RESPIRATORY SYSTEM

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Topics

1- Pulmonary function test – S د. أسيل
2- Asthma – L د.أسيل
3- COPD – L د. أسيل
4- Diffuse parenchymal lung disease – L د. أسيل
5- Pneumonia –S د. سري
6- Sarcoidosis – S د. جعفر الشياب
7- TB - S د. سري
8- Pulmonary hypertension- L د. فردوس
9- VTE+ PE – S د. فردوس

بداية هذا التلخيص يشمل سلايدات الدكاترة مع أهم النوتات التي تم ذكرها في المحاضرة بناءا على محاضرات الفصل الأول وأي اضافة جديدة سيتم التعديل على الملف .

L-lecture / S- seminar

بالنسبة للسمينارات فسيتم ذكر النقاط التي تم التركيز عليها من قبل الدكتور في المحاضرة فقط ، وبالتالي يجب دراستها من المصدر المعتمد لديكم ثم قراءة أي شيء ركز عليه الدكتور .

1- Pulmonary function test :

بالنسبة لهاد السمينار فالدكتورة حكت ال chart تبع ال lung volume and capacity جدا مهم
علقت على جهاز ال spirometryإنه بقيسلنا أغلب الشغلات يلي بدنا اياها ما عدا ال RVلأنه هاد بنحسبه حساب عن طريق معادلة ما بنقدر نعمله measurement

وال human variation مهمة رح أحطها هون . وغير هيك الدكتورة ما علقت على اشي بتقدروا تشوفوا الموضوع من أي مكان معتمدينه للدراسة وبنصح من board and byoned

Human variations
Lungs volumes and capacities are not identical in every human being and they vary according to different variables:

• Age : lung measurements and functions decline with age.
• Height : size varies with height.
• Gender : males have larger lungs compared to females.
• Race : white people have much larger lungs than black people

Asthma

Definition: Asthma is an inflammatory disorder of the airways characterized by cough, wheezing, chest tightness, dyspnea, and variable airflow obstruction.

Pathogenesis:

- inflammation due to irritant ---recurrent exposure--- chronic air way inflammation - edema ---airway narrowing ---fibrosis –smooth muscle hypertrophy
- Mucus hypersecretion
- Airway smooth muscle constriction causing bronchial hyperreactivity in response to various stimuli.

Risk Factors

Host factors: genes predisposing to atopy; bronchial hyperreactivity; and airway inflammation have been identified.

Environmental factors:

● Exposure to indoor allergens (mites, furred animals, cockroaches, molds)
● Outdoor allergens (pollens, molds)
● Tobacco smoke
● Occupational sensitizers and allergens,
● Viral respiratory infections
● Air pollution.
● Obesity

Symptoms are :

● **Intermittent** and occur in response to various potential stimuli include: allergens, infections, dusts, fumes, and exercise.
● Have a **diurnal variation**, worsening in the evening and early morning.
● **Variability of symptoms** (both improvement and worsening of symptoms over time) is a key diagnostic feature of asthma.
● Symptoms often occur with or **worsen with viral infection**

Diagnosis:

- **HISTORY :** ask about Smoking history , Pets exposure ,Work place , Personal or family history of atopy or allergic sinus disease and **Presence of nasal polyps, sensitivity to aspirin, and wheezing** is known as the "**asthmatic triad.**

- **Physical examination:** Wheezing , Reduced airflow, Prolonged expiratory phase . **Patients may also have a completely normal respiratory exam, particularly when they are symptom-free. "**This doesn't exclude asthma**"**

- **Peak flow**
- **Spirometry** "gold standard" Confirmation of reversible airflow obstruction with Bronchodilators

  FEV1/FVC <70 --- obstructive lung disease

  • Asthma: reversible obstruction

  ↑ FEV1 with bronchodilators (most common B2 agonist –salbutamol)

  FEV1 increases by > 200 mL AND >12% of the baseline value

  • COPD: partial/no change bronchodilators

ملاحظة : ال 70 % بنسميها ال lower limit of normal range ما تحكي عنها ال cut off point لأنهاا
عند 5% من الناس بتكون أعلى أو أقل .

- **Bronchial challenge test (methacholine challenge)**

الفكرة هون مثلا مريض عند couphوبتصيحه من النوم أو بتصير لما يعمل تمارين ف على ال physical طلع
معك normal وعلى ال spirometerطلع normal بنلجأ لهاد الفحص عشان أشوف هل عنده أزمة ولا لا
وبعطيه مادة بتعمل bronchoconstriction

A negative test **excludes asthma**

Positive test : if there is 20% decrease in FEV1 from the baseline

يلي بهمني هو ال negativeلأنه بقدر أستثني ال asthma أما ازا طلع positive فهون بحكي إنه عندي
hyperactivity of airway could be asthma require additional testing

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Asthma Syndromes

1) Allergic Asthma – usually in childhood + FH of atopy

2) Cough-Variant Asthma – role out other most common causes of chronic cough **(GERD)**

3) Exercise-Induced Bronchospasm -

بهاد النوع يكون المريض ما عنده اشي at rest بس بتصير لما يعل exercise وغالبا تفسيرها الهوا البارد
وهو بيركض بحيث بعمله irritant of air wayوبصير معه bronchospasm والعلاج هون رح يكون عن
طريق أشياء يعملها المريض مثلا يلبس ماسك بحيث يخفف من الهوا البارد والناشف وكمان ممكن
- salbutamal قبل 50 دقيقة من التمرين أو ازا كانت الأعراض عنده يومية ياخد long acting inhaler
أو leukotriene receptor antagonist أو oral corticosteroid

4) Occupational Asthma - هاي خلال الأسبوع بتكون الأعراض سيئة وبنهاية الأسبوع بتتحسن

5) Reactive Airways Dysfunction Syndrome - exposure to huge amount of irritant .

مثلا وحدة نضفت البيت بكميات كبيرة من الفلاش وبعد هيك اجت بأعراض مستمرة لمدة بتزيد عن 3 أشهر

6) Aspirin-Exacerbated Respiratory Disease - Asthma, chronic rhinosinusitis, nasal polyposis • Chronic asthma/rhinosinusitis symptoms • Acute exacerbations after ingestion aspirin or NSAIDs.

7) Allergic Bronchopulmonary Aspergillosis

Common Comorbidities

Comorbidities in asthma are common and should be considered and actively managed to reduce symptoms and potentially improve asthma control.

- Gastroesophageal Reflux Disease PPI + FOLLOW UP AFTER 1 MONTH
- Sinus Disease allergic rhinitis , sneezing and post nasal drip --- worsing the symptoms

Ttt : nasal spray (corticosteroid)

- Obstructive Sleep Apnea
- Vocal Cord Dysfunction
- Obesity

Asthma in Pregnancy

- Pregnant patients should be advised that the advantages of treatment are significantly greater than the potential risk to the fetus from asthma therapies or exacerbations.
- Pregnancy can affect asthma control, **leading to either worsening or improvement**, and patients should be closely monitored for signs of exacerbation, which occurs most frequently during the second trimester.
- Inhaled glucocorticoids, oral glucocorticoids, SABAs, leukotriene-receptor antagonists (montelukast, zafirlukast), and LABAs have ALL been used extensively during pregnancy without data to suggest fetal harm.

Asthma treatment

Chronic therapy

- Goals : Control chronic asthma symptoms, Prevent acute exacerbations and Minimize risks of developing fixed airway obstruction/ prevent respiratory failure.

#هلأ أي مريض chronic asthmaلازم بكل زيارة تتأكد من إنه الأعراض عنده قاعدة بتتحسن ولا لا ؟ عن طريق 4 أشئلة بتسأله اياهم بجاوب عليهم ب yes or no .

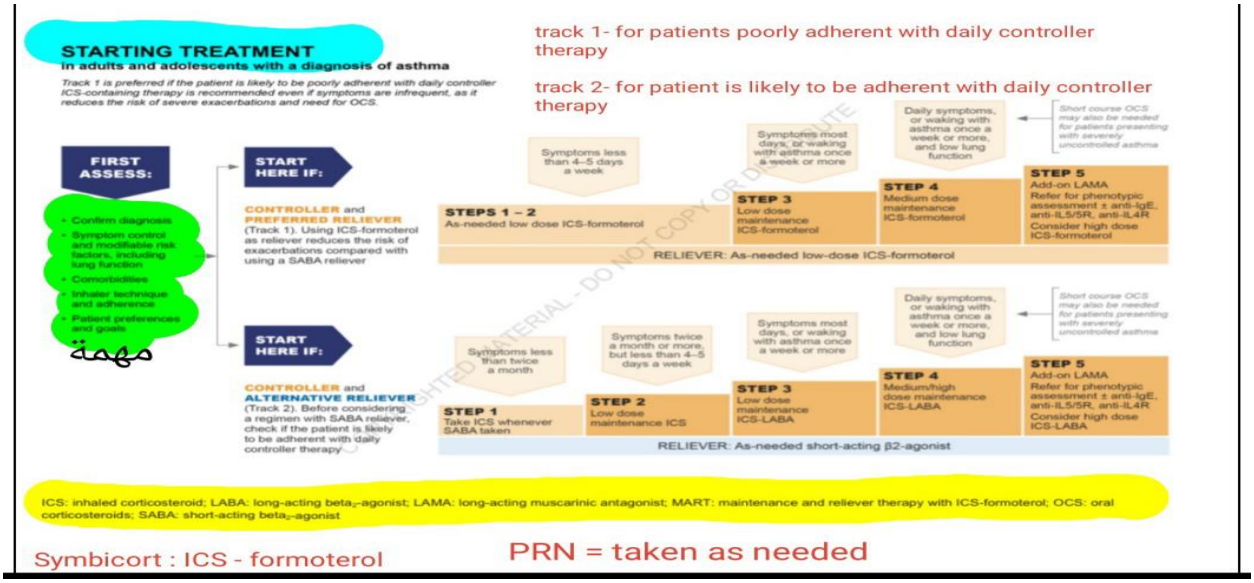
- Day time symptoms more than twice / week ?
- Any night waking due to asthma ?
- SABA reliever for symptoms more than twice/ weeks ?
- Any activity limitation due to asthma ?
  - Well control : none of these
  - Partly control: 1-2 of these
  - Uncontrolled : 3-4 of these

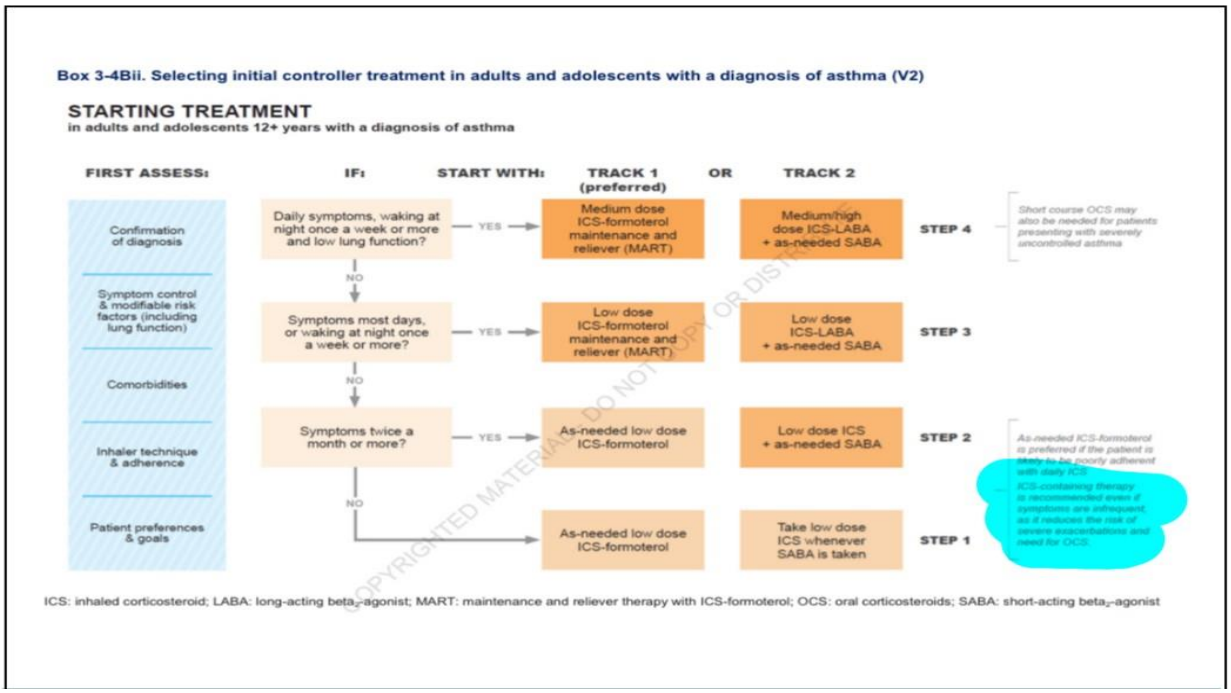
وكمان بكل زيارة لازم تسأل المريض عن ال Risk factors of exacerbation :

- Uncontrolled symptoms
- Modifiable risk factors : High SABA use – more than 2 inhaler/ month, Inadequate ICS , Poor adherence , Incorrect inhaler technique , Obesity , GERD , food allergy , pregnancy , Smoking , air pollution , Psychological or socioeconomic problem and FEV1 < 60 .

- Independent risk factor : Ever intubated or in ICU for asthma , > or = 1 severe exacerbation in last 12 months .

START TREATMENT هدول السلايدين جدا مهمين والدكتورة ركزت على كل اشي فيهم





Management of Asthma Exacerbations

Asthma exacerbation refers to an acute worsening in symptoms or lung function from baseline that necessitates a step-up in therapy.

● All asthma patients should have a written asthma **management plan** that helps them to recognize the symptoms of an exacerbation and begin self-treatment.

كل مريض لازم تكون موضحله شو يعمل اذا الأعراض صارت أسوأ عنده وكيف يزيد ال inhaler تبعونه وازا كنت مروحه على OCS كيف ياخدهم ومتى يجي على الطوارئ إزا هاي الأعراض ما تحسنت .

● Clinicians should screen for patient factors that contribute to an increased **risk of death** from asthma and counsel patients appropriately.

إزا كان عنده أي اشي منهم ما بتقدر هدول المرضى تعالجهم ك out patient ولا إنك تعالجهم بالطوارئ وتروحهم لازم تعمللهم دخول .

1.	A history of near-fatal asthma requiring intubation and mechanical ventilation
2.	Hospitalisation or emergency visits for asthma in the past year
3.	Currently using or having recently stopped using oral corticosteroids
4.	Overuse of SABAs (>1 canister of salbutamol or equivalent monthly)
5.	A history of psychiatric disease or psychosocial problems
6.	Not currently using inhaled corticosteroids
7.	A history of psychiatric disease or psychosocial problems
8.	Poor adherence with asthma medications and/or poor adherence with (or lack of) a written asthma action plan
9.	Food allergy in a patient with asthma
10.	several comorbidities including pneumonia ,…

هلأ بغض النظر اجاك المريض على العيادة أو الطوارئ لازم تسأل حالك 3 أسئلة :

- If symptoms due to asthma or others differential (copd, HF,MI,….)
- If there is any factors of asthma – related death
- Asses the severity of exacerbation (mild or moderate , sever , life threatening)
  - Life threatening : drowsy, confused , silent chest
  - Sever : talks in words , using accessory muscle , RR more than 30 /min , pulse more than 120 bpm, o2 sat less than 90 , Peak expiratory flow less or equal 50.
  - Mild to moderate : talks in phrases , not using accessory muscle, RR increase but not more than 30 , pulse 100- 120 bpm, o2 sat 90- 95 , Peak expiratory flow more than 50.

هلأ الحالة الأولى إزا كان المريض mild to moderate وما عنده ال factors of death هون ممكن تحكيله يزيد ال inhaler تبعونه ويبلش ياخد ocs بالبيت . وهاد المريض لازم ترجع تعمله assessment after 1 h إزا المريض بعد ساعة ما تحسن لازم توديه على الطوارئ إزا كان هو أصلا بالطوارئ هون ممكت يحتاج يدخل ال ICU .
وهون بالطوارئ ممكن تعطيه : SABA , Ipratropium , O2 , Oral corticosteroids

إزا المريض دخل ER فلازم أول شي تعمل ال ABC .

الحالة التانية إزا كان المريض عنده sever أو life threatening هون لازم ادخله على ال ICU وهون ممكن تعطيه

- SABA , Ipratropium , O2 , Oral or iv corticosteroid , Consider iv magnesium and high dose ICS.

ملاحظة : هون بما يخص ال exacerbation كتبت فقط ما ذكرته الدكتورة ، للي بده تفاصيل أكتر يرجع للمخطط بالسلايد .

COPD

Definition : Chronic Obstructive Pulmonary Disease (COPD) is a *common*, *preventable* and *treatable* disease that is characterized by persistent respiratory symptoms and airflow limitation or obstruction that is not fully reversible .

FVV1 progression over the time :

هون الدكتورة حكت إنه بضل يزيد لحد ما يوصل لل peakعمد عمر ال20 سنة ومع عمر ال30 ببلش ينزل وعند عمر ال70 بكون كتير نزل بس بالرغم من هيك بالناس ال normal ما رح ينزل عن ال80 بينما بمريض ال copd رح يزل عن ال 80 .

- COPD is now one of the top three causes of death worldwide.
- The most common respiratory symptoms include dyspnea, cough and/or sputum production. These symptoms may be under reported by patients.
- The main risk factor for COPD is tobacco smoking but other environmental exposures such as biomass fuel exposure and air pollution may contribute.
- Other host factors : genetic abnormalities, abnormal lung development and accelerated aging.

Diagnosis and initial assessment :

- **Pathways to diagnosis:**
  - **Symptoms:**
    1- Dyspnea :progressive , persistent and worse with exercise .
    2- Chronic cough : intermittent , may be unproductive and recurrent wheeze.
    3- Sputum : any pattern of chronic production .
  - **Risk factors :** recurrent lower respiratory tract infection , host factors , tobacco , occupation , indoor /outdoor pollution, family history of copd and childhood factors (low birthweight , respiratory infection).
  - **Spirometry:** required to establish diagnosis , the presence of a **post** - bronchodilator **FEV1/FVC < 0.70** confirms the presence of persistent airflow limitation.

    كلمة post مهمة جدا بالتعريف لأنه احنا ما بنشخص المريض بال pre- bronchodilator وإنما بنشخصه على ال post وهاي مهمة وبتيجي سؤال بالامتحان .

    مريض عمره أقل من 40 وعنده ال typical symptoms تبعت ال copd وعلى ال spirometry

    أكدلك التشخيص , شو رح يكون سبب ال copd عند هاد المريض ؟ alpha 1 antitrypsin deficiency

- **Initial assessment of severity :**

  Determined by using a combination of :

  1- Symptoms :

  عشان أحدد ال severity بناءا على ال symptoms عنا استبيانين ومش مطلوب حفظهم بس ناخد فكرة عنهم ... الاستبيان الأول اسمه **CAT** وهو مجموعة أسئلة بتسألهم للمريض ضمن score معين وبتشوف كم طلع معك وهاد ال score بنعتمد عليه بتحديد ال severity . أما الاستبيان التاني هو **MRC dyspnea scale** بصنف المريض ل grade 0-4 بناءا على متى بتصير معه ال shortness of breath .

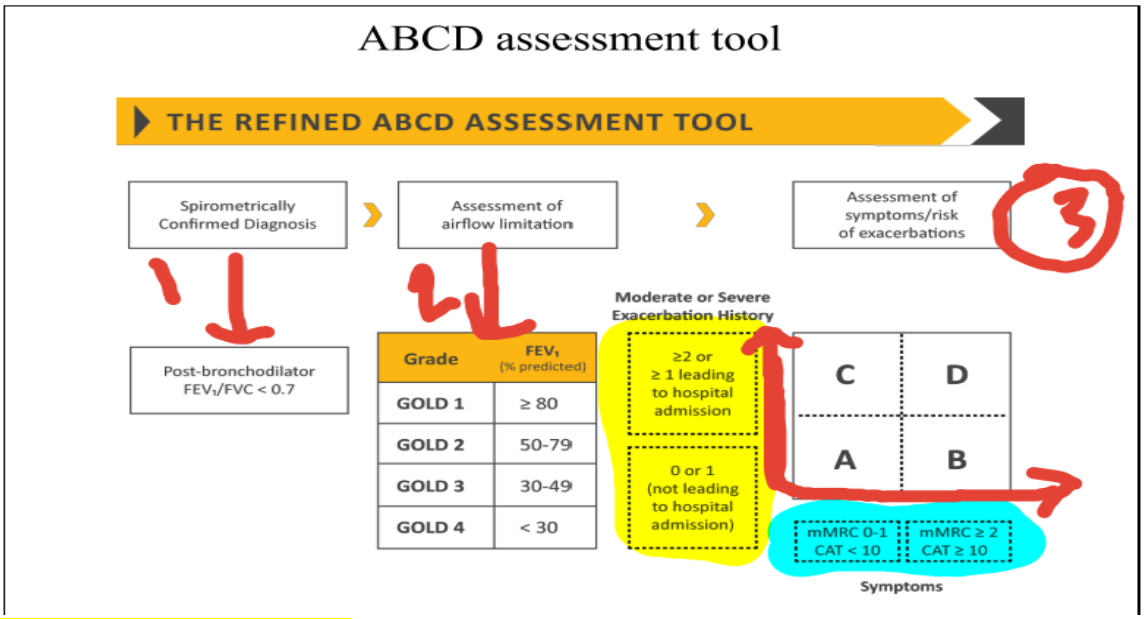
2- Degree of flow obstruction on spirometry :

هلأ الشغلة التانية يلي بنعتمد عليها لتحديد ال severity هي ال spirometry وبالتالي لما يطلع معك ال **ratio أقل من 0.70** بنروح بنحدد على أساس ال **post- bronchodilator FEV1** وبناءا عليه بنصننفهم (GOLD1 mild ---GOLD 4 very severe)

3- History of acute exacerbation and presence of comorbid condition :

تالت شغلة بنعتمد عليها هي كم مرة صار عند المريض exacerbation وهل دخلته المستشفى ولا لا ؟

والتلت شغلات هدول حطوهم كلهم ب box واحد لحتى نقدر نحدد ال severity ونصنف المريض هل هو بمجموعة A ولا B ولا Cولا D وعليه بنقرر شو هي ال MANAGEMENT لهاد المريض .



Management of Stable COPD

- **Goals:**
  Reduce symptoms (relieve symptoms , improve exercise tolerance , improve health status) .
  Reduce risk (prevent disease progression , exacerbation and reduce mortality) .
- **Pharmacological treatment**

Group A – bronchodilator
Group B – long acting bronchodilator (LABA or LAMA)
Group C – LAMA
Group D – LAMA or(LAMA+ LABA)if highly symptomatic or(ICS+LABA)if eos >300

Most commonly used maintenance medication in COPD :

1- **BETA2 – AGONIST**
   SABA such as (albuterol) + LABA such as (formoterol)
2- **ANTICHOLINERGICDS**
   SAMA such as (ipratropium bromide)+ LAMA such as (aclidinium bromide)

- **Non – pharmacological treatment**
  1- Smoking cessation .
  2- Vaccination, all COPD patients should have annual influenza immunization and the pneumococcal vaccine
  3- Oxygen therapy with
  - Arterial Po2 of 55 mm Hg or less , or oxygen saturation is 88% or less.
  - Arterial Po2 is 59 mm Hg or less ,or the oxygen saturation is 89% or less And patient has one of these (cor pulmonale, heart failure, erythrocytosis).

أول 3 يلي بالأحمر مهمين جدا وسؤال امتحان إنه شو هي الأشياء يلي بتقلل الmortality لمرضى ال COPD ؟

  4- Others : Education and self-management, Physical activity, Pulmonary rehabilitation programs, Exercise training, Self-management education, End of life and palliative care, Nutritional support.

ACUTE EXACERBATION

DEFINITION

An acute exacerbation of COPD is a change in a patient's typical symptoms that leads to a change in medical therapy or requires hospitalization.

● Most commonly, exacerbations are manifested by an increase in the severity or frequency of cough, worsening dyspnea, and an increase in the amount or change in the character of sputum produced.

● Most exacerbations are triggered by a respiratory infection (either viral or bacterial), smoking, and environmental exposures.

Medical history

● Time course of the symptoms ● Comparison to baseline level of symptoms
● Severity of respiratory compromise (eg, dyspnea at rest, dyspnea climbing stairs)
● Delineation of sputum characteristics (eg, amount, purulence, blood).

Physical exam

➢ wheezing ➢ tachypnea

➢ May include features of respiratory compromise such as : difficulty speaking due to respiratory effort, use of accessory respiratory muscles, and paradoxical chest wall/abdominal movements.

➢ Decreased mental status could reflect hypercapnia or hypoxemia and asterixis could indicate increased hypercapnia.

Evaluation

• Pulse oxygen saturation
• Chest XR to exclude pneumonia, pneumothorax, pulmonary edema, pleural effusion
• Laboratory studies (complete blood count and differential, serum electrolytes and glucose)
• Electrocardiogram
• ABG , can determine the presence of hypercapnia or hypoxemia.

Goals

Relieve acute symptoms + Prevent future exacerbations.

- **Oxygen therapy :**
  Supplemental oxygen should be used to maintain oxygen saturation between **89% and 92%.**

ما بدنا نوصل لأعلى من هيك لحتى ما يصير عند المريض respiratory depression والفكرة هون إنه بال normal people ال main respiratory driver هو ال co2 بحيث لو صار فيه build up رح ال brain يحفز ال respiratory center ويصير hyperventilation . ولأنه مرضى ال copd أصلا عندهم ال copd عالي ومتعودين عليه فبصير يلي يحدد هو ال hypoxia .

  ● **Venturi masks** are the preferred – fixed amount of o2
  ● **Nasal cannula**
  ● When a higher FiO2 is needed, **simple facemasks** can provide an FiO2 up to 55% using flow rates of 6 to 10 L per minute.
  ● **Noninvasive mechanical ventilation** may be required if oxygenation or ventilation cannot be maintained
  ● **Mechanical intubation:** If they cannot tolerate noninvasive mechanical ventilation , Have an altered mental status, Have worsening hypercapnic or hypoxemic , respiratory failure despite the use of noninvasive , mechanical ventilation , Profound academia(cutoff point 7.25) .

- **Therapeutic Management**

  ● **Short-acting B2-agonists with or without anticholinergic agents** should be used to relieve acute symptoms.
  ● The use of **glucocorticoids** during acute exacerbations has been shown to decrease the frequency of treatment failures, length of stay, and the time to subsequent exacerbations while improving FEV1 and hypoxemia.
  ● **Antibiotics** should be prescribed in cases of moderate or severe exacerbations or for patients with mild exacerbations who have noted an increase or change in sputum production.

  ● The most common infectious triggers are **viruses**, but bacterial causes include Streptococcus pneumoniae, **Haemophilus influenzae**, Moraxella catarrhalis, and Mycoplasma pneumoniae.

سؤال امتحان مهم :
لو اجاك مريض بأعراض ال exacerbation شو هي ال most likely bacterial infection ؟
Haemophilus influenzae

Diffuse Parenchymal Lung Disease

(interstitial lung disease)

DEFINITION :

Diffuse parenchymal lung diseases (DPLDs) are a group of disorders based on similar clinical, radiographic, physiologic, and pathologic changes that affect the alveolar walls and often the related small airways and distal pulmonary vasculature.

❖ inflammatory disorder of alveolar wall---fibrosis---restriction

❖ Like other lung diseases, these disorders present primarily with shortness of breath.

❖ Imaging studies will typically demonstrate bilateral rather than unilateral lung disease.

EPIDEMIOLOGY

❖ DPLD is uncommon, compared to other pulmonary diseases such as asthma/ COPD

❖ The true prevalence of DPLDs is unknown; however, the literature estimates the prevalence at approximately 70 per 100,000 persons, with idiopathic cause accounting for 30% to 40% of disease in these patients.

in systemic sclerosis --- most common causes of death in it is interstitial lung disease

CALSSIFICATION

- **Known causes**
  - Drug induced; examples: amiodarone, methotrexate, nitrofurantoin and chemotherapeutic agents.
  - Smoking-related: "Smokers'" respiratory bronchiolitis characterized by gradual onset of *persistent cough and dyspnea*. Radiograph shows ground-glass opacities and thickened interstitium. Smoking cessation improves prognosis. Desquamative interstitial pneumonitis and pulmonary Langerhans cell histiocytosis are other histopathologic patterns associated with smoking and DPLD .
  - Radiation : may occur 6 weeks to months following radiation therapy.
  - Chronic aspiration : Aspiration is often subclinical and may exacerbate other forms of DPLD.
  - Pneumoconiosis: Asbestosis, silicosis, berylliosis
  - Connective tissue disease.
  - Hypersensitivity pneumonitis.
- **Unknown causes (idiopathic)**
  - Idiopathic interstitial pneumonia
    1- Idiopathic pulmonary fibrosis
    2- Acute interstitial pneumonia : dense bilateral acute lung injury similar to acute respiratory distress syndrome; 50% mortality rate.
    3- Cryptogenic organizing pneumonia: may be preceded by flu-like illness, radiograph shows focal areas of consolidation that may mimic infectious pneumonia or may migrate from one location to another.
  - Sarcoidosis : variable clinical presentation, ranging from asymptomatic to multi-organ involvement

هون بالأول بتفكره pneumonia وبنبلش نعالجه ب antibiotic
بس ما بتحسن عليه أو ممكن تعيد الصورة وتلاقي ال
consolidation صار ب lobe ثاني ورح تلاقي recurrent
symptoms فهون بنشك بهاد المرض وطبعا علاجه بكون عن
steroid طريق

هلا الدكتورة بالسلايد ركزت على أربع أنواع منهم فقط ورح نحط شرحهم هون :

Hypersensitivity Pneumonitis

- **Definition :**
  Repetitive inhalation of antigens in a sensitized patient can result in hypersensitivity pneumonitis (HP). It is an immunologic response that results in noncaseating granulomas and peribroncial mononuclear cell infiltration with giant cells.
  The antigens are typically complex proteins, which can come from several sources, including agricultural dusts, thermophilic fungi, and bacteria.
- **Forms :**

1- Acute form :

- most easily identified, results after **a large exposure to an inciting antigen**.
- The patient will develop **fevers, cough, and fatigue**, typically within 12 hours of exposure.
- Physical examination will reveal **inspiratory crackles**.
- Chest radiography can demonstrate **diffuse micronodular disease** but may be normal.
- HRCT will demonstrate **diffuse centrilobular micronodules and ground-glass opacity.**
- After removal from the offending antigen, **symptoms will resolve** within approximately 48 hours.

2- Subacute and chronic form :

- occur after more **prolonged lower level antigen exposure.**
- Bird fanciers disease is an example of a chronic disorder. These patients have a chronic low-level exposure to avian antigens within the home and will ultimately experience **cough, fatigue, weight loss, and shortness of breath**.Similar to the acute form, the HRCT will show micronodules and ground-glass opacities, **but there is also evidence of septal line thickening and fibrosis.**
- In its most severe and chronic form, significant traction bronchiectasis and honeycomb changes will be evident.
- **Removal of exposure** to the offending antigen is essential in the treatment of HP.
- **Glucocorticoids** are often used for those with more severe symptoms. Response to this therapy is variable.

Idiopathic Pulmonary Fibrosis

- most common idiopathic form of DPLD.
- It typically presents in patients between 50 and 70 years of age who have a greater than 6-month duration of a **dry cough and dyspnea on exertion**.
- History will reveal no potential cause for the development of fibrosis.
- Lung examination is noticeable for Velcro inspiratory **crackles** that are predominant **at the bases** and may be subtle in early disease.
- **Clubbing** is present in up to 50% of patients**.**
- The diagnosis of IPF is challenging because it is uncommon and indolent.

- **The best diagnostic test is HRCT**, which may show abnormalities ,such as bilateral, peripheral, and basal predominant septal line thickening with honeycomb changes.
- IPF is progressive, with a median survival of 3 to 5 years after diagnosis.
- FDA-approved therapy with **nintedanib and pirfenidone** decreases the rate of progression of idiopathic pulmonary fibrosis but is not curative.

Asbestos-Related Lung Disease

- **Affected basal part of lung**
- Asbestos includes a group of minerals that, when crushed, will break into fibers. These fibers are chemically heterogeneous hydrated silicates that are used in industry because of their high tensile strength, heat resistance, and acid resistance.
- In the past, asbestos fibers were widely used in insulation, brake linings, flooring, cement paint, and textiles.
- **Asbestos-associated diseases have a prolonged latency period (15 to 35y)**
- **Duration and extent of exposure** are key risk factors for the development of disease.
- The most common symptom is **exertional dyspnea.**
- Findings of parietal **pleural calcifications or plaques** on chest radiograph should alert the clinician to the possibility of asbestos exposure.
- Most patients with pleural plaques are asymptomatic.
- Asbestos exposure increases the risk for development of lung cancer regardless of smoking status, but the risks are substantially higher in smokers, also risk of mesothelioma (pleural malignancy) .

Silicosis

- Silicosisis a fibrotic lung disease caused by the **inhalation of silica dust.**
- **Affected apical part of the lung**
- It is associated with altered cell immunity and **macrophage function**. Patients with silicosis are at increased risk for the development of mycobacterial infection and connective tissue disease.

مثلا مريض شخصنا ب silicosis وضل ماشي على symptomatic ttt for 3 years واجاك على العيادة وحكالك بالأسابيع لأخيرة بلشت الأعراض تسوء هون لازم تفكر انه صار عنده superinfection ويلي هو غالبا رح يكون ال TB

- Once fibrosis develops in silicosis, there is little evidence that any therapies alter disease course.
- If individuals have continued exposure, removal from the environment will prevent further lung injury.
- Silica exposure is associated with increased risk of lung cancer, particularly for smokers. Smoking cessation remains an essential intervention.
- **There are four main types of silicosis:** depend on the amount of silica and how acute manifestation .
  1- **acute silicosis:** large amount of exposure , manifestation within weeks to month , mainly cough and SOB , sever consolidation / fibrosis on chest x rays **.**
  2- **chronic simple silicosis:** asymptomatic , exposure to small amount over years , nodules on x rays or HRCT .

3- **chronic complicated silicosis:**

الفكرة هون إنه ال nodules ممكن يتجمعوا مع بعض وبيعطينا large nodules وببلش هدول المرضى يصير عندهم symptoms

4- **accelerated silicosis:**

هاي نفسها ال chronic بس بتصير ب shorter time

Diagnostic Approach and Evaluation

Symptoms

❖ **Nonproductive cough** and **dyspnea** are the most common presenting symptoms of a DPLD.

❖ Dyspnea that comes on suddenly and is of short duration is more likely due to respiratory infection, asthma, pulmonary embolism, or heart failure than DPLD.

❖ In contrast, patients presenting with **subacute or chronic dyspnea** lasting weeks to months without response to treatment should be evaluated for DPLD.

❖ As opposed to the **typical nonproductive cough** of DPLD, a long history of cough with sputum production can suggest an underlying chronic infection, airways inflammation such as chronic bronchitis, or bronchiectasis.

HISTORY

❖ When DPLD is suspected, questions should focus on determining the onset of symptoms, the disease course (improving or worsening), medications, and exposures.

❖ The most common identifiable etiologies of DPLDs are those associated with **exposures**, and the history should include a thorough review of occupations, home environment, hobbies, and other activities.

❖ **Medication** review should include current medications as well as those taken before the onset of symptoms.

❖ **Connective tissue diseases** can lead to the development of DPLD; therefore, the review of systems should assess for symptoms of arthralgia, myalgia, arthritis, tenosynovitis, dry eyes, dry mouth, dysphagia, gastroesophageal reflux, and unexplained rash.

❖ **A family history of DPLD** due to connective tissue disease should substantially increase clinical suspicion.

PHYSICAL EXAMINATION

❖ Physical examination **findings differ depending on the underlying cause of DPLD**.

❖ In patients with connective tissue disorders, findings may include Raynaud phenomenon, skin thickening, sclerodactyly, malar rash, inflammatory arthritis, or tenosynovitis.

❖ **Lung examination** findings are variable and may be normal. This is more likely early in disease or in those with imaging findings of ground-glass opacity or micronodules .

❖ Decreased breath sounds and dullness to percussion may suggest a pleural effusion, which is atypical for many DPLDs.

❖ Wheezes may suggest small airways disease, while inspiratory dry "Velcro" crackles are more suggestive of fibrosis.

❖ The physical examination should include **resting and exertional pulse oximetry**.It is common for patients with DPLD to have normal resting pulse oximetry.

❖ individuals with DPLD will often demonstrate desaturation when ambulating. Desaturation of greater than 4 % while ambulating is consistent with adiffusion limitation, which is a hallmark of interstitial lung disease.

INVESTIGATION

Patients with a clinicalsuspicion of DPLD should undergo full pulmonary function testing, including lung volumes and DLCo.

The vast majority of DPLDs have restrictive physiology.

❖ **Serologic testing** for diffuse parenchymal lung disease is most appropriate in young patients, those with symptoms of rheumatologic disease, or those with a family history of rheumatologic conditions.

IMAGING

❖ Plain chest radiography is an appropriate initial test for the evaluation of dyspnea and cough in patients suspected of having DPLD.

❖ Chest radiography may show various findingsin patients with DPLD, including diffuse reticular and reticulonodular patterns, increased septal line thickening, consolidation, pleural effusions with or without pleural calcification, bronchiectasis, and hilar or mediastinal lymphadenopathy.

❖ Chest radiograph can be normal in patients with minimal disease, and a normal chest radiograph does not rule out DPLD.

❖ High-resolution CT (HRCT) scan of the chest (slice thickness 1-2 mm) is the best imaging study to identify abnormalities that can help diagnose the underlying disease.

❖ **The findings on HRCT highly correlate with the histopathology identified on open lung biopsy.**

❖ **The diagnosis of idiopathic pulmonary fibrosis can be made without lung biopsy based on the results of HRCT.**

Pneumonia – seminar

بهاي السمينار الدكتور بعد ما خلصوا الطلاب شرح حكى كم شغلة مهمة عن الموضوع وهمه يلي كتبتهم هون .

Classification of pneumonia

According to microorganism (infectious / non infectious)

وفيه عنا كثير factors بتلعب ور بنوع ال pneumonia يلي رح يكون عند المريض مثلا ازا كان immunocompromised

Also according to clinical classificatioى

- community acquired
- nosocomial : hospital acquired , ventilator acquired and healthcare – associated pneumonia

when assess the patient with pneumonia ---there is risk factors for certain pathogen , for example :

klebsilla وخصوصا ال more risk for aspiration بكون عند heavy alcohol use بالمريض يلي بكون
وبمريض ال COPD بنتوقع يكون عنده PSUDOMONAS أو H.influenza أو legionella
وكمان بالنسبة لل age بكون عندهم risk not only to bacterial pneumonia وانما أحيانا بكون عندهم
bacterial infection secondary to viral infection وعشان هيك مهم بالهستوري تعرف إزا المريض اخد ال influenza vaccine ولا لا ؟ وكمان مهم تسأل عن ال animal contact or exposure فمثلا ال
chlamydia ممكن تكون related to Birds وهكذا وكمان legionelella هي atypical وبتكون drinking water containing Legionella

- **severity of pneumonia**
  1- **CURB65**
  CURB65 criteria for hospitalization
  • Confusion (new onset)
  • BloodUrea nitrogen greater than 19mg/dL
  • Respiratory rate of 30 or greater
  • SystolicBlood pressure < 90 mmHg systolic
  • Or diastolic blood pressure < 60 mmHg
  • Age65 or older
  • CURB65 > 2 = hospitalize (2 inpatient /+3 ICU)
  2- **Pneumonia severity index (PSI) or PORT Score موجود الجدول بكتاب ستب اب**
  3- **Criteria by ATS = American Thoracic Society and IDSA = Infectious Diseases Society of America.**

وهدول التلاتة بشتركوا بال age و presence of comorbidities و ال vital sign تحديدا ال
(HR + RR+ BP+T وبرضه ال PHYSICAL EXAMINATION وتحديدا ال CNS زي ال alter
أو ال mental status أو ال confusion وبرضه عندك اللاب زي ال urea أو ال leukocytosis أو ال
leukopenia أو po2 بال ABG
وكل هدول الشغلات بخلوك قادر على تحديد ال SEVERITY وكمان بخلوك تعرف وين ممكن ادخل المريض فمثلا
ممكن يجي على الطوارئ وتروحه as outpatient وممكن يكون inpatient / non ICU وممكن يكون
. inpatient in ICU

وك RADIOLOGICALLY ممكن يكون عنا أو lobar pneumonia أو multiple infiltrative
أو bronchopneumonia interstitial infiltrates يلي بنشوفها بال mycoplasma pneumonia
أو ممكن تكون lung abscess, cavitary lung lesion

عادة ازا كان عندي بالهستوري aspiration والمريض صار عنده right lower lobe pneumonia هون بتخلينا
نفكر بال anaerobes
لو كان فيه Empyema فهون ممكن يكون عندي staph أو strep
لو كان cavitary أو abscess هون ممكن تكون staph

- **complications of pneumonia**
  1- **pulmonary complication :**non resolving pneumonia **,** pleural effusion , pleural empyema , ARDS, respiratory failure , Lung abscess.
  2- **Neurological complication :** delirium , confusion , decrease level of consciousness
  3- **Bone marrow can be affected ,** Thrombocytopenia, thrombocytopenia
  4- **Heart -- ischemia --- acute kidney injury**
  5- **Septic shock**

ركزوا على ال microbiology بحيث تعرف شو ال most likely microorganism وفيه بعضهم بكون اله
specific feature : مثلا ال presentation of atypical mycoplasma بيجي معها ال hemolytic
anemia وال legionella بيجي معها elevation in liver enzymes وبكون مرات عندهم
. hyponatremia

Sarcoidosis – seminars

هاد السمينار انشرح مع الدكتور جعفر رح أحط الأسئلة يلي سألها للطلاب أو أي اشي علق عليه

Lofgren's syndrome

acute form of sarcoidosis characterized by a traid of:
1)erythema nodosum
2)Polyarthalgia(Arthritis)
3)Bilateral Hilar Lymphadenopathy

staging of sarcoidosis on cxr : فيه عليهم صور بالسمينار شوفوهم مهم لل ميني أوسكي
stage 1: bilateral hilar adenopathy without parenchymal infiltrates (highest rate of remission)
stage 2 : hilar adenopathy with parenchymal infiltrates
stage 3 : diffuse parenchymal infiltrates without hilar adenopathy (least favorable prognosis)
stage 4 : pulmonary fibrosis with honeycombing and fibrocystic parenchymal changes

رح أحط رابط السمينار شوفوا الصور
https://docs.google.com/presentation/d/19eGN5Q1r6ZxouiqMHI1VFVa6PwzmBuEg/edit?usp=sharing&ouid=117760599540334916618&rtpof=true&sd=true

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TB – SEMINARS

هلأ عنا واحد من ال diagnostic test يلي هو interferon gamma release assay هاد يدل على وجود ال infection وعادة يستخدم في ال latent TB وببعض الأحيان لما يكون عندي ال Mantoux tuberculin skin test (TST) --- false positive وخصوصا ازا كان المريض اخد BCG vaccine فلازم تشيك بال (IGRA) .

وبالنسبة لموضوع ال INFECTION CONTROLES مهم تعرف إنه ال mode of transmission هو airborne
ال BCG vaccine بكون contraindication لل pregnancy لأنه هو live attenuated vaccines

When we stope isolation ?

هلأ المرضى يلي عندهم TB بضل ماشي على ال medication in hospital لمدة اسبوعين بعده بنرجع نعمله Sputum culture for acid fast bacilli وبنعمل 2 consecutive sample خلال 48 ساعة إزا طلع negative والمريض كان afebrile وما عنده respiratory symptoms especially cough هون بنقدر نعمل stop isolation وبيقدر المريض يكمل ال regimen لمدة معينة حسب البروتوكول يلي الفريق الطبي حطله اياه.

دائما بالاضافة لل history والفيزيكال بدك تعرف هل المريض جاي من منطقة فيها risk for TB ولا لا

ذكروا الطلاب بالسمينار لمحة تاريخية عن المرض بأول سلايدين و الدكتور مدح فيها وحكى إنه حبها فمروو عليها
هي رابط السمينار
https://docs.google.com/presentation/d/1n5z0L7R1kf1o_5JN3V3XGmb7XCKuQDUD/edit?usp=sharing&ouid=117760599540334916618&rtpof=true&sd=true

pulmonary HTN

Resting mean PAP of 25 mm hg or greater , or >=30 mm hg with exercise
• Leads to RV failure and may directly contribute to death
• Pathophysiology determined by the specific cause , vast majority due to left sided heart disease(volume and pressure overload) and hypoxic respiratory disorders(vasoconstriction)
وفيه عنا حالات ما بكون الها سبب بنسميها Idiopathic pulmonary arterial hypertension . ومهم كتير تعرف السبب عشان تعرف شو هي ال management .

We have 5 groups according to underlying cause :
Group 1 : pulmonary arterial hypertension /(idiopathic) PHT
وهدول المرضى الهم management مختلفة عن باقي المرضى ممكن أستخدم معهم VD. وبعد ال right heart بعمل اشي اسمه cath VD test وازا كان المريض عنده استجابه اله بعدها بقدر أعطيه ال VD
Group 2 : Pul HTN due to left sided heart disease. So management of right sided HF can improve these patient
Group 3 : due to lung disease and hypoxia (COPD , ILD, others with mixed restrictive and obstructive , sleep disorders , alveolar hypoventilation , high attitude developmental abnormalities
Group 4 : chronic thromboembolic pul HTN
هدول المرضى بصير عندهم pulmonary embolism وبتكون ا chronic and recurrent وبالتالي بصير عندي as secondary -- PH وهدول بتكون ال management تبعتهم عن طريق إني أعطيهم anticoagulation
Group 5 : unclear or multifactorial causes
- Hematologic disorders (myeloproliferative , splenectomy)
- Systemic disorders (sarcoidosis , vasculitis ..)
- Metabolic disorders (thyroid , glycogen storage dis ..)
- Others : tumoral obstruction , fibrosing mediastinitis , ckd on dialysis

• Group 2-5 : treat underlying cause
• Group 4 (CTEPH) : anticoagulation and possible surgery
• PAH : vasodilators , rt heart cath , vasidilator test ,

Diagnosis
• Mainly present with SOB and exercise intolerance.
• Physical signs – right ventricular hypertrophy (lift parasternal heaves) , Loud p2 , graham steel murmur due to pulmonary regurgitation.
• ECHO, right heart cath to confirm dx

Venous thromboembolism- seminar

هلأ Anticoagulation therapy المتوفرة لهدول المرضى هيه ال novel oral + Heparin
warfarin + anticoagulants (NOACs)
وبشكل عام بنستخدم ال LMWH ما بنستخدم ال Unfractionated heparin لأنه بنعطى IV وبده
mentoring كل 6 ساعات .

ال surgical option / endovascular surgical option للمرضى يلي عندهم DVT أو PE بس بتكون
للمرضى يلي عندهم sever massive DVT زي الناس يلي عندهم Phlegmasia cerulea dolens (PCD)
وبالاضافة للمرضى يلي عندهم massive PE

ال massive PE هي يلي بتعملك hemodynamic instability وليس لها علاقة بحجم ال PE
يعني حجمها ما اله دخل بتحديد هل هي MASSIVE ولا لا ، وإنما يلي بحددلك هو ال effect on
hemodynamic instability

يعني لو صار عنده hypotention هون بتعتبرها massive وال mangment بهاي الحالة رح تكون
thrombolytic therapy وازا ما كانت كافية ممكن نلجأ لل others endovascular ttt
ودور ال surgery بهاي الحالات محدود .

Pulmonary embolism – seminar

ال Westermarck's sign هي نفسها يلي بنسميها oligemia يعني عندي less blood vessels

ملاحظة : إزا المريض ما عنده hypoxia --- this is not role out PE
يعني مش كل PE رح يجيك ب hypoxia

Conventional pulmonary angiography هاد ما بنستخدمه الا في حالة
patient is hemodynamically unstable and embolectomy may be required.

هاد السمينار الدكتورة كتير مدحت فيه وحكت ممكن تعتمدوه ك مصدر للدراسة .