COPD ILLNESS SCRIPT

Hello, clinical problem solvers! This is Rafael Medina dos Santos. I'm a 5th year medical student at State University of Maringá from Brazil. This time, I'll walk you through an illness script for chronic obstructive pulmonary disease (COPD).

First of all, what is COPD?

COPD can be defined as persistent respiratory symptoms and airflow limitation due to airway and/or alveolar abnormalities. It is usually caused by significant exposure to noxious particles or gases.

To understand the importance of COPD, we must comprehend its global burden. So, how COPD affects the world?

According to the World Health Organization, the top 3 global causes of deaths are: ischemic heart disease, stroke, and COPD. While the global disease mortality of cardiovascular diseases, such as MI and stroke, has been decreasing, COPD-related mortality has increased in the last few years. But how does it affect the USA specifically? Along with the CDC, COPD is the third leading cause of death in the USA, and mortality from COPD has increased during the past 30 years in both men and women.

The age of onset is generally older than 35 years old and women and men are equally affected.

Most patients with COPD present with two distinct disease phenotypes, both related to smoking: Chronic Obstructive Bronchitis (COB) and Pulmonary emphysema.

To be classified as chronic bronchitis, patients must have a cough and mucus most days for at least 3 months a year, for 2 years in a row. Also, other causes of symptoms, such as tuberculosis or other lung diseases, must be ruled out. Pathologically, chronic bronchitis consists of an abnormal enlargement of the mucous glands within the central cartilaginous airways. There are hypertrophy and hyperplasia of the submucosal glands along with an increase in the number of goblet cells in the mucosa with bronchial wall thickening resulting in a narrowing of the bronchial lumen (aka bronchiolitis obliterans).

The second disease phenotype, pulmonary emphysema refers to an abnormal enlargement of air space distal to the bronchioles, due to the progressive destruction of the alveolar septa.

By far, the most important risk factor is smoking tobacco. However, some nonsmokers with other risk factors including exposure to dust and or air pollutants and genetic factors (like α1 antitrypsin deficiency), that lead to a chronic lung inflammation can develop COPD. α1 antitrypsin is an important enzyme that reduces lung tissue damage since it is the major inhibitor of neutrophil elastase, an enzyme that leads to acinar wall destruction. An α1 antitrypsin deficiency leads to unchecked elastase-mediated tissue damage in the lungs resulting in emphysema in young patients.

COPD can manifest clinically in several ways but there are 3 cardinal features: dyspnea, chronic productive cough, and chronic sputum production.

Physical exam depends on disease course. Early in the disease, you hear a prolonged expiration phase and wheezes. As the disease progresses overtime, you might hear decreased air movement or wheezes, rhonchi, and rales. You might even see barrel chesting. In end-stage disease, FEV1 / FVC ratio is less than 70% and FEV1 is less than 30%. In this state, patients often adapt instinctively during episodes of respiratory distress dyspnea-relieving position such as tripod position and pursed lip breathing.

On imaging, common signs of severe COPD include hyperinflated lungs, flattened diaphragms, blebs, and increased retrosternal clear space.

While this is a chronic disease, most patients seek medical attention during an acute exacerbation which is marked by an acute worsening of respiratory symptoms- specifically an increase in dyspnea, cough, or sputum production. These exacerbations are most frequently due to respiratory tract infections and exposures to dust/pollution. Remember to consider and rule out other diagnoses like a pneumothorax, pulmonary embolism, heart failure, and myocardium infarction that might present similarly. Treatment in the acute phase requires bronchodilators, oral steroids, and antibiotics if the exacerbation was due to a bacterial infection.

But what is used to reach the diagnosis? It is made in the outpatient setting through spirometry. It shows a FEV1 / FVC ratio lower than 70% of predicted, without significant change after using a bronchodilator which shows the existence of a “fixed” airway obstruction. Staging is made using GOLD criteria which assess the FEV1 predicted, intensity of the symptoms, risk of exacerbations, and the presence of comorbidities. In this way, the treatment and follow-up can be made individually providing what is best for each patient.

Differential diagnosis includes asthma, bronchiectasis, lung cancer, heart failure, and interstitial lung disease. For a more comprehensive detail for each disease, please check the corresponding illness scripts on our website.

Regarding management, smoking cessation is key! As physicians, it is essential to aid the patient quitting smoking, if it is the case. If O2 saturation is less than 88%, patients need supplemental O2. Bronchodilators, inhaled steroids, pulmonary rehabilitation, and relevant vaccinations are also important.

Remember, COPD is a severe debilitating disease! Making sure we detect it early, treat it adequately, and seek change in patient’s health-style is essential!

I hope you all enjoyed it! See you next time!