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A Systematic Review of Neurogenic Pulmonary Edema in Traumatic Brain Injury

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Background	Neurogenic pulmonary edema (NPE) is a rare but serious complication of central nervous system (CNS) injury, characterized by acute pulmonary edema without a secondary cause. We present a case of NPE followed by a systematic review and analysis focused on traumatic brain injury (TBI), and consider potential mortality-associated risk factors.
Summary	We conducted a systematic search across PubMed, Cochrane, Google Scholar, and SCOPUS using PRISMA guidelines. After reviewing 1,399 articles based on established criteria, 29 studies were included in the final analysis (n=45 patients). We performed frequency analysis, independent samples t-tests, and calculated odds ratios to investigate risk factors for mortality.
Conclusion	NPE related to TBI carries a significant mortality rate of 42.2% (n=19) and substantial morbidity. Median hospital stay was 11 days, and 78.3% of survivors were discharged home. Initial vital signs, including systolic blood pressure (mean [SD], 113.13 [20.17], p = 0.29) and heart rate (97.93 [19.89], p = 0.16), were not significant predictors of mortality. While most patients were male (83.7%), sex, age, and admission vitals were not associated with mortality risk. This study represents the first systematic review of NPE, specifically in TBI. However, currently, there is insufficient evidence to conclusively identify risk factors associated with mortality.
Key Words	neurogenic pulmonary edema; trauma; lung edema; trauma surgery

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Case Description

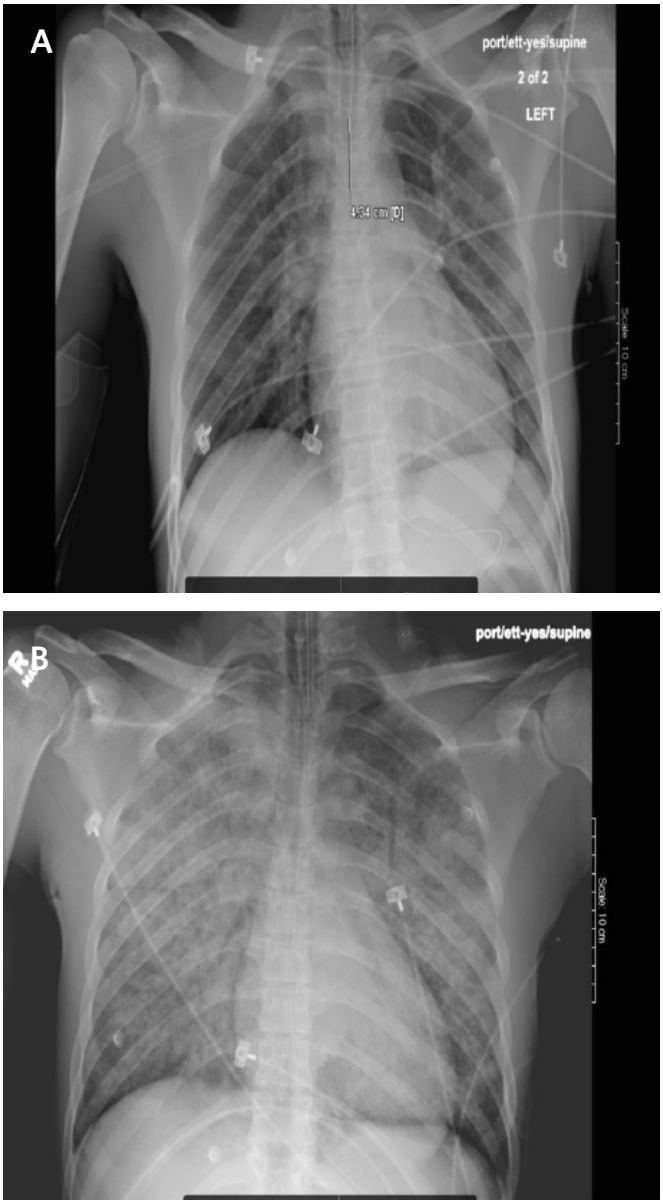
Neurogenic pulmonary edema (NPE) is a clinical syndrome of acute pulmonary edema triggered by a major central nervous system (CNS) insult. Its exact pathophysiology remains unclear, but a sudden catecholamine surge following the CNS injury is a leading hypothesis. Despite its frequent description in the medical literature, diagnosing NPE is challenging due to the lack of specific markers and its multisystem presentation. Common etiologies include seizures, traumatic brain injury (TBI), and intracranial hemorrhage.[1] Onset is rapid, occurring within minutes to hours of the inciting event. NPE can mimic several conditions, including aspiration pneumonitis, acute respiratory distress syndrome (ARDS), cardiogenic pulmonary edema, transfusion-associated lung disorders, and sepsis.

A previously healthy 22-year-old African American male presented with a single gunshot wound (GSW) to the head. On arrival, he was minimally responsive (Glasgow Coma Score [GCS] of 6: E4V1M1), with labored, agonal respirations. Despite 100% oxygen saturation on bag valve ventilation, his initial blood pressure was 80/30 mm Hg, and his heart rate was 62 beats per minute in sinus rhythm. Pupils were 2 mm, equal, and reactive. Rapid sequence intubation was performed within 10 minutes of arrival.

Focused assessment with sonography in trauma (FAST) revealed a diffusely hypokinetic left ventricle without tamponade or pericardial effusion. Initial chest X ray (CXR) demonstrated mild bilateral pulmonary edema (Figure 1A). CT scan of the head showed a ballistic fragment entering the left posterior parietal area, terminating in the left parasagittal anterior frontal lobe without contralateral injury (Figure 2). Imaging also revealed subarachnoid and subdural hematomas with a 3 mm midline shift.

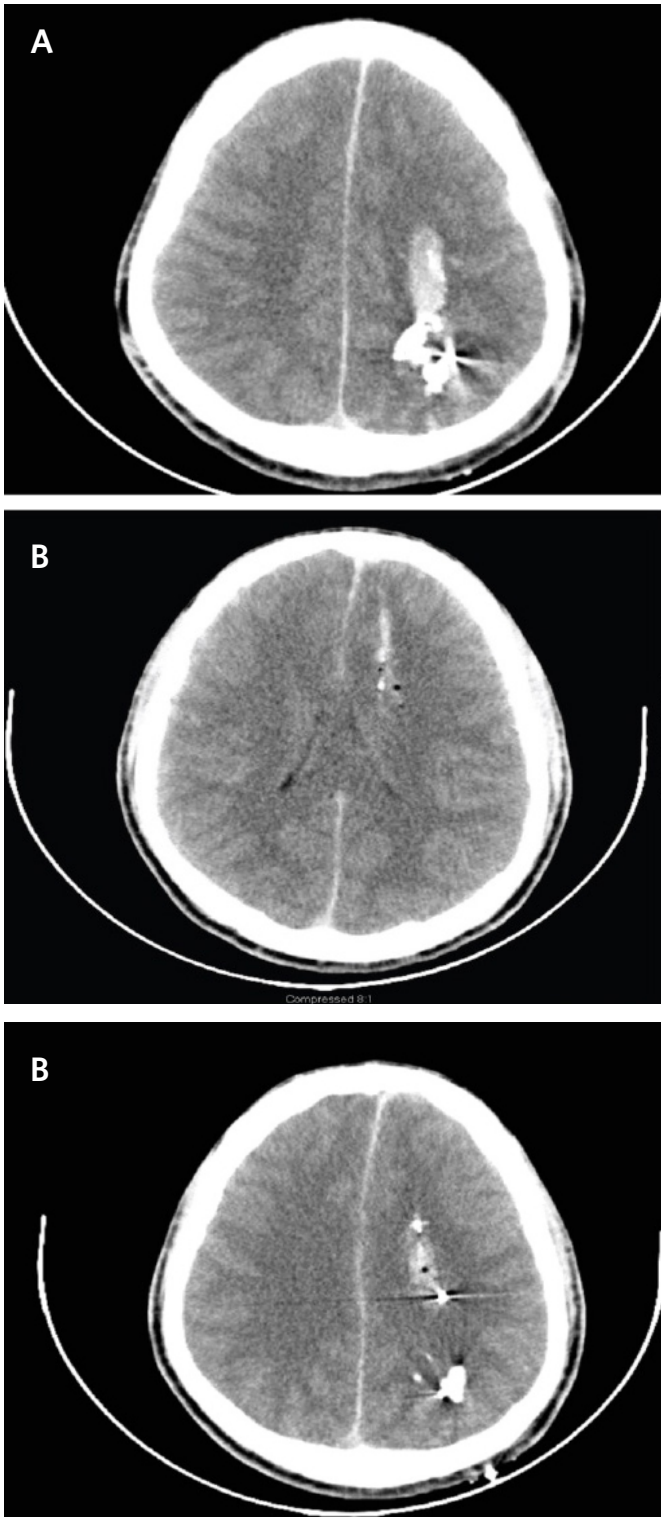
Repeat CXR less than an hour later showed worsening pulmonary edema (Figure 1B). The patient developed copious pink frothy secretions within 30 minutes. In the OR, serial bronchoscopies extracted approximately 700cc of serosanguinous fluid. He also underwent a left-sided decompressive hemicraniectomy, revealing diffuse brain edema.

Figure 1. Chest X-rays Illustrating Progression of NPE. Published with Permission



A) Initial CXR within ten minutes of arrival shows clear lung fields. B) Repeat CXR one hour later reveals marked bilateral pulmonary edema consistent with NPE.

Figure 2. Head CT (superior to inferior views). Published with Permission



Bullet trajectory through the left posterior parietal area, terminating in the left parasagittal anterior frontal lobe. No contralateral injury.

Despite intervention, the patient's neurological status did not improve over 72 hours, meeting brain death protocol criteria. He expired on hospital day 4 following confirmation of brain death.

Discussion

Neurogenic pulmonary edema is a rare but serious complication of CNS injury. While reported following infectious CNS events and aneurysmal hemorrhage, data on NPE-related TBI is limited to case reports and series, with mortality rates estimated at 60% to 100%.[2] This lack of strong evidence prompted our systematic review to provide an analysis of demographics and mortality in NPE caused by TBI.

The purpose of this study was to perform a systematic review of literature to provide an evidence-based analysis of demographics and mortality related to NPE caused by TBI. We adhered to PRISMA guidelines, searching PubMed, Cochrane, Google Scholar, and SCOPUS for articles on NPE (Figure 3). Two reviewers screened the initial 1,399 results, applying rigorous exclusion criteria. The final analysis included 29 articles (all case reports and series) with objective data, representing 45 TBI patients.[3–32] Data collection covered demographics, vital signs, time to pulmonary edema onset, hospital length of stay (LOS), discharge status, and initial oxygenation levels. Given the limited amount of studies on this topic, case reports and case series comprised all of the literature (Table 2).

Figure 3. Prisma Diagram

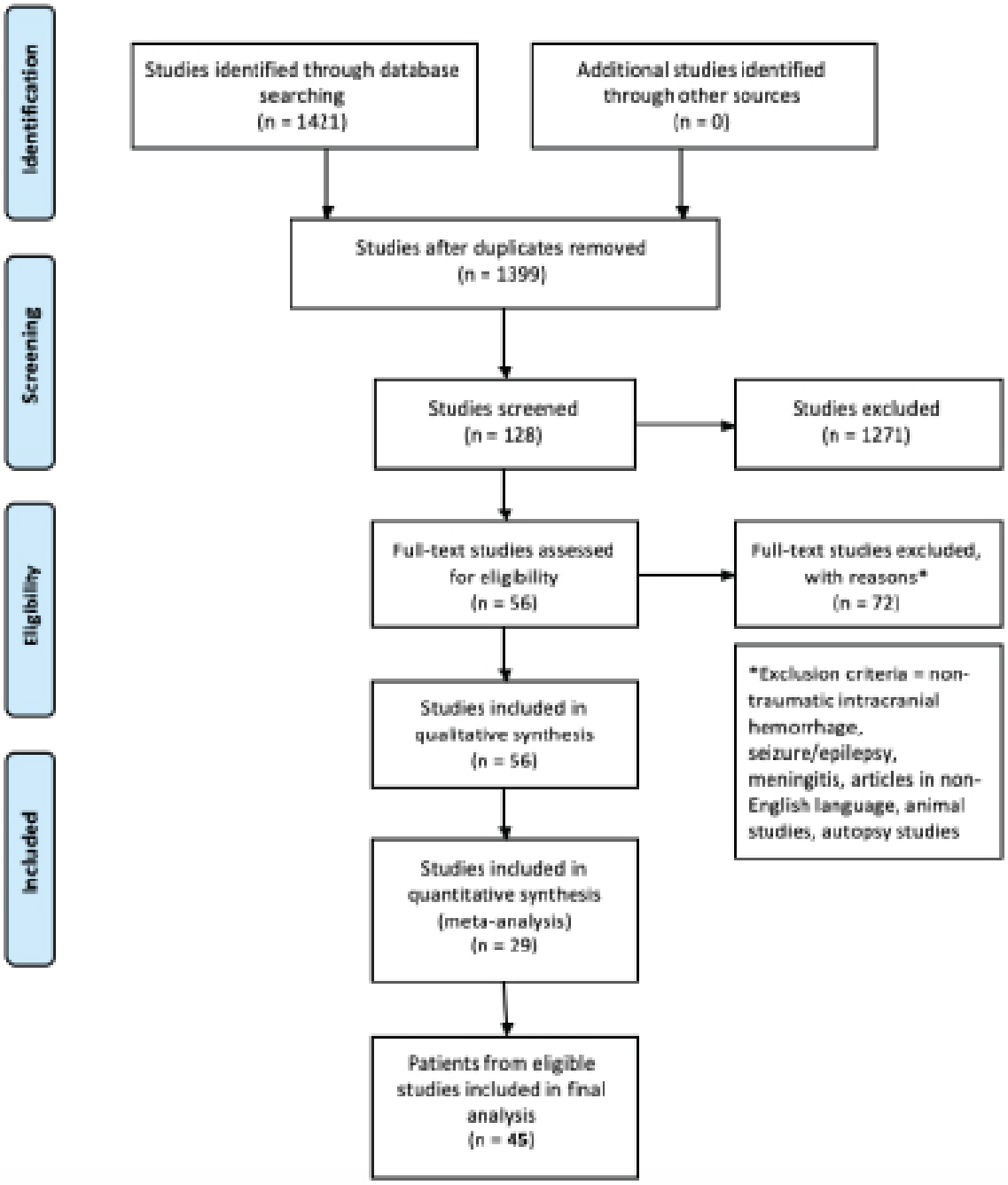


Table 1. Review and Quality of Included Studies

Study	Design*	# Male	# Female	Mortality (yes/no)	Time to first CXR	DC Neuro Status**	Hospital LOS
Bejalokovic 2006	CR	1	0	n	1.5 hours	2	20 days
Casey 1983	CR	0	0	y			
Chaari 2015	CR	1	0	y	4 hours		
Cohen 1977	CR	2	2	y	2 hours, 1 hour	1	21 days
Felman 1971	CS	2	1	y		1	
Fontes 2003	CR/LR	1	1	y	1 hour, 1 hour		
Gary 1998	CR	1	0	n		2	
Hegde 2017	CR	0	1	n	2 hours	1	8 days
Hideki 2018	CR	1	0	n	1 hour		
James 1978	CR	1	0	y	5 hours		
Jeyashanmugaraja 2019	CR	1	0	n	1 hour	2	
Lagerkranser 1982	CS	1	0	n		1	7 days
Loughnan 1980	CR	1	0	n	1 hour	1	10 days
Matsuyama 2007	CR	1	0	n		1	7 days
Mehmet 2015	CR	2	0	y	12 hours, 1 hour	1	35 days
Qin 2005	CS	0	0	y			
Rajagopal 2017	CR	1	0	n			5 days
Rubin 2001	CR	2	0	n	3 hours	1, 3	5 days, 11 days
Schranz 1981	CR	1	0	n		1	
Shah 2013	CR	1	0	n	1 hour	1	3 days
Sherr 2009	CR	0	1	n	1 hour	2	
Shigemori 1981	CR	0	1	n		1	
Simmons 1969	CS	9	0	y			
Sisikht 2020	CR	1	0	y	24 hours		
Wauchob 1984	CR	2	0	n	4 hours, 4 hours	2, 2	45 days, 30 days
Wohns 1985	CR	1	0	n	45 hours	2	60 days
Xiaoping 2016	CR	1	0	n		2	
Yamagishi 2014	CR	0	1	n			

CR = case report, CS = case series, LR = literature review
***DC Neuro status. 1 = no deficit, 2 = mild deficit (one neurological deficit one discharge), 3 = severe deficit (two or more neurological deficits on discharge)*

Table 2. Patient Data

Characteristic	Survived (n = 24)	Deceased (n =19)	*P*
Age in years median (Q1, Q3)	15.5 (8.75, 28.75)	20 (16, 29)	0.40
Gender, n (%)			
Male	21 (87.5%)	15 (83.3%)	
Female	3 (12.5%)	3 (16.7%)	
Blood pressure, systolic (mmHg) median (Q1, Q3)	130 (115, 165)	110 (100, 125)	0.29
Blood pressure, diastolic (mmHg) median (Q1, Q3)	67 (53, 97.5)	76 (60, 80)	0.85
Admission heart rate (beats per minute) median (Q1, Q3)	110 (86, 163)	100 (82, 118)	0.16
Admission PaO_2 (mmHg) median (Q1, Q3)	64 (49, 68)	53 (28, 56)	
Time to first Chest X-Ray (hours) median (Q1, Q3)	1 (1, 3.5)	4 (1, 12)	
Hospital length of stay (days) median (Q1, Q3)	11 (6.5–31.3)	0.71 (0.26–8.25)	
Neurological status at discharge, n (%)			
Full recovery	14 (63.6%)		
Mild deficit*	7 (31.8%)		
Severe deficit**	1 (4.5%)		
Discharge disposition, n (%)			
Home	18 (78.3%)		
Rehab	5 (21.7%)		

**Mild deficit was defined as one neurologic deficit on discharge*
***Severe deficit was defined as two or more neurologic deficits on discharge*

Descriptive statistics (means, standard deviations, frequencies) were calculated using SPSS Version 26. We compared patients who survived versus those who died, with mortality as the primary outcome. Independent samples t-tests and odds ratios were used to assess risk factors, including admission vitals, gender, age, and time to first chest X-ray. Secondary outcomes were discharge neurological status, disposition, and LOS.

Our analysis revealed an overall NPE mortality rate of 42.4% (n=19) in these TBI cases. Most survivors (78.3%) were discharged home, with 63.6% returning to baseline neurological function. Admission vitals, gender, and time to pulmonary edema onset did not appear to significantly affect mortality risk (Table 2). The median LOS for survivors was 11 days. (Table 3).

As seen in our case, a single high-velocity GSW to the head likely triggered a sudden rise in intracranial pressure, leading to a catecholamine surge. This cascade of events potentially caused both direct myocardial stunning and subsequent diffuse pulmonary edema. The observed severe hypoxia, acute bilateral infiltrates, and copious pulmonary secretions further support the diagnosis of NPE.

While pre-intubation aspiration remains a potential contributing factor, the onset of hypoxia related to aspiration pneumonitis (hours to days after the inciting event)[31] contrasts with the acute presentation observed in this case. This timing makes NPE a more compelling diagnosis. Acute respiratory distress syndrome (ARDS) warrants consideration due to the acuity of respiratory distress. However, the clear neurologic insult combined with evidence of cardiac hypokinesis, in the absence of a primary cardiac or pulmonary cause, strengthens the case for NPE as the predominant mechanism.

Transfusion-associated circulatory overload (TACO) must also be carefully weighed in the differential. While TACO and NPE can present with similar acute respiratory distress, the extremely rapid onset of pulmonary edema in this patient, following ten units of blood products, deviates from TACO's typically slower progression (less than six hours after a blood transfusion).[33] This further supports NPE as the more likely primary process.

Our systematic review highlights a high mortality rate associated with NPE. While insufficient evidence exists to link age or gender definitively to NPE risk, our analysis and presented case highlight a predominance of young males among those affected. Notably, 63.6% of survivors experienced a full recovery, suggesting the importance of prompt NPE identification and management for better outcomes.

Furthermore, we observed a prolonged average delay of 1.7 hours between admission and the first chest X-ray. The cause of this delay is unclear; however, it may be due to prioritization of other injuries or limited awareness of pulmonary complications. Due to the small sample size (n = 45) and limited number of published cases, statistical analysis of risk factors associated with mortality did not yield significant results. Outliers in the data likely skewed averages within this dataset. We advocate for continued case reporting and data collection on NPE to enable a more robust investigation into risk factors for mortality.

While the precise mechanism of NPE remains under investigation, four leading theories propose an initial intracranial event triggering a "catecholamine surge" that ultimately leads to acute onset pulmonary edema.[34]

1. **Neuro-Cardiac Model:** Acute neurological injury elevates intracranial pressure (ICP), causing massive sympathetic catecholamine release.[2] This induces direct myocardial injury, paralleling processes seen in Takutsubo's cardiomyopathy. Resultant cardiac dysfunction contributes to pulmonary edema.
2. **Neuro-Hemodynamic Model:** The catecholamine surge primarily increases afterload and systemic pressure rather than direct myocardial injury. This sudden vasoconstriction impairs contractility, with backflow leading to elevated pulmonary hydrostatic pressure and edema.[2]
3. **Blast Theory:** Expanding on the neuro-hemodynamic model, this theory posits a combination of hydrostatic pressure changes and direct pulmonary barotrauma due to rapid pressure shifts. The presence of red blood cells and protein in the alveolar fluid of NPE patients suggests a mechanism beyond simple hydrostatic pulmonary edema. This observation supports the theory of alveolar capillary cell death, which would explain the capillary leak and exudative nature of the effluent. This phenomenon aligns with our case, where bronchoscopy revealed copious amounts of pink, frothy fluid.[2]
4. **Pulmonary Venule Adrenergic Hypersensitivity:** This theory proposes that widespread, hypersensitive alpha and beta-adrenergic receptors in pulmonary vasculature suffer direct injury from the catecholamine surge, leading to vascular damage and edema.[2]

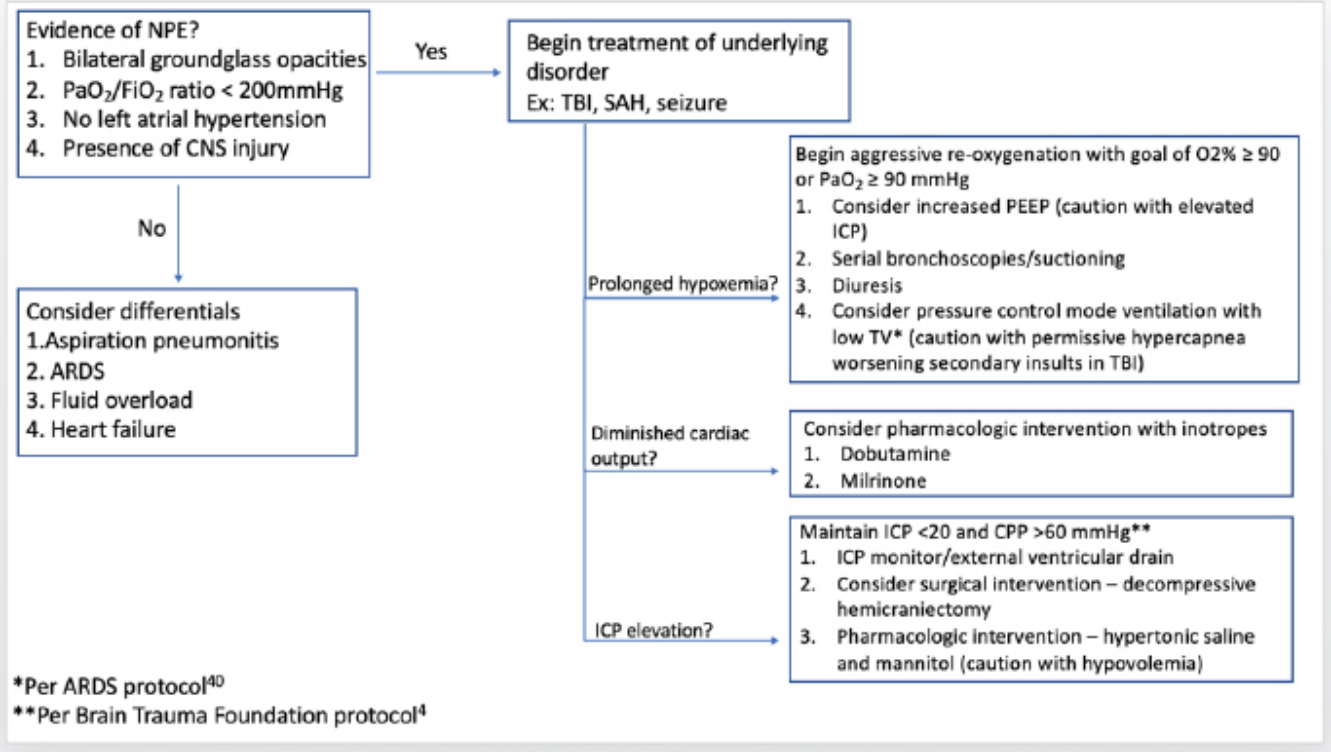
NPE remains a diagnosis of exclusion, requiring careful differentiation from aspiration pneumonitis, TACO, and ARDS. Key distinguishing features include rapid onset, absence of primary cardiopulmonary injury, and evidence of CNS trauma (Figure 4), and there are several proposed diagnostic criteria:

- Bilateral chest X-ray infiltrates
- PaO_2/FiO_2 ratio <200
- No left atrial hypertension
- CNS injury
- Absence of other ARDS triggers[1]

Alongside diagnostics, clinical presentation aids in identifying NPE.[34] Symptoms typically manifest within 30 to 60 minutes of CNS injury (occasionally delayed up to 12 to 24 hours).[1] Symptoms present concurrently with neurogenic cardiac disturbances. Dyspnea, pink frothy sputum, tachypnea, cyanosis, hypertension, and fever are common at presentation.[35] Notably, NPE's rapid resolution can lead to some patients being asymptomatic upon arrival.[1]

Treatment prioritizes addressing the primary CNS insult, with supportive care for symptomatology. Decompressive hemicraniectomy may be indicated in cases of elevated ICP, as seen in our case presentation. Respiratory management aligns closely with ARDS protocols: abiding by Brain Trauma Foundation Guidelines,[36] mechanical ventilation with a target O_2 saturation >90% or PaO_2 >90 mmHg is generally acceptable for TBI patients. Serial bronchosco-

Figure 4. Diagnosis and Treatment



py aids in excessive fluid removal, and inotropes may be necessary to support compromised cardiac output. While aggressive suctioning of pulmonary edema and positive end-expiratory pressure (PEEP) can be useful in oxygen management,[37] these interventions must be used cautiously due to their potential to worsen ICP.[38] Lung-protective ventilation strategies are critical.[40] Pressure control ventilation may become necessary, but clinicians must be vigilant in avoiding permissive hypercapnia, as the resultant cerebral vasodilation can exacerbate secondary brain injury.

Though various pharmacological interventions have been used in case reports for NPE, robust evidence for their efficacy is lacking. Inotropes like dobutamine and milrinone might improve cardiac contractility where it is compromised,[35] while diuretics and nitrates can help decrease pulmonary fluid volume by enhancing diuresis and promoting pulmonary vasodilation. However, using diuretics in acute trauma requires extra caution due to the potential for hypovolemia due to hemorrhagic causes. Some case studies suggest that positive inotropes, combined with diuretics and nitrates, may provide both symptom relief and a mortality benefit in severe pulmonary edema.[35] However, whether this benefit extends to patients with complicated NPE remains unclear.

This review's value relies on the quality of available evidence. Unfortunately, high-level evidence on NPE caused by TBI is scarce, necessitating the inclusion of case reports and case series. Incomplete data across studies and a focus on in-hospital complications were also limitations. We acknowledge the rarity of TBI-induced NPE, which hinders the collection of robust data. The small sample sizes inherent to this clinical area limited the power needed to draw definitive conclusions about mortality risk factors. Retrospective reviews of patient data, structured for survival vs. mortality comparisons, hold promise for identifying key risk factors influencing outcomes in this complex clinical scenario.

Conclusion

Neurogenic pulmonary edema, a rare complication of acute neurological insult like trauma, is characterized by the acute onset of pulmonary edema. Currently, there is insufficient data to definitively identify risk factors associated with its significant mortality and morbidity.

Lessons Learned

In trauma patients presenting with head injuries, consider the possibility of NPE as a potential complication, even in the absence of primary lung injury. Early identification, though challenging due to its rarity, is essential for guiding management and potentially improving outcomes.

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