

Original Article

Analysis of Incidence and Outcome of Acute Lung Injury in Patients with Traumatic Brain Injury at a Tertiary Care Hospital

Anand Prakash¹, Jyoti Gupta², CB Sahay³

¹Associate Professor, Department of Neurosurgery, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India.

²Senior Consultant, Department of Anesthesia, CCL Hospital, Gandhinagar, Ranchi, Jharkhand, India.

³Professor, Department of Neurosurgery, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India.

Corresponding Author

Dr. Anand Prakash,

Associate Professor,

Department of Neurosurgery,

Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India.

Email: dr.anand.prakash123@gmail.com

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ABSTRACT

Background: To assess the incidence and outcome of acute lung injury in patients with traumatic brain injury. **Materials & Methods:** A total of 200 patients with presence of traumatic brain injury were enrolled. Complete demographic and clinical details of all the patients was obtained. Emergency care was done and radiographic analysis of all the patients was done. CT and MRI findings were separately recorded. Chest radiographs were carried out. Assessment of incidence of acute lung injury was done. **Results:** Out of 200 patients, acute lung injury was seen in 20.5 percent of the patients. Out of 41 patients with presence of acute lung injury, mean hospital stay was 16.2 days while good neurological outcome was seen in 18.29 percent of the patients. Overall mortality was seen in 19.77 percent of the patients. **Conclusion:** The mechanism of TBI-associated acute lung injury is multifactorial. Hence, adequate screening of pulmonary functions with prompt treatment should be done.

Key words: Acute Lung Injury, Traumatic Brain Injury.

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INTRODUCTION

Traumatic brain injury (TBI) presents in various forms ranging from mild alterations of consciousness to an unrelenting comatose state and death. In the most severe form of TBI, the entirety of the brain is affected by a diffuse type of injury and swelling. Treatment modalities vary extensively based on the severity of the injury and range from daily cognitive therapy sessions to radical surgery such as bilateral decompressive craniectomies.¹⁻³

The fact that acute injury can lead to long-term consequences, including progressive brain atrophy and an increased vulnerability to neurodegenerative disorders, is a critical problem. In some models of TBI, immunocytochemical evidence for chronic traumatic encephalopathy (CTE) has been presented. Indeed, several studies have emphasized that months to years after injury, evidence for progressive gray and white matter (GM/WM) atrophy is observed after TBI. Similar observations have been shown in TBI patients using computed tomography

and magnetic resonance imaging (MRI) modalities.⁴⁻⁶ Acute lung injury (ALI) and the acute respiratory distress syndrome (ARDS) describe clinical syndromes of acute respiratory failure with substantial morbidity and mortality. Even in patients who survive ALI, there is evidence that their long-term quality of life is adversely affected. Recent advances have been made in the understanding of the epidemiology, pathogenesis, and treatment of this disease. However, more progress is needed to further reduce mortality and morbidity from ALI and ARDS.^{7,8} Hence; the present study was conducted for assessing the incidence and outcome of acute lung injury in patients with traumatic brain injury.

MATERIALS & METHODS

The present study was conducted in Department of Neurosurgery, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand (India) for assessing the incidence and outcome of acute lung injury in patients with traumatic

brain injury. A total of 200 patients with presence of traumatic brain injury were enrolled. Complete demographic and clinical details of all the patients was obtained. Emergency care was done and radiographic analysis of all the patients was done. CT and MRI findings were separately recorded. Chest radiographs were carried out. Assessment of incidence of acute lung injury was done. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software.

RESULTS

A total of 200 patients with presence of traumatic brain injury were analysed. Mean age of the patients was 46.26 years. Out of these 200 patients, 136 patients were males while the remaining 64 were females. median APACHE II Score on admission was 23. Fifty six percent of the patients had Glasgow Coma Scale (GCS) score between 3 to 5. Out of 200 patients, acute lung injury was seen in 20.5 percent of the patients. Out of 41 patients with presence of acute lung injury, mean hospital stay was 16.2 days while good neurological outcome was seen in 18.29 percent of the patients. Overall mortality was seen in 19.77 percent of the patients.

Table 1: Demographic data of patients with traumatic brain injury

| Variable | Number | Percentage | |
|---|---------|------------|----|
| Gender | Males | 136 | 68 |
| | Females | 64 | 32 |
| Mean age (years) | 46.25 | | |
| Median APACHE II Score on admission | 23 | | |
| Patients with GCS score between 3 to 5 on admission | 112 | 56 | |

Table 2: Incidence of acute lung injury

| Acute lung injury | Number | Percentage |
|-------------------|--------|------------|
| Present | 41 | 20.5 |
| Absent | 159 | 79.5 |
| Total | 200 | 100 |

Table 3: Outcome of patients with acute lung injury

| Outcome variables | Number | Percentage |
|---------------------------|-----------|------------|
| Length of hospital stay | 16.2 days | |
| Good neurological outcome | 15 | 18.29 |
| Mortality | 16 | 19.77 |

DISCUSSION

TBIs are classified as focal or diffuse based on the presence or absence of focal lesions. Although injuries may be considered predominantly focal or diffuse, most injuries are heterogeneous with both focal and diffuse components. Mass lesions, such as contusion, subdural hematoma, epidural hematoma, and intraparenchymal hemorrhage are considered focal injuries, whereas diffuse injury encompasses axonal injury, hypoxic-ischemic injury, and microvascular injury that affect widely distributed anatomic regions. The mortality rate for severe focal injuries is reported to be approximately 40% and, for severe diffuse injuries, approximately 25%.⁶⁻⁸

TBI is commonly classified into primary and secondary brain injuries. Primary brain injury involves damage to brain tissue resulting from the transfer of kinetic energy. Secondary brain injury is a term used to describe the

aggravation of TBI over subsequent minutes to hours as a consequence of various factors, such as hypoxemia, hypotension, hypo- or hyper-carbia, hypo- or hyper-glycemia, hypo- or hyper-thermia, and seizures. Prevention of secondary brain injury is the primary concern of therapeutic interventions following TBI, which can best be managed by an anaesthesiologist, preferably a neuroanesthesiologist.^{8, 9} Hence; the present study was conducted for assessing the incidence and outcome of acute lung injury in patients with traumatic brain injury

A total of 200 patients with presence of traumatic brain injury were analysed. Mean age of the patients was 46.26 years. Out of these 200 patients, 136 patients were males while the remaining 64 were females. median APACHE II Score on admission was 23. Fifty six percent of the patients had Glasgow Coma Scale (GCS) score between 3 to 5. Out of 200 patients, acute lung injury was seen in 20.5 percent of the patients. Kerr, Nadine A et al, summarized that TBI is associated with higher rates of various medical complications, in particular pulmonary and CNS (central nervous system) dysfunction. In this study, we show that (a) severe TBI increases HMGB1 and inflammasome protein expression in cortical and lung tissue and induces changes in lung morphology consistent with ALI; (b) TBI results in pyroptosis in lung tissue and increased expression of inflammasome proteins in type II alveolar epithelial cells; and (c) adoptive transfer of EV from TBI mice activates the inflammasome and induces ALI, indicating that brain injury induces the release of EV containing a cargo of inflammasome proteins that are then carried to the lungs resulting in ALI. These findings support the concept of a neural-respiratory-inflammasome axis that contributes to the pathology in TBI-induced ALI.¹⁰ Mortality after severe TBI results not only from direct brain damage but also from pulmonary edema and bacterial infection, which is, in part, due to systemic immunosuppression. Specifically, TBI increases the risk for nosocomial pneumonia secondary to neuronal deficits, including altered mental status, dysphagia, impaired gag and cough reflexes, and inability to clear secretions. A variety of retrospective and prospective clinical trials have been performed that examine the relationship among the severity of TBI, the incidence of nosocomial pneumonia [including early vs. late ventilator-associated pneumonia (VAP)], and the microorganisms that increase morbidity and mortality in post-TBI VAP.^{11, 12}

Out of 41 patients with presence of acute lung injury, mean hospital stay was 16.2 days while good neurological outcome was seen in 18.29 percent of the patients. Overall mortality was seen in 19.77 percent of the patients. Bratton, S. L et al determined the incidence of acute lung injury (ALI) in comatose patients after isolated traumatic brain injury. Descriptive epidemiology and a case-control study using the Traumatic Coma Data Bank was performed to evaluate clinical features and brain lesions associated with ALI in patients with isolated head trauma. Twenty of 100 comatose patients developed ALI. Patients with ALI were almost three times more likely to die or survive in a vegetative state. Specific anatomic brain lesions diagnosed by cranial computed tomographic scans were not associated with ALI. However, patients with more severe injuries, i.e., large nonevacuated mass lesions, and those with midline shift demonstrated a 10- and 5-fold increased risk of ALI. ALI was common in comatose victims with an isolated traumatic brain injury and was associated with an increased risk of death or a severe neurological morbidity.¹³

CONCLUSION

The mechanism of TBI-associated acute lung injury is multifactorial. Hence; adequate screening of pulmonary functions with prompt treatment should be done.

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