MUNI
MED

Pulmonary edema

Marek Lukeš

Learning outcomes

- Student will learn the basics of pathophysiology, diagnostics and management of the different types of pulmonary edema

Pulmonary edema

- **abnormal accumulation of extravascular fluid in the lung parenchyma**

- **clinically, there are two fundamentally different types of pulmonary edema:**

  a) cardiogenic pulmonary edema (CPE)
  b) non-cardiogenic pulmonary edema (NCPE)

Major pathophysiologic mechanisms

1. **Imbalance of starling forces**
   - increased pulmonary capillary pressure
   - decreased plasma oncotic pressure
   - increased negative interstitial pressure

2. **Damage to the alveolar-capillary barrier**

3. **Lymphatic obstruction/insufficiency**

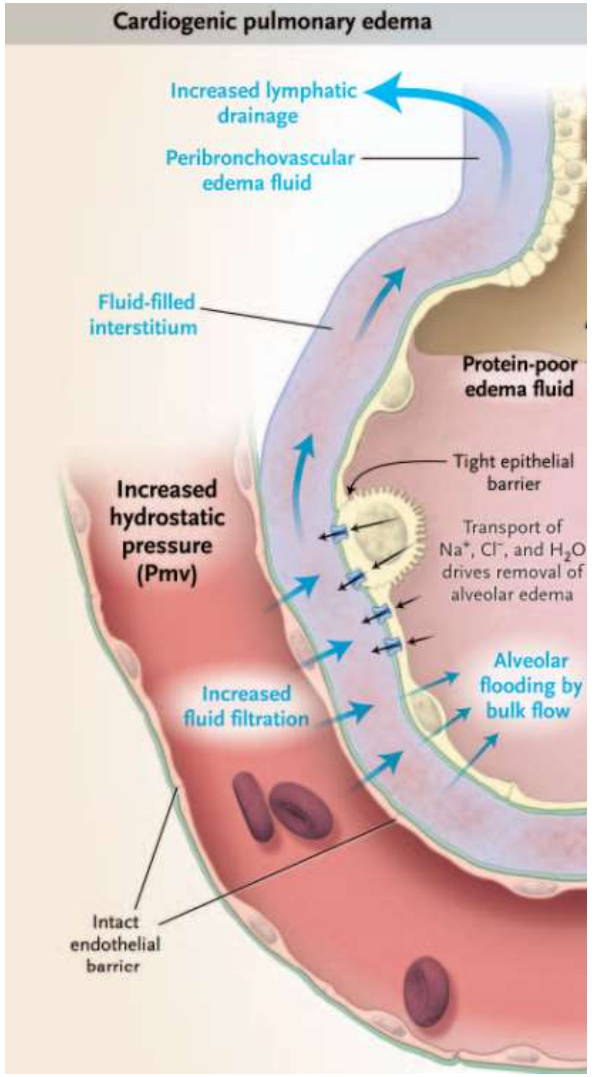
Increased pulmonary capillary pressure

- systolic and diastolic dysfunction of the left ventricle (LV)
- pulmonary venous outflow obstruction
- left ventricle volume overload

→ increased pulmonary capillary pressure is the hallmark of cardiogenic (or volume overload) pulmonary edema!

Increased pulmonary capillary pressure

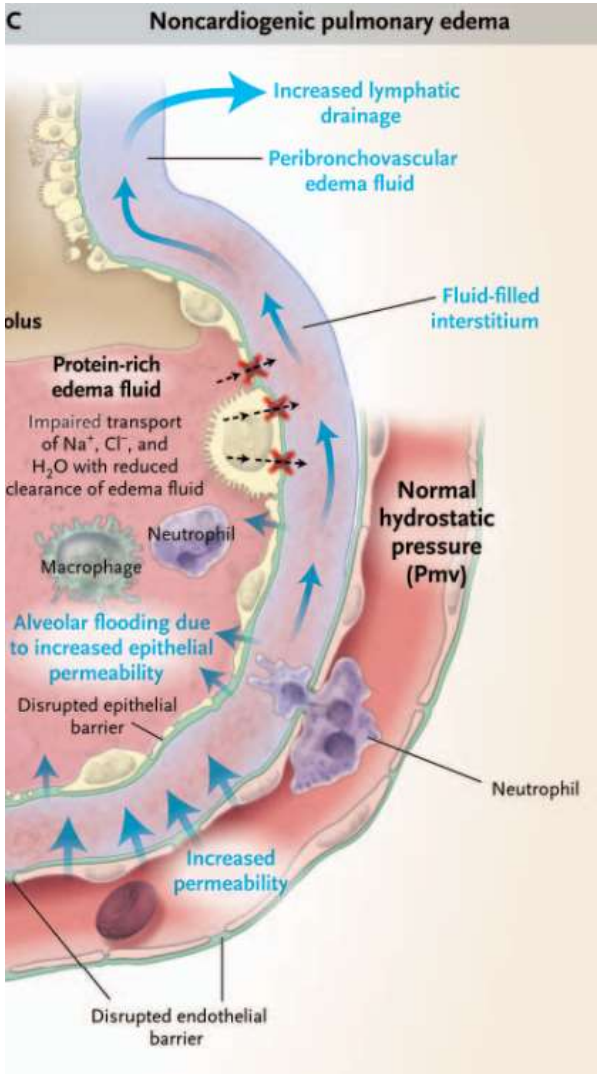
- elevation of pulmonary venous pressure
  ↓
- elevation of hydrostatic pressure in the pulmonary capillaries
  ↓
- increase of transvascular fluid filtration → flooding the alveoli (protein-poor fluid)



MUNI MED

Injury of the alveolar-capillary membrane

- increase in vascular permeability
  ↓
- incresed flux of fluid into interstitium and air spaces
  ↓
- flooding the alveoli (protein-rich fluid)



MUNI MED

Injury of the alveolar-capillary membrane

Direct lung injuries

- pneumonia
- aspiration
  (gastric content, drowning)
- contusion
- inhalation injury
  (heat, smoke, chemical irritants)
- radiation

Indirect lung injuries

- shock
- sepsis
- acute pancreatitis
- multiple transfusions (TRALI)
- anaphylaxis
- toxin exposure
  (opioids, salicylate)

Acute Respiratory Distress Syndrome

- early ARDS (exsudative phase) is characterized by increased capillary permeability and the influx of protein-rich fluid into the alveolar space

→ **early ARDS is the most severe form of NCPE !**

Specific (sub)types on NCPE

- **Neurogenic pulmonary edema (NPE)**
- massive sympathetic overreactivity and capillary leak due to an acute central nervous system pathology
- traumatic brain injury, intracranial hemorrhage, epileptic seizures

- **High altitude pulmonary edema (HAPE)**
- breakdown in the bloog-gas barrier triggered by hypobaric hypoxia
- risk depends upon individual susceptibility, altitude attained and time spent at high altitude

Common symptoms of CPE

- clinical features may appear suddenly or develop over time and can be very dramatic

- short of breath (on exertion, at rest, orthopnoea)
- cough with frothy sputum (pink or tinged with blood)
- feelings of drowning, restlessness, anxiety
- other symptoms depend upon the underlying cause (chest pain, palpitations, etc.)

Common signs of CPE

- tachypnoea
- tachycardia
- hypertension (symptom of the hyperadrenergic state)
- hypotension (symptom of the LV dysfunction or cardiogenic shock)
- diaphoresis
- agitation, confusion
- cool extremities and peripheral cyanosis
- jugular venous distention
- cracles on chest auscultation

Laboratory testing

- **arterial blood gases** → respiratory failure (hypoxia, hypercapnia)
  → hyperlactataemia / metabolic acidosis
- **full blood count** → anemia, white blood cell elevation
- **renal function** → urea, creatinine, electrolytes
- **liver function**
- **cardiac troponin** → myocardial ischemia / damage of myocytes
- **BNP/ NT pro BNP** → marker of heart failure

- troponin and BNP levels can be confounded in critically ill patients!

Electrocardiography

- **LV hypertrophy**
  → increased QRS voltage
  → increased QRS duration
  → left axis deviation

- **myocardial ischemia /infarction**
  → new ST depression /elevation
  → new left bundle branch block

- **tachydysrhytmia**
- **bradydysrhytmia**

Chest radiography

CPE likely

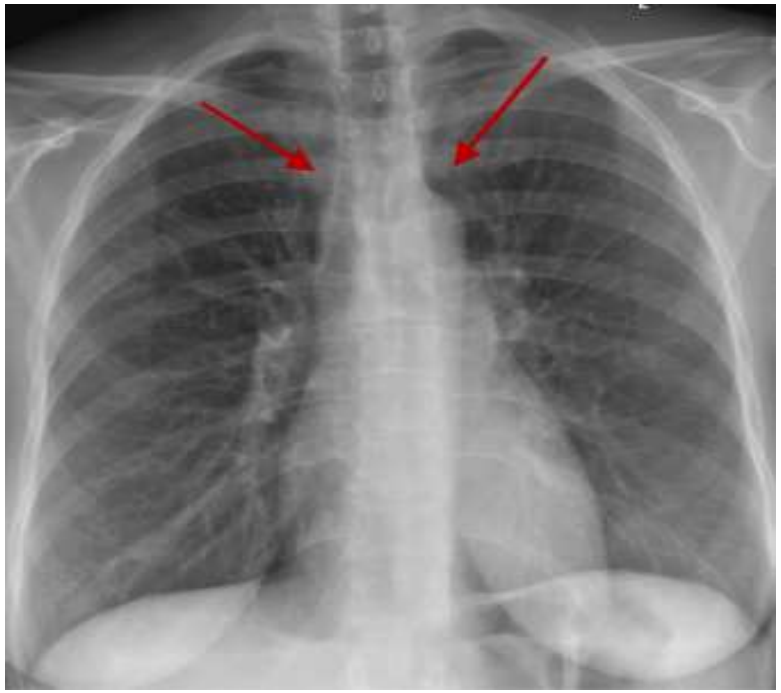
- enlarged cardiac silhouette
- enlarged vascular pedicle width
- enlarged peribronchovascular spaces
- central infiltrates
- Kerley´s B lines (septal lines)
- no air bronchograms

NCPE likely

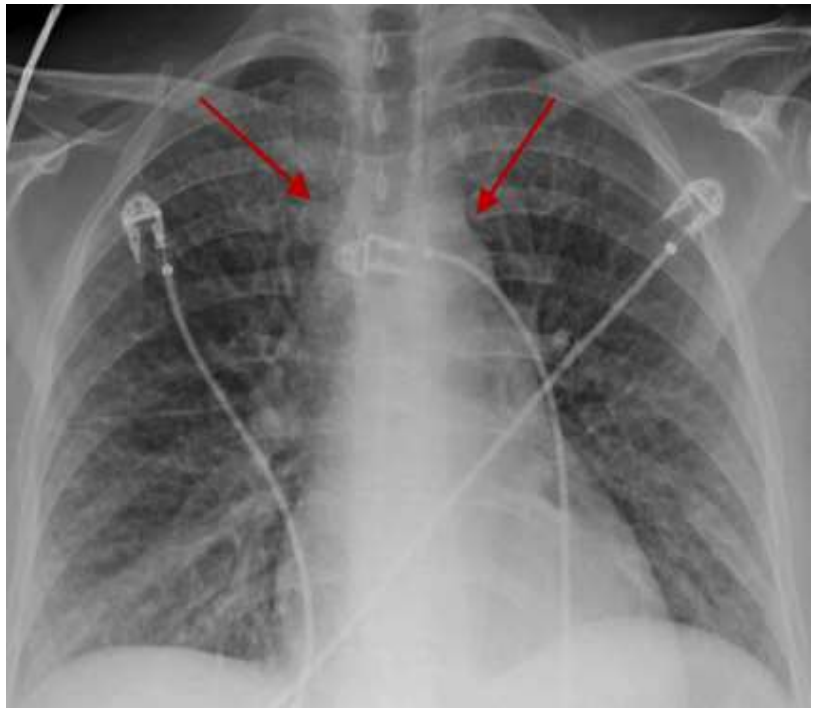
- normal cardiac silhouette
- normal vascular pedicle width
- normal peribronchovascular spaces
- peripheral / diffuse infiltrates
- no Kerley´s B lines
- air bronchograms

Chest radiography

normal silhouette & vascular pedicle (red arrows)

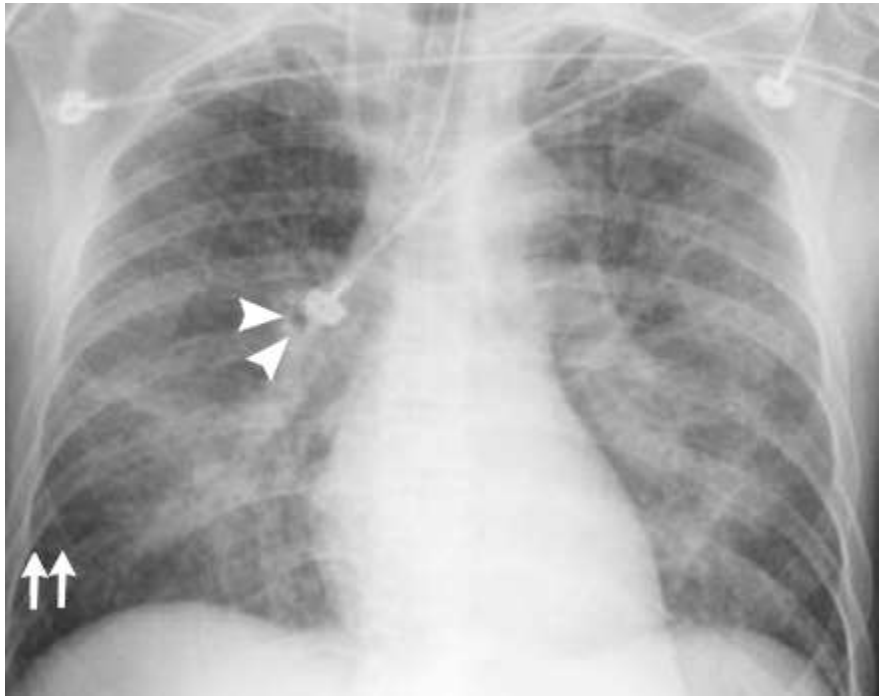


cardiomegaly & widening of the vascular pedicle

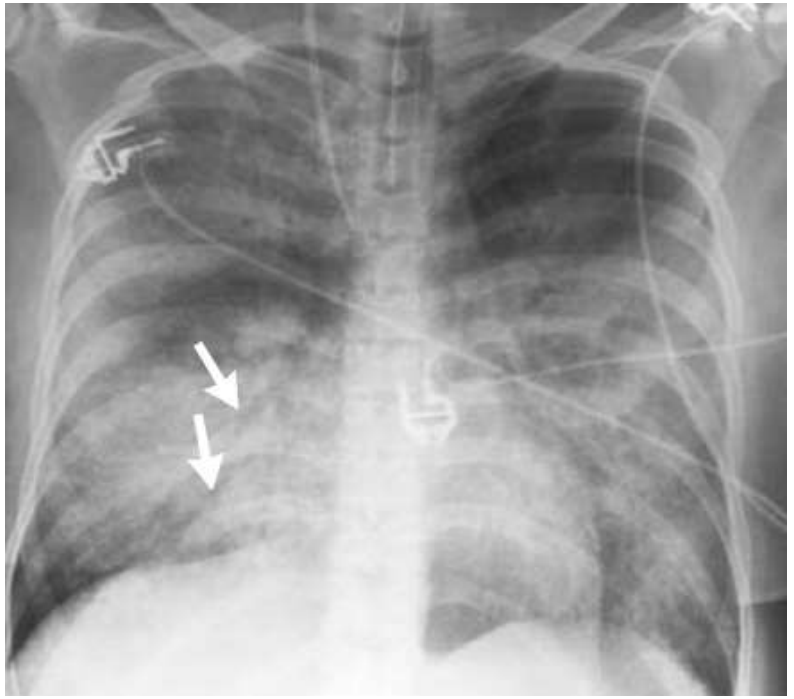


Chest radiography

enlargement of peribronchovascular space & prominent septal lines



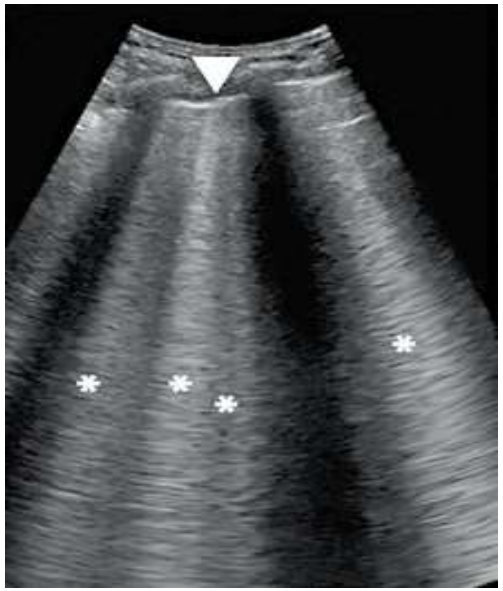
air bronchogram



Ultrasound imaging

Lung ultrasonography

- B line artefacts (*)
- pleural effusion



Echocardiography

- etiology of CPE

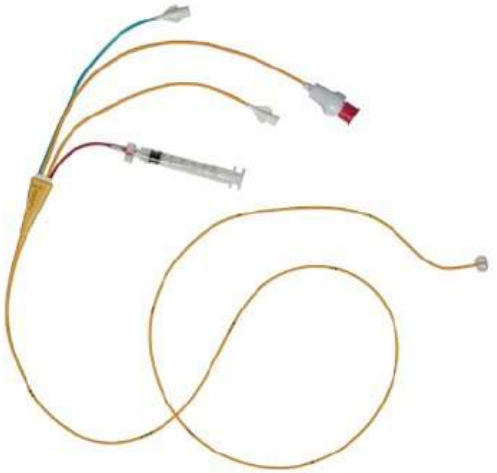
→ LV systolic function
→ LV diastolic function
→ wall motion abnormalities
→ valvular function
→ papillary muscle rupture

Pulmonary artery catheterization

- **definitive determining the cause of acute pulmonary edema**

- **pulmonary capillary wedge pressure (PCWP) / pulmonary artery occlusion pressure (PAOP) enables derivation of LV filling pressure**

- **PCWP / PAOP > 18 mmHg indicates CPE**
- **PCWP / PAOP < 18 mmHg indicates NCPE**



Management and treatment of CPE

1) **treatment of the underlying disease**

2) **haemodynamic optimization**

3) **supportive care**

- **initial management of the criticaly ill patients always according to ABC-approach!**

Respiratory support

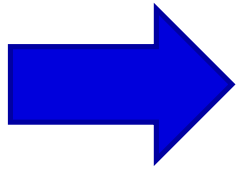
- **oxygen therapy is indicated to keep a SpO2 > 90%**
- face mask
- non-invasive ventilation (NIV)
- mechanical ventilation (MV)

- **hyperoxia is detrimental to coronary vascular tone, ventricular contractily and may worsen outcome of the patients !**

Ventilatory support

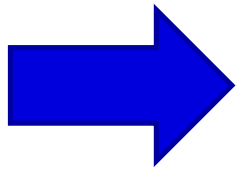
- **Effect of positive airway pressure (NIV, MV)**

↑ alveolar recruitment
↓ extravascular lung water
↓ work of breathing



↑ pulmonary air exchange
↑ global O2 supply/demand balance

↑ intrathoracic pressure



↓ LV preload
↓ LV afterload

Medical treatment

1) preload reduction

2) afterload reduction / optimization

3) contractility improvement

4) symptomatic relief

Preload reduction

- **Effect of preload reduction:**

↓ pulmonary capillary hydrostatic pressure
↓ left ventricular end diastolic pressure
→ ↓ fluid transudation
↓ myocardial wall stress

1) venous tone reduction → vasodilators *(nitrates)*

2) circulating volume reduction → loop diuretics *(furosemide)*
→ renal replacement therapy

Afterload reduction / optimization

- **Effect of afterload reduction:**

↓ systemic vascular resistance → ↑ cardiac output
↑ tissue perfusion

- **Initial target systolic pressure should be ≈ 100mmHg**
- hypertension = vasodilators *(nitroprusside)*
- hypotension = vasopressors *(noradrenaline)*

Contractility improvement

- **inotropic support**
- sympathomimetics *(dobutamine)*
- phosphodiesterase III inhibitors *(milrinone)*
- calcium sensitizers *(levosimendan)*

Symptomatic relief

- **opioids *(morphine)***
- anxiolysis and dyspnea reduction

Take home message

- Shock is a circulatory failure
- It is a life threating condition
- It could be divided into four types: hypovolemic, cardiogenic, obstructive and distributive

MUNI
MED

Lékařská fakulta Masarykovy univerzity
2021