Doctor.021

no. 1

RS P.B.L

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Adult Respiratory cases

Case 1

History:

- **45 years old lady, previously healthy.**
- **Presented to emergency department with fever for 5 days, reaching 39.5 C.**
- **Associated with productive cough and shortness of breath**

-the clues are:

-fever for 5 days: is indicative of acute illness (any symptom that continues less than 2 weeks is considered an acute illness and the DDx are acute causes)

-productive cough, shortness of breath

-you must think about LRTIs (pneumonia) as a main diagnosis.

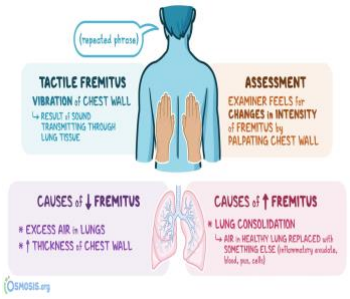
Physical examination

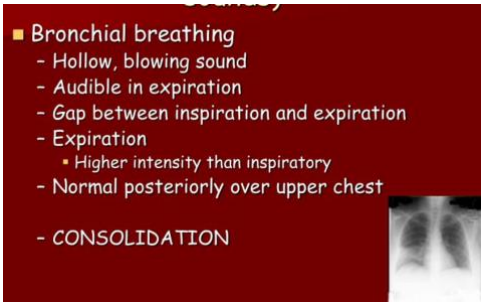
- **General: looks unwell, has increased WOB (work of breathing). (RR (respiratory rate) 40 b/m, PR (pulse rate) 110, temp 39). subcostal and intercostal retractions.**
- **Chest:**
  Auscultation: decreased air entry on Rt lower side.
  Bronchial breathing, increased tactile vocal fremitus (TVF), few inspiratory crackles Rt side.
- **Percussion: dull to percussion**

Extra: Let's remember the normal ranges and medical definitions to compare with:

-RR normally=12-20 breath/min in adults

-PR=60-100 beats/min





You can read about lung physical examination in this link: https://www.slideserve.com/masao/respiratory-examination?fitview=true#ssShare

-RR is increased→tachypnea, PR is increased→tachycardia, temp is increased, the patient is febrile, subcostal and intercostal muscles indicate that she is in respiratory distress (**extra**: hence accessory muscles of expiration: Subcostal, intercostal muscles and sternocleidomastoid. Accessory muscles of expiration work minimally during tidal (normal) breathing, when the patient becomes tachypneic, they assist in breathing, so in physical examination, subcostal and intercostal muscles enter between the ribs in respiration (because they try to work to compensate for the work of breathing), while in resting, the major working muscle is the diaphragm).

What are the Clinical Investigations needed?

- **CXR, CBC, Blood culture, inflammatory markers ,…etc.**

1)CBC: complete blood count, WBCs and especially neutrophils are expected to be increased in some types of pneumonia secondary to infections more precisely the bacterial ones. On the other hand, it is expected to decrease in cases of anemias, like mycoplasma pneumonia infection, which is known with IgM cold agglutination causes hemolytic anemia. Also, it is expected to decrease significantly in cases of severe sepsis.

2)blood culture: it is indicated only in specific cases culture decision is taken, examples: if the patient is febrile at the time of presentation or not responding to treatment. This determination is due to low diagnostic yield of culture about 20-30%, and because in practice we start empirical antibiotics based on clinical presentation.

Extra: Low yields in blood cultures for pneumonia can be attributed to several factors, and it's important to consider various aspects of the patient, the infection, and the diagnostic process. Here are some potential reasons for a low yield in blood cultures for pneumonia: Pathogen Characteristics: Some bacteria causing pneumonia may not consistently or efficiently enter the bloodstream, resulting in a lower chance of detection in blood cultures. For example, certain atypical pathogens may not be readily found in blood cultures. Prior Antibiotic Use: If a patient has received antibiotics before blood cultures are taken, it can significantly reduce the likelihood of detecting the pathogen. Antibiotics may kill or suppress the growth of bacteria, making them harder to isolate in culture. Timing of Blood Culture Collection: The timing of blood culture collection is crucial. Bacteria may not be present in the bloodstream at all stages of the infection, and there can be fluctuations in bacterial load. Collecting blood cultures at the right time is essential for improving sensitivity. Inadequate Volume of Blood Sample: Collecting an insufficient volume of blood for culture can reduce the chances of detecting the pathogen. Adequate blood volume is necessary to increase the sensitivity of blood culture tests. Fastidious Organisms: Some bacteria have specific nutritional and environmental requirements for growth and may not thrive well in standard blood culture media. Patient Population: The likelihood of positive blood cultures may vary among different patient populations. For example, elderly patients and those with underlying health conditions may have different patterns of bacteremia compared to healthy individuals. Microbiological Techniques: The sensitivity of blood cultures is also dependent on the laboratory techniques used. The choice of culture media, incubation conditions, and the duration of incubation can influence the ability to detect pathogens. Host Immune Response: In some cases, a robust host immune response may limit the systemic spread of the infection, reducing the chances of detecting the pathogen in the bloodstream. Viral Pneumonia: Not all pneumonias are bacterial; some are caused by viruses. Viral pneumonia may not result in bacteremia, and therefore, blood cultures may not yield positive results in these cases.

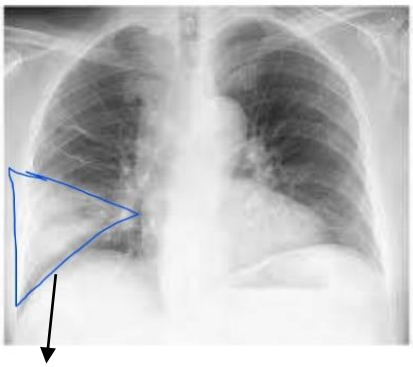
3)inflammatory markers: are very important indicators

-raised CRP→indicates infection

-patients who present with other differential diagnosis that are similar in terms of symptoms to pneumonia (like pulmonary embolism) will be afebrile or maybe low-grade fever with normal to mildly elevated CRP.

4)CXR: patchy opacity involving mid and lower zones of right lung (in this image)

-note: With X-rays, we prefer describing the lung by zones instead of lobes; the lung is divided into 3 equal zones.



Lobar infiltration is more characteristic to pneumonia

So according to previous history, clinical presentation (fever, dyspnea, cough) and CXR**, What is your diagnosis?** It should be pneumonia or LRTI.

Pneumonia

- **Definition**

Inflammation of the parenchyma of the lungs. (Alveoli and terminal airspaces in response to invasion by an infectious agent introduced into the lungs through direct invasion, hematogenous spread or inhalation.)

- **Causes:**

1-Infectious, mostly (Strept Pneumonia, staph aureus, Mycoplasma.p)
2-Noninfectious: less likely cause:
a-aspiration of food or gastric juice: especially in patients who drink alcohol, have stroke, or neurological disorders, and are unable to stay conscious at all.
b-hypersensitivity reactions
c-foreign bodies (children)
d-Hydrocarbons and lipoid substances: like cigarettes and vapes (also they are well-known to cause hypersensitivity pneumonia)
e-radiation-induced pneumonitis (in cancer patients)

COMPLICATIONS

-When patients admit to the hospital on the 2nd or 3rd day of IV antibiotics administration and not improving, so he might develop one of the following:

• **Pleural effusion:** comes with worsening dyspnea, fever recurrence, chest pain, and inflammatory markers will go up after being down in the first few days. In addition to physical findings including dull percussion note and absent TVF.

• **Direct invasion: Empyema, pericarditis**

- The direct infection is caused by the infection itself, and causes infection in the pleural space by the microorganisms or the infectious process
- So , empyema is direct invasion of the bacterial Ag to the pleural space in inflammation, while pleural effusion is a reactive process to pneumonia.

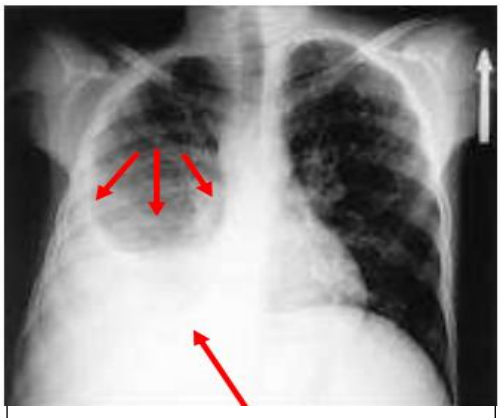
• sometimes the patient has distant invasion for the bacteria or virus**:** **Hematogenous (through bloodstream) spread: meningitis, suppurative arthritis and osteomyelitis (rare).**

-however, it is rare because most patients of pneumonia complain early and because pneumonia treatment is established empirically.

- **Extra**: Empirical treatment means initiating therapy based on clinical judgment and available information before a specific diagnosis is confirmed.

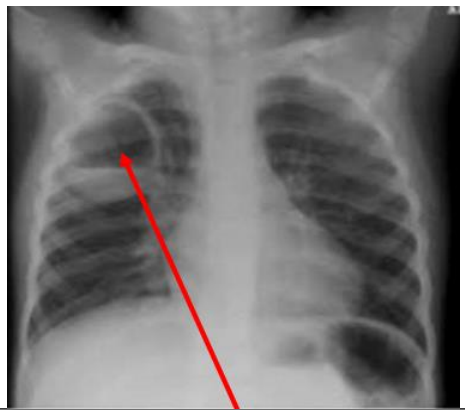
-So you need to start antibiotics ASAP, once symptoms appear and new infiltration on CXR, you don't need to wait for culture or blood test.

Pleural effusion



Red arrow =collection of fluid in the pleural space with a sign called: Meniscus sign: Fluid rises higher along the edge of pleural effusion, producing an upside down "U" or meniscus shape

Necrotizing pneumonia: cavitation



There is a cavity filled with fluid indicates abscess formation, a complication of untreated or delayed treatment of pneumonia.

-there is only one study that shows that mortality increases significantly if you start Abx after 6 hrs of presentation to ED, so txt should be initiated before 6hrs because all what is needed is PE, history, CXR, and sometimes blood test - which should take less than 6hrs

Typical vs atypical pneumonia

Typical pneumonia	Atypical pneumonia
caused by microorganisms can be identified by gram staining and culture (however we don't do this in practice) example: strep pneumoniae, staph.,...	Caused by microorganisms that can't be detected by gram stain or culture, so you have to do specific tests Example: Legionella pneumonia: legionella urine Ag Mycoplasma p: serology to show IgM
Present with similar symptoms regardless of typical pneumonia microorganism, so all present very sick after 3-5 days of illness With high CRP and WBC	Present with what called'' walking pneumonia'', have you heard about it before? -they are not sick -Looking -they present less suddenly after 5-7 days of their illness -usually don't have elevation in their WBCs or neutrophil left shift
Appear sicker than what their CXR shows	Their CXR appears worse than their presentation in term of significant multilobar infection like COVID pneumonia
mild illness: out -patient Mx (no admission): oral amoxicillin, cefuroxime, amoxicillin/clavulanic acid.	If mild, we treat them as an out-patient **Txt: macrolide like azithromycin or Levofloxacin**

-They are categorized according to CURB-65, CRB-65, [you can read more about this here;)]

-Sick, hospitalised patients; parenteral cefuroxime. If staph. aureus suspected (pneumatocele, empyema) clindamycin or vancomycin (special category)

Case 2

History:

• 45-year-old gentleman presents for evaluation of dyspnea of 6 months duration, associated with chronic minimally productive cough. He is police officer. He is current smoker of 40 pack year. He has unremarkable past medical, surgical and drug history. He has no history of childhood Asthma, atopy or family history of Asthma.

The clues are:

-6 months duration of cough, dyspnea: it indicates chronic disease (>2 weeks) can be: Acid reflux (GERD), Asthma, COPD (Chronic bronchitis + Emphysema)

-smoker -significant smoking history -.

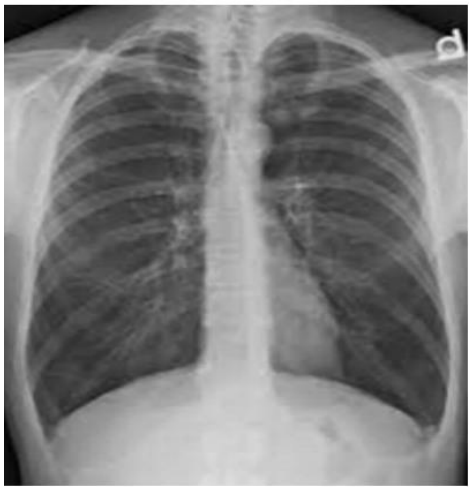
-no previous history of asthma, atopy, childhood asthma: doesn't seem to have risk factor for asthma though it doesn't negate it - patients can develop asthma without any previous history-.

Physical examination

- **Afebrile, RR 35 (20-30** /min normally**):** tachpnea**, Pulse rate 100:** tachycardia**.**
- **SPO2 89%.** (low O2 saturation level at room air), normal almost 98% according to Dr Yanal's lec😊
- **Intercostal and subcostal retractions.**
- **Chest: diffuse Expiratory** (indicative to an obstructive disease) **wheeze, prolonged expiratory phase with decreased air entry.**
- **CVS: normal,**
- **liver not palpable** (no evidence of abdominal mass),
- **hands: no finger clubbing. (extra** it negates IPF**)**

What is the next investigation?

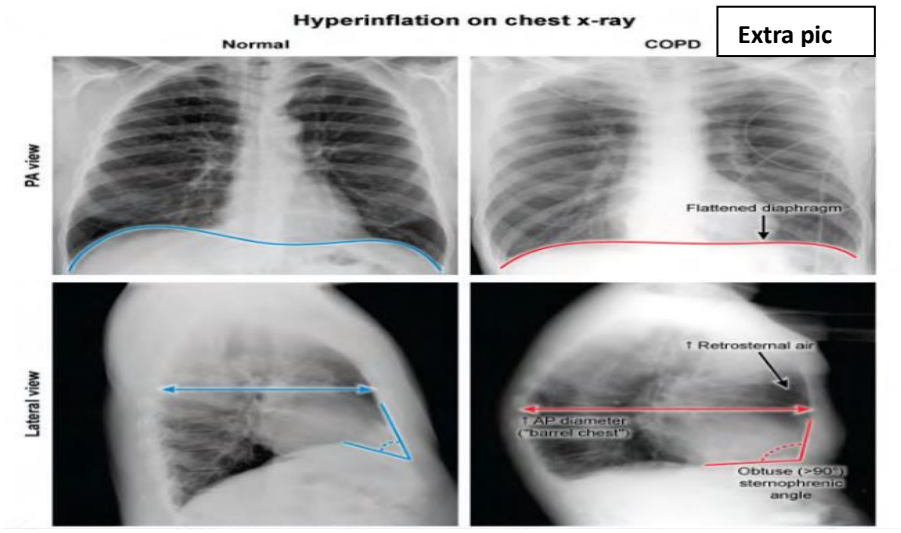
1-chest x-ray: Hyperinflation is seen, which can be recognized by:



1- We count the number of posterior ribs (the oblique ones), here in this example we can see 9 ribs, Normally, we should see 6 ribs anteriorly, and 8 ribs posteriorly, anything more indicates overinflation

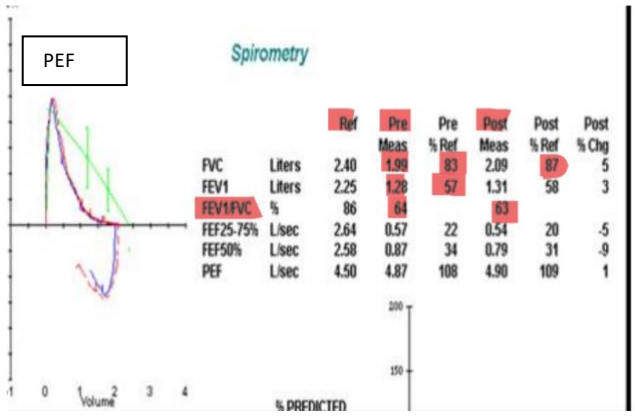
2- Another clue is that the diaphragm is pushed downward

3- small heart,



2-spirometry: It is one of lung function measurements, which is an instrument in which the patient exhales forcefully to measure expiratory flow, it is helpful in airway and restrictive lung diseases diagnosis.

This part is physio lab revision:



-the straight black line: air expelled out of large airways, starts from the TLC at the X-axis until it reaches the Peak flow.

-the green line: air expelled out of small airway (normal), continues until the residual volume.

-the curve below the x-axis is the inspired air

-in the patient, the green line is deviated with more curving (coving) giving the red curve: indicates that the problem is in expired air of small airways→ airway narrowing mainly which would cause taking more time to exhale → good clue to think of COPD - airway obstruction-.

-Then look at FEV1R pre-bronchodilators = 64%, and post-drug = 63%--> both are less than 70 %.

-also, pre- and post-FVC are normal (83 % and 87%).

-for FEV1: It is 57% less than normal (80-120%) and after the reversibility test (using bronchodilator) it stays low, indicating definite airway obstruction.

What is the diagnosis:

After the history, physical examinations, X-ray and spirometry, we diagnose the patient **as COPD**

Definition

- **is a common, preventable and treatable disease.**
- **It is characterized by persistent respiratory symptoms and airflow limitation that is due significant exposure to noxious particles or gases.**
- obstructive spirometry -obstructive airflow-: FEV1/FVC < 70%, caused by specific risk factors like smoking.

- **The chronic airflow limitation that is characteristic of COPD is caused by a mixture of small airways disease (e.g., obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person in term of clinical presentation.**
- **Extra**: Obstructive lung diseases:

1- COPD (chronic bronchitis and emphysema)
2- Asthma
3- Bronchiectasis

Treatment

- **Reducing risk factor exposure:**
- **vaccination, smoking cessation, pulmonary rehabilitation**
- **Appropriate assessment of disease**
- **Patient education**
- **Pharmacological and non-pharmacological management of stable COPD**
- **Prevention and treatment of acute COPD exacerbations:**

-exacerbation is defined by the worsening of clinical symptoms that were stable and require a change in medications.
-You need to treat acute exacerbations because it is linked to mortality, morbidity, and lung function reduction.

-patients with COPD who come having exacerbations or more flares up of their disease are more likely to die, they have very bad outcomes and very severe disease, and even if they are stable, sometimes, they tend to progress very quickly because every time they have exacerbation, there will be more worsening of their disease and lung function.

-COPD patients always complicate persistent dyspnea and cough when they exacerbate, they will suffer more cough, more sputum and change in sputum color, worsening dyspnea and they usually seek medical evaluation or go to ED for treatment change, or steroids administration, they also may be admitted.

- **Pharmacological treatment**

-should be started with inhaled medications: B2 agonists and anticholinergics

-both have short acting and long acting effects

-short-acting drugs are used in exacerbations, while long acting are used in stable chronic patients

-we usually start with LAMA then add LABA, or the combination of both may be the first course.

-but we don't usually start with ICS, because if you have asthmatic patient, you would start with ICS at the upfront treatment.

-so, in asthma we start with ICS, then add other inhaled bronchodilators, while in COPD, we start with muscarinic antagonist (anticholinergics)and B2 agonists then add ICS if necessary.

-so when is ICS usage indicated in COPD? If the patient has suffered from 2 exacerbations in the last 12 months, if the FEV1 is less than 50%, if peripheral eosinophilia is presented, and with overlapping asthma.

-Inhaled B2 agonist (short acting) (SABA)
-Inhaled B2 agonist (long acting) (LABA)
-Inhaled anticholinergic (short acting) (SAMA)
-Inhaled anticholinergic (long acting) (LAMA)
-Inhaled corticosteroid (ICS)
-Combination inhalers
-Methylxanthine:
Like theophylline, is indicated in special cases but in general, we don't use them because of their toxicities.

-Phosphodiesterase-4 inhibitor:
Have been used recently for exacerbation management, so it is added to inhaled bronchodilators.

*the steps are inhaled bronchodilators initiation then ICS +/- phosphodiesterase inhibitors addition in case of more complicated exacerbations.

Case 3

64 years old female patient with longstanding history of type 2 DM and recently treated breast cancer presented to the ER with fever, cough and dyspnea. Her COVID19 swap is positive

-the background of the patient: DM, breast cancer survivor

-symptoms: fever, cough, dyspnea

-covid patient → she has COVID pneumonia.

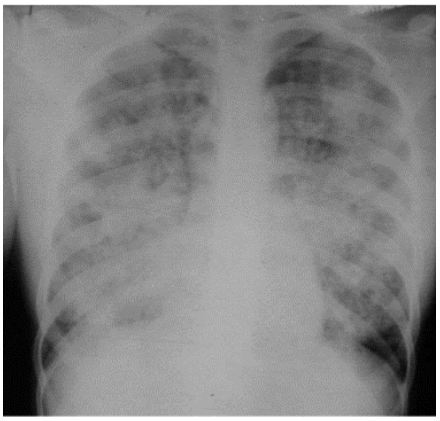
Physical examination

- **BP is 130/70, RR 18, HR 98, SO2 86% on room air. temp 38.6 C.**
- **Chest: bilateral inspiratory crackles and bronchial breath sounds.**

Increased TVF and dull percussion to auscultation

- These findings are characteristic for bilateral pneumonic consolidation.

Investigation



-CXR: which indicated the following:

-bilateral patchy opacities involving most of the lung fields

-normal heart

-acute symptoms

*You must think of ARDS,

Investigation
ABG on room air
PH: 7.42
PaCO2: 33 mmHg
PaO2: 40 mmHg
SPO2: 80%
-PF ratio: Pao2/Fio2 → 40/0.21=190

-we use PF ratio to define ARDS, PaO2 is obtained from ABGs, and FiO2 depends on the O2 breathed by the patient

-If it is room air, it will equal 0.21, if we let the patient expire 32% of O2 FiO2=0.32

Diagnosis: ARDS

Adult respiratory distress syndrome Acute respiratory distress syndrome (ARDS)

Definition

It is a clinical syndrome characterized by an acute, diffuse, inflammatory form of lung injury resulting from diffuse injury to the alveolo-capillary

membranes., (characterized by increased pulmonary vascular permeability, and loss of aerated tissue, increased work of breathing and impaired gas exchange.)

ETIOLOGIES AND PREDISPOSING FACTORS

-ARDS is related to inflammatory cytokines releasing and systemic response from the body

DIRECT LUNG INJURY	INDIRECT LUNG INJURY
Pneumonia	Sepsis
Aspiration of gastric contents	Multiple trauma
Pulmonary contusion (caused by trauma, blunt injury)	Cardiopulmonary bypass
Fat, amniotic fluid, or air emboli	Drug overdose
Near-drowning	Acute pancreatitis
Inhalational injury (CO inhalation=chemical pneumonitis, inhalation to flames of burn)	Transfusion of blood products
Reperfusion pulmonary edema	

seen usually when we drain pleural effusion that has been there for long time or drain too much fluid at the same time, so the lung that has just expanded is reperfused-->increasing alveolar capillaries permeability(ARDS cause)

Treatment

Treatment for ARDS typically aims to:
Increase blood oxygen levels.
Provide breathing support.(ventilators)
Treat the underlying cause of the disease.
IV fluids support
Anti-virals in case of viral pneumonia
Steroids in case of severe ARDS

The end

1- A case about a patient with covid who developed ARDS, which of the is not expected to be in this patient:

A. the PF ratio is 190
B. expiratory wheeze with prolonged expiratory phase
C. the patient has Adult respiratory distress syndrome

Ans: B