

Shortness of breath

Emergency medicine



What is shortness of breath?

- AKA dyspnea, it is the subjective feeling of difficult, labored, or uncomfortable breathing. There is no single pathophysiologic mechanism that causes dys-pnea and its etiology may be secondary to pulmonary, cardiac, or neurological dysfunction.



Etiology of shortness of breath

Table 18. Differential Diagnosis for Dyspnea

	High Mortality/Morbidity	Usually Less Emergent
Respiratory	Airway obstruction (foreign body, epiglottitis, abscess, anaphylaxis) Pneumo/hemothorax Gas exchange – pulmonary edema, PE, pneumonia, Acute exacerbations of COPD	Chronic obstructive, interstitial or restrictive lung disease Pleural effusion
Cardiac	CHF, MI, valvular disease, tamponade, arrhythmia	Chronic CHF, angina
Metabolic	Metabolic acidosis NYD, carbon monoxide inhalation	Anemia, Hemoglobinopathy
Neuromuscular	Myasthenia gravis, diaphragmatic paralysis	CNS lesion, primary muscle weakness
Other		Anxiety, deconditioning

Clinical features

- The initial assessment of any patient with dyspnea should be directed toward identifying respiratory failure, which requires more immediate action. Dyspnea alone is a subjective complaint often difficult to quantify; therefore, objective findings are often needed to aid in diagnosis. Assess for signs of impending respiratory failure which include tachycardia, tachypnea, stridor, the use of accessory respiratory muscles, lethargy, agitation, altered mental status, and inability to speak due to breathlessness. If present, oxygen should be administered immediately. Early use of noninvasive ventilation is often helpful in reversing the downward trajectory, although the need for aggressive airway management and mechanical ventilation should be anticipated.



History and physical examination

- acute SOB is often due to a relatively limited number of conditions; associated symptoms and signs are key to the appropriate diagnosis
- substernal chest pain with cardiac ischemia
- fever, cough, and sputum with respiratory infections
- urticaria with anaphylaxis
- wheezing with acute bronchospasm
- environmental or occupational exposures (patient profile)
- dyspnea may be the sole complaint and the physical exam may reveal few abnormalities (e.g. PE, pneumothorax)
- check vitals including pulse oximetry (rapid but insensitive screen for disorders of gas exchange)
- wheeze and stridor (airway) vs. crackles (parenchymal), JVP, and murmurs



Investigations

- blood work:
- CBC and differential (hematocrit to exclude anemia), electrolytes, consider ABG/VBG
- serial cardiac enzymes and ECG if considering cardiac source
- Wells scores to consider appropriateness of D-dimer
- imaging:
- CXR (hyperinflation and bullous disease suggestive of obstructive lung disease, or changes in interstitial markings consistent with inflammation, infection, or interstitial fluid)
- CT chest usually is not indicated in the initial evaluation of patients with dyspnea, but can be valuable in patients with interstitial lung disease, occult emphysema, or chronic thromboembolic disease (i.e. PE)



Emergency Department Care and Disposition

- the history and physical exam lead to accurate diagnoses in patients with dyspnea in approximately two-thirds of cases; the decision to admit or discharge should be based on the underlying disease process identified.
- The goal of oxygen therapy is to maintain the PaO_2 above 60 mm Hg or the oxygen saturation above 90%. Lower PaO_2 , or saturation may be appropriate in those with longstanding lung disease such as chronic obstructive pulmonary disease (COPD).
- After oxygenation has been ensured with an appropriate delivery device (nasal cannula, facemask, non-rebreather, BVM, NIV, or intubation) and the patient is stabilized, disorder-specific treatment and evaluation can be pursued.
- Patients with hypoxia and an unclear cause of dyspnea will require hospital admission.



Emphasis on:



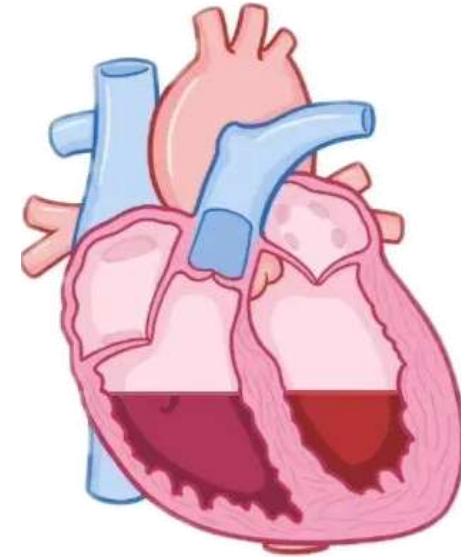
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- Congestive heart failure : is a chronic condition in which the heart is unable to pump blood effectively, leading to fluid buildup in the lungs(precipitates pulmonary edema) and other tissues.
 - Asthma: A chronic condition leading to swelling and narrowing of airways.
 - COPD: Chronic obstructive pulmonary disease caused by Smoking, pollutants or genetics.

Congestive heart failure and pulmonary edema

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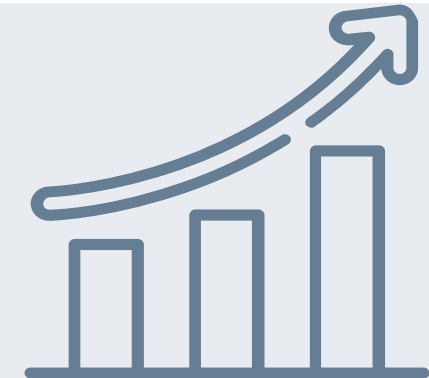
Introduction

CHF is a clinical syndrome that occurs when cardiac pump function does not adequately meet the circulatory demands of the body

Pulmonary edema should be conceptualized as an extreme on the continuum of severity of CHF and is often an acute worsening of chronic heart failure.

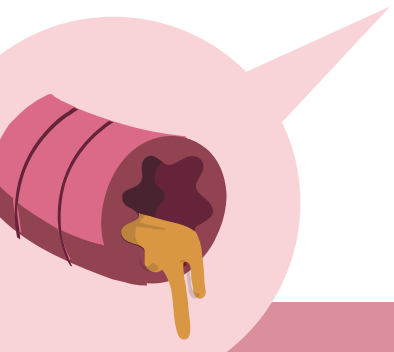
Cardiovascular diseases, one of the main NCDs, cause 42% of these deaths. In 2019, data from Jordan's national STEPwise survey for NCDs risk factors.

- In Jordan, noncommunicable diseases (NCDs) pose a heavy burden on health as they are responsible for nearly 80% of all deaths.



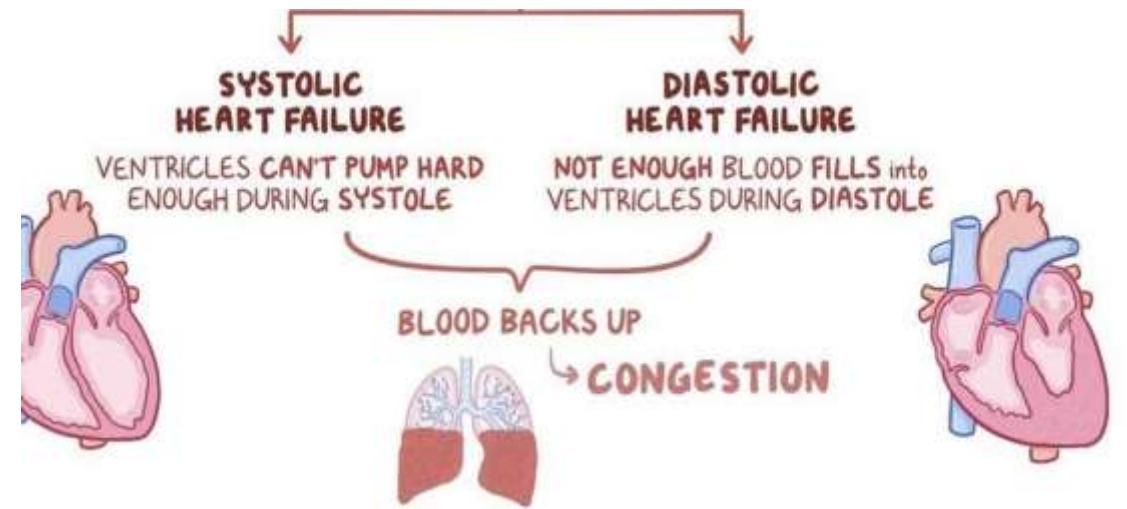
Pathophysiology

cardiac output decreases, reflex arterial vasoconstriction redistributes blood flow so that the brain and heart remain well perfused. The resulting drop in renal blood flow activates the renin-angiotensin-aldosterone system, causing increased sodium and fluid retention. Decreased cardiac output also results in an increase in left atrial and left ventricle filling pressures, which is in turn reflected in elevated pulmonary capillary pressures resulting in pulmonary edema which impairs base exchange and increase respiratory drive. The consequence of these elevated pressures is the transudation of protein-poor fluid into the pulmonary interstitium and alveolar spaces. As the right ventricular volume and pressure increases, the systemic venous and capillary pressures rise and fluid transudates from the vascular to the interstitial space causing peripheral edema.



Pathophysiology

Heart failure can be subdivided into systolic and diastolic dysfunction. Systolic dysfunction is characterized by a dilated left ventricle with impaired contractility. Diastolic dysfunction refers to an increase in ventricular stiffness (reduced compliance) and impaired relaxation that impedes ventricular filling during diastole. The net effect of diastolic dysfunction is an increase in end-diastolic pressure.

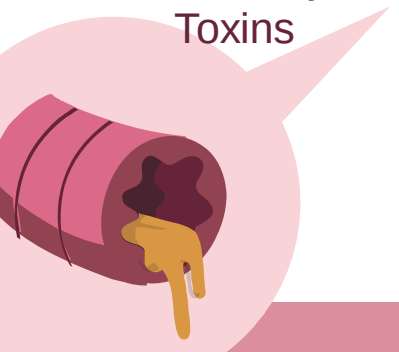


Etiology

Remember that acute CHF is not the primary problem; it is the consequence of an acute decompensation due to an underlying cause.

Causes of CHF:

- Coronary artery disease
- Hypertension
- Valvular heart disease
- Cardiomyopathy (dilated, hypertrophic, restrictive)
- Pericardial disease
- Metabolic disorders (e.g. hypothyroidism)
- Viral myocarditis
- Toxins



Precipitating factors

FAILURE

1. Forgot medication
2. Arrhythmia (Dysrhythmia)/Anemia
3. Ischemia/Infarction/Infection
4. Lifestyle (e.g. high salt intake)
5. Upregulation of cardiac output
6. (e.g. pregnancy, hyperthyroidism)
7. Renal failure
8. Embolism (pulmonary)

Cardiac

Ischemia/infarction

Tachy- or bradyarrhythmias

Mechanical complication of acute MI (e.g. papillary muscle rupture)

Medications

Negative inotropes (beta-blocker, calcium channel

blocker)

NSAIDS

Steroids

Pharmacological non-compliance

Dietary

Increased sodium intake

High output

Anemia

Infection

Pregnancy

Hyperthyroidism

AV fistula

Iatrogenic fluid overload (e.g. blood transfusion)

Renal failure

Hypertensive crisis

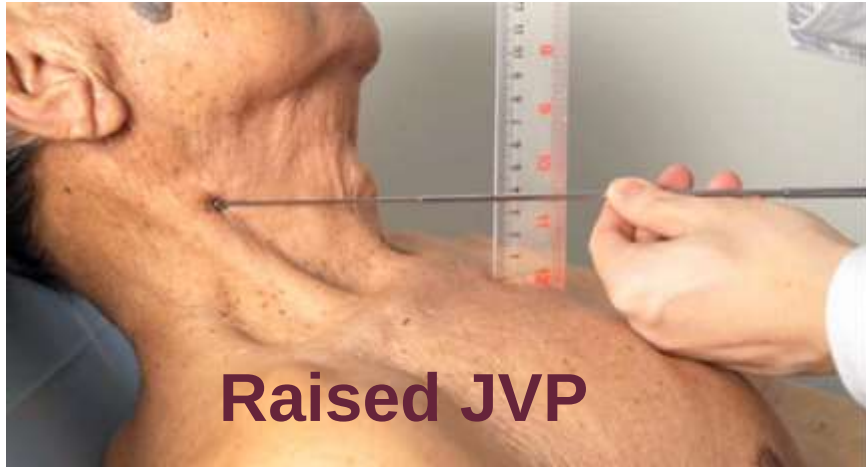
Symptoms.

- Dyspnea
- Orthopnea, paroxysmal nocturnal dyspnea
- Cough, wheezing
- Fatigue, generalized weakness
- Edema of lower limbs, increased abdominal girth

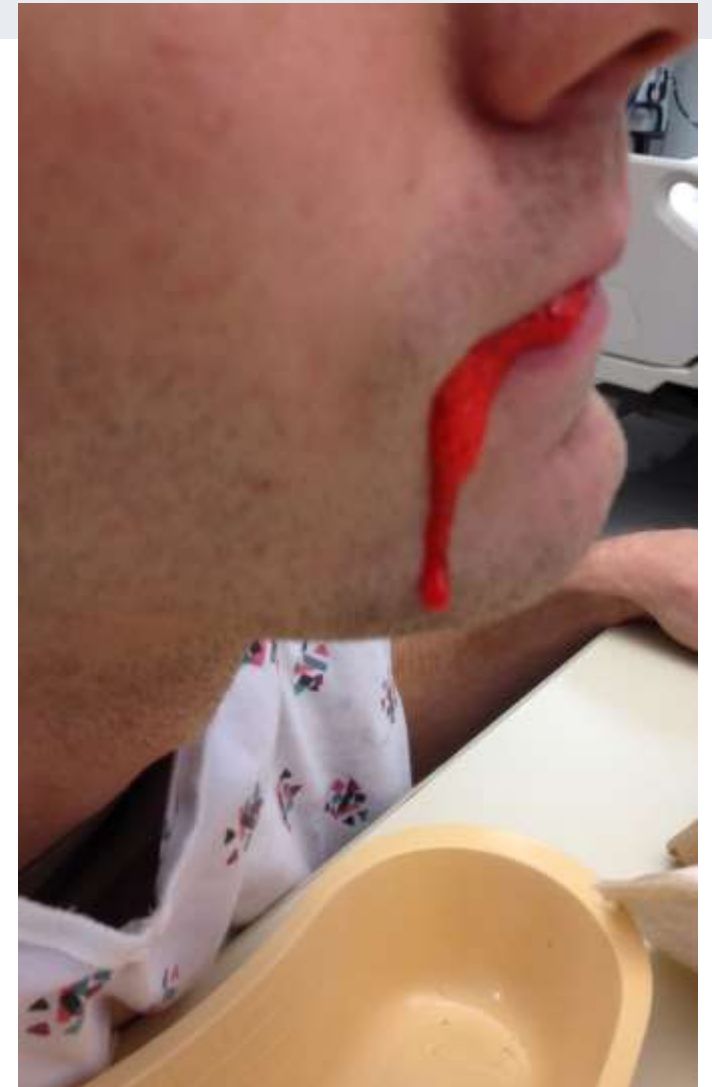
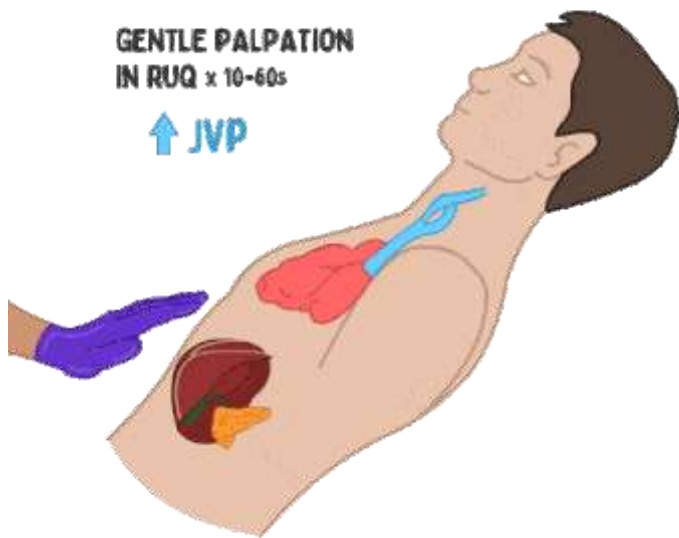


Sign(physical examination)

- Tachypnea, tachycardia, hypertension or hypotension, hypoxia
- Crackles, wheezes
- S3, S4
- Jugular venous distension, hepatojugular reflux
- Hepatomegaly
- Dependent edema
- Weak peripheral pulses, cool extremities, i.e. hypoperfusion



HEPATOJUGULAR REFLUX



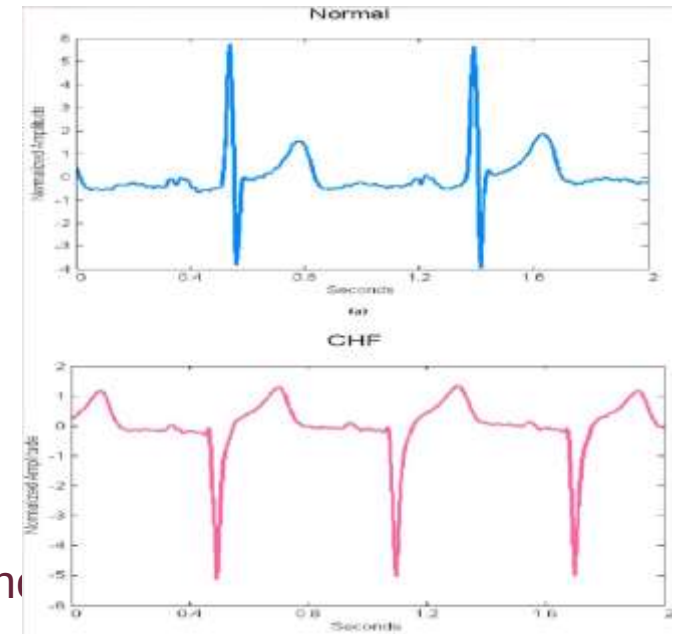
Investigations

- Complete Blood Count (CBC): Checks for anemia or infection.
- • Electrolytes and Renal Function Tests: Assess sodium, potassium, creatinine, and blood urea nitrogen levels
- Chest X-Ray
 - cardiomegaly,
 - increase in prominence of upper lung fields (pulmonary vascular redistribution),
 - interstitial edema and obliteration of vasculature structures,
 - kerley B lines (1-2 cm lines perpendicular to the pleura and continuous with it, representing edematous septal lines),
 - pleural effusions (usually absent in acute pulmonary edema).



Investigations

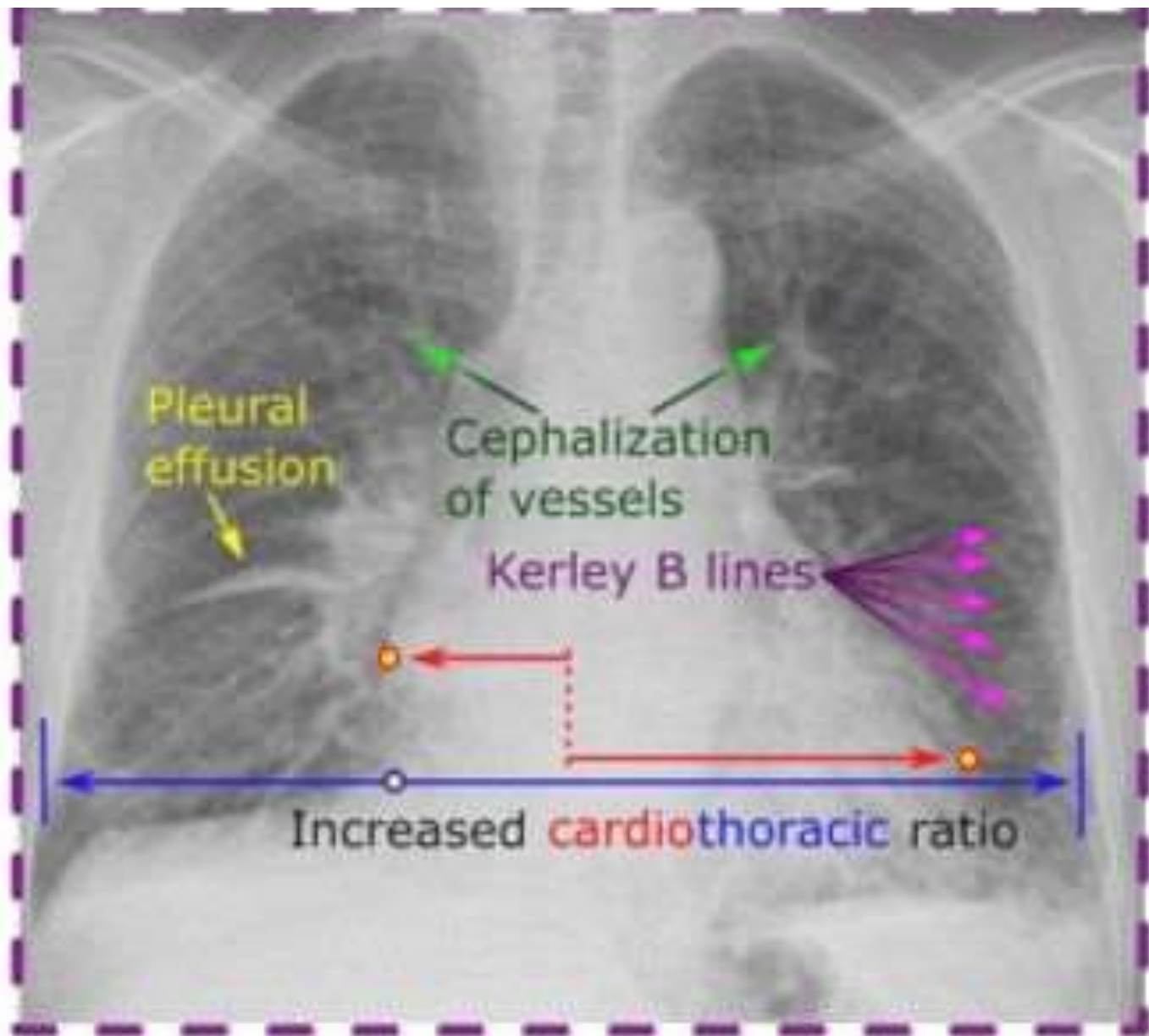
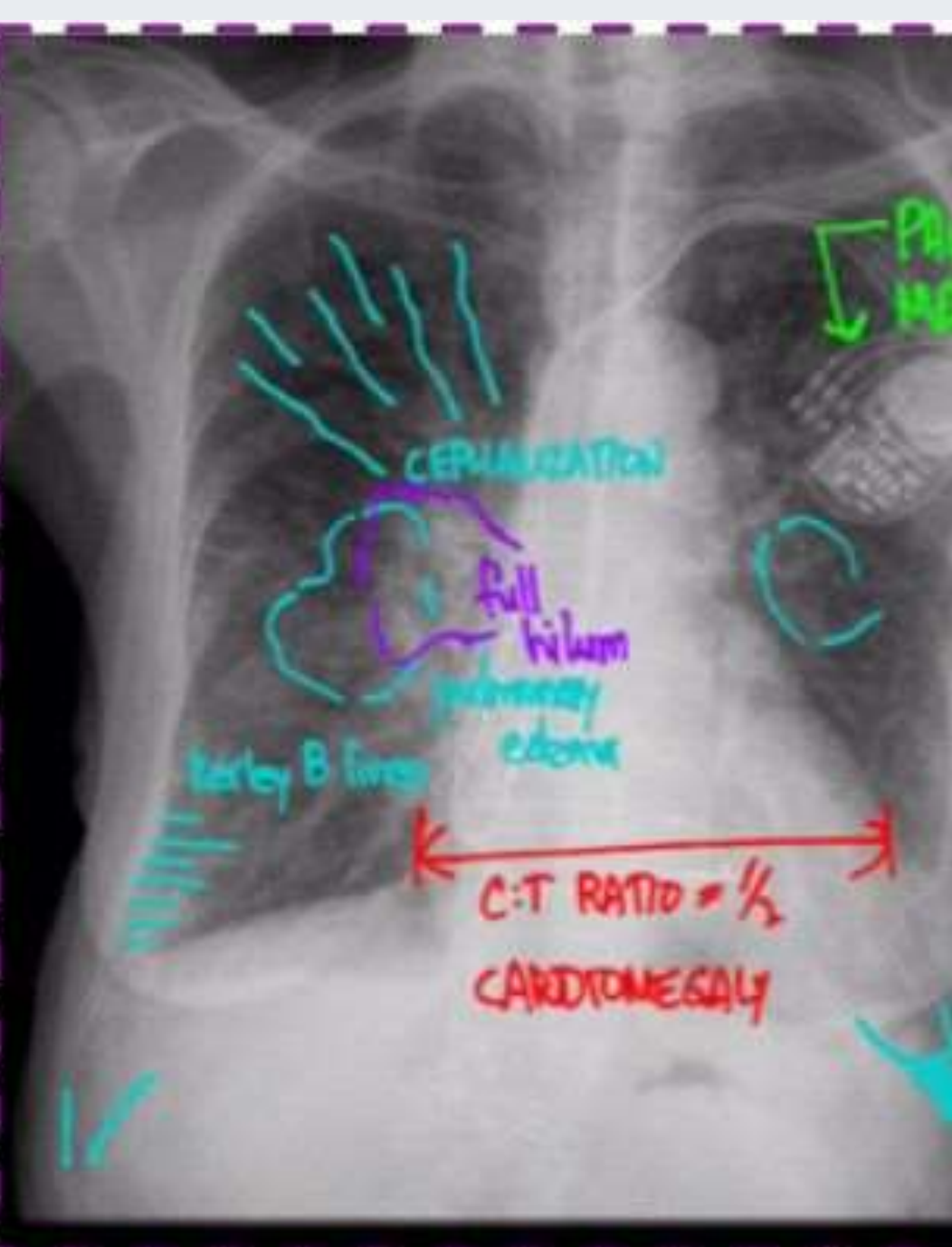
- ECG
 - left ventricular hypertrophy
 - left atrial abnormalities,
 - myocardial ischemia, or
 - the presence of an arrhythmia (e.g. atrial fibrillation).
- cardiac enzymes, brain natriuretic peptide
 - Troponins to rule out myocardial infarction.
 - Brain natriuretic peptide (BNP) and amino-terminal fragment of the pro-BNP are elevated in heart failure. These tests are most useful in patients whose clinical assessment and CXR are suggestive, but not clearly diagnostic of heart failure.
- Echocardiogram
 - Assess ejection fraction,
 - wall motion abnormalities,
 - valvular function.

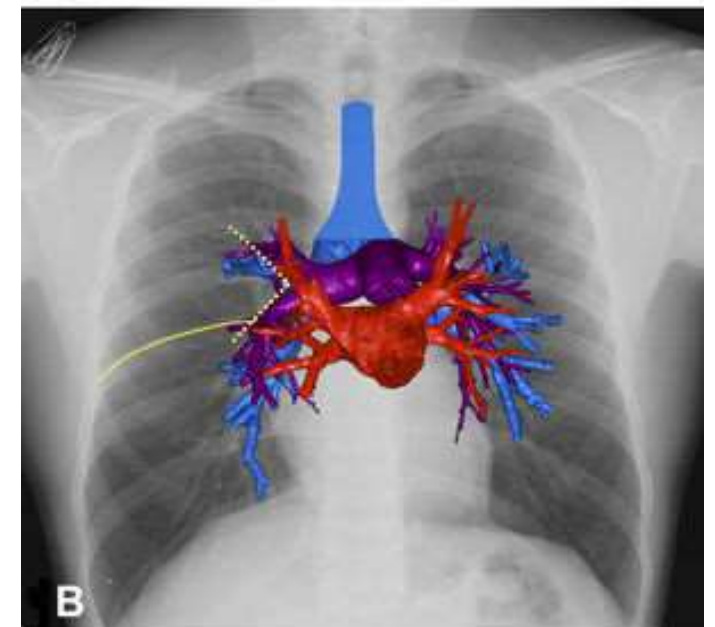
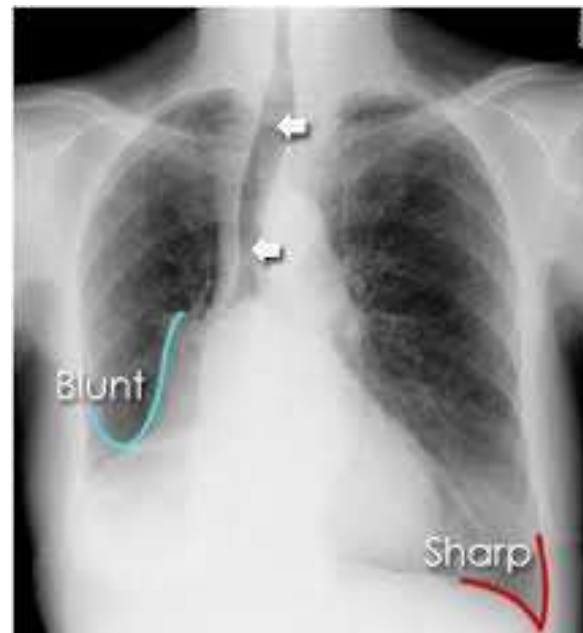
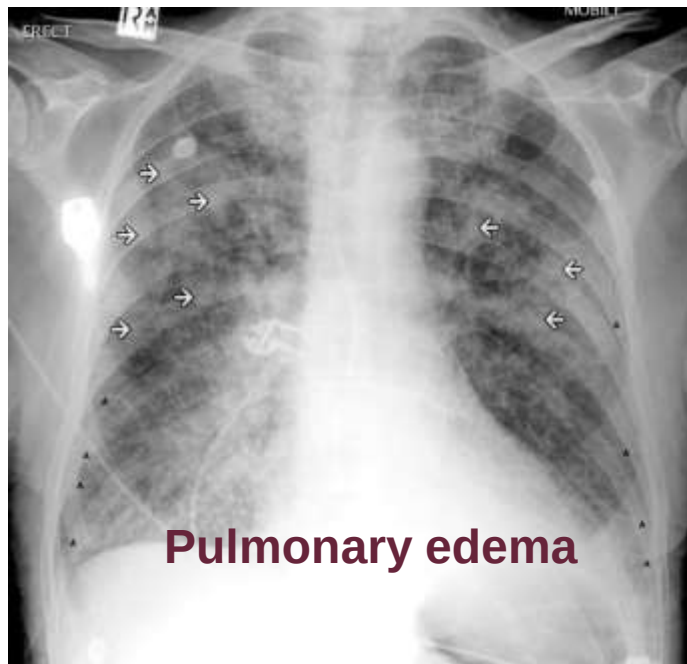
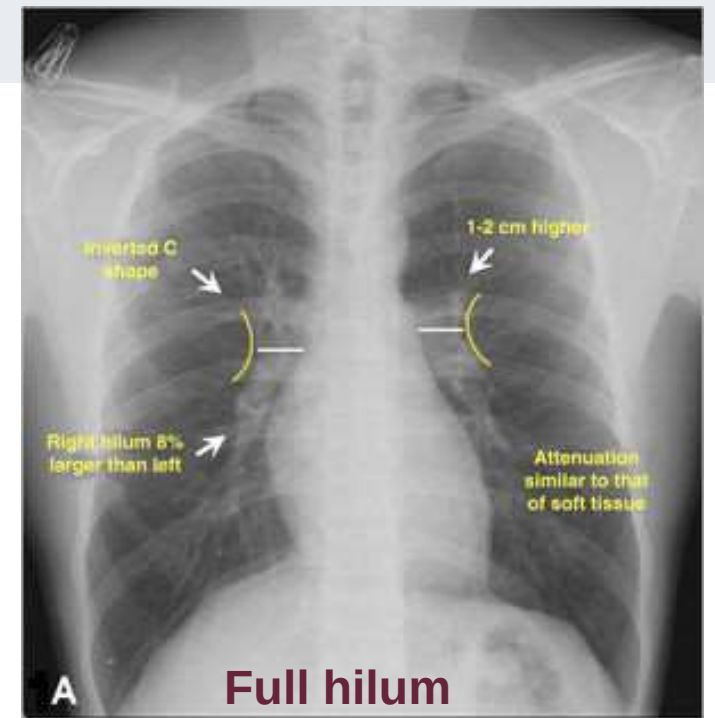
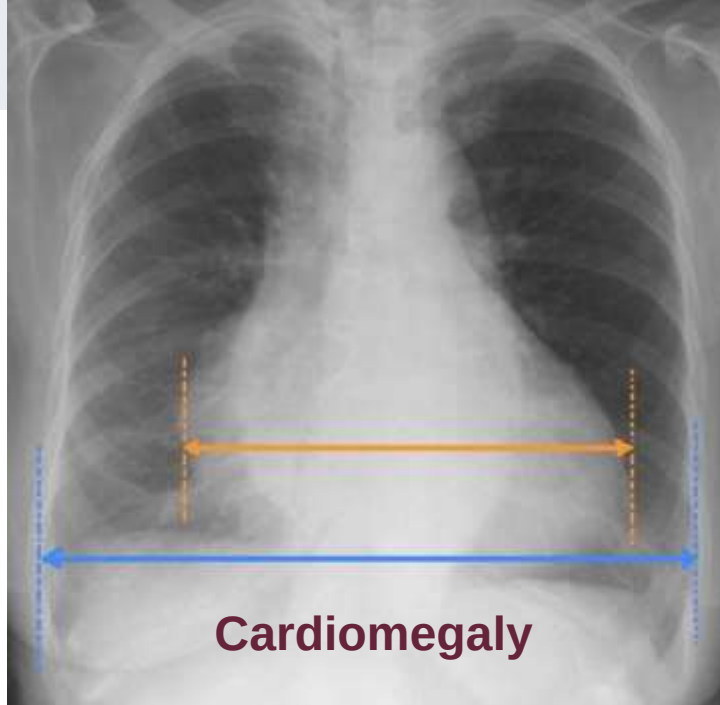


B-type natriuretic peptide

- hormone released primarily by the heart's ventricles in response to increased pressure and volume overload, because the heart can't pump blood efficiently . So it secrete more BNP to counteract the stress by promoting vasodilation, reducing blood volume, and encouraging sodium excretion.
- BNP levels (>100 pg/mL) suggest the presence of heart failure.
- elevated BNP can also occur in conditions such as renal failure, pulmonary hypertension, and acute coronary syndromes.
- NT-pro BNP: This is a more stable precursor of BNP and is also used for diagnostic purposes , > 300 pg /mL may suggest heart failure.





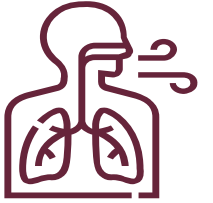


Management

Rescuing

- ABCs, may require intubation if severe hypoxia
- sit upright,
- Monitoring: cardiac monitoring, and continuous pulse oximetry
- IV access , Foley catheter due to diuretic therapy
- 100% O₂ by mask
 - if poor response or RDS, may require Bi-PAP or intubation medical



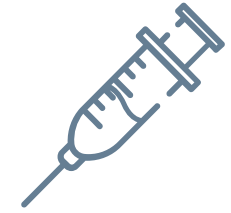


Management

Medication

- **diuretic** (if volume overloaded): furosemide 0.5-1 mg/kg IV.
- **vasodilators** (if sBP >100 mmHg): nitroglycerin 0.4 mg SL q5 min PRN +/- topical Nitrodur' patch
- (0.4-0.8 mg/h)
 - if patient not responding to treatment or showing signs of ischemia (angina): nitroglycerine 5-10 ng/min IV, titrate to response.
- inotropes/vasopressors (if sBP <90 mmHg)
 - without signs of shock: dobutamine 2.5 ug/kg/min IV, titrate up to sBP >90 mmHg
 - with signs of shock: norepinephrine 8-12 pg/min, titrate up to sBP >90 mmHg
- treat precipitating factor - eg. rate control (β -blocker, calcium channel blockers) or rhythm-control (electrical or chemical cardioversion) if new AFib
- Morphine (cautious use): Can help reduce anxiety, preload, and oxygen demand, though its use has become more selective due to risks of respiratory depression.

Management



Medication

- Diuretics and vasodilators are key treatments for acute congestive heart failure.
- Treatment intensity varies based on the patient's volume and hemodynamic status.
- **Hypertensive** flash pulmonary edema: Requires aggressive vasodilator therapy.
- **Normal blood pressure with volume overload**: Best treated with both diuretics and vasodilators.
- **Hypotension with intravascular overload**: May respond to diuretics alone or with vasopressors and inotropes for additional support.

Dobutamine is recommended in patients with hypotension who do not have clinical evidence of shock.

Dopamine is recommended in patients with hypotension who also have clinical evidence of shock.

Diltiazem may be used in acute CHF when rapid atrial fibrillation is also present

Hospitalizations

SHADOW HIM ACT

- S: Severe decompensated CHF
- H: Hypotension
- A: Altered mentation
- D: Dyspnea at rest
- O: Oxygen (hypoxia)
- W: Worsening renal function
- H: Hemodynamically significant arrhythmia
- I: ICD firings
- M: Major electrolyte disturbance
- A: Acute coronary syndrome
- C: Comorbid conditions (pneumonia, PE, DKA)
- T: TIA or stroke



Case 1: A 76-year-old man presents to the emergency department with complaints of severe shortness of breath, fatigue, and swelling in his legs. He has a history of hypertension and was diagnosed with congestive heart failure (CHF) one year ago. His current vitals include a heart rate of 110 beats per minute, blood pressure of 150/90 mm Hg, and an oxygen saturation of 88% on room air. On physical examination, he has jugular venous distention, bilateral pitting edema, and crackles in both lungs. Which of the following should be the primary focus in his immediate management?

- a) Administering oxygen and starting IV diuretics
- b) Checking his BNP levels
- c) Administering an ACE inhibitor
- d) Obtaining a chest X-ray



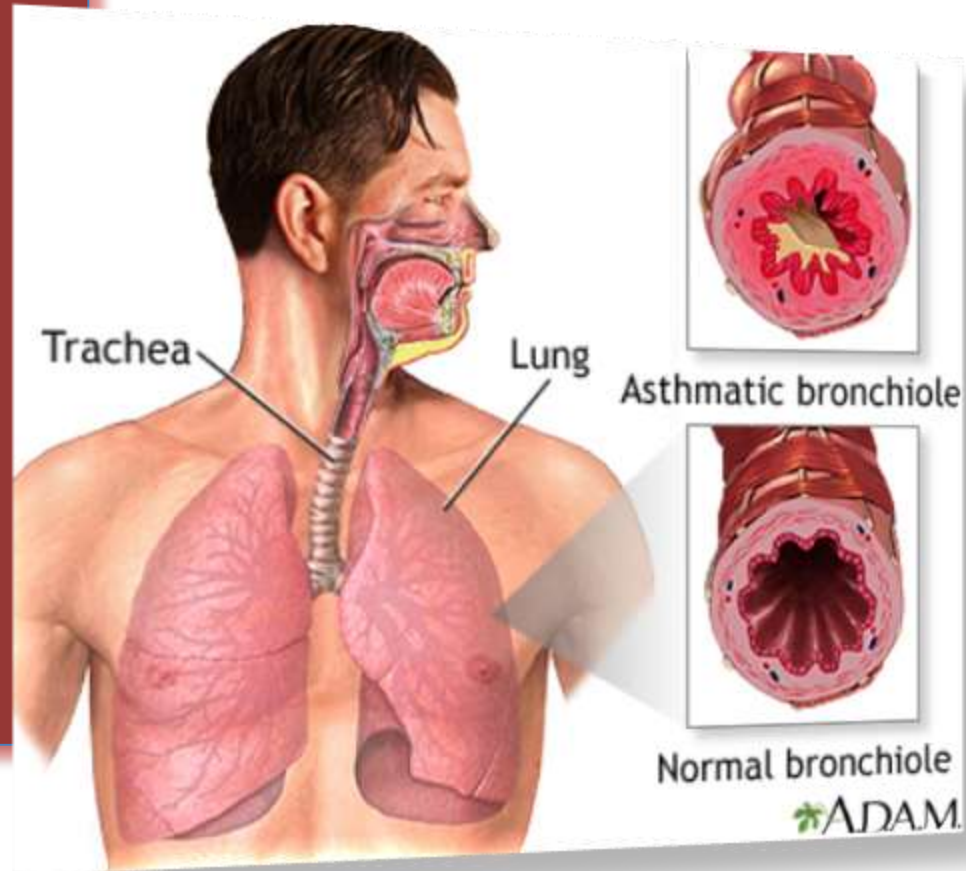
A 58-year-old man presents to the ED with progressive dyspnea over the course of 1 week. Upon arrival, he is able to speak in full sentences and states that he stopped taking all of his medications recently. Initial vitals include a HR of 92 beats per minute, a BP of 180/100 mm Hg, and an RR of 16 breaths per minute with an oxygen saturation of 94% on room air. Upon physical examination, the patient has bibasilar crackles, jugular venous distention, and pedal edema. Which of the following medication regimens was the patient most likely on?

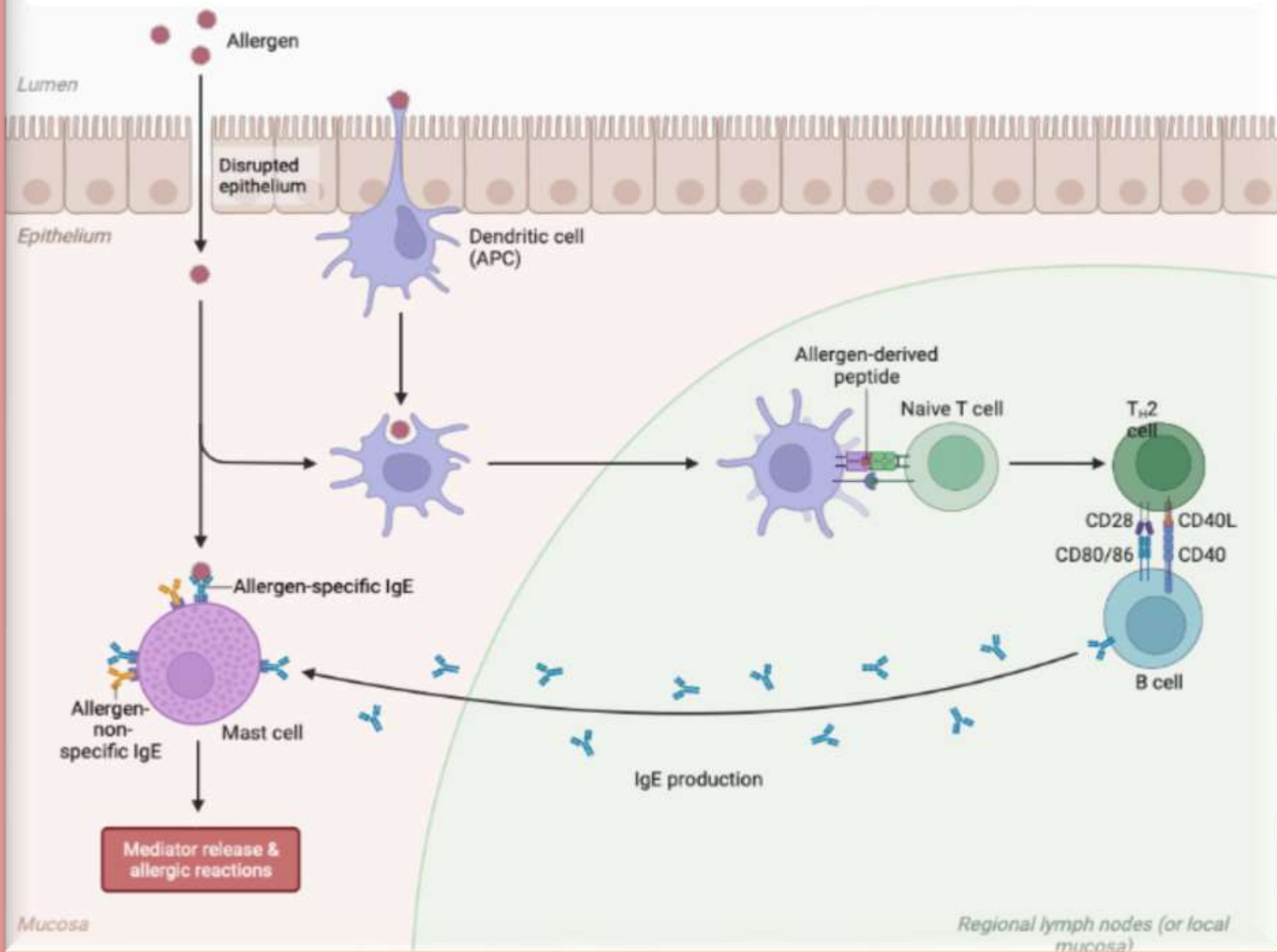
- a) Loop diuretic only
- b) Aspirin only
- c) Loop diuretic and β -blocker
- d) Calcium channel blocker
- e) Loop diuretic, β -blocker, and angiotensin-converting enzyme (ACE) inhibitor



Asthma

**Asthma → chronic inflammatory airway disease with episodes of Bronchospasm and inflammation resulting in reversible airflow obstruction.
Type 1 Hypersensitivity reaction.**





Clinical Presentation

01

Wheezing, a high-pitched whistling sound during breathing, especially during exhalation (hyper resonance on percussion)

02

Coughing, worse at night or early morning can be persistent or episodic

03

Shortness of breath, Difficulty breathing, especially during physical activity.

04

Chest tightness: a sensation of pressure or constriction in the chest.

05

Difficulty sleeping

06

Respiratory distress (- rapid breathing -use of accessory muscles - nasal flaring – pursed lip breathing)

07

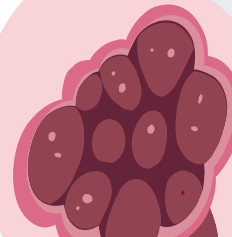
Prolonged expiratory phase

Note: Symptoms can vary in severity and frequency. Severe exacerbations may require urgent medical attention.

Wheezing does not correlate with degree of airflow obstruction.

Silent chest in asthma exacerbations:

This is a medical emergency and may require emergency intubation. It is a life-threatening asthmatic attack.



History and Physical Examination

•History:

Triggers

Symptom assessment

Past Medical History (PMH)

Family history,
medication use,

•PE (Physical Examination):

Respiratory rate plus effort (Respiratory distress)

Auscultation: Wheezing or prolonged expiration

Vital signs

Examine allergic rhinitis and eczema

Investigations:

CXR,ABG

PFT (spirometry)

FEV₁/FVC ratio < 70%

Peak flow meter (Asthma exacerbation)

< 40% = Bad Asthma exacerbation

ASTHMA



Pollution



Genetic



Smoking



Pets



Bacteria and
Viruses



Dust



Household
Chemicals



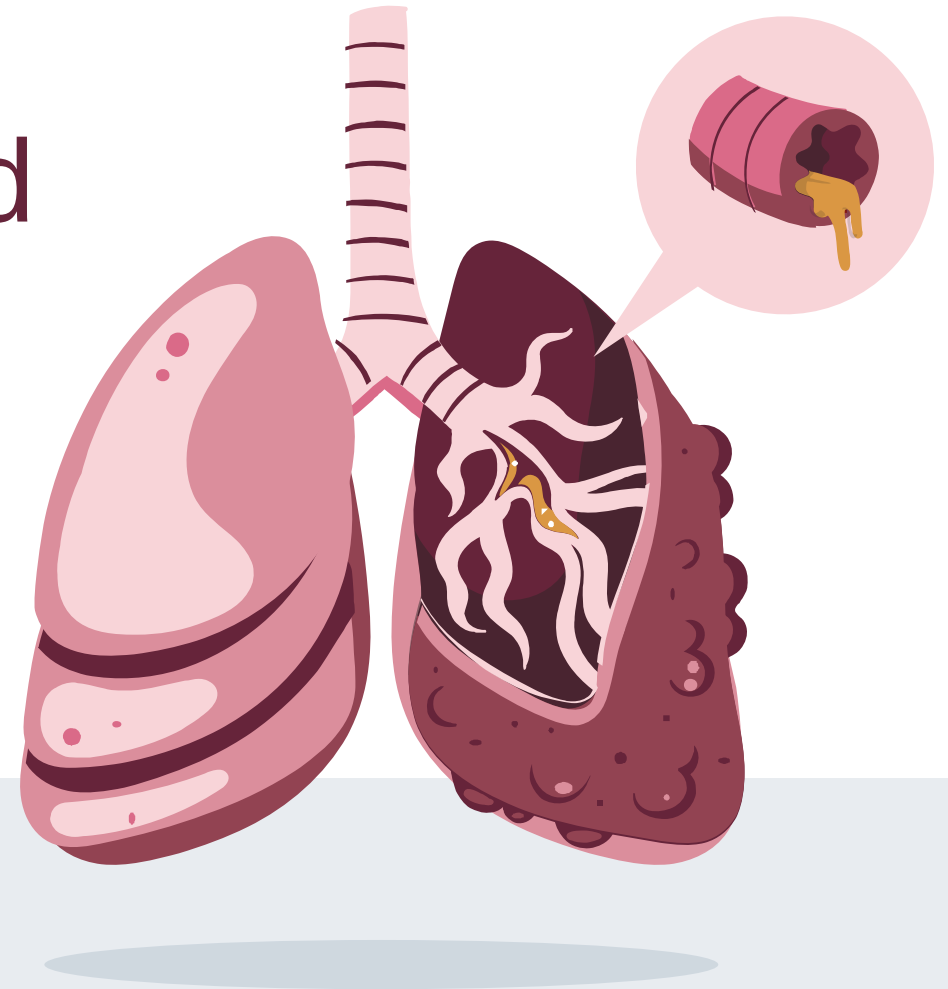
Some common
causes and triggers
that everyone
should know

Pseudo Asthma

- **Pseudo Asthma:** A disorder that mimics asthma but actually not asthma .
Several conditions such as:
 1. Chronic Obstructive Pulmonary Disease (COPD)
 2. Allergic Rhinitis
 3. Vocal Cord Dysfunction
 4. Gastroesophageal Reflux (GERD)
 5. Pneumonia
 6. Heart Failure
 7. Pulmonary Embolism
 8. Pulmonary Edema
 9. Airway Foreign Body
 10. Exercise-Induced Dyspnea
 11. anxiety and panic attack

Asthma management and treatment

Managing an acute asthma exacerbation involves several key steps to quickly relieve symptoms and restore normal breathing. Here's a general approach:



General approach :

01

Assesment of the severity either by the PT ability to talk or by history of exposure of any antigen

02

Immediate Treatment

03

Monitor

04

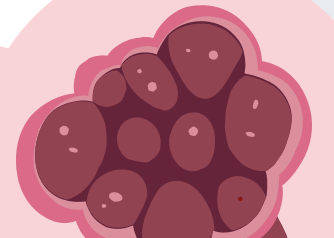
Adjunctive Treatments

05

Re-evaluate

6

Discharge Planning

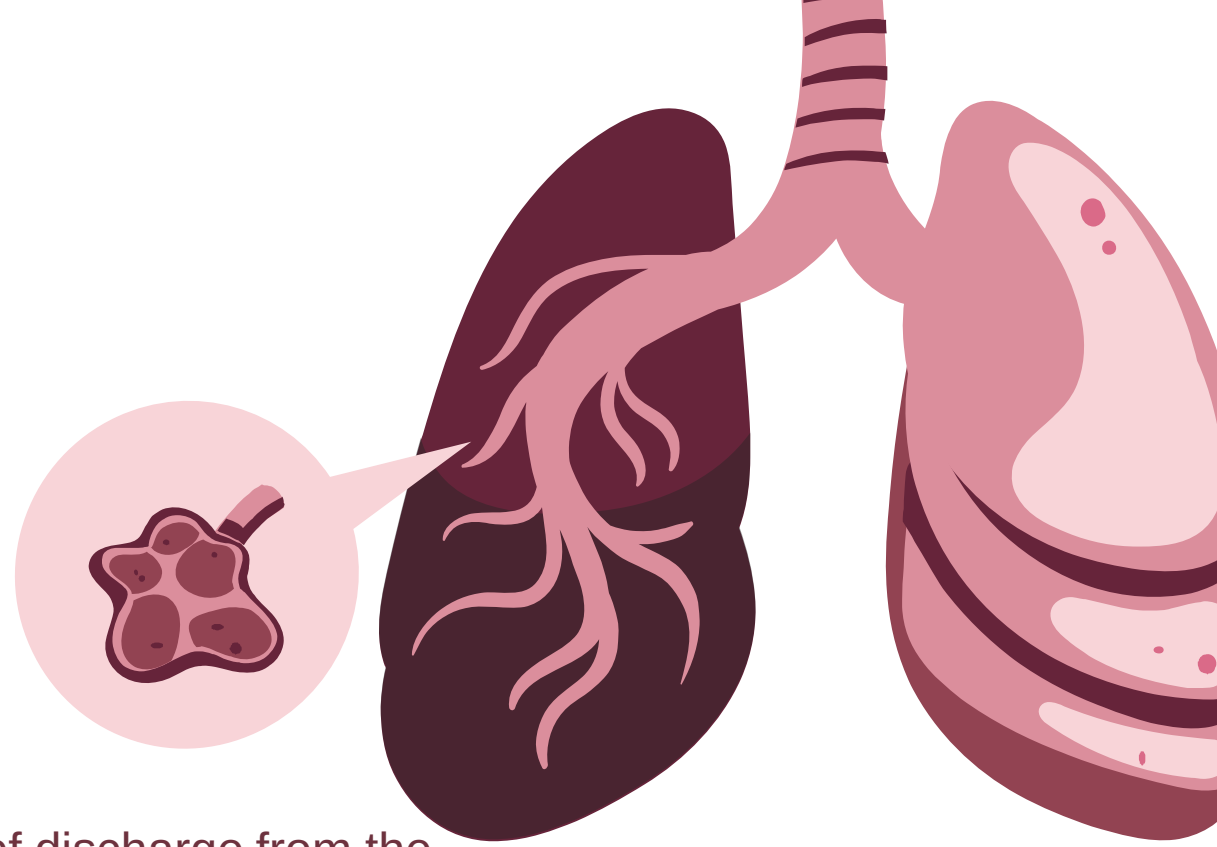


start your Management by :

<u>Oxygen</u>	Start ABC Titrate O2 to achieve sat of at least 92% and continuous monitoring by oximetry
<u>RAPID-ACTING INHALED BRONCHODILATORS</u>	Back to back salbutamol with ipratropium are first line therapy in acute asthma after performing ABC ,salbutamol work by relaxing the muscles around the airways and ipratropium block M3 to inhibit bronchial secretions all these work to decrease the narrowing of the airway
<u>Anticholinergic bronchodilators (M3)</u>	Anticholinergic treatment is recommended for mild to moderate severe exacerbations rather than for mild cases. Given alongside SABA. •MDI Ipratropium bromide (20mcg/puff) with spacer.4 to 8 puffs q 20 minutes x 3 doses . •Nebulized Ipratropium bromide 500mcg (with salbutamol) q20 minutes x 3 doses, then q2-6 prn.
<u>Systemic Steroids</u>	Oral prednisone(50mg) is given to patients with exacerbations of asthma if oral not possible IV methylprednisolone (40-125mg)
<u>intravenous Magnesium sulfate</u>	Effective for patients with no response to first line therapy and with severe airflow obstruction in ED , Given as bolus - 2 g IV over 20 min
Aminophylline and helium-o2 mixtures and empirical AB	<u>They are not effective in managing acute asthma exacerbation attack</u> <u>,antibiotics are not effective since most of the triggers are viral</u>

01

Disposition



Response to initial treatment is a better predictor of safety of discharge from the ED than the apparent severity of the attack at presentation . Pretreatment FEV1 or PEF <25-30% predicted(PEF<100L/min), or post-treatment <40%predicted (PEF<200L/min) - admission usually necessary Post-treatment FEV1 or PEF 40-60% predicted(PEF 200-300L/min) - discharge possible based on risk factors for recurrence of severe attackFEV1 or PEF >60% predicted (PEF>300L/min)- discharge usually safe

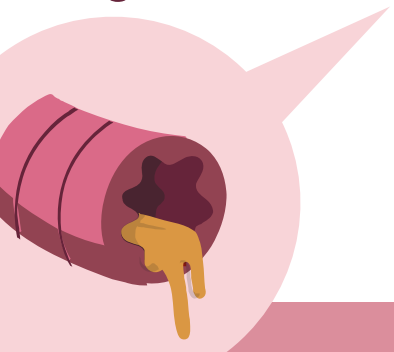
Disposition

PEATIENT AT RISK FOR RELAPSE :

- previous near death episode
- Frequent ED visits
- Frequent hospitalization
- Steroid dependence or recent use
- "Flash" attacks
- Prolonged duration of recent exacerbation
- Poor compliance or understanding of self-management
- Prolonged use of high dose beta-agonists

DISCHARGE INSTRUCTIONS :

- Continue short acting beta-agonist - salbutamol 2-4 puffs q4h, then prn once symptoms controlled and return if use required more than q1-2h
- Oral corticosteroids for patients with moderate to severe attack - prednisone 30-60 mg/day for 7-14days (no need to taper)
- Begin inhaled steroid - Beclomethasone or Budesonide (500-1000 ug/d) or continue inhaled steroid (if patient already on inhaled steroid) even if taking prednisone
- Review written asthma plan to prevent and manage future exacerbations
- Review use and role of different medications and avoidance of triggers
- Ensure follow-up with primary care physician or asthma specialist

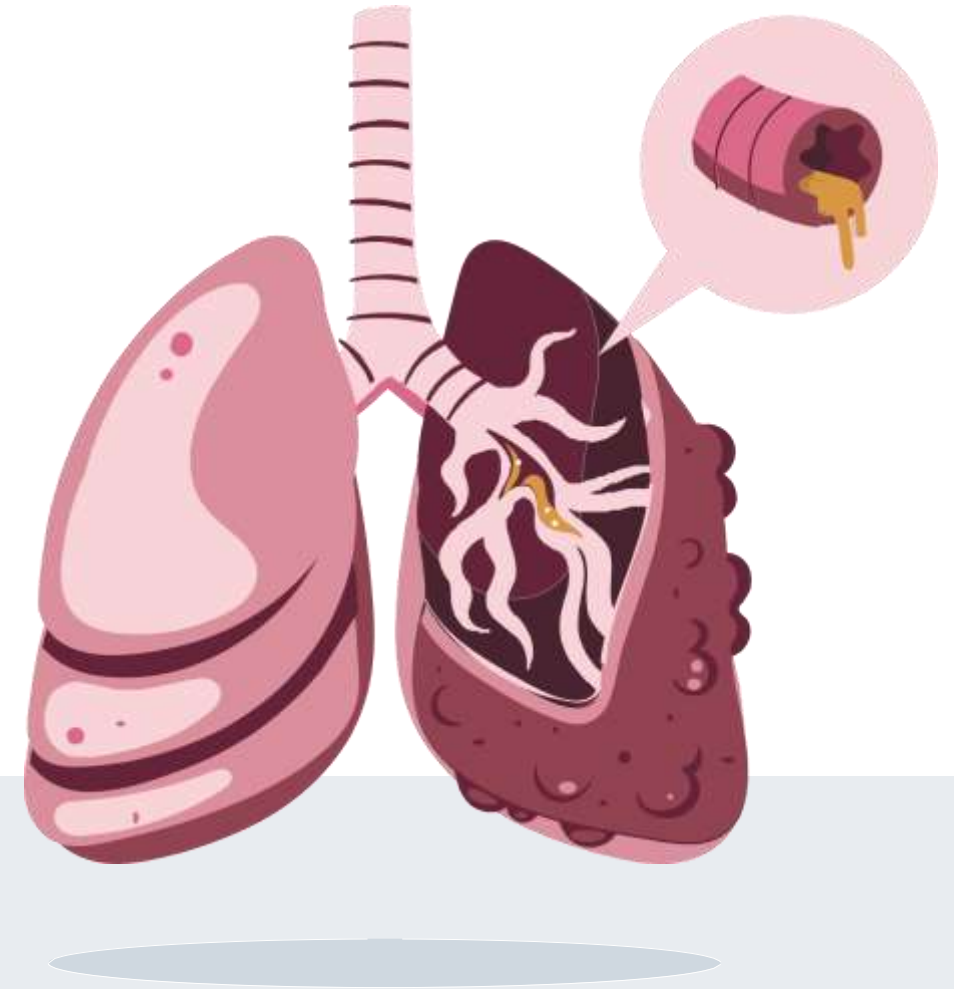


COPD

Exacerbation

Leen AL-Qalqili (2136543)

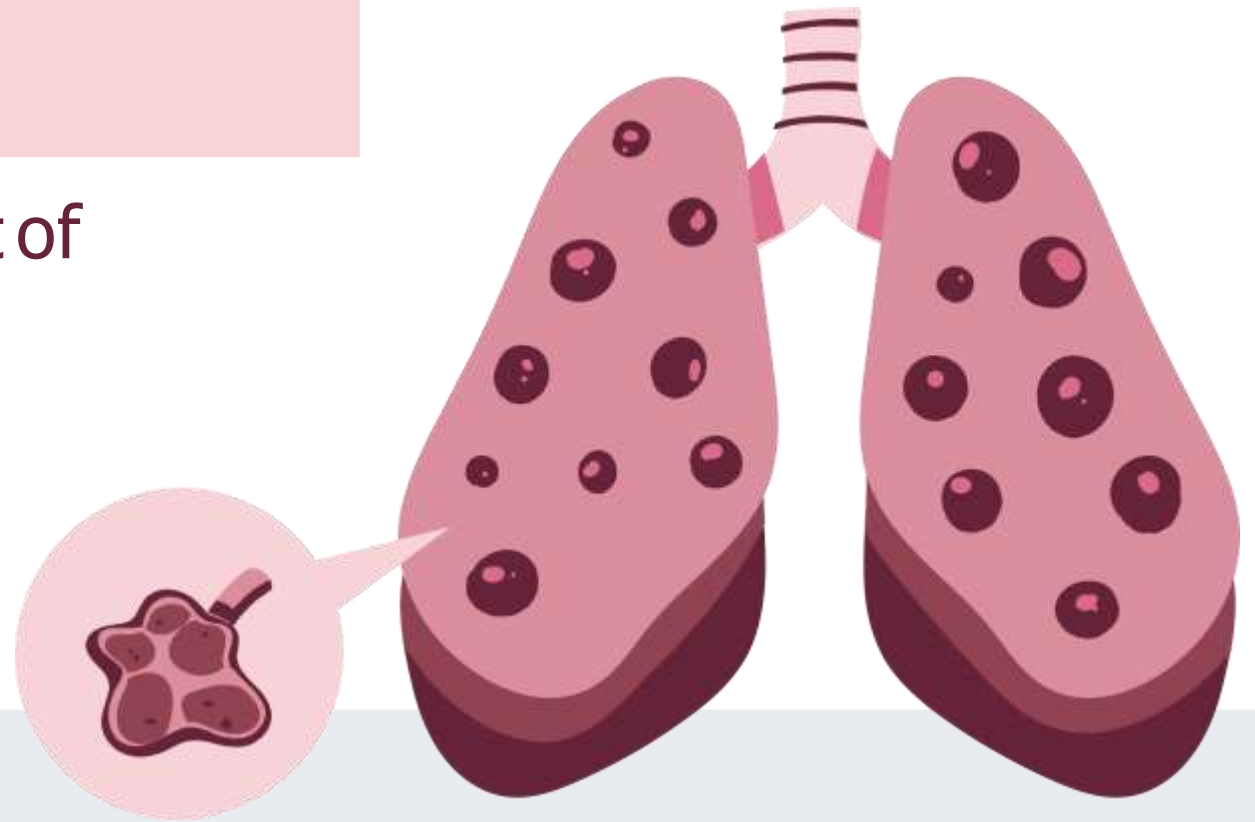
Walaa Azzam (2143237)



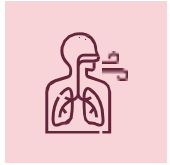
COPD

Exacerbation

Acute progressive development of irreversible airway obstruction, typically caused by smoking



Typical patient presentation (Cardinal symptoms)



Increased
coughing
frequency



Increased
sputum volume
or purulence



Increased
dyspnea

By history and physical exam



Triggers

Virus Pneumonia

Urinary tract infection PE ,CHF ,MI, drugs



Vital signs

Tachypnea Tachycardia Hypoxia



Previous episodes and hospitalizations , smoking history



Signs of respiratory distress

Cyanosis
Grunting
Nose flaring
Wheezing

COPD and Hypercapnia

- Oxygen is probably the most commonly prescribed drug in the emergency setting and is a life-saving modality as well.
- Patients with chronic obstructive pulmonary disease (COPD) may develop hypercapnia during supplemental oxygen therapy, particularly if uncontrolled, can lead to seizures and altered LOC
- Various mechanisms leading to the development of oxygen- induced hypercapnia are the abolition of 'hypoxic drive', loss of hypoxic vasoconstriction, and absorption atelectasis leading to an increase in dead-space ventilation and the Haldane effect.

Management:

Immediate interventions may include bronchodilators, corticosteroids, and, if necessary, adjustments to oxygen therapy to prevent worsening hypercapnia.



Treatment of COPD Exacerbation



Oxygen

Keep O₂ saturation 88-92% Be aware of chronic hypercapnic CO₂ retention but don't withhold O₂ if hypoxic

Antibiotics



Trimethoprim/sulfamethoxazole, cephalosporins, respiratory quinolones (given if all 3 or 2 cardinal symptoms present with increased sputum purulence or mechanical ventilation)

- No antibiotic for mild exacerbation (only one of three cardinal symptoms)



Steroids

- prednisone 40-60 mg PO for 7-14 day
- or Methylprednisolone 1-2mg/kg IV if severe exacerbation or unable to take PO

Non Invasive positive pressure ventilation



NIPPV may be required if patient is experiencing respiratory failure (hypercapnia, acidosis or severe dyspnea)

1. BiPAP
2. CPAP



Treatment of COPD Exacerbation

Bronchodilator

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graph TD; A[Bronchodilator] --> B[1]; A --> C[2]; B --> D[Short acting beta agonists]; C --> E[Short acting anticholinergics]; D --> F["Example: salbutamol, Albuterol, levalbuterol"]; E --> G["Example: ipratropium"]; F --> H["- inhaled via MDI with spacer 4-8 puffs via q15 min *3PRN"]; G --> I["-0.5 mg via MDI q30 *3PRN"];
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1

Short acting
beta agonists

Example: salbutamol
, Albuterol , levalbuterol

- inhaled via MDI with spacer 4-8 puffs via q15
min *3PRN

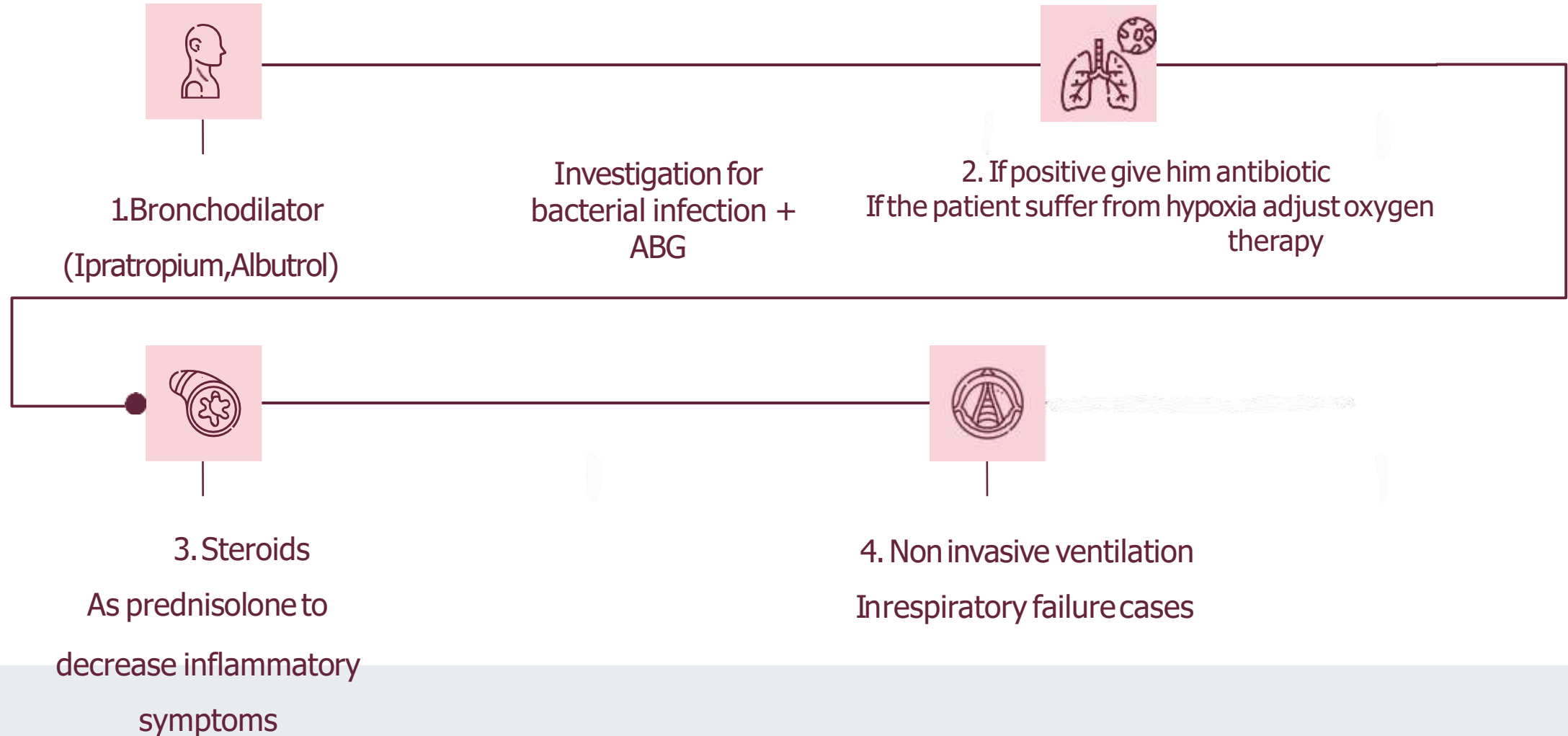
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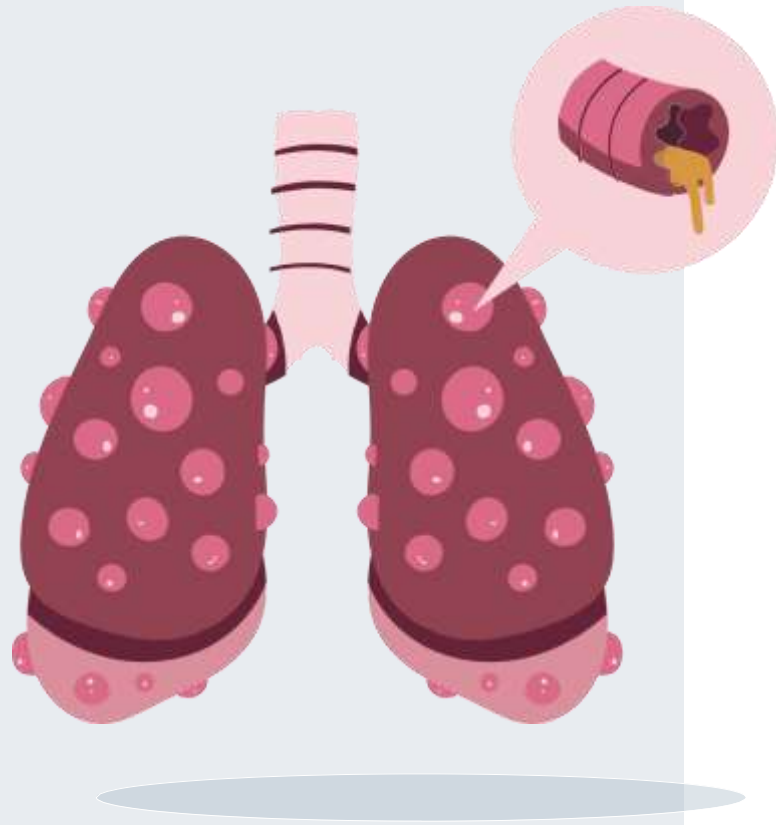
Short acting
anticholinergics

Example:
ipratropium

-0.5 mg via MDI q30
*3PRN

Treatment of COPD Exacerbation





Question

A 40 year old man, chronic smoker and a known diabetic presented with sudden onset breathlessness since 1 day. O/E peripheral extremities are cool, B/L crepts in the chest and neck veins are engorged. **MOST LIKELY DIAGNOSIS**

- A) Spontaneous pneumothorax
- B) Pulmonary embolism
- C) Cardiac tamponade
- D) Myocardial infarction

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