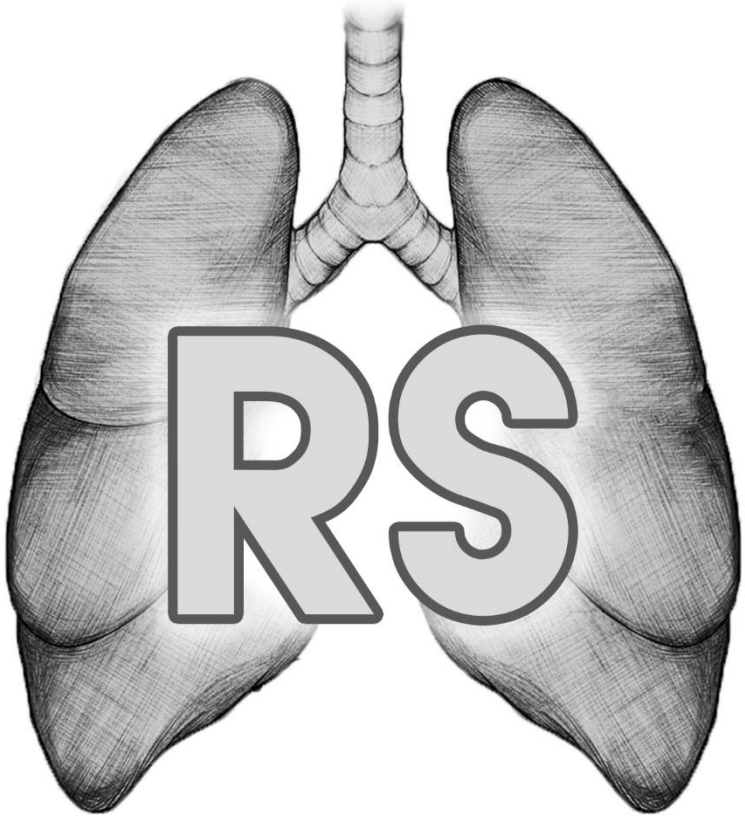

PATHOLOGY

☑ Sheet ○ Slide ○ Handout

Number

4

Subject

Bronchiectasis, Bronchial Asthma

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Date: 1/12/2016

Price:

بسم الله الرحمن الرحيم

Salam … this sheet was written according to section 2 record, and I tried to do my best to make everything clear.

Last lecture we talked about Obstructive lung diseases and restrictive lung diseases.

obstructive diseases are:

1) COPD, with both types emphysema and chronic bronchitis. (we talked about them).

2) Bronchiectasis

3) Bronchial asthma

- **BRONCHIACTESIS:** (توسع القصبات الهوائية)

The name has two parts:

bronch :it is in the bronchi

iachtesis :it means dilation , so as the name implies , it is dilatation of the large air ways .

Definition : it is a permanent irreversible dilatation of the bronchi associated with infection – *so there should be an infection in this disease* - .

Pathogenesis: -

We said before that Bronchiectasis is a secondary disease not primary one, usually to an obstructive disease, as an example it is secondary to Chronic Bronchitis. ***So it caused by Obstruction and infection*** (don't forget).

- A student QUESTION: how did the dilatation occur?

Answer: the dilatation is secondary to destruction followed by Infection.

- What occurs first? Obstruction or Infection?

In some cases, the infection occurs first, but in other cases obstruction occurs first and one of them can lead to other one. so (نرجع لقصة البيضة والجاجة مين فيهم أجا اول محدش بعرف): ***P***
HOWEVER WE NEED BOTH (Infection and obstruction)

- What type of infection do we need to develop bronchiectasis?

We need an infection that lead to DESTRUCTION of the wall in order to develop dilation of the bronchi, so it should be **necrotizing invasive infection** →→→necrosis of the wall →→ the wall becomes weak →→ dilatation occurs.

- So what diseases predisposes to bronchiectasis, in other words what diseases increases the risk of developing bronchiectasis?

ANYTHING THAT LEADS TO INFECTION OR OBSTRUCTION. Examples:

1. **COPD**
2. **Cystic fibrosis**, it increases the mucus secretion →→thick mucus layer →increases the susceptibility to infection and obstruction.
3. **Bronchial asthma**
4. **Kartagener syndrome**: it is a syndrome characterized by immobility of the cilia → susceptibility to infection increases because of inability to get rid of the mucus and foreign bodies because the cilia is not moving. This syndrome also affect sperms, so the patient become sterile.
5. **Pneumonia**, but it should be SEVERE and NECROTIZING.

NOTE: we don't talk about ACUTE diseases (like pulmonary edema) that causes infection, the disease needs something chronic and continues to cause destruction.

6. **Tumors, causes** obstruction →**bronchiectasis**
7. **TB,** its necrotizing chronic infection**.**
8. **Smoking.**
9. **Immunodeficiency,** with both types acquired or genetic that can predispose to severe infections**.**

SO, to remember anything that causes obstruction or infection can predisposes to this precious cycle leads to BRONCHIACTEISIS.

Symptoms:

1. Productive cough with sputum.
2. Large thoracic wall as a result of the dilatation of the air ways.
3. Hemoptysis THE MOST IMPORTANT symptom, the disease is caused by destruction of the wall and necrosis to the capillary near it so he will cough blood.
4. Clubbing of the fingers may develop

The patient will come to you with a history of COPD, suddenly started coughing with blood !! so you have to think about bronchiectasis, cancer and TB,

BUT if he has long history and SUDDENLY developed hemoptysis usually he is having bronchiectasis or Cancer!

NOW we will discuss the last disease in obstructive lung diseases

- **BRONCHIAL ASTHMA:**

We have types of BRONCHIAL ASTHMA and we will talk about each one of them:

1) **Atopic asthma,** related to allergy**.** (immunologic reaction) -most common-.
2) **Non-atopic asthma,** not related to allergy**,** and it is subdivided in to:
    a) viral induced
    b) drug induced
    c) due to occupational inhalation of certain substances.

**pathogenesis of Atopic asthma:

Its type 1 hypersensitivity reaction

the first cell to be stimulated by an allergen is T– helper 2 cell, when it is stimulated it releases IL-4,5,13.

IL-4 →causes the B-cells and plasma cells to produce and release IgE.

IL-5 →activates EOSINOPHILES which are the most important in allergic rxn.

IL-13 → mucus production.

- From where did the mediators that causes the obstruction and bronchoconstriction come from?

From Mast cells, IgE attaches to mast cell in the first exposure to the allergen without causing symptoms but in the second exposure the allergen will react with the IgE that found already on the mast cell causing degranulation and releasing of Histamine.

Note: we studied 2 Rxns that produces mediators:

a) Acute phase Rxn: PRODUCES Histamine –stored mediator - (بيعمل كل المشاكل :P), causes bronchoconstriction ,increase permeability , mucus production and vasodilation.

b) Late phase Rxn: PRODUCES other mediators –mediators need to be synthesized at time of exposure (needs more time) –as an example Arachidonic acid metabolites**.**

Types of arachidonic acid metabolites are:

1) **Cyclooxygenase**, which will produce prostaglandin.
2) **Lipoxygenase**, which will produce leukotriene so the effect of them will be bronchoconstriction and vasodilation (caused by leukotriene B4, D4, E4 and C4).

We said that we have Atopic type of asthma and NON-atopic type, we've done with first one so let's begin with the second one.

NON-ATOPIC asthma:

i) **Caused mainly by Viral infection. here** we have the same pathogenesis as before but with deferent triggering factor.

ii) **Drug induced asthma,** especially ASPIRIN, why? Because it affects arachidonic acid metabolism. Aspirin affect cyclooxygenase so it prevents the production of PGs, but the Lipoxygenases are not affected so leukotrienes may produce bronchospasm.

iii) **Occupational inhalation** (as a part of environmental causes) like working in astrologers (مناجم) ,carpentry(النجارة) , etc.((here the occupational inhalation actually is allergic))

Q: if a patient came with signs and symptoms of asthma how to decide which type of asthma he has?

A: by history also we can do an allergic screening test,

**Notice that allergic asthma has

*more familial history

*more common in children. However, in some cases, asthma that occurs in childhood can go with age.

WHY??

(1) Desensitization

(2) They'll try to avoid the causes of their allergy.

(3) And may be due to transforming from IgE to IgM!

(4) Their Bronchi become larger. (<u>most important</u>) so allergic Rxn didn't affect it as it was obstruction ↓ →symptoms become less severe.

SIGNS and SYMPTOMS:

1) **Wheezing,** as a result of bronchoconstriction.
2) **Difficulty of breathing.**
3) **Cough, sputum** because of sanna3 al mashakel :P **(Histamine).**
4) **Later they'll develop Cyanosis.**

NOTE1: in asthma, the symptoms are localized not generalized as systemic allergic shock, so here no Hypotensive shock.

NOTE2: these symptoms are related to mediators, which are short lived, so the symptoms is *reversible.*

To conclude, asthma is characterized by reversible obstruction due to bronchospasm. other obstructive diseases are irreversible because they're related to structural *damage.*

- Does the reversibility stay forever?

If the patient develops attack after attack for years, it will end up with damage, why?

Because of inflammatory process that'll produces the chemical mediators, inflammatory cells (neutrophils, lymphocytes, macrophages) and proteases (elastases) → tissue damage.

So, every time the patient develops an attack he will have minor tissue damage, → minor damage over thousands minor damage ends up with major damage → structural changes → constriction in bronchi →in this case histologically we say that the patient has **air way remodeling**, the muscles hypertrophies permanently (not only spasm) and fibrosis because the infection. as a result, →permanent damage → permanent obstruction

SO, the disease begins as reversible bronchoconstriction but with time, it will transform to irreversible

In the reversible stage, we can prevent the disease from progression to irreversibility state by giving drugs .but in the irreversible stage -we already had structural damage- so it's useless to give drugs .

- In Bronchiectasis, we said in order to develop it needs OBSTRUCTION and INFECTION, do asthma needs these too??

NO, we said inflammation and obstruction related to bronchi, hypertrophy of the muscles and mucus secretion.

- How much time does the asthmatic attack stays?

It may stay for hours, however in some patients it could be prolonged up to days!! In this case, it is called (**status asthamaticus)** it is very dangerous because it affects breathing for long time even with treatment.

The doctor didn't talk about morphology, she will continue it in the next lecture.

اهداء الى كل من يضحي بوقته بكتابة الشيتات

الى عائلتي و اهلي الاعزاء في فلسطين دعواتكم لي ولهم .....دمتم سالمين:

(مقامك حيث أقامك , فاجتهد فيما أقامك)

sorry for any mistake (: