



Respiratory emergencies

Dr. Hatem Elhussieny

Emergency Medicine Board

Al-Shifa Medical Complex

Acute asthma

an initial assessment of the severity of acute asthma based upon a combination of clinical features, peak flow measurement, and pulse oximetry, as outlined below.

Moderate exacerbation of asthma

- symptoms.
- $\uparrow$ Peak flow 50– 75% best or predicted.
- No features of acute severe asthma.

Acute severe asthma (Any one of):

- Peak flow 33– 50% best or predicted.
- RR $\geq$ 25/ min.
- Heart rate $\geq$ 110/ min.
- Inability to complete sentences in one breath.

Acute asthma

Life- threatening asthma (patient with severe asthma with any one of):

- Peak flow $<33\%$ best or predicted.
- SpO₂ $<92\%$.
- pO₂ $<8\text{kPa}$.
- Normal pCO₂ (4.6– 6.0kPa).
- Silent chest.
- Cyanosis.
- Poor respiratory effort.
- Arrhythmia.
- Exhaustion.
- Altered conscious level.
- Hypotension.

↑
Near- fatal asthma

- pCO₂ and/ or requiring mechanical ventilation with i inflation pressures.

Investigations in asthma

Peak flow is most useful when expressed as a percentage of that patient's previous best, but the percentage of predicted is a rough guide.

Pulse oximetry (SpO₂) determines the adequacy of O₂ therapy and the need for ABG measurement. Use O₂ to aim for SpO₂ of 94– 98%.

Obtain ABG if SpO₂ <92% or if there are other features of life- threatening asthma.

Obtain a CXR (without delaying treatment) if there is:

- Suspected pneumomediastinum or pneumothorax.
- Suspected consolidation.
- Life- threatening asthma.
- Failure to respond to treatment satisfactorily.
- Requirement for ventilation.

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Acute asthma

Initial assessment

Brief history, physical examination (auscultation, use of accessory muscles, heart rate, respiratory rate), PEF or FEV₁, oxygen saturation, and other tests as indicated.

FEV₁ or PEF ≥40 percent (mild-to-moderate)

- Oxygen to achieve SaO₂ ≥90 percent
- Inhaled SABA by nebulizer or MDI with valved holding chamber, up to 3 doses in first hour
- Oral systemic corticosteroids if no immediate response or if patient recently took oral systemic corticosteroids

FEV₁ or PEF <40 percent (severe)

- Oxygen to achieve SaO₂ ≥90 percent
- High-dose inhaled SABA plus ipratropium by nebulizer or MDI plus valved holding chamber, every 20 minutes or continuously for 1 hour
- Oral systemic corticosteroids

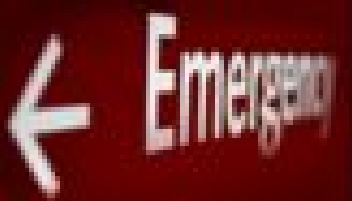
Impending or actual respiratory arrest

- Intubation and mechanical ventilation with 100 percent oxygen
- Nebulized SABA and ipratropium
- Intravenous corticosteroids
- Consider adjunct therapies

Repeat assessment

Symptoms, physical examination, PEF, O₂ saturation, other tests as needed

Admit to hospital intensive care (see box below)



Acute asthma

Moderate exacerbation

FEV₁ or PEF 40-69 percent predicted/personal best
Physical exam: moderate symptoms

- Inhaled SABA every 60 minutes
- Oral systemic corticosteroid
- Continue treatment 1-3 hours, provided there is improvement; make admit decision in <4 hours

Severe exacerbation

FEV₁ or PEF <40 percent predicted/personal best
Physical exam: severe symptoms at rest, accessory muscle use, chest retraction
History: high-risk patient
No improvement after initial treatment

- Oxygen
- Nebulized SABA + ipratropium, hourly or continuous
- Oral systemic corticosteroids
- Consider adjunct therapies

Good response

- FEV₁ or PEF ≥70 percent
- Response sustained 60 minutes after last treatment
- No distress
- Physical exam: normal

Incomplete response

- FEV₁ or PEF 40-69 percent
- Mild-to-moderate symptoms

Poor response

- FEV₁ or PEF <40 percent
- PCO₂ ≥42 mmHg
- Physical exam: symptoms severe, drowsiness, confusion

Individualized decision re: hospitalization



Acute asthma

Discharge home

- Continue treatment with inhaled SABA
- Continue course of oral systemic corticosteroid
- Consider initiation of an ICS
- Patient education
 - Review medications, including inhaler technique
 - Review/initiate action plan
 - Recommend close medical followup

Admit to hospital ward

- Oxygen
- Inhaled SABA
- Systemic (oral or intravenous) corticosteroid
- Consider adjunct therapies
- Monitor vital signs, FEV₁ or PEF, SaO₂

Admit to hospital intensive care

- Oxygen
- Inhaled SABA hourly or continuously
- Intravenous corticosteroid
- Consider adjunct therapies
- Possible intubation and mechanical ventilation

Improve



Discharge home

- Continue treatment with inhaled SABAs
- Continue course of oral systemic corticosteroid
- Continue on ICS. For those not on long-term control therapy, consider initiation of an ICS
- Patient education (eg, review medications, including inhaler technique and, whenever possible, environmental control measures; review/initiate action plan; recommend close medical followup)
- Before discharge, schedule followup appointment with primary care provider and/or asthma specialist in 1-4 weeks

Improve



COPD

characterized by chronic airflow limitation due to impedance to expiratory airflow, mucosal edema, infection, bronchospasm, and bronchoconstriction due to d lung elasticity.

Smoking is the main cause, but other causes are chronic asthma, α -1 antitrypsin deficiency, and chronic infection

COPD

Exertional dyspnoea, cough, and sputum are usual complaints. Ask about:

- Present treatment: including inhalers, steroids, antibiotics, theophyllines, nebulizers, opiate analgesia, and home O2 treatment.
- Past history: enquire about previous admissions and comorbidity.
- Exercise tolerance: how far can they walk on the flat without stopping? How many stairs can they climb? Do they get out of the house?
- Recent history: ask about wheeze and dyspnoea, and sputum volume and colour. Chest injuries, abdominal problems, and other infections may cause respiratory decompensation.
- Read the hospital notes: have there been prior ICU assessments? Has the respiratory consultant advised whether ICU would be appropriate?

COPD

Examine for dyspnoea, tachypnoea, accessory muscle use, and lip- pursing.

Look for hyperinflation ('barrel chest'), and listen for wheeze or coarse crackles (large airway secretions).

Cyanosis, plethora (due to secondary polycythaemia), and right heart failure (cor pulmonale) suggest advanced disease.

Look for evidence of hypercarbia: tremor, bounding pulses, peripheral vasodilatation, drowsiness, or confusion.

COPD

Investigation

- SpO₂, RR, pulse rate, BP, T°, and peak flow (if possible).
- CXR (look for pneumothorax, hyperinflation, bullae, and pneumonia).
- ECG.
- ABG (or VBG), documenting the FiO₂. Use pCO₂ to guide O₂ therapy.
- CBC, U&E, glucose, theophylline levels, and, if pneumonia is suspected, blood cultures, CRP.
- Send sputum for microscopy and culture if purulent.

COPD

Treatment

- Give oxygen
- Give bronchodilators and steroids
- Give antibiotics (eg amoxicillin, doxycycline, or clarithromycin) if the patient reports i purulent sputum or there is clinical evidence of pneumonia and/ or consolidation on CXR.
- Only consider IV aminophylline if there is an inadequate response to nebulized bronchodilators.
- Non- invasive ventilation. Start BiPAP at 10cmH2O inspiratory positive airway pressure (IPAP)/ 5cmH2O expiratory positive airway pressure (EPAP),

Pneumonia

Pneumonia involves symptoms and signs of lower respiratory tract infection (breathlessness, productive cough, and fever) usually associated with CXR abnormalities.

Pneumocystis pneumonia may occur with minimal or no CXR changes.

Consider pneumonia in patients with septicaemia or acute confusional states.

Pneumonia

Causes

- Bacterial (80– 90%) *Streptococcus pneumoniae* is the most common cause of community- acquired pneumonia. Others include *Mycoplasma pneumoniae*, *Haemophilus influenzae*, *Legionella*, *Chlamydia psittaci*, and *Staphylococcus aureus* (can cause fulminant pneumonia in patients with influenza).
- Gram –ve and anaerobic infections are rare.
- Always consider TB, particularly in chronic alcoholism, poor social circumstances, immigrants and those travelling to developing countries, or individuals not BCG-vaccinated.
- Immunosuppressed patients (eg HIV, steroid therapy) are at i risk of TB and *Pneumocystis jirovecii* pneumonia.
- Viral (10– 20%) Predominantly COVID-19, influenza A and B, RSV, rarely varicella and SARS.
- Rickettsial (1%) Rarely *Coxiella burnetii*.

Pneumonia

Signs and symptoms

- Fever, cough, and production of sputum are common complaints.
- Breathlessness, pleuritic chest pain, myalgia, rigors, or haemoptysis may occur.
- Pneumonia can present without obvious chest signs.
- Mycoplasma pneumonia may present in children and young adults with sore throat, headache, nausea, abdominal pain, and diarrhoea.
- Legionella can present with constitutional upset, diarrhoea, or confusion, particularly in the elderly.
- Pneumocystis pneumonia in immunosuppressed patients may present with cough, dyspnoea, and marked hypoxia, with relatively few other findings.

Pneumonia

Examination and investigation

- Assess for signs of severe sepsis.
- Check RR, pulse, and BP.
- Auscultation may reveal a patch of inspiratory crackles; signs of consolidation are present in <25%.
- Check SpO₂ (obtain ABG if <90% or known to have COPD).
- Take blood for U&E, CBC, and CRP, and blood cultures before giving IV antibiotics.
- Obtain a CXR. Look for patchy or lobar opacification, mass lesions, or an air bronchogram.
- In early pneumonia, the CXR may be normal.
- Obtain blood cultures and sputum cultures, and consider urinary pneumococcal and Legionella antigen testing.
- If suspicion of COVID-19 infection, take a nasopharyngeal swab for viral testing.

Pneumonia

Pneumonia

Treatment

Patients deemed suitable for discharge

- Give analgesia, oral antibiotics for 5 days, and follow- up (including a decision about the need to repeat CXR in 6 weeks to confirm changes have cleared).

Patients admitted, but not severely unwell

- Start either PO or IV antibiotics, as follows:
- Either amoxicillin 0.5– 1g PO tds + erythromycin 500mg PO qds (or clarithromycin 500mg bd).
- Or if IV therapy is needed: ampicillin 500mg IV qds + erythromycin 500mg IV qds (or clarithromycin 500mg bd).
- Monitor SpO₂ and provide O₂ accordingly.
- Provide simple analgesia.

Pulmonary embolism

The mortality of diagnosed and treated PE is 7%.

Pulmonary embolic disease can result in a variety of symptoms often misdiagnosed as asthma, anxiety, pneumonia, and ACS.

Pulmonary embolism

Most patients with PE experience dyspnoea, commonly without other symptoms.

Syncope with cyanosis, cardiac arrest, or angina are signs of massive PE.

A minority present with pleuritic chest pain, some with additional haemoptysis.

Always consider PE in patients with unexplained hypoxia or breathlessness.

Take a full history of concurrent illness, surgical procedures, recent hospital admission, past history, including DVT and PE, and travel and family history.

Pulmonary embolism

Examination may be normal.

Tachycardia and tachypnoea are common.

Pyrexia following lung infarction is common.

30% of all patients with PE have normal SpO₂.

Always record BP. Hypotension indicates massive PE.

Perform a full respiratory and cardiovascular examination.

Always examine the legs for signs of DVT.

Pulmonary embolism

Any patient scoring ≥ 4.5 on the Wells score OR who has an elevated D- dimer requires pulmonary imaging.

Only a normal D- dimer AND a 'PE unlikely' Wells score will safely exclude PE.

Table 3.9 Modified Wells clinical probability assessment score for PE

Clinical feature	Score
Signs of DVT (minimum of objective leg swelling and tenderness)	3.0
IV drug use	3.0
PE is the most likely diagnosis	3.0
Heart rate >100	1.5
Prior PE or DVT diagnosis	1.5
Bedridden for >3 days or surgery within the past 4 weeks	1.5
Cancer (treated actively or with palliation within last 6 months)	1.0
Haemoptysis	1.0

Total score ≤ 4.0 = PE unlikely; score ≥ 4.5 = PE likely.

Pulmonary embolism

Table 3.10 Choice of anticoagulant drug for PE in the ED

Anticoagulant	Dose
Rivaroxaban	15mg PO bd for 21 days, then 20mg PO daily
Apixaban	10mg PO bd for 7 days, then 5mg PO bd
Enoxaparin	1mg/kg SC bd, max 100mg bd
Dalteparin	200U/kg SC daily, max 18,000U
Tinzaparin	175U/kg SC daily, max 18,000U
Unfractionated heparin	IVI—arrange admission for warfarinization Give if estimated glomerular filtration rate (eGFR) <20mL/min

Pleural effusion

Under normal circumstances, each pleural cavity contains <20mL of fluid.

An exudate is diagnosed if the pleural fluid:serum protein is >0.5, fluid:serum lactate dehydrogenase (LDH) >0.6, or fluid LDH more than two-thirds the upper limits of the laboratory normal value for serum LDH.

Pleural effusion

Table 3.6 Causes of pleural effusion

Exudates	Transudates
Pneumonia	Cardiac failure
Malignancy	Nephrotic syndrome
TB	Hepatic failure
PE with pulmonary infarction	Ovarian hyperstimulation
Collagen vascular disease	Peritoneal dialysis
Abscess (subphrenic and amoebic liver)	Ovarian fibroma (Meig's syndrome)
Pancreatitis	
Chylothorax (thoracic duct injury)	

Pleural effusion

Symptoms are usually due to the underlying disease process.

- A mild dull ache and dyspnoea (initially on exercise, later at rest) may occur if the effusion is large.
- A history of vomiting, followed by chest pain, points to a ruptured oesophagus— a surgical emergency.

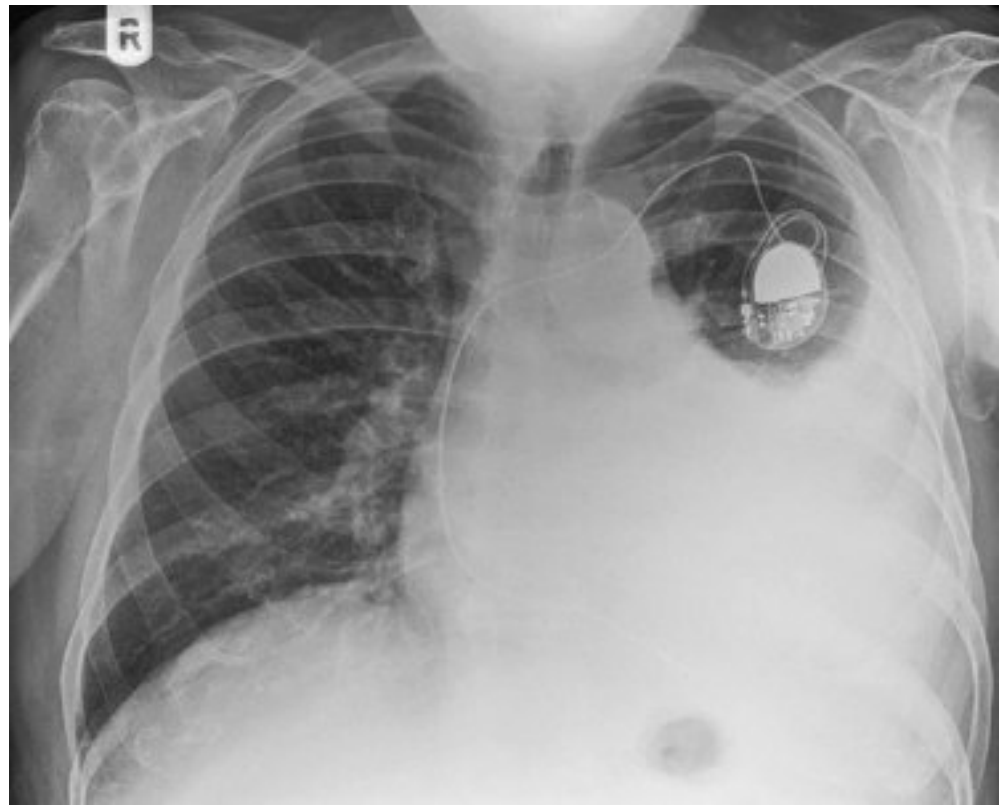
Signs are not apparent until >500mL are present.

- Dyspnoea and stony dullness to percussion, with absent breath sounds over the effusion, are characteristic.
- Bronchial breathing may be heard just above the effusion.
- Very large unilateral effusions may produce evidence of mediastinal shift.

Pleural effusion

Investigation

- CXR can demonstrate pleural effusions as small as 250mL as blunting of the costophrenic angle.
- Other investigations depend on the likely cause.



Pleural effusion

Treatment

- Provide O₂ and resuscitate as necessary, according to the underlying pathology.
- Emergency therapeutic pleural aspiration is rarely required in the ED, except where haemothorax is suspected.
- Refer to the medical team for further investigation (including diagnostic pleural aspiration).

Cardiogenic pulmonary edema

Left heart failure results in LV[↑]end-diastolic pressure, causing pulmonary capillary hydrostatic pressure.
↑

Fluid collects in extravascular pulmonary tissues faster than the lymphatics clear it.

Cardiogenic pulmonary edema

Causes

- Often an acute complication of MI and IHD, or an exacerbation of pre- existing cardiac disease (eg hypertension, aortic/ mitral valve disease).
- Arrhythmias.
- Failure of prosthetic heart valve.
- Ventricular septal defect.
- Cardiomyopathy.
- Negatively inotropic drugs (eg β - blockers).
- Acute myocarditis.
- Left atrial myxoma (may cause syncope, fever, i ESR))— very rare.
- Pericardial disease.

Cardiogenic pulmonary edema

History

- Dyspnoea and distress may prevent a full history from being taken.
- Find out the length of the history and whether there is any chest pain.
- Check current drug therapy/ allergies, and establish what emergency prehospital treatment has been administered.

Cardiogenic pulmonary edema

Examination Usually reveals a tachypnoeic, tachycardic, and anxious patient.

If pulmonary oedema is severe, the patient may be cyanosed, coughing up frothy pink sputum and unable to talk.

Check pulse and BP; auscultate the heart for murmurs and third/ fourth heart sounds of gallop rhythm.

Look for i JVP (also a feature of PE and cardiac tamponade).

Listen to the lung fields— fine inspiratory crepitations (crackles) may be limited to the bases or be widespread.

Wheeze may be more prominent than crepitations. Cardiogenic pulmonary oedema is associated with evidence of d cardiac output (sweaty, peripherally cool, and pale).

Consider other diagnoses (eg sepsis) in patients with warm, flushed extremities.

Cardiogenic pulmonary edema

Investigations

Commence treatment before completing investigations:

Attach a cardiac monitor and check SpO₂ with a pulse oximeter.

Obtain an ECG. Check for arrhythmias, LAD, LVH, LBBB, and recent or evolving MI.

Send blood for U&E, glucose, CBC, troponin, and B- type natriuretic peptide (BNP).

If severely ill or SpO₂ <90% on air, obtain an ABG.

Cardiogenic pulmonary edema

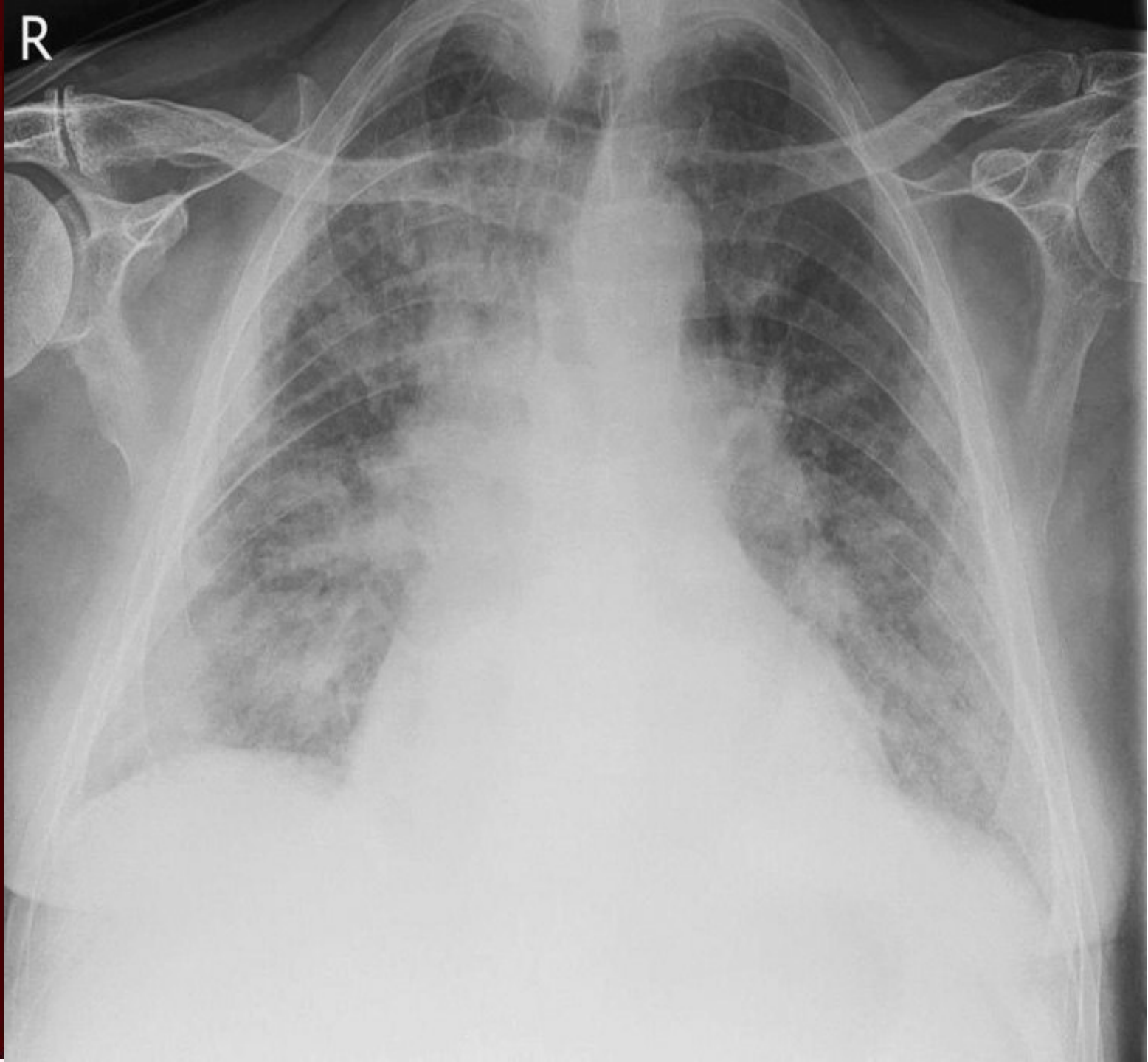
Obtain a CXR and look for features of cardiogenic pulmonary oedema:

- Upper lobe diversion (distension of upper pulmonary veins).
- Cardiomegaly (LV and/ or left atrial dilatation).
- Kerley A, B, or C septal lines
- Fluid in interlobar fissures.
- Peribronchial/ perivascular cuffing and micronodules.
- Pleural effusions.
- Bat's wing hilar shadows.

Request old hospital notes/ ECGs. In newly diagnosed heart failure, an urgent transthoracic echo will identify the presence or absence of cardiac abnormalities.

← Emergency

R



Cardiogenic pulmonary edema

Treatment

- Check that the airway is clear.
- Raise the trolley to sit the patient up (support with pillows, if needed).
- Provide high- flow O₂
- Consider NIV (continuous positive airway pressure (CPAP)
- Treat the underlying cause and associated problems (arrhythmias, MI, cardiogenic shock, acute prosthetic valve failure).

Non- cardiogenic pulmonary edema

Pulmonary oedema may occur in the absence of pulmonary venous pressure.

The following mechanisms may be responsible:

- • capillary permeability.
- • plasma oncotic pressure.
- • lymphatic pressure.

Changes in capillary permeability, secondary to a variety of triggers, is the mechanism most frequently implicated in non- cardiogenic pulmonary oedema, when it occurs as adult respiratory distress syndrome (ARDS).

Since the mechanisms producing cardiogenic and non- cardiogenic pulmonary oedema differ, so does the approach to treatment.

Non- cardiogenic pulmonary edema

Causes

- ARDS (sequel to sepsis, trauma, pancreatitis, COVID-19).
- Intracranial (especially subarachnoid) haemorrhage.
- IV fluid overload.
- Hypoalbuminaemia (liver failure, nephrotic syndrome).
- Drugs/ poisons/ chemical inhalation.
- Lymphangitis carcinomatosa.
- Smoke inhalation.
- Near drowning incidents.
- High altitude mountain sickness.

Non- cardiogenic pulmonary edema

Distinguishing non- cardiogenic from cardiogenic pulmonary oedema is usually apparent from the history.

Evaluate the patient and resuscitate according to ABCs.

Direct treatment towards the underlying cause and according to the physiological disturbance.

Use NIV early and consider urinary, intraarterial, and central venous lines. Involve ICU early and provide appropriate IV fluids and inotropes— deterioration may require intubation, whilst being mindful of the risk of hypotension afterwards.

Thank you

