Acute (Adult) Respiratory Distress Syndrome

Synonyms: Adult respiratory distress syndrome (ARDS); Acute lung injury (ALI).

Acute respiratory distress syndrome (ARDS) is a common and devastating condition which affects both medical and surgical patients. It occurs when non-cardiogenic pulmonary oedema (secondary to acute damage to the alveoli) leads to acute respiratory failure.

Epidemiology

Incidence is uncertain, but was reported as 17.9 per 100,000 for acute lung injury and 13.5 per 100,000 for ARDS in a Scandinavian study in 1994.

Causes - Pulmonary & non-pulmonary:

Commonest

- Sepsis
- Massive trauma + shock & multiple transfusions
- Hypovolaemic shock
- Pneumonia
- Gastric aspiration

Other

- Smoke inhalation
- Burns
- Near drowning
- Diabetic ketoacidosis
- Eclampsia
- Amniotic fluid embolus
- Drugs - Paraquat, heroin, aspirin
- Acute liver failure
- Acute pancreatitis
- DIC
- Head injury/↑ICP
- Fat emboli
- Transfusions of blood products
- Heart/lung bypass
- Tumour lysis syndrome
- Pulmonary contusion
- Vasculitis

Pathophysiology - Increased permeability of pulmonary microvasculature causes leakage of proteinaceous fluid across the alveolar-capillary membrane. This may be one manifestation of a more generalized disruption of endothelium, resulting in hypoxia and multiple organ failure.

Clinical features

- **Symptoms**: History of relevant injury and increasing dyspnoea which may occur some time after the precipitating event.
- **Signs**: Cyanosis (reflecting hypoxia refractory to oxygen therapy), tachypnoea, tachycardia, peripheral vasodilatation; bilateral fine inspiratory crackles.

Investigations

- FBC, UEC, LFTs, amylase, clotting, CRP, blood cultures, ABG.
- CXR shows bilateral alveolar shadowing, often with air bronchograms.
- Pulmonary artery catheter to measure pulmonary capillary wedge pressure (PCWP).

Diagnostic criteria

One consensus requires these 4 to exist:

1. Acute onset
2. CXR: bilateral infiltrates
3. PCWP <18mmHg or a lack of clinical evidence of LVF
4. Refractory hypoxaemia: acute lung injury is present when ratio PaO2:FiO2<300; ARDS is present when PaO2: FiO2<200.

20-50% of acute lung injury patients will develop ARDS within 7 days.

Management Admit to ITU, give supportive therapy and treat the underlying cause.

Respiratory support

In early ARDS, CPAP with 40-60% O_2 may be adequate, but most req IPPV

Indications for ventilation:

- PaO_2: <60mmHg (8.3kPa) despite 60% O_2;
- $PaCO_2$: >45mmHg (6kPa)

Cx of conventional ventilation:

- Large tidal volumes (10-15mL/kg) plus reduced lung compliance in ARDS can → high peak airway pressures ± PTX.
- PEEP increases PO_2 at the expense of venous return, cardiac output, and organ perfusion
- Newer approaches include low-tidal-volume techniques e.g. inverse ratio ventilation (Tinsp > Texp), permissive hypercapnia, prone position & high-frequency jet ventilation

Example settings for lung protection (basically all but those with Asthma/COPD):

- *Ventilator Mode:* Volume Assist Control or SIMV modes
- *Lung protection:* TV 6-8ml/kg (aim Plateau Pressure<30cmH_2O)
- *Comfort:* Insp Flow rate 60-80ml/min
- *Ventilation:* RR 16-18 (adjust to keep pH7.3-7.45)
- *Oxygenation:* FiO_2 100% & PEEP 5cmH_2O (drop FiO_2 to 30-40% if ABG adequate and increase PEEP in 2-3cmH_20 per 10% increase in FiO_2 up to max PEEP of 20-24cmH_2O)

Circulatory support

- Invasive haemodynamic monitoring - arterial line and Swan-Ganz, PICCO
- Maintain CO and oxygen delivery with inotropes, vasodilators and blood transfusion.
- ?Consider treating pulmonary hypertension with low-dose (20-120ppm) nitric oxide
- Haemofiltration may be needed in renal failure and to achieve a negative fluid balance.

Experimental therapies

- ?activated protein C,
- ?granulocyte-macrophage colony-stimulating factor
- beta agonists to enhance alveolar fluid clearance.

Sepsis

- Identify organism(s) and treat accordingly.
- If clinically septic, but no organisms cultured, use empirical broad spectrum antibiotics.
- Avoid nephrotoxic antibiotics.

Nutritional support

Enteral is better than parenteral feeding.

Steroids do not improve mortality in the acute phase but may be of benefit later on (>7 days)

Prognosis

- Overall mortality is 50-75%. Depends on:
    - Age of patient
    - Cause of ARDS (pneumonia 86%, trauma 38%)
    - Number of organs involved (3 organs involved for >1 week is invariably fatal).
- Survivors' lung function returns to almost normal within 6-12 months.
- Patients with acute lung injury have reduced exercise capacity up to two years