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Severe Traumatic Brain Injury with Multiorgan Dysfunction: Case Report of A Critical Case

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ABSTRACT

Background:

Severe traumatic brain injury (TBI) predisposes patients to secondary systemic complications such as sepsis, acute respiratory distress syndrome (ARDS), and acute kidney injury (AKI). These complications significantly worsen neurological outcomes and increase ICU mortality. Early recognition, evidence-based critical care, and continuous nursing vigilance are crucial for survival. This case report highlights the multidisciplinary management of a young female with severe TBI who developed sepsis, severe ARDS, and sepsis-associated AKI requiring renal replacement therapy.

Case Report:

We report the multidisciplinary management of a complex critical care case of a 30 yrs old female admitted with closed head injury and cerebral edema following trauma. She demonstrated rapid neurological deterioration requiring intubation and mechanical ventilation. Her ICU course was complicated by multiple issues making her condition more fragile.

Conclusion:

This case demonstrates the complex interplay between severe TBI and secondary multisystem complications. Coordinated multidisciplinary critical care interventions, combined with vigilant nursing surveillance, played a central role in preventing further deterioration and achieving a favorable outcome despite severe ARDS and AKI.

Keywords: Traumatic Brain Injury, Sepsis, Acute Kidney Injury, Ventilator-associated Pneumonia, Multidisciplinary Care, Nursing Care.

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INTRODUCTION

Traumatic brain injury (TBI) remains a leading cause of acute neurological admission and disability in young adults worldwide. Although males account for the majority of traumatic injuries, women experience TBI and its complications in substantial numbers and may have different clinical trajectories and outcomes. Severe TBI frequently triggers a cascade of systemic complications — most importantly acute respiratory distress syndrome (ARDS), sepsis and sepsis-associated acute kidney injury (AKI) — all of which markedly increase morbidity, length of ICU stay and mortality. The present case, a 30-year-old female with closed head injury complicated by ARDS, Ventilator-associated infection, sepsis and AKI requiring dialysis, exemplifies this high-risk cascade and the key role of vigilant nursing and multidisciplinary care in achieving survival with functional recovery.[1]

A traumatic brain injury (TBI) is defined as an alteration in brain function or evidence of brain pathology resulting from an external physical force. It may occur due to a strong blow, jolt, or impact to the head or body, although not every mechanical insult leads to neural injury. Penetrating trauma in which an object breaches the skull and enters intracranial tissue can also produce significant damage. In clinical terminology, TBIs are frequently grouped under the broader categories of head injury and brain injury.[2]

Early manifestations of traumatic brain injury usually include headache, dizziness and confusion which tend to appear immediately after the incident and gradually resolve. Emotional or behavioral disturbances such as irritability, mood fluctuations and frustration may evolve during the healing phase. Prompt medical evaluation is essential if any concerning symptoms arise within the first day following the injury. Patients may progress to conditions ranging from a minimally conscious state to unresponsive wakefulness syndrome (UWS), coma, or eventually brain death, depending on the severity and extent of damage.[3]

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The neuropathological effects of TBI vary by type of insult. Damage may be confined to a well-defined region (focal injury) or spread across multiple brain areas (diffuse injury). Traumatic brain injury may cause intracranial hemorrhage, cerebral parenchymal edema or diffuse axonal injury which usually arise secondary to stretching or tearing of neuronal fibers. These primary injuries in cases of traumatic brain injuries often trigger a cascade of secondary processes such as neuroinflammation, cerebral edema and metabolic deregulation. All of these consequences may further worsen neurological outcomes.[4]

Optimal management of these cases relies on early airway protection, lung-protective ventilation and strict adherence to sepsis bundles.[5] Additional measures may include timely tracheostomy, and coordinated multidisciplinary care including intensivists and neurosurgeons and highly skilled nursing teams. Evidence suggests that early tracheostomy in selected patients can reduce ventilator days and may improve survival, while multidisciplinary ICU involvement is associated with better overall outcomes.

CASE REPORT

A 30 yrs old female patient was admitted in a drowsy condition with a Glasgow Coma Scale (GCS) of 8/15 following a closed head injury. An urgent CT scan was done which showed mild cerebral edema (Figure 1).

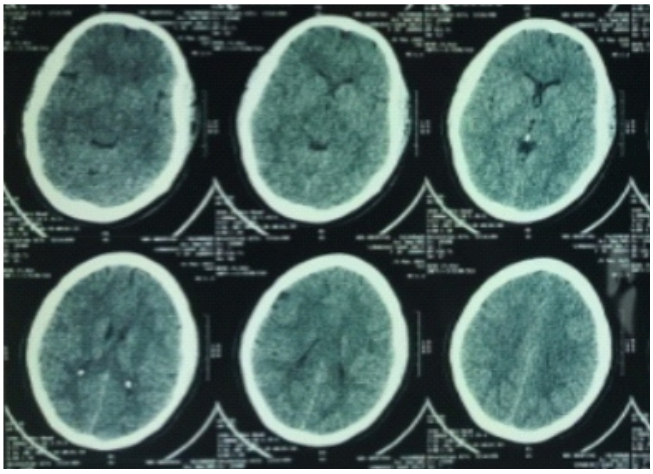

Figure 1: Non-contrast CT brain (axial sections) showing features suggestive of early mild diffuse cerebral edema, evidenced by subtle reduction in gray–white matter differentiation and mild effacement of cortical sulci without significant midline shift or ventricular compression.

She was kept nil per os (NPO) with intravenous fluids initiated. Initial laboratory tests revealed leukocytosis (WBC 17,800/µL), hyponatremia (Na129 mEq/L), elevated lactate (3.8 mmol/L), and metabolic acidosis (pH 7.31, $HCO_3$18 mEq/L). Serum procalcitonin was 1.8ng/mL, indicating early systemic inflammation (Table 1).

Parameter	Reference Range	Day 1 (Admission)	Day 7	Day 8	Day 18	Remarks
WBC (cells/µL)	4,000–11,000	17,800 ↑	19,200 ↑	16,400 ↑	13,900 ↑	Leukocytosis suggestive of infection/inflammation
Hemoglobin (g/dL)	12–15	10.2 ↓	9.1 ↓	8.0 ↓	9.6 ↓	PRBC transfusion on Day 8
Sodium (mEq/L)	135–145	129 ↓	131 ↓	134 (improving)	138	Hyponatremia likely due to cerebral injury/SIADH
Serum Lactate (mmol/L)	0.5–2.0	3.8 ↑	2.9 ↑	2.2 ↑	1.6	Improvement with resuscitation
Procalcitonin (ng/mL)	<0.05	1.8 ↑	5.6 ↑	4.9 ↑	0.8	Indicative of VAP/aspiration pneumonia
Creatinine (mg/dL)	0.6–1.1	1.0	1.2	1.3	3.2 ↑	AKI requiring haemodialysis started on Day 18
ABG – pH	7.35–7.45	7.31 ↓	7.29 ↓	7.33 ↓	7.38	Mild metabolic acidosis improving
ABG – HCO_3^- (mEq/L)	22–26	18 ↓	19 ↓	20 ↓	22	Trend suggests correction
PaO_2/FiO_2 Ratio	>300	260 ↓	120 (ARDS)	150 ↑	260 ↑	Severe ARDS on Day 7

during an episode of agitation and was promptly re-intubated. Sedation, neuromuscular blockade and appropriate sedation was initiated in view of previous episode of self extubation. Her hemodynamics were stable. In view of stable hemodynamic status early high-protein Enteral nutrition was started.

Toward the end of the first week of admission, the patient developed high-grade fever and oxygen desaturation. Procalcitonin increased significantly, and her PaO_2/FiO_2 ratio dropped to 120, consistent with acute respiratory distress syndrome (ARDS) secondary to aspiration pneumonia or ventilator-associated pneumonia. On day 8 of admission, her hemoglobin fell to 8 g/dL, requiring transfusion of one unit of PRBC. In view of worsening condition ARDS protocol (prone positioning, lung-protective ventilation, and systemic corticosteroids) was initiated. Aviptadil infusion was started as an adjunctive therapy and antibiotics were optimized based on culture sensitivity reports.

A tracheostomy was done on day 10 of admission in anticipation of prolonged ventilatory support. Central lines were placed to minimize catheter-related sepsis risk and complication associated with peripheral venous catheters. Neuroimaging was repeated which demonstrated hypoxic-ischemic changes. On day 18 of admission, the patient developed acute kidney injury (creatinine 3.2 mg/dL) with oliguria in view of this hemodialysis sessions were initiated. By day 20 of admission, she became afebrile, and after two dialysis sessions her urine output normalized allowing removal of the femoral dialysis catheter. Her creatinine gradually returned to normal by 21st day of admission.

From day 22 of admission onward, she underwent CPAP trials and progressively weaned off the ventilator. By day 27 of admission, assisted ambulation was initiated with beginning of physiotherapy. She showed steady neurological and functional improvement. The patient was eventually discharged on day 47 of admission. At the time of discharge she was able to walk, communicate, and demonstrating significant neurological recovery after a prolonged and complex ICU course (Table 2).

Day of Admission	Clinical Event / Intervention
Day 1	Patient admitted; drowsy (GCS 8/15). Diagnosed with closed head injury with cerebral edema. Kept NPO. IV fluids initiated.
Day 1	Developed fever and agitation; further drop in GCS. Serum procalcitonin sent. Electively intubated for airway protection and started on mechanical ventilation.
Day 2	Self-extubated due to agitation; immediately re-intubated by intensivist. Sedation + muscle relaxation started. Daily sedation breaks initiated. Early high-protein enteral feeding begun.
Day 7	Developed high-grade fever and desaturation. Elevated procalcitonin. Diagnosed with ARDS with probable aspiration pneumonia / VAP.
Day 8	Hb 8 g/dL → 1-unit PRBC transfused. ARDS protocol started: prone ventilation, low tidal volumes, steroids. Aviptadil infusion initiated (adjunctive therapy). Antibiotics adjusted per cultures.
Day 10	Tracheostomy performed for prolonged ventilatory support.
Day 12	Central lines changed to prevent line-related sepsis. Routine labs sent.
Day 15	MRI Brain showed hypoxic-ischemic changes with water-shedding effect.
Day 18	Developed acute kidney injury with oliguria → haemodialysis initiated.
Day 20	Became afebrile. After two dialysis cycles, femoral dialysis catheter removed. Urine output normalized.
Day 22 onwards	CPAP trials initiated; progressive ventilator weaning.
Day 27	Ambulation started with physiotherapy until day of discharge; steady neurological recovery.
Day 47	Discharged home walking, talking, and neurologically stable.

Table 2:- Clinical events and interventions done in the patient from day of admission till discharge.Nursing care played a critical and continuous role in management of this case. This included airway management, strict measures for prevention of hospital acquired infections , monitoring of neurological status, hemodynamic surveillance, urine output monitoring, dialysis support and safe execution of position changes. All these measures contributed to the patient's successful recovery and eventual discharge (Table 3).

Domain	Medical Management	Nursing Management
Airway & Ventilation	Early intubation for GCS ≤ 8; lung-protective ventilation (TV 6 mL/kg, Pplat < 30 cmH_2O); ARDS protocol	Ventilator bundle care (head elevation 30–45°), aseptic suctioning, cuff-pressure checks, oral hygiene, monitoring ventilatory parameters
Cerebral Edema Control	Osmotic therapy (mannitol/hypertonic saline); neurosurgical guidance	Hourly GCS and pupil assessment, sedation breaks, early detection of raised ICP
Sepsis Management	Surviving Sepsis Bundle: early cultures, broad-spectrum antibiotics, vasopressors for MAP < 65 mmHg	Early sepsis recognition, timely cultures, administering antibiotics within 1-hour, titrating inotropes, close hemodynamic monitoring
ARDS Care	Prone positioning, low tidal-volume ventilation, steroids where appropriate, Aviptadil as adjunctive therapy	Coordinated proning with team, pressure-area protection, eye care, securing lines/tubes, continuous SpO_2 and ABG monitoring
Hemodynamic Support	Noradrenaline infusion; invasive BP monitoring via arterial line	Preparing/titrating vasopressors, hourly MAP documentation, line-site infection/extravasation vigilance
Renal Support	Hemodialysis for sepsis-associated AKI	Dialysis catheter asepsis, monitoring intradialytic complications (hypotension/cramps), strict intake–output, daily weight charting
Tracheostomy Care	Tracheostomy for prolonged ventilation and facilitated weaning	Daily stoma care, suctioning, humidification, cuff care, supporting communication and early mobilization
Nutrition	Early high-protein enteral feeding	NG tube care, checking gastric residuals, aspiration prevention, gradual feed advancement
Multidisciplinary Care	Involvement of intensivists, neurosurgery, nephrology, physiotherapy, dietetics	Coordinating rounds, implementing care plans, identifying subtle clinical changes promptly

Table 3. Integrated Medical and Nursing Management of Severe Head Injury with Sepsis, ARDS, and AKIle Sclerosis

DISCUSSION

Traumatic brain injury (TBI) is frequently complicated by systemic secondary insults that influence overall prognosis. In moderate–severe TBI, complications such as sepsis, acute respiratory distress syndrome (ARDS), and sepsis-associated acute kidney injury (SA-AKI) markedly increase morbidity and mortality. The present case demonstrates this typical high-risk trajectory.[6]

The onset of ARDS following aspiration pneumonia/VAP is consistent with reported incidence rates of 17–20% in acute brain injury populations. Management required strict application of lung-protective ventilation, prone positioning, and corticosteroids, all supported by current ARDS guidelines. Adjunctive use of Aviptadil was guided by emerging evidence showing improved oxygenation in severe ARDS, although definitive survival benefit remains uncertain.[7]

Systemic infection and inflammatory burden led to AKI, a complication occurring in 50–60% of septic ICU patients and strongly associated with poor outcomes.[8] Rapid implementation of the sepsis prevention and treatment —including early cultures, prompt antibiotics, hemodynamic resuscitation, and vasopressor titration—was critical for stabilization.[9]

Given prolonged ventilatory needs, early tracheostomy facilitated airway management and supported progressive weaning, consistent with evidence promoting tracheostomy in selected ICU patients.[10] Equally important was the multidisciplinary approach, involving intensive care, neurosurgery, nephrology, physiotherapy, dietetics, and highly coordinated nursing care, all of which have been shown to improve ICU outcomes.

This case highlights that even in the presence of severe multi-organ complications, timely protocol-driven ICU care—combined with vigilant nursing surveillance—can result in favorable neurological and functional recovery.

CONCLUSION

This case highlights the complex and rapidly evolving course of traumatic brain injury, especially when it's complicated by sepsis, acute respiratory distress syndrome, and sepsis-associated acute kidney injury. Despite the severity of these multisystem complications timely implementation interventions such as measures for early airway protection, lung-protective ventilation, prone positioning, sepsis bundle adherence, tracheostomy at an appropriate time and renal replacement therapy resulted in meaningful neurological and functional recovery. The patient's outcome underscores the essential role of continuous nursing vigilance, multidisciplinary collaboration and protocol-driven ICU management.

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