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 can affect all adult patients - eg, medical, surgical and obstetric patients. It occurs when non-cardiogenic pulmonary oedema (secondary to acute damage to the alveoli) leads to acute respiratory failure. Although the terms ARDS and ALI are used interchangeably, the American-European Consensus Conference Committee describes them as different entities - ALI having less severe hypoxaemia than ARDS.[1]

Epidemiology

Incidence is uncertain, but was reported as 190,000 in the US population in 1999-2000 and has been declining since.[1, 2]

Aetiology

The more risk factors present, the greater the chance of ARDS.

Most common risk factors[2]

- Sepsis
- Massive trauma with shock and multiple transfusions
- Hypovolaemic shock
- Pneumonia
- Gastric aspiration

Other risk factors

- Smoke inhalation
- Burns
- Near drowning
- Diabetic ketoacidosis
- Pregnancy
- Edampsia
- Amniotic fluid embolus
- Drugs - paraquat, heroin, aspirin
- Acute pancreatitis
- Disseminated intravascular coagulation (DIC)
- Head injury/raised intracranial pressure (ICP)
- Fat emboli
- Transfusions of blood products
- Heartlung bypass
- Tumour lysis syndrome
- Pulmonary contusion

Pathophysiology

Increased permeability of pulmonary microvasculature causes leakage of proteinaceous fluid across the alveolar-capillary membrane.[2, 3] This may be one manifestation of a more generalised disruption of endothelium, resulting in hypoxia and multiple organ failure. There is also evidence of inflammation in the lung tissue which can be seen on metabolic imaging methods.[2, 4]

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, U&E, LFTs, amylase, clotting, CRP, blood cultures, ABG.
- CXR shows bilateral alveolar shadowing, often with air bronchograms.
- Other investigations as deemed by the clinical scenario - eg, echocardiography.

**Diagnostic criteria**

The current consensus requires these three to exist:\[5, 6\]

- Acute onset: 20-50% of acute lung injury patients will develop ARDS within seven days.
- CXR shows bilateral infiltrates.
- Refractory hypoxaemia: acute lung injury is present when the ratio \(\text{PaO}_2:\text{FiO}_2<300\); ARDS is present when \(\text{PaO}_2:\text{FiO}_2<200\).

Previously pulmonary capillary wedge pressure (PCWP) \(\leq 18\) mm Hg was part of the diagnostic criteria, but this has since been removed. Clinical judgement based on each individual case is deemed important; thus ARDS risk factors should be sought and other causes should be excluded.

ARDS can be categorised into mild, moderate or severe based on \(\text{PaO}_2/\text{FiO}_2\) made using a ventilator.\[5, 7\]

**Management**

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

**Respiratory support**

In early ARDS continuous positive airway pressure (CPAP) with \(40-60\%\) oxygen may be adequate to maintain oxygenation. But most patients need mechanical ventilation.

The large tidal volumes (10-15 mL/kg) produced by conventional ventilation plus reduced lung compliance in ARDS may lead to high peak airway pressures ± pneumothorax. Positive end-expiratory pressure (PEEP) increases oxygenation but at the expense of venous return, cardiac output, and perfusion of the kidneys and liver. Low tidal volume ventilation, ie \(\leq 6\) mL/kg predicted body weight is the only form of ventilation associated with improved survival.\[8, 9\] Newer approaches include inverse ratio ventilation (inspiration > expiration), permissive hypercapnia, prone position and high-frequency jet ventilation, and other low-tidal-volume techniques.\[8, 10, 11, 12\]

Prone ventilation has been shown to improve alveolar gaseous exchange.\[13\] This has also been used with good effect in a pregnant patient who experienced blunt chest trauma.\[14\]

**Circulatory support**

Invasive haemodynamic monitoring with an arterial line and Swan-Ganz catheter may be helpful in monitoring pulmonary capillary wedge pressure and cardiac output.

Maintaining cardiac output and thus oxygen delivery usually needs inotropes (eg, dobutamine), vasodilators and blood transfusion. Fluid rehydration needs to be carefully balanced and in some cases negative fluid balance is the objective. This may require haemofiltration in extreme cases.

**Pulmonary hypertension** has been treated with low-dose (20-120 ppm) nitric oxide, a selective pulmonary vasodilator.\[15\] However, a systematic review and meta-analysis found that nitric oxide only provides short-term improvement and does not affect survival. Furthermore, nitric oxide was associated with renal dysfunction.\[16\]

**Other therapies**
Steroids, such as methylprednisolone were used a number of years ago but due to adverse effects are no longer recommended. Earlier evidence suggested that low doses of steroids are associated with a reduced duration of ventilation in early ARDS (duration less than seven days), but without overall improvements in survival rates.\[1,\] ARDS which persists beyond seven days is mediated by an ongoing inflammatory process and small studies had shown improvements in mortality with methylprednisolone in this group. The presence of eosinophilia in the blood or in bronchial-alveolar-lavage (BAL) identified those most likely to respond.\[11\] However corticosteroid use was associated with a significant number of adverse effects such as, septicemia and hyperglycaemia, laying doubt on the benefits. The Late Steroid Rescue Study was undertaken in an attempt to clarify the role of steroids in ARDS persisting beyond 7 days.\[11\] There was a significant increase in mortality at 60 days with methylprednisolone, especially if treatment was begun after 14 days. This was despite improvements in secondary measures such as, changes in markers of inflammation and intensive care unit-free days.

The role of ketoconazole has also proved to be disappointing.\[11\] More novel therapies currently include activated protein C, granulocyte-macrophage colony-stimulating factor and the use of beta agonists to enhance alveolar fluid clearance.\[15\]

Sepsis

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

Other supportive care\[17\]

- Nutritional support - enteral feeding is better than parenteral feeding.
- Venous thromboembolism prevention with low molecular weight heparin.
- Gastric ulcer prevention with prophylactic medications.

Prognosis

- Overall mortality is 50-75%. and prognosis varies with age of patient, cause of ARDS (pneumonia 86%, trauma 38%), and number of organs involved (three organs involved for >1 week is invariably fatal).\[18\]
- In most cases, survivors' lung function returns almost to normal within 6-12 months. However, some may have persistent reduced vital capacity and even obstructive lung disease, although these are usually asymptomatic.\[19\]
- Interestingly, patients with ALI have reduced exercise capacity up to two years after the episode (despite normal lung function) and there is evidence to suggest long-term neurocognitive impairment.\[17,19\]

Further reading & references


18. Longmore, JM, Wilkinson, IB, Rajagopalan, SR; Mini Oxford Handbook of Clinical Medicine, Oxford University Press, 2006


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