MANAGEMENT OF ARDS IN A POLYTRAUMA PATIENT

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Abstract:
This was a case of polytrauma involving musculoskeletal system and lungs. During initial evaluation lung contusion was not evident. As the injury worsened patient became hypoxic which manifested as low saturation and irritability in the post operative period. Associated hypotension could have aggravated the cerebral ischemia. Patient was diagnosed as having ARDS by clinical, biochemical and radiological findings. Onset of ARDS following the lung injury, complicated the condition further. HIE was diagnosed by neurophysician. Cerebral ischemia in this case, probably resulted from primary lung injury, ARDS and hypotension. Presence of long bone fracture explains the hypotension. Inotropic support was given along with aggressive volume resuscitation with blood and crystalloids. There was improvement in the hemodynamic status. Lung protective ventilation strategy was followed to manage ARDS. CVP guided fluid therapy was given. Glycemic control was maintained. Gradual improvement in lung injury and oxygenation was indicated by clinical examination, ABG analysis and serial chest X-ray. Though cerebral ischemia took sometime for recovery, it was almost a complete one.

Keyword: polytrauma, lung contusion, ARDS, hypoxic ischemic encephalopathy

27 year old male patient was admitted in the trauma ward with alleged history of road traffic accident. On examination GCS 15/15, PR-112/min, BP-110/70mmHg, SpO2-94%. Left femur fracture was diagnosed, and patient was taken up for emergency orthopaedic surgery under subarachnoid block after routine basic investigations, which were normal. Intra operative period was uneventful and patient was shifted to post-operative ward. 6 hours later he became dyspnoeic and irritable, on examination, PR-132/min, BP-80/60mmHg, RR-30/min, SpO2 90% in room air, 92% with supplemental oxygen, bilateral crepitations present. Dopamine infusion
was started at 10mics/kg/min rate. Fat embolism was suspected and as the saturation decreased further, patient was intubated and connected to ventilator in SIMV mode, SpO2 improved to 95% with FiO2-0.7. Patient was given mild sedation. Investigations including CXR were ordered. On day three patient became pale and saturation was 86% with FiO2-1, crepitations increased, bilateral air entry diminished, GCS deteriorated -E2VTm5, PR-140/min, BP was 70/50mmHg with inotrope. Subcutaneous emphysema was noticed in the neck. CT-chest was taken which showed bilateral extensive lung contusion with hemopneumothorax. ICD tube was inserted on both sides. In this condition patient was received in the Post Anesthesia Care Unit (PACU) for further management.

**ICD tube insitu**

3 units of blood was transfused, dopamine support was increased to 15mics/kg/min. Patient was paralysed with vecuronium 4mg and put on CMV mode with TV-500ml, RR-14bpm. Saturation improved to 94%. Right subclavian vein was cannulated. Bed side chest X-ray taken. ABG- pH 7.21, PaCO2-51mmHg, PaO2-86mmHg with FiO2 0.7, PaO2/FiO2-122. CXR showed bilateral fluffy infiltrates. CVP was 12cmH2O. Hemoglobin -8gms/dl, Blood sugar-104mg/dl, urea-38mg/dl, serum creatinine-1.4mg/dl, ECG -normal. Patient was diagnosed as having ARDS with underlying lung contusion by clinical, biochemical and radiological findings. ARDS lung protective ventilatory management was started. Patient was kept in CMV mode with VT-6ml/kg, RR-20/min, Fio2-0.5, PEEP-8 with target plateau pressure (Ppl) as less than 30cms H2O. Patients Ppl was 21cms H2O, PIP-20cms of H2O. Fluid therapy was planned to maintain CVP at 10-12mmHg.

In the next four days there was improvement in hemodynamic status and respiratory parameters, but the GCS deteriorated to E1VTm5 with sluggish pupillary reaction. Hypoxic ischemic encephalopathy was diagnosed by the neurophysician. Glycemic status was monitored for cerebral protection. TPN was started. Tracheostomy was done. Patient was in the SIMV mode with very minimal crepitations. ABG was PaO2/FiO2-335, pH -7.47, PaO2-134mmHg, PaCO2-37mmHg, HCO3-24mmol/L. ICD was removed.

On day 8, there was improvement in GCS-E1VTM5 and on day 9 it was E4VTM6. CPAP trial was followed by T-piece ventilation. On day 10 tracheostomy site was closed and the patient was shifted to ward with 15/15GCS, stable hemodynamic status and normal blood gas values fulfilling Aldrete discharge criteria. **Discussion ARDS**

Acute respiratory distress syndrome (ARDS) is an inflammatory response of the lung to both direct and indirect insults(1). It is characterized by inflammation of the lung parenchyma leading to impaired gas exchange. This condition is often fatal, usually requiring mechanical ventilation. A less severe form is called acute lung injury. **Epidemiology**

The annual incidence of ARDS is 1.5–13.5 people per 100,000 in the general population. Prevalence
of acute lung injury is 16.1\% percent in ventilated patients admitted for more than 4 hours. More than half these patients may develop ARDS.

Observational studies generally report 50–60\% mortality

**Aetiology/Trigger**

Pneumonia and sepsis are the most common triggers, and pneumonia is present in up to 60\% of patients. Pneumonia and sepsis may be either causes or complications of ARDS. Mechanical ventilation, sepsis, pneumonia, shock, aspiration, **trauma (especially pulmonary contusion)**, major surgery, massive transfusions, smoke inhalation, drug reaction or overdose, fat emboli and reperfusion pulmonary edema after lung transplantation or pulmonary embolectomy may all trigger ARDS (2).

**Pathophysiology**

The pathology most commonly associated with ARDS is diffuse alveolar damage (DAD). The triggering insult usually results in an initial release of cytokines and other inflammatory mediators which is amplified by neutrophils and some T-lymphocytes(3). Typical histological presentation involves diffuse alveolar damage and hyaline membrane formation in alveolar walls. **Phases** 1. Exudative phase- characterized by diffuse alveolar damage. Cardinal features are a) accumulation of extravascular lung water, protein, inflammatory cells- precipitates of which results in , characteristic intra-alveolar “hyaline membranes”. b) type 1 alveolar cell necrosis .c) intra-alveolar haemorrhage (4). 2. Proliferative phase- features include increased number of type 2 pneumocytes, clearing of alveolar edema and debris, improving gas exchange, eventually liberation from ventilator. 3. Fibrotic phase- This stage is characterized by progressive interstitial and alveolar fibrosis, appearance of large bullae, prolonged ventilator dependency with increased morbidity and mortality.

**Signs and symptoms** People usually present with dyspnoea, tachypnea , tachycardia and occasionally with confusion resulting from low oxygen levels. Other symptoms of aetiology and complications may be present. **Diagnosis** Clinical evaluation, arterial blood gas analysis and chest X-ray allow diagnosis of ARDS by the following criteria(1):

1. Acute onset with direct or indirect pulmonary insult, 2. Bilateral infiltrates on chest radiograph sparing costophrenic angles 3. Pulmonary artery wedge pressure < 18 mmHg (obtained by pulmonary artery catheterization), if this information is available; if unavailable, then lack of clinical evidence of left ventricular failure suffices 4. if PaO2:FiO2 < 300 mmHg (40 kPa) acute lung injury (ALI) is considered to be present and if PaO2:FiO2 < 200 mmHg (26.7 kPa) to be present.
Acute respiratory distress syndrome (ARDS) is considered the most reliable method for confirming or excluding the diagnosis of ARDS is bronchoalveolar lavage analyzing neutrophil density and protein concentration.

Progression
If the underlying disease or injurious factor is not removed ARDS may progress to SIRS and MODS.

Treatment
Acute respiratory distress syndrome is usually treated with mechanical ventilation in the Intensive Care Unit. Treatment of the underlying cause is important. Appropriate antibiotic therapy must be administered as soon as microbiological culture results are available. **Lung protective ventilation strategy**

(6) Goals

- PaO2- 55-80mmHg, SpO2- 88-95%, FiO2 < 0.6, Plateau pressure < 30cmH2O, pH > 7.3-7.45.
- Calculate patients predicted body weight (PBW) initial tidal volume - 8ml/kg PBW, reduce tidal volume by 1ml/kg every 2 hours until TV-6ml/kg PBW. This strategy reduced the mortality rate by 22% in a study (7).
- Allowing hypercapnia to persist in favour of maintaining lung protective low volume ventilation is known as permissive hypercapnia (8).

**Plateau pressure (Ppl)**

If Plateau pressure > 30cmH2O or TV down to 4ml/kg .pH

If pH 7.15-7.30 increase respiratory rate until pH > 7.3 or respiratory rate = 35bpm. If pH < 7.15 increase respiratory rate to 35bpm. If pH is still < 7.15 increase tidal volume at 1ml/kg increments until pH >7.15. FiO2

**Fluid management**

Fluid management often needs care, preferably CVP guidance as both overloading (pulmonary edema) (6) and volume depletion (hemodynamic instability) are dangerous.

**Role of steroids**

Conflicting evidences are available. A Meduri et al. study has found significant improvement in ARDS using modest doses of corticosteroids during fibrinoproliferative phase (9). This was a study involving a small number of patients in one center. But ARDSnet LAZARUS study of corticosteroids for ARDS found that they are not efficacious in ARDS. **Promoting oxygen transport** To maintain O2 delivery, cardiac output (5-6L/min) and haemoglobin level (10g/dl) have to be maintained.

**Adjunct approaches to ARDS treatment**

1. inverse ratio ventilation 2. airway pressure release ventilation 3. proportional assist ventilation 4. prone position ventilation (10) 5. high frequency ventilation 6. tracheal gas insufflation 7. extracorporeal membrane oxygenation

**New approaches to ARDS treatment**


**Complications**

1. Pulmonary: barotrauma (volutrauma), pulmonary embolism, ventilator-associated pneumonia
2. Cardiac: arrhythmias, myocardial dysfunction
3. Renal: acute renal failure, positive fluid balance

**Pulmonary injury** - Pulmonary contusion is the most common type of potentially lethal chest trauma.
It occurs in 30–75% of severe chest injuries. No treatment is known to speed the healing of a pulmonary contusion; the main care is supportive.

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**New Hypoxic ischemic encephalopathy**

Treatment of hypoxic-ischemic encephalopathy (HIE) is largely supportive and should focus on adequate ventilation and perfusion, careful fluid management, avoidance of hypoglycemia and hyperglycemia and treatment of seizures.

**Conclusion**

Chest injury is common in polytrauma and pulmonary contusion is a common and potentially lethal form of chest injury. It may lead on to ARDS which in turn can cause hypoxic brain injury if severe. Though the mortality is very high in ARDS (50-60%), early and appropriate intervention may save the patient. Similarly hypoxic ischemic encephalopathy is also sometimes reversible to a large extent especially when the brainstem is spared.

**References:**


