e-ISSN: 2807-6257

Open Access Indonesian Journal of Medical Reviews

[OAIJMR]

https://hmpublisher.com/index.php/oaijmr

Navigating the Triad of Trauma: A Case Report on Managing Concurrent Pulmonary Contusion, Traumatic Brain Injury, and Cervical Fracture in a Geriatric Patient

Yasyfie Asykari[1*], Septian Adi Permana[2], Eko Setijanto[2]

[1]Anesthesiology and Intensive Care Specialist Education Program, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

[2]Department of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

ARTICLE INFO

Keywords:
Cervical spine injury
Geriatric trauma
Mechanical ventilation
Pulmonary contusion
Traumatic brain injury

***Corresponding author:**
Yasyfie Asykari

E-mail address:
Yasyfie1993@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/oaijmr.v5i5.771

A B S T R A C T

The management of geriatric patients with multiple severe injuries presents a formidable clinical challenge due to reduced physiological reserve and the complex interplay of competing therapeutic goals. This report details the case of a geriatric patient suffering from the triad of pulmonary contusion (PC), moderate traumatic brain injury (TBI), and an unstable cervical spine fracture, highlighting the intricate balance required in neuroprotective and lung-protective ventilatory strategies. A 68-year-old male was admitted following a 10-meter fall, sustaining a moderate TBI with a temporoparietal subdural hemorrhage, a complete C3 vertebral fracture, and significant bilateral pulmonary contusions. His hospital course was marked by acute respiratory distress and neurological deterioration, with a Glasgow Coma Scale (GCS) score of E3V4M5 and hypoxemia requiring intubation and mechanical ventilation in the intensive care unit (ICU). Management focused on the cautious application of positive end-expiratory pressure (PEEP) to improve oxygenation without exacerbating intracranial pressure (ICP), alongside strict cervical spine immobilization and neuro-monitoring. After eight days of complex critical care, the patient's prolonged need for mechanical ventilation and significant sputum retention necessitated a percutaneous dilational tracheostomy (PDT) to facilitate respiratory weaning and improve pulmonary toilet. In conclusion, this case underscores the profound difficulty of managing concurrent lung and brain injuries in the context of cervical instability. The successful navigation of this trauma triad hinged on a highly individualized, multidisciplinary approach, with judicious ventilator management and timely procedural intervention like PDT being pivotal. It affirms the need for integrated care protocols that can dynamically balance competing organ-system priorities in complex geriatric trauma.

1. Introduction

Trauma remains a significant cause of morbidity and mortality worldwide, with geriatric patients representing a uniquely vulnerable demographic.[1] Advancing age is associated with a host of physiological changes, including decreased cardiopulmonary reserve, altered immune responses, and underlying comorbidities, which collectively amplify the impact of traumatic injuries and complicate management. Elderly patients are more susceptible to severe outcomes even from seemingly low-impact mechanisms, and falls are a predominant cause of injury in this population. Blunt chest trauma, a frequent consequence of such events, is particularly lethal, contributing to 20-25% of all blunt trauma-related deaths.[2]

Among the spectrum of injuries resulting from blunt chest trauma, pulmonary contusion (PC) is a common and serious pathology.[3] It is characterized by direct parenchymal lung injury leading to alveolar hemorrhage and interstitial edema, which impairs gas exchange and reduces lung compliance. While the

incidence of PC in the elderly is reported to be around 3.9% to 5.2%, it is frequently associated with other injuries like rib fractures, which significantly increases the risk of complications such as pneumonia, acute lung injury (ALI), and acute respiratory distress syndrome (ARDS), thereby worsening prognosis. The clinical course of PC is often insidious, with maximal respiratory compromise typically developing 24 to 48 hours post-injury, necessitating vigilant monitoring, often in an intensive care unit (ICU) setting.[4]

The complexity of managing a geriatric trauma patient is magnified when PC is concurrent with a significant traumatic brain injury (TBI). TBI independently triggers a systemic inflammatory response, with the intracranial release of pro-inflammatory cytokines that can leak into the systemic circulation, predisposing the patient to secondary organ dysfunction, including ALI/ARDS.[5] It is estimated that 20-25% of patients with isolated brain injuries develop ALI/ARDS. This creates a profound clinical dilemma, as the standard management strategies for these two conditions are often diametrically opposed. Lung-protective ventilation for ARDS advocates for low tidal volumes (around 6 ml/kg of ideal body weight) and the use of positive end-expiratory pressure (PEEP) to recruit collapsed alveoli and improve oxygenation.[6] Conversely, traditional neuroprotective ventilation guidelines for TBI have historically focused on preventing intracranial hypertension by maintaining normocapnia or inducing mild hypocapnia, and have cautioned against high levels of PEEP due to concerns about impeding cerebral venous outflow and increasing intracranial pressure (ICP). The application of PEEP can increase intrathoracic pressure, which may be transmitted to the central venous system, thereby increasing cerebral venous pressure and, consequently, ICP, especially when baseline ICP is low. Furthermore, PEEP-induced alveolar overdistension can increase physiological dead space, leading to a rise in arterial partial pressure of carbon dioxide ($PaCO_2$), a potent cerebral vasodilator that can further elevate ICP.[8]

Adding a third layer of complexity is the presence of an unstable cervical spine fracture. A complete fracture of the third cervical vertebra (C3), as seen in this case, poses an immediate threat to the spinal cord and necessitates strict immobilization. This has significant implications for airway management, requiring advanced techniques like manual in-line stabilization (MILS) during intubation to prevent secondary neurological injury. It also complicates routine patient care, such as positioning for pressure sore prevention and performing adequate pulmonary hygiene, which are critical in a patient with PC. The combination of these three life-threatening injuries—PC, TBI, and a C3 fracture—in a geriatric patient constitutes a "triad of trauma" that tests the limits of modern critical care. Each injury demands specific and often conflicting interventions, requiring the clinical team to navigate a narrow therapeutic window where the treatment for one condition could potentially worsen another.[9]

The novelty of this case report lies in its detailed exploration of the day-to-day clinical decision-making involved in managing this specific triad of injuries, a combination that is severe and not extensively detailed in existing literature from a management-dilemma perspective.[10] Therefore, the aim of this study was to present and meticulously discuss the management strategies, particularly the challenges of balancing competing ventilatory demands for lung and brain protection, in a geriatric patient with concurrent pulmonary contusion, moderate TBI, and an unstable high cervical fracture, to provide insights for clinicians facing similar complex trauma scenarios.

2. Case Presentation

A 68-year-old male was brought to the Emergency Department (ED) of Dr. Moewardi Regional General Hospital two days after falling approximately 10 meters from a tree. He was complaining of a severe headache and incapacitating back pain. The patient had a past medical history of hypertension, for which he was intermittently compliant with amlodipine, but no other significant comorbidities. Upon arrival in the

ED, an initial assessment following the advanced trauma life support (ATLS) protocol was conducted. The patient was immobilized with a rigid cervical collar and a spinal board. His airway was patent but noisy, with gurgling sounds suggestive of secretions. A summary of the patient's initial clinical and diagnostic findings is presented in Table 1.

The primary survey revealed a complex clinical picture. The patient's airway was patent but compromised by secretions, while his breathing was tachypneic at a rate of 28 breaths per minute, with decreased bilateral air entry and coarse crepitations. His oxygen saturation was 94% on 10 L/min of oxygen. Circulation was stable, with a blood pressure of 145/90 mmHg and a heart rate of 95 beats per minute. On disability assessment, he was drowsy but rousable, with a Glasgow Coma Scale (GCS) of 14 (E4, V4, M6) and equal, reactive pupils. A full exposure of the patient revealed bruising over the chest wall and significant tenderness upon palpation of the cervical spine and posterior thorax, with a core temperature of 37.1°C.

Two large-bore intravenous cannulas were secured, and a 1-liter bolus of warmed crystalloid solution (Ringer's Lactate) was initiated. Blood samples were drawn for a complete blood count, coagulation profile, renal and liver function tests, electrolytes, and arterial blood gas (ABG) analysis. Given the mechanism of injury and clinical findings, a multi-detector computed tomography (CT) scan series was performed (Figure 1). The detailed results from these investigations are summarized in Table 1. The scans confirmed the multi-system nature of his injuries, revealing a right-sided acute subdural hemorrhage, an unstable C3 burst fracture, and severe bilateral pulmonary contusions with associated rib fractures. The findings on the thorax X-ray were consistent with these contusions (Figure 2). Initial laboratory results were significant for a mild leukocytosis and an uncompensated respiratory acidosis with significant hypoxemia, as noted in Table 1.

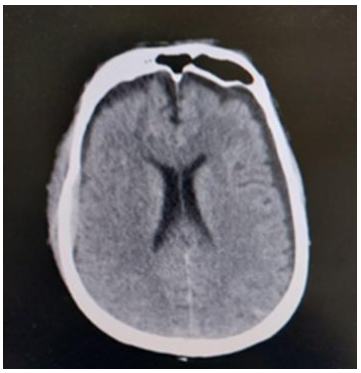

Figure 1. Patient's multi-slice computed tomography (MSCT) results.

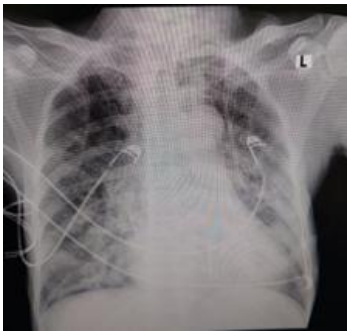

Figure 2. Patient's thorax X-ray imaging.

Table 1. Summary of patient's clinical findings on admission.

Parameter	Finding
Demographics	68-year-old male
Mechanism of injury	Fall from 10-meter height, 2 days prior
Presenting complaints	Severe headache, back pain
Initial vital signs (ED)	BP: 145/90 mmHg, HR: 95 bpm, RR: 28/min, SpO_2**:** 94% on 10L O_2 mask, Temp**:** 37.1°C
Neurological status (ED)	GCS**:** 14 (E4, V4, M6). Pupils 3 mm, equal and reactive to light. Drowsy but rousable.
Physical examination	Bruising on chest wall, tenderness on palpation of cervical spine. Decreased breath sounds at the lung bases.
Initial arterial blood gas	pH: 7.32, $PaCO_2$: 48 mmHg, PaO_2: 68 mmHg, HCO_3^-: 23 mEq/L, SaO_2: 93%
MSCT Head findings	Right temporoparietal acute subdural hemorrhage (8 mm thickness). 4mm midline shift to the left. Effacement of cortical sulci. Traumatic pneumocephalus in the frontal region.
CT cervical spine findings	Complete, unstable three-column burst fracture of the third cervical vertebra (C3). Significant retropulsion of vertebral body fragment into the spinal canal, causing ~40% stenosis.
CT chest findings	Extensive, patchy, non-segmental ground-glass opacities and consolidation in both lungs, consistent with severe bilateral pulmonary contusions. Non-displaced fractures of right 4th-7th ribs.
CT abdomen/Pelvis	No evidence of acute intra-abdominal or retroperitoneal injury.

Following stabilization, the patient was admitted to the high care unit (HCU) for close monitoring under the care of the neurosurgery and orthopedic teams. A non-operative approach for the TBI and strict immobilization for the C3 fracture were the initial plans. However, over the subsequent 24 hours, the patient's condition deteriorated significantly. He became progressively somnolent (GCS drop to 12: E3V4M5) and developed severe respiratory distress, with a respiratory rate of 33 breaths per minute and use of accessory muscles. His SpO_2 fell to 91% on a 15 L/min Non-Rebreather Mask, and a repeat ABG confirmed worsening acute hypercapnic respiratory failure (pH 7.25, $PaCO_2$ 60 mmHg, PaO_2 55 mmHg).

This coupled decline in respiratory and neurological status necessitated emergent transfer to the ICU for airway protection and mechanical ventilation. The intubation was performed by a senior anesthesiologist using a video laryngoscope and manual in-line stabilization (MILS). An arterial line and central venous catheter were placed. He was kept deeply sedated with infusions of propofol and fentanyl, and a norepinephrine infusion was initiated to maintain a mean arterial pressure (MAP) > 80 mmHg to ensure adequate cerebral perfusion pressure (CPP).

The first week in the ICU was a continuous balancing act between supporting his failing lungs (which had progressed to moderate ARDS with a PaO_2/FiO_2 ratio of 110) and protecting his injured brain. This involved meticulous, day-by-day adjustments to therapy, as detailed in Table 2. Management focused on lung-protective ventilation, targeting a tidal volume of 6-7 mL/kg PBW. This was coupled with cautious PEEP titration, with each adjustment evaluated against its effect on oxygenation, respiratory mechanics, hemodynamics, and neurological status. Crucially, as PEEP was optimized from 5 to 12 cmH_2O, the driving pressure (ΔP) was maintained at ≤15 cmH_2O, and the static respiratory system compliance (Crs) improved from a low of 26 mL/cmH_2O to 38 mL/cmH_2O, indicating successful alveolar recruitment without

overdistension. The MAP target was achieved without escalating vasopressor requirements, and central venous pressure (CVP) remained stable, suggesting minimal hemodynamic impact from the PEEP adjustments. Concurrently, a strategy of active de-resuscitation was employed after initial stabilization, achieving a negative cumulative fluid balance by day 5, which aided both pulmonary and cerebral edema management.

By day eight, despite significant improvements in oxygenation, it was clear that his weak cough reflex and inability to manage copious secretions were preventing ventilator liberation. After a multidisciplinary team discussion, a percutaneous dilational tracheostomy (PDT) was performed at the bedside to facilitate weaning and improve pulmonary toilet. The procedure was successful, and in the following days, the patient was rapidly weaned from mechanical support and sedation. His neurological status improved significantly, and he was transferred from the ICU to a surgical step-down unit on day 15 for continued rehabilitation and planning for definitive cervical spine fixation.

Table 2. Daily physiological and therapeutic parameters in the intensive care unit.

Hospital day	**Key clinical events / Status**	**Ventilator parameters**	**Gas exchange**	**Hemodynamics**	**Fluid balance**	**Neuro**
		FiO_2 / PEEP	**VT (mL/kg PBW)**	**Pplat (cmH_2O)**	**ΔP (cmH_2O)**	**Crs (mL/cmH_2O)**
Day 2 (ICU Adm)	Moderate ARDS, post-intubation, deep sedation	1.0 / 5	6.3	26	21	26
Day 3	High O_2 requirement, copious secretions	0.8 / 8	6.3	28	20	27
Day 4	Cautious PEEP titration, initiating de-resuscitation	0.7 / 10	6.3	28	18	30
Day 5	PEEP optimized, neuro status stable	0.6 / 12	6.6	27	15	38
Day 6	Oxygenation improving, failed SBT (secretions)	0.5 / 10	6.6	24	14	41
Day 7	Weaning failure due to poor airway clearance	0.4 / 8	6.8	22	14	43
Day 8	PDT Procedure performed at the bedside	0.4 / 8	(PSV)	-	-	-
Day 9	Weaning sedation, tolerating PSV post-PDT	0.3 / 5	(PSV)	-	-	-
Day 12	Weaned to Trach Mask, alert and following commands	Off Vent	-	-	-	-
Day 15	Medically stable for transfer	Off Vent	-	-	-	-

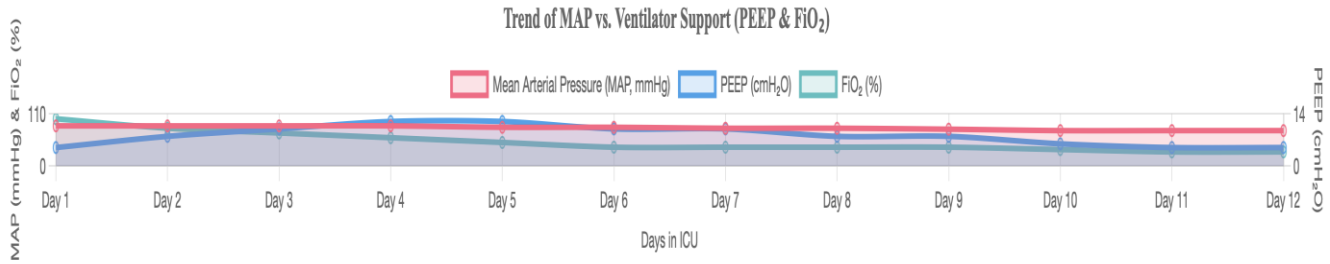

Figure 3. Intensive care unit hemodynamic and ventilator follow-up.

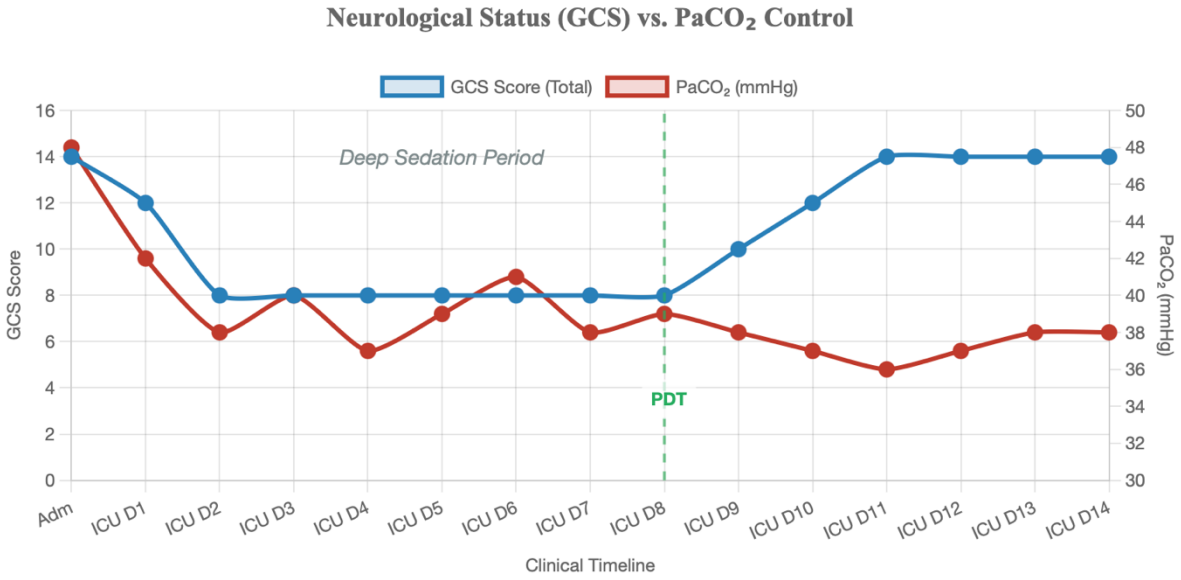

Figure 4. Neurological assessment and correlated physiological parameters.

3. Discussion

This case report presents a formidable clinical scenario that lies at the intersection of several critical care disciplines, demanding a nuanced and highly integrated management approach. The patient, a 68-year-old male, embodied a "triad of trauma"—severe pulmonary contusion, a moderate traumatic brain injury, and an unstable high cervical fracture—a combination that creates profound physiological conflicts. The successful navigation of his clinical course from near-fatal respiratory failure to ICU discharge provides a valuable framework for discussing the intricate pathophysiology of these concurrent injuries and the therapeutic tightrope that clinicians must walk.[9] The discussion will delve deeply into the specific challenges posed by each injury and, most importantly, the synergistic dilemmas that arose from their coexistence, focusing on the underlying theories and physiological principles that guide the day-to-day management decisions.

The patient's chronological age of 68 is not merely a demographic data point; it is a critical determinant of his entire clinical trajectory. The field of geriatric traumatology is founded on the principle that elderly patients are not simply "older adults." They are a distinct population with a unique physiology defined

by the concept of homeostenosis, the progressive, age-related decline in the functional reserve of every organ system. While a younger individual might possess the cardiovascular, pulmonary, and immunological reserve to buffer the immense stress of a 10-meter fall, the geriatric patient operates much closer to their physiological limit at baseline.[10] The initial insult, therefore, can rapidly push them beyond their capacity to compensate, leading to a precipitous decline, as was witnessed in this patient's rapid deterioration in the HCU.

This vulnerability extends to the cellular and immunological level through a process known as immunosenescence. The aging immune system is characterized by a state of chronic, low-grade inflammation (termed "inflammaging") and a diminished capacity for robust, targeted immune responses.[11] Following trauma, this dysregulated system often mounts an exaggerated and ineffective inflammatory response. The systemic inflammatory response syndrome (SIRS) can be more pronounced, while the compensatory anti-inflammatory response syndrome (CARS) can also be excessive, leaving the patient susceptible to secondary infections, a major cause of late mortality in geriatric trauma. The leukocytosis noted on this patient's admission is a non-specific marker, but it represents the initiation of an inflammatory cascade that, in an older individual, is less controlled and more likely to contribute to secondary organ injury, such as the progression of pulmonary contusion into full-blown ARDS.[11]

Furthermore, age-related changes in body composition (sarcopenia), pharmacokinetics (altered drug distribution and clearance), and the high prevalence of comorbidities (in this case, hypertension) all conspire to complicate management. Sarcopenia contributes to a weak cough reflex and difficulty weaning from ventilation. Altered clearance of sedative agents like propofol and opioids like fentanyl can lead to prolonged sedation and delayed neurological assessments.[12] The patient's pre-existing hypertension, while seemingly controlled, signifies a degree of endothelial dysfunction and vascular stiffness that compromises the ability to autoregulate blood flow, a critical function for both the injured brain and the kidneys. The decision to admit this patient directly to a high-surveillance unit was, therefore, not just appropriate but essential, as the risk of sudden decompensation in this population is exceptionally high.

The bilateral pulmonary contusions were the engine driving the patient's respiratory failure. The pathophysiology of PC is a two-hit process.[12] The primary injury occurs at the moment of impact. The thoracic cage is rapidly compressed and re-expanded, creating shear forces at the interface between tissues of different densities—specifically, the gas-filled alveoli and the more solid capillary membranes. This tearing of the alveolar-capillary interface leads to immediate hemorrhage and edema into the interstitial and alveolar spaces. This process directly obliterates functional lung units, creating areas of shunt (perfusion without ventilation) and V/Q mismatch, which was the cause of the patient's initial hypoxemia. The secondary injury unfolds over the subsequent 24-48 hours and is far more insidious. The damaged lung tissue and extravasated blood trigger an intense, localized inflammatory response. Alveolar macrophages release a flood of pro-inflammatory cytokines, including Tumor necrosis factor-alpha (TNF-α) and Interleukin-1 (IL-1), IL-6, and IL-8. These cytokines act as potent chemoattractants for neutrophils, which migrate from the circulation into the lung parenchyma. Once activated, these neutrophils release a toxic arsenal of reactive oxygen species (free radicals), proteases, and leukotrienes. This onslaught further damages the delicate alveolar-capillary membrane, increasing its permeability and leading to a protein-rich exudate flooding the alveoli. This inflammatory exudate inactivates surfactant, leading to widespread alveolar collapse (atelectasis) and a dramatic decrease in lung compliance (a "stiffening" of the lungs). This secondary inflammatory wave is what transforms a localized contusion into the widespread, bilateral infiltrates characteristic of ARDS, which was confirmed in this patient by his

PaO_2/FiO_2 ratio of 110. The non-displaced rib fractures, while not causing mechanical instability, would have contributed significantly to this decline by causing pain, splinting, and atelectasis, further compounding the effects of the contusion.[13]

The management of PC and ARDS is fundamentally supportive, aimed at maintaining oxygenation while allowing the lung to heal and mitigating the secondary inflammatory injury. The two cornerstones of this support are judicious fluid management and lung-protective ventilation.[13] In trauma patients, aggressive fluid resuscitation is often necessary to maintain hemodynamic stability. However, in the setting of PC, where capillary permeability is high, excess fluid can easily leak into the lung interstitium, worsening pulmonary edema. This necessitates a careful fluid strategy, often termed "de-resuscitation" or "permissive hypovolemia" after the initial resuscitation phase, aiming for a net even or slightly negative fluid balance. The other cornerstone, lung-protective ventilation (LPV), was central to this patient's care. The principles of LPV are designed to minimize VILI. This involves using low tidal volumes (4-6 mL/kg of ideal body weight) to prevent overdistension of the remaining healthy alveoli (volutrauma) and maintaining a plateau pressure (a surrogate for peak alveolar pressure) below 30 cmH_2O. The second component of LPV is the application of PEEP, which serves to keep alveoli open at the end of expiration, preventing the shear stress injury caused by their repeated collapse and re-opening (atelectrauma) and recruiting collapsed lung units to improve oxygenation.[14] It was the application of this second component, PEEP, that created the central therapeutic conflict of this case.

Concurrent with his pulmonary failure, the patient suffered a moderate TBI, evidenced by his initial GCS, the subdural hemorrhage, and the CT signs of increased ICP (midline shift and sulcal effacement). The pathophysiology of TBI, much like PC, is a two-stage process. The primary injury is the direct physical damage that occurs at the moment of impact—the tearing of bridging veins leading to the SDH, and the contusion of brain tissue. This damage is irreversible. The entirety of neurocritical care is therefore focused on preventing the secondary injury, a complex and devastating biochemical cascade that unfolds in the hours and days following the initial trauma.[14]

This secondary cascade is initiated by a massive release of excitatory neurotransmitters like glutamate (excitotoxicity), which leads to an influx of calcium into neurons.[15] This calcium overload activates a host of intracellular enzymes, including proteases and phospholipases, that degrade cellular structures, and it triggers mitochondrial dysfunction. This leads to energy failure and the production of reactive oxygen species, which cause lipid peroxidation and further membrane damage. Ultimately, these pathways lead to neuronal apoptosis and necrosis, causing cerebral edema, increased ICP, and a worsening of the initial injury.

The management of TBI revolves around mitigating these secondary insults. This is achieved by maintaining physiological homeostasis and optimizing cerebral perfusion. According to the Monro-Kellie doctrine, the intracranial volume is a fixed sum of three components: brain parenchyma, cerebrospinal fluid (CSF), and blood. As the SDH and cerebral edema added volume, the body first compensated by shunting CSF and venous blood out of the skull. The CT findings of sulcal effacement and ventricular compression showed that these compensatory mechanisms were being exhausted. Once compensation fails, any small increase in volume leads to a logarithmic rise in ICP.[16] The goal of management is to keep ICP below 20-22 mmHg and, more importantly, to maintain an adequate cerebral perfusion pressure (CPP), which is the difference between mean arterial pressure (MAP) and ICP (CPP = MAP - ICP). CPP is the pressure gradient driving blood flow to the brain, and a CPP between 60-70 mmHg is typically targeted to prevent ischemia.

Our management was a direct application of these principles. We targeted a higher MAP of >80 mmHg with norepinephrine to ensure that even if the unmonitored ICP was moderately elevated to a level of

15-20 mmHg, the CPP would remain adequate. We maintained the head of the bed at 30 degrees to promote cerebral venous outflow. And crucially, we took control of his ventilation not just for his lungs, but for his brain. This is because the most powerful and rapid modulator of cerebral blood volume, and therefore ICP, is the arterial partial pressure of carbon dioxide ($PaCO_2$). Hypercapnia (high $PaCO_2$) is a potent cerebral vasodilator, increasing cerebral blood volume and ICP.[16] Conversely, iatrogenic hyperventilation to induce hypocapnia causes vasoconstriction, which can rapidly lower ICP. However, this is a double-edged sword; excessive vasoconstriction can lead to cerebral ischemia. Therefore, the modern guideline-based approach, which we followed, is to maintain strict normocapnia ($PaCO_2$ 35-45 mmHg), using $PaCO_2$ as a stable variable rather than a therapeutic tool, unless there is evidence of impending herniation.

The crux of this case was the direct physiological conflict between the optimal ventilator strategies for the patient's ARDS and his TBI. To save his lungs, we needed to recruit alveoli with PEEP. To save his brain, we needed to avoid any maneuver that could potentially raise his ICP. The cautious upward titration of PEEP from 5 to 12 cmH_2O was the most critical intervention of his ICU stay.

The mechanism by which PEEP can increase ICP is multifaceted. The primary pathway is hemodynamic transmission. PEEP increases the mean intrathoracic pressure. This pressure can be transmitted to the right atrium, increasing central venous pressure. Because the cerebral venous system is a low-pressure, valveless circuit that drains via the jugular veins, this increased downstream pressure can impede cerebral venous outflow, increasing cerebral blood volume and ICP. The efficiency of this transmission is highly dependent on lung and chest wall compliance. In a patient with very stiff, non-compliant lungs (like in severe ARDS), a greater portion of the applied PEEP is dissipated within the lung parenchyma itself and is less efficiently transmitted to the pleural space and the heart.[17] This patient's ARDS, paradoxically, may have offered a degree of protection against this hemodynamic transmission.

The second, more subtle mechanism involves respiratory mechanics and gas exchange. PEEP is beneficial when it recruits collapsed alveoli, improving V/Q matching and oxygenation. However, if PEEP is set too high, it can overdistend the healthier, more compliant lung regions. This overdistension increases alveolar dead space (ventilation without perfusion). An increase in dead space ventilation impairs the elimination of CO_2, causing $PaCO_2$ to rise. As discussed, this hypercapnia would directly increase ICP through cerebral vasodilation. This is a critical point: even if the hemodynamic transmission of PEEP is minimal, its effects on gas exchange can still have a profound impact on the brain.[17]

Our management navigated this dilemma through a strategy of personalized, physiology-guided titration. We did not adhere to a dogmatic cap on PEEP. Instead, we used a stepwise approach, making small changes and then carefully observing the integrated response of the patient. Each increase in PEEP by 1-2 cmH_2O was a clinical experiment. We assessed the benefit by looking at the PaO_2/FiO_2 ratio and SpO_2. We assessed the cost by monitoring for key warning signs, including hemodynamic instability, such as a drop in MAP suggesting impaired cardiac preload; worsening gas exchange indicated by a rise in $PaCO_2$; and neurological decline. In the absence of an invasive ICP monitor, we used the pupil as a crude but vital surrogate, where any new or worsening anisocoria or sluggishness after a PEEP increase was interpreted as a sign of worsening intracranial hypertension, prompting an immediate reduction in PEEP. We found this patient's "sweet spot" at a PEEP of 10-12 cmH_2O, where his oxygenation improved significantly, his MAP remained stable, his $PaCO_2$ was controllable, and his pupils remained reactive. This iterative, cautious approach is the cornerstone of managing these complex patients.

The unstable C3 burst fracture was a constant and menacing background presence that constrained nearly every aspect of care. A high cervical fracture is

uniquely dangerous due to the proximity of the phrenic nerve nucleus (C3-C5), which controls the diaphragm. While the patient did not have overt diaphragmatic paralysis, the spinal canal stenosis and potential for cord edema could have contributed to his respiratory muscle weakness.[18]

The most acute challenge posed by the C3 fracture was airway management. Securing the airway in a patient with an unstable cervical spine is a high-stakes procedure. The primary goal is to avoid any movement of the cervical spine, as extension or flexion could cause the fractured vertebra to shift and catastrophically injure the spinal cord.[18] The technique of manual in-line stabilization (MILS), where an assistant holds the patient's head and neck in a neutral position, is mandatory. The use of a video laryngoscope, as was done in this case, is now considered the standard of care. It allows for visualization of the glottis with minimal head and neck movement compared to the neck extension required for traditional direct laryngoscopy, thereby increasing both the safety and success rate of intubation.

Beyond the initial intubation, the unstable spine complicated routine ICU care. The patient had to be maintained in a rigid cervical collar at all times, which can increase ICP by compressing the jugular veins and also carries a high risk of causing occipital pressure ulcers. Log-rolling for pressure care and linen changes required a team of 4-5 staff members to be performed safely. Crucially, the instability precluded the use of prone positioning. Prone ventilation is a powerful, evidence-based intervention for moderate-to-severe ARDS that improves oxygenation by recruiting dorsal lung regions and improving V/Q matching. For a patient with a PaO_2/FiO_2 ratio hovering around 110, prone positioning would normally have been a primary consideration. The inability to use this therapy meant we had to rely entirely on optimizing conventional ventilation, placing even greater importance on the careful titration of PEEP and aggressive pulmonary hygiene.[19]

After eight days of invasive ventilation, it became evident that the primary barrier to weaning was not a failure of gas exchange but a failure of airway competence. The patient had a weak, ineffective cough reflex, a common sequela of critical illness polyneuropathy, prolonged sedation, and the mechanical inefficiency caused by his chest wall injuries. This led to the retention of thick secretions, which would rapidly obstruct his airways during spontaneous breathing trials, causing desaturation and distress.[20]

The decision to proceed with a percutaneous dilational tracheostomy (PDT) was a turning point. For patients requiring prolonged mechanical ventilation, tracheostomy offers numerous advantages over continued endotracheal intubation. It improves pulmonary hygiene by providing a more direct and effective route for suctioning, reduces the work of breathing due to its lower resistance, and enhances patient comfort, which allows for a significant reduction in sedative infusions. Less sedation is vital for allowing reliable neurological assessments, preventing delirium, and promoting active participation in physical therapy.[20] The potential for oral communication and nutrition further improves the patient's quality of life. The decision on day 8 was based on a clear clinical trajectory, and the bedside procedure avoided the risks of transport. The immediate post-PDT course confirmed the correctness of the decision, as the patient was successfully weaned from the ventilator within four days, a process that would have been impossible with the endotracheal tube still in place.

4. Conclusion

This case of a geriatric patient with a traumatic triad of pulmonary, neurological, and cervical spine injuries exemplifies the pinnacle of critical care complexity. Its successful resolution was not the result of a single intervention but rather the synthesis of numerous, meticulously managed physiological variables. The core lesson from this case is the imperative of abandoning a siloed, organ-specific approach in favor of a holistic, integrated strategy that acknowledges and adapts to the profound interplay

between injured systems. The management demonstrated that established protocols, while essential, must serve as a guide, not a directive, and that the best outcomes are achieved through personalized, bedside assessments that continuously weigh the therapeutic benefits against the potential for iatrogenic harm.

The judicious and monitored titration of PEEP, balancing the conflicting demands of the lung and the brain, was the central therapeutic challenge and a testament to physiology-guided care. Furthermore, the timely implementation of a percutaneous tracheostomy was pivotal, breaking the cycle of ventilator dependency driven by poor airway clearance and unlocking the patient's potential for recovery. Ultimately, this case powerfully affirms that in the realm of severe, multi-system trauma, especially in the vulnerable geriatric population, success is predicated on a dynamic, vigilant, and highly collaborative multidisciplinary approach that skillfully navigates a narrow and ever-shifting therapeutic path.

5. References

1. Van Diepen MR, Wijffels MME, Verhofstad MHJ, Van Lieshout EMM. Classification methods of pulmonary contusion based on chest CT and the association with in-hospital outcomes: a systematic review of literature. Eur J Trauma Emerg Surg. 2024; 50(6): 2727–40.
2. Kashani RA, Hatamian S, Maghsoudi MR, Zoghi M. Comparative evaluation of lung contusion assessment using ultrasound and computed tomography scan in the first 24 hours of intensive care unit admission. Dubai Med J. 2024; 7(4): 325–39.
3. Merkl T, Astapenko D, Štichhauer R, Šafus A, Dušek T, Kotek J, et al. Exogenous surfactant for lung contusion causing ARDS: a systematic review of clinical and experimental reports. Clin Respir J. 2024; 18(5): e13776.
4. Kozaci N, Aydın İE, Ersahin DA, Yüksel Y. Transient complete right bundle branch block due to lung contusion: case report. J Emerg Med Case Rep. 2024; 15(2): 38–41.
5. Nakatsutsumi K, Choi W, Johnston W, Pool K, Park DJ, Weaver JL, et al. Lung contusion complicated by pneumonia worsens lung injury via the inflammatory effect of alveolar small extracellular vesicles on macrophages and epithelial cells. J Trauma Acute Care Surg. 2025; 98(1): 55–63.
6. Elsayed RG, Hafez AF, Maarouf MM, AbdElAziz FK. Impact of the early use of high-flow nasal cannula in patients with post-traumatic lung contusion: a randomized clinical trial. Indian J Crit Care Med. 2025; 29(2): 117–24.
7. Kuzucuoğlu M, Balta C, Altun E, Yekdeş AC, Acar A. Effects of different treatment modalities on lung injury in experimental pulmonary contusion model. J Surg Res. 2025; 307: 100–6.
8. Rix AC, Störmann P, Vollrath JT, Hörauf J-A, Eichler K, Marzi I, et al. The neuronal biomarker neuron-specific enolase correlates with the volume of lung contusion in polytraumatized patients. Shock. 2025; 63(3): 428–34.
9. Sharma S, Shrestha PS, Acharya SP. Can early use of HFNC through improved study design make a statistically significant difference in the rate of intubation in patients with post-traumatic lung contusion? Indian J Crit Care Med. 2025; 29(4): 396–7.
10. Nakatsutsumi K, Park DJ, Choi W, Johnston W, Pool K, Kezios J, et al. Lung contusion drives lung injury by modifying miRNA cargo in alveolar small extracellular vesicles. Inflamm Res. 2025; 74(1): 83.
11. Ali-Osman F, Mangram A, Sucher J, Shirah G, Johnson V, Moeser P, et al. Geriatric (G60) trauma patients with severe rib fractures: Is muscle sparing minimally invasive thoracotomy rib fixation safe and does it

improve post-operative pulmonary function? Am J Surg. 2018; 216(1): 46–51.
12. Ito S, Nakashima H, Segi N, Yokogawa N, Sasagawa T, Funayama T, et al. A deep learning-based prediction model for prognosis of cervical spine injury: a Japanese multicenter survey. Eur Spine J. 2025.
13. Zhang Z, Li N, Ding Y, Cheng H. An integrative nomogram based on MRI radiomics and clinical characteristics for prognosis prediction in cervical spinal cord Injury. Eur Spine J. 2025; 34(3): 1164–76.
14. Azad TD, Kartal A, Shafi M, Lubelski D, Theodore N, Bydon A, et al. Duraplasty in acute spinal cord injury: a systematic review. Eur Spine J. 2025.
15. Burke MJ, Ahmed YE, Li Z, Sanchez K, Winston A, Broadhurst P, et al. Identifying mild traumatic brain injury in the post-acute polytrauma setting: a scoping review of diagnostic approaches and screening tools. Brain Inj. 2025; 39(6): 445–56.
16. Jung E, Lee J-H, Park H-K. Association between serum magnesium level and long-term prognosis of traumatic brain injury. Brain Inj. 2025; 1–8.
17. Matsoukas S, Inzerillo S, Wedderburn RV, Legome EL, Margetis K, Camara D. Unraveling key determinants of cranial decompression in isolated severe traumatic brain injury: insights from a national trauma database. Brain Inj. 2025; 1–8.
18. Rollins R, Altman J, Diemer K, Smith A, Clugston JR, Silvestri P, et al. Pulmonary contusions in a collegiate football player with same-game return-to-play. Cureus. 2025; 17(2): e79752.
19. İşevi M, İşevi C, Çolak ÖY, Akman TS, Pirzirenli MG, Ünal Akdemir N. Mortality and morbidity in rib fracture surgery: Pulmonary contusion versus extrathoracic trauma. J Surg Res. 2025; 310: 8–16.
20. Lagazzi E, Panossian VS, Argandykov D, de Roulet A, Nzenwa IC, Rafaqat W, et al. Rib fixation in severe isolated chest trauma with pulmonary contusion: rib fixation in pulmonary contusion. Injury. 2025; 56(6): 112292.