CASE REPORT

ROLE OF ANTI-INFLAMMATORY DRUGS IN TRAUMATIC ARDS

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ABSTRACT: ALI/ARDS is an acute pathological process with dynamic and complex inflammatory response. In traumatic ARDS inflammatory response plays major role in outcomes. Anti-inflammatory agents may play major role in recovery. We report successful management of early traumatic ARDS with lung protective strategy along with Anti-inflammatory agents. Larger trials are needed to assess Anti-inflammatory agents in ALI/ARDS.

KEYWORDS: ALI/ARDS, Inflammatory Response, Steroids, Ulinastatin.

INTRODUCTION: ARDS is a dynamic and complex inflammatory response. ARDS in trauma patients, though identified in 1967, by Ashbaugh, and Levine², Sepsis ARDS, is more evaluated than trauma ARDS in many studies. The trauma population constitutes a more homogeneous ARDS group with greater physiological reserve because of their lower age and lesser co-morbidity. Recent analysis of National Trauma Data Bank shows, the incidence of Trauma related ARDS to be 6.5 %, ¹ requiring Ventilation for more than 48 hours. Two forms of ARDS have been described 1) Early ARDS: during first 48 hours after Trauma and related to Haemorrhagic Shock 2) Late ARDS: after 48 hours and more related to Organ Failure and Sepsis. Here we present early ARDS without Haemorrhagic Shock.

CASE REPORT: A male patient of 31 year old with no co morbidity, sustained crush injury with both bone fracture of right leg when a stationary vehicle rolled on its own. There are no other external injuries seen. Patient GCS in ER in 15/15 with stable hemodynamics and maintaining saturation on room air His routine investigation including chest x-ray are within normal limits. Immediate fixation of bones was not considered as patient wants to get surgery done through government sponsored programme.

Patient was managed symptomatically and shifted to floors. After 3 hours patient started complain of SOB and tachypnea his saturation came down to 84% and with oxygen supplementation with 10 liters, SpO₂ improved to 90% patient but, patient continue to have tachycardia and tachypnea with normal blood pressure. ABG and Chest x-ray, ECG along with CBC, CRP, and D-dimer immediately sent. His ABG showed PO₂ 48 and pCO₂ 36 pH 7.416 Hco₃ is 23, chest x-ray showed bilateral infiltrates, his ECG is normal, CRP level positive – 11.0mg/dl with no drop in Hb% and D-dimer negative.

A provisional diagnosis of Fat Embolism Syndrome / ARDS was considered. Patient was kept on NIV S/T with 15 liter of Oxygen and IPAP 14 EPAP 10, after one hour his ABG was Po₂ 50.7 Pco₂ 38.3 pH 7.24 with respiration rate more than 35 per minute. Patient was intubated and connected to ventilator, he is ventilated in pressure control mode with lung protective strategy and PEEP of 12, his CBC did not show thrombocytopenia no rash was observed, his 2D Echo was normal. His fat embolism score is low. He is ventilated on first day with fentanyl infusion 50 – 100 mic per hour and vecuronium 4 to 8mg, whenever needed.

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Patient was kept on Inj. Ceftriaxone one gm bid, Injection Hydrocortisone 100 milligram bolus followed by 10 milligram/hr infusion, injection Ulinastatin one lac units Tid, and LMWH 0.4 SC OD. His 2nd day ABG PO₂ 121 PCO₂ 19.4 and HCO₃ 25.6 with FiO₂ 0.6 and PEEP 12, his "crp" level is positive 6.1 mic/dl hydrocortisone infusion continued.

On 3rd day his ABG showed Po₂ 85.7 with Fio₂ 0.4 and PEEP 8, crp level is 4.8mg/dl he is maintained with light sedation and spontaneous breath trail given, injection hydro cortisone to converted to 100 milligram to TID, bronchoscopy was done, which is within normal limit and BAL showed no growth and fat globules. On 5th day patient was converted to spontaneous mode with PEEP six and FiO₂ .35, his ABG showed pO₂ 106 and he is Extubated and shifted to floor after one day, his CRP level is 3.4 micro grams /dl on day 5.

DISCUSSION: Trauma has been identified to constitute 7%⁴ of ARDS cases the incidence of ARDS did not differ between blunt and penetrating mechanism of injury. The incidence of ARDS in trauma was higher in older age group and female population.⁹ The mortality of trauma – ALI is estimated at 24%. The 60 day mortality day rate of trauma patient in ARDS network trial hospital is a 10%, it has not seen any further decrease in recent times.

The mortality between female and male patients with ARDS after traumatic injury does not appear to differ. The mortality of ARDS in victims of trauma is lower than those with ARDS from other causes after adjusting for baseline differences. Over the last decade³ mortality rate have continue to decrease in the general population with ALI / ARDS, this changes might be because of change in ventilation strategies , fluid management and overall organ specific management.

A 2006 retrospective review of trauma ICU data¹ at the University of Southern California showed an overall complication rate of 43% in patient with ARDS. Complication included Pneumonia (50%), deep venous thrombosis, Pulmonary Embolism, acute renal failure and DIC. ARDS patient had longer hospital stage (32days), longer ICU stays (22days), crude mortality rate is 19% and higher hospital charges.

Independent risk factors for the development of ARDS after trauma are those with an injury severity score (ISS) > 25, increasing Acute Physiology and Chronic Health Evaluation II (APACHE II) scores, the presence of pulmonary contusion, a large transfusion requirement, hypotension on admission, age >65 years, duration of mechanical ventilation along with delayed stabilization of femoral bone fracture.

ARDS following trauma is different in many respects from ARDS related to other causes. Patients with ARDS after injury tend to be younger and have fewer comorbid medical conditions. Endothelial activation factor levels are lower in patients with ARDS related to trauma as opposed to septic insults.

Following cellular injury, endothelial cells can become activated, a principal mechanism in the complex pathologic events that result in ARDS. Subgroup analysis of ARDS Network data⁴ has shown significantly lower levels of serum biomarkers (von Willebrand factor antigen [vWF], surfactant protein D [SP-D], tumour necrosis factor receptor-1 [TNFr-1], and intercellular adhesion molecule-1 [ICAM-1]), associated with poor outcome in trauma patients.

This patient had crush injury with long bone fracture and no other injury he had stable vitals and conscious and coherent. He developed symptoms within 4hours from inciting injury and developed hypoxia and bilateral infiltrates on chest X-ray, his 2D Echo is normal.

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Differential diagnosis of FES considered but except for hypoxia, tachycardia and tachypnea no other criteria met (Gurd and Wilson criteria). Lung contusion and pulmonary haemorrhages are considered but no feature of chest trauma seen. Patient didn't receive any blood transfusion or massive fluid resuscitation.

Patient was managed with lung protective strategy, conservative fluid management. His CRP a nonspecific marker of inflammation is high, so patient is kept on hydrocortisone infusion and Ulinastatin,^{7,8} a serine protease inhibitor which inhibits several pro-inflammatory proteases and decreases inflammatory cytokine levels, to decrease the inflammatory changes. It is observed that the CRP levels decreased have the patient conditions improved. Therapeutic usage of Ant-inflammatory agents might help in decreasing complications.

The role of steroids^{5,6} in traumatic ARDS, not much evidence is available, but the recent meta-analysis showed that early steroids treatment in ARDS decreased ventilator days, ICU days, and hospital stay. The use of Ulinastatin is initiated with hope of decreasing SIRS response. The role of Ant-inflammatory needs consideration in early ARDS. Larger studies and controlled studies regarding efficacy of steroid with or without Ulinastatin is required.

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