macrophages can cause inflammation & fibrosis

There is 2 types of macrophages !! , which one play a role here ??

M1 → for inflammation

M2 → for fibrosis

So both types will play a role , **But to have a restrictive lung disease & fibrosis , we need M2 & production of TGF-β & the rest of fibrogenic molecules** .

But , How macrophages recognize the presence of potentially harmful agents ??

They **express inflammasome receptors** that designed to sense the presence of these agents .

## Types

- ✓ Coal Worker's Pneumoconiosis
- ✓ Silicosis
- ✓ Asbestosis

## Coal Worker's Pneumoconiosis

Coal dust is relatively **inert** & it is mainly carbon .



Inert >> so NOT easy for fibrosis to develop

Inert >> neither affects the DNA , nor causes mutations so

**NO association between coal & cancer ☺**



At the beginning patient has only accumulation of this carbon pigment → **Black lung ( we call it anthracosis )**

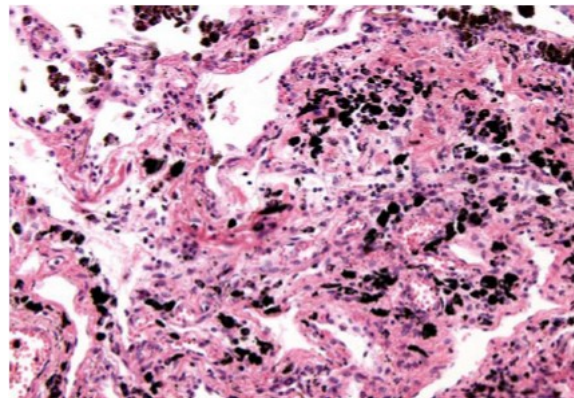
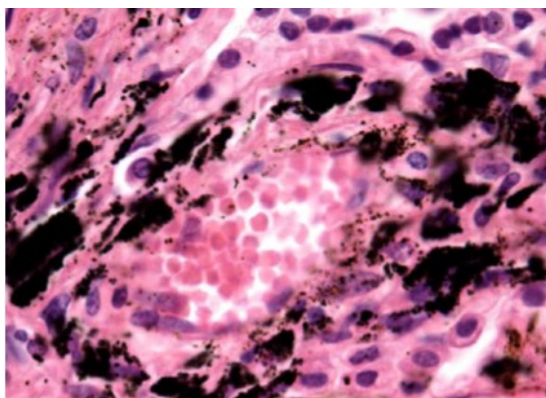
So anthracosis mean accumulation of carbon pigment within the macrophages in the alveoli & interstitium **without fibrosis** >>> so its asymptomatic step

Q : What's the other things that might cause black lung ??

Smoking & air pollution >>> so all of us have a degree of this blackness of the lung

This step needs very long time to become fibrogenic & so lead to fibrosis !!

Under the light microscope we see only **black pigment**



With time this carbon pigment will increase , collected in **macules**- if larger called nodules- & so **minimal collagen fibers ( minimal fibrosis )** will develop

→ we call this stage : **Simple coal workers pneumoconiosis** ( CWP )

From slides : -Centrilobular emphysema can complicate these macules

-These are found mainly in the **upper lung lobes**

If the fibrosis increases a lot , which takes many years then → ONLY 10% of CWP will progress to **Complicated CWP** & here there is extensive fibrosis (**progressive massive fibrosis** (PMF) )

Progressive >>> so even if you stop the exposure , it will progress as we reach a stage of **NO return !!**

The term **progressive massive fibrosis** refer to any end stage in the conditions that we will discuss (Silicosis, Asbestosis , Coal Worker's Pneumoconiosis )

So when we reach the stage of extensive fibrosis then this means >> **progressive massive fibrosis** whatever the cause is .

From slides : Complicated CWP Histologically : • There is coalescence of coal nodules .  
• Multiple black scars

So , simply there is 3 stages here :

Asymptomatic anthracosis → Simple CWP → Complicated CWP

From slides : Simple CWP > little effect on lung function , while the complicated one causes dysfunction , pulmonary hypertension and cor pulmonale.

## Silicosis

Silica is found in different forms in nature such as quart and powder .And is used in sand blasting ( السنفرة ) and many different uses .

### **Silicosis is most prevalent chronic occupational disease in the world.**

When silica accumulates in the lung of the individuals who work in occupations that uses silica they will develop fibrosis in the lungs called SILICOSIS , as an example glass manufacturing, because actually glass is made from sand that contains this material .

- Silica is found in nature in two forms :
  1. Amorphous silica, which is more soluble .
  2. Crystallized silica (quartz) , tiny particles, crystals at the particle level .

Which one of them is more fibrogenic ??

**The Crystalline or the Quartz is more fibrogenic** , especially when it is very **pure** Quartz .

SO , when a person develops silicosis he will have *fibrosis* and **nodules** that contain fibrous tissue that looks like onions.

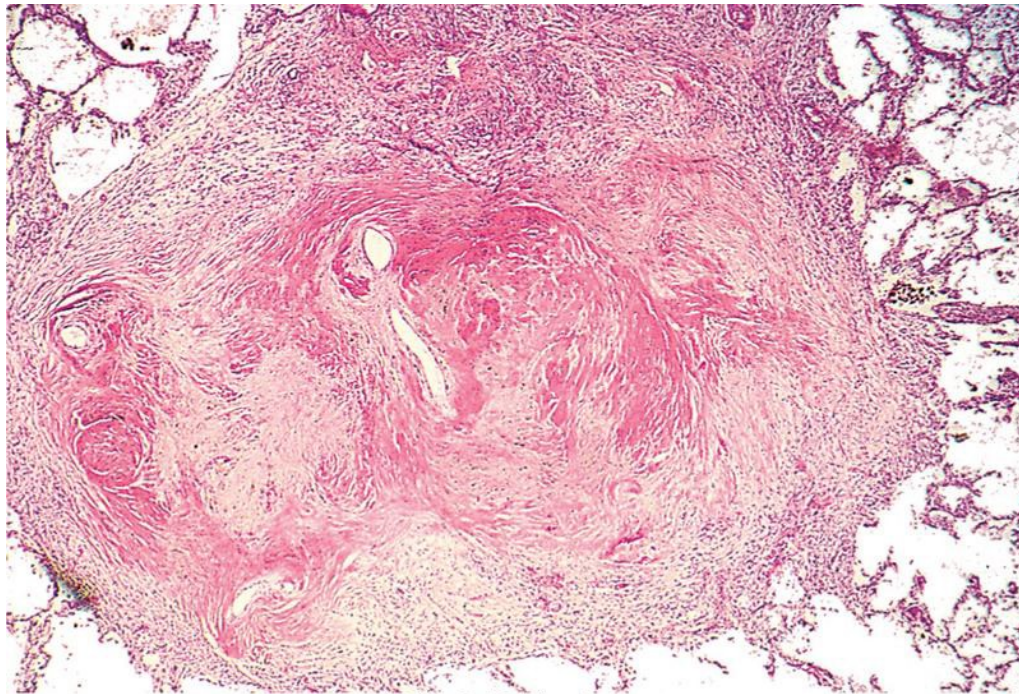
From slides : nodules in the **upper zones of the lungs**.

Histologically : look at picture in the next page :-

There are **concentrically arranged hyalinized collagen fibers surrounding amorphous center** ,and this arrangement is characteristic of silicosis that we can't see in other forms of fibrosis .

And notice that we can't see pigments here , because the patient is not working with coal or a smoker . But if there is some association to coal or smoking we can see pigments with this features .



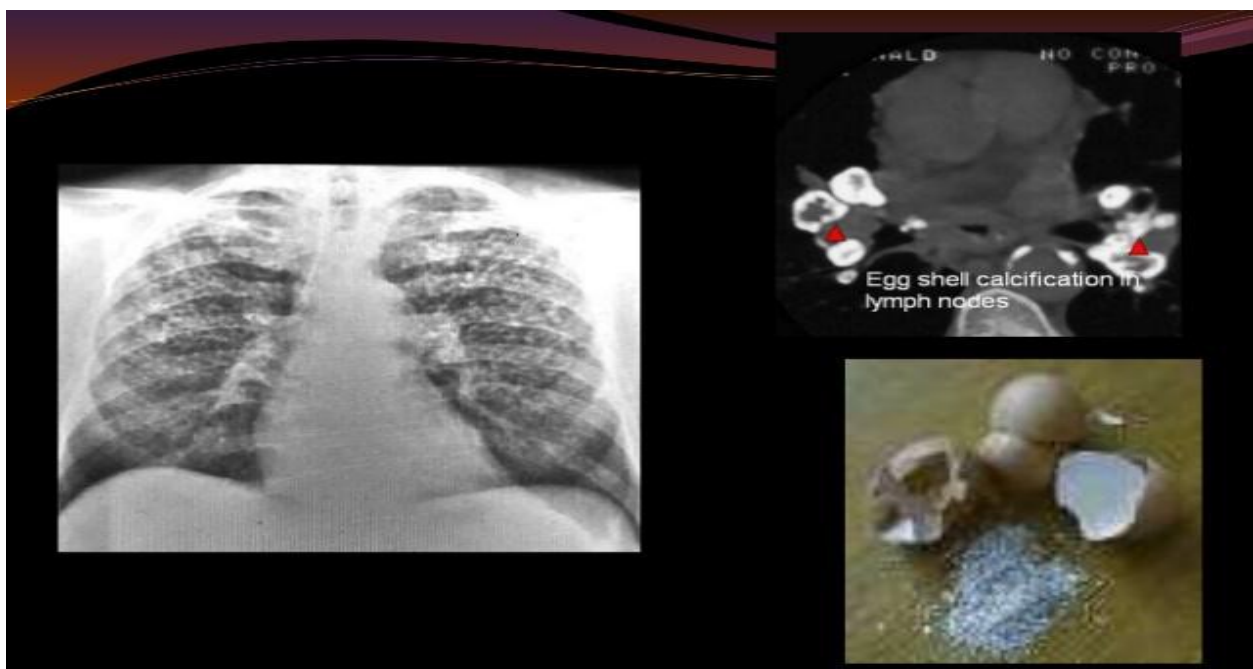


Kumar et al: Robbins Basic Pathology, 9e.  
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If we do a chest X-ray to a silicosis patient we can see infiltrates in the lungs that are fibrotic, and sometimes we can see Egg shell calcification (its white color calcification and empty in the middle)



Egg shell calcification is also seen in other conditions, but in lungs it mainly indicates silicosis.





**IMPORTANT :** there is association between silicosis and cancer, keep in mind that we said that coal doesn't has, so silicosis increases the incidence of lung carcinomas .

**SILICA also increases the risk of TB**, they are more susceptible to develop TB more than normal individuals . WHY???

Because silica particles when they reach the Macrophages they decrease their ability to respond and recognize TB Bacilli , so **less immunity to fight TB** .

From slides : Nodules of silicotuberculosis often contain a central zone of **caseation**.

What is the receptor that recognizes the particles on the macrophages ??

Inflammosome .

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### Asbestosis

Asbestos is used a lot in houses and many areas around us , because it is heat resistant and used for insulation .

**Asbestosis** , is fibrotic lung disease related to exposure to Asbestos , and is **strongly associated with cancer** .



Asbestos is found in two forms :

1. **Amphiboles** , they are straight and brittle crystals ,not soluble .
2. Serpentine , they are Curly , flexible , soluble crystals .

Which is **more dangerous** ? more fibrogenic ?

**Amphiboles** because they are not soluble and straight .

From slides : **In contrast with CWP and silicosis, asbestosis begins in the lower lobes and subpleurally**

We said these diseases are related to occupational settings , but here people could have asbestosis by not working with it , people living in old houses can develop it . And actually if one member of the family works in asbestos industry ,his family members have increase risk of developing the disease or the cancers related to .



Most cases of Mesotheliomas (nearly 100% of them) are related To asbestos .

Which is the most common of the all disease we have mentioned ??

Silicosis , because there is more silica industry and more glass industry .

Coal waaaaas the most common but nowadays no , and for Asbestos now they are using different substances for insulation and heat resistant so its reducing .

Asbestos harms the pleura, causing plural fibrosis “plural plates” that’s leads to plural effusion .

And can cause plural tumor which is “ plural mesothelioma ” and in any other places it can cause mesothelioma (in peritoneum ,pericardium) . that can cause restrictive lung disease and affects lung function even if there is no lung fibrosis.

In the lung it causes fibrosis which is “ Asbestosis “ , lung Carcinoma and can cause Nasopharyngeal and laryngeal carcinoma .

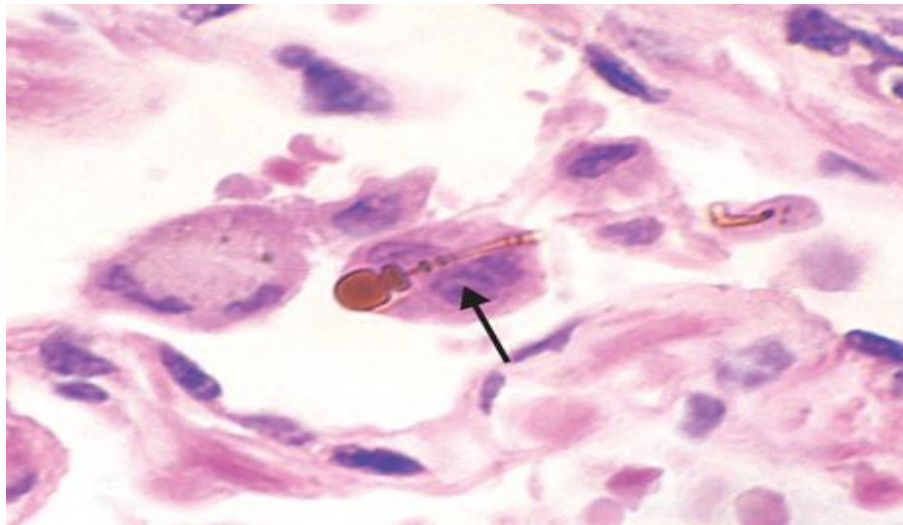
Pleural plaques are most common with asbestosis patients , it can occur in other situations but rarely . ( shown in the picture below).



If we have a patient and he has shown to have fibrosis , how to know that if he has asbestosis or not??

**If he had Asbestos bodies most likely he developed asbestosis.** These bodies are fibers around them there is brown color which is Hemosiderin, hemosiderin coats the fibers and gives this appearance of asbestos bodies.

▲ Notice that these bodies could be found in normal individuals, however, the load or the concentration of them is low and not associated with fibrosis (if they are not related to heavy asbestos). And these **appear in the lungs not in pleura.**



So, coal does not cause cancer, Silica does, and Asbestosis related to many cancers.

IF a patient is a smoker and he deals with asbestos, HE is at risk of developing what disease??

Lung cancer but not mesothelioma.

**So smoking is not related to mesothelioma .**

Sorry for any mistakes.