CASE REPORT

Rapid Oxygenation Recovery in Drowning-Associated ARDS Managed With Early Lung-Protective Ventilation

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ABSTRACT

Background: Drowning is a major cause of accidental injury worldwide, accounting for an estimated 236,000 deaths annually, and may lead to acute respiratory distress syndrome (ARDS) due to aspiration-induced lung injury. Epidemiological data from Indonesia remain limited; retrospective studies from Bali and North Sulawesi report drowning-related mortality rates ranging from 0.18 to 1.73 per 100,000 population, reflecting provincial-level data and suggesting potential underestimation of the national burden. Although freshwater and seawater aspiration have traditionally been considered pathophysiologically distinct, clinical outcomes appear to be influenced more by the severity of hypoxemia and early respiratory management than by water salinity.

Case: We report two cases of drowning-associated ARDS in young adult males. The first patient developed moderate ARDS following seawater drowning, while the second presented with severe ARDS after freshwater drowning. Both patients underwent early endotracheal intubation and were managed with lung-protective mechanical ventilation using low tidal volumes and appropriate positive end-expiratory pressure (PEEP). In both cases, oxygenation improved rapidly, with PaO_2/FiO_2 ratios increasing to greater than 250 within hours of ventilation. Serial chest radiographs demonstrated progressive resolution of bilateral alveolar infiltrates, allowing successful ventilator weaning and extubation on day four and day six, respectively.

Discussion: Despite differences in initial ARDS severity and the type of aspirated water, both patients demonstrated similar patterns of rapid oxygenation recovery and radiological improvement. This supports the concept that the extent of aspiration and early disruption of the alveolar–capillary membrane, rather than water composition, are the primary determinants of clinical severity. Early lung-protective ventilation likely mitigated further ventilator-induced lung injury, promoted alveolar recruitment, and facilitated rapid reversibility of hypoxemia in both cases.

Conclusion: In this two-patient case series, drowning-associated ARDS following freshwater and seawater aspiration showed rapid improvement in oxygenation and radiological findings during management with early airway control and lung-protective

mechanical ventilation. These observations support the potential reversibility of drowning-related lung injury but should be interpreted cautiously given the limited sample size.

Keywords: Acute respiratory distress syndrome; Drowning; Freshwater; Lung-protective ventilation; Seawater

INTRODUCTION

Drowning is a major cause of accidental injury worldwide and remains a significant contributor to morbidity and mortality, particularly in regions with extensive exposure to natural and recreational water environments. The primary pathophysiological consequence of drowning is respiratory impairment due to aspiration, which may rapidly progress to acute respiratory distress syndrome (ARDS). Pulmonary injury following drowning is characterized by surfactant dysfunction, alveolar–capillary membrane damage, inflammatory response, and severe ventilation–perfusion mismatch.[1]

Epidemiological data on drowning in Indonesia remain limited. A retrospective study conducted at Sanglah Hospital, Bali, between 2010 and 2014 reported 209 drowning cases, corresponding to a mortality rate of 1.73 per 100,000 population. Similarly, data from Prof. Dr. R. D. Kandou Hospital, collected between 2007 and 2011, demonstrated an average drowning-related mortality rate of 0.18 per 100,000 population. However, these findings are restricted to provincial-level data, and the absence of nationwide epidemiological studies suggests that the true burden of drowning in Indonesia may be substantially underreported.[2]

Traditionally, drowning-related lung injury has been described differently according to the type of aspirated water. Freshwater aspiration is thought to cause surfactant washout and alveolar collapse, whereas seawater aspiration results in hypertonic injury, alveolar flooding, and pulmonary edema. Despite these theoretical differences, clinical evidence demonstrating distinct respiratory outcomes between freshwater and seawater drowning remains inconsistent. Increasingly, the severity of hypoxemia and extent of aspiration, rather than water composition alone, appear to determine clinical course and prognosis.[3]

Early airway management and appropriate ventilatory support are critical in drowning- associated ARDS. Current guidelines recommend lung-protective ventilation strategies, including low tidal volume ventilation and appropriate positive end-expiratory pressure (PEEP), to minimize ventilator-induced lung injury and improve outcomes in ARDS of various etiologies. However, data focusing on the early application of lung-protective ventilation in drowning-related ARDS, particularly in comparative freshwater and seawater cases, remains limited.[4,5]

We report two cases of drowning-associated ARDS: one following seawater drowning, presenting with moderate ARDS, and the other following freshwater drowning, presenting with severe ARDS. Both patients were managed with early intubation and lung-protective mechanical ventilation, resulting in rapid improvement in oxygenation, radiological resolution, and successful extubation. These cases

illustrate the reversibility of drowning-associated ARDS and highlight the importance of early lung-protective ventilation, regardless of drowning medium or initial severity.

CASE

Case 1 – Saltwater Drowning

A 19-year-old male with a predicted body weight of 78 kg was admitted after seawater drowning complicated by cardiac arrest secondary to submersion. Return of spontaneous circulation (ROSC) was achieved following cardiopulmonary resuscitation at the scene. No reliable submersion time could be established because of the chaotic circumstances surrounding the rescue. On arrival at the emergency room, he presented with a decreased level of consciousness and severe respiratory distress. Initial examination revealed a Glasgow Coma Scale score of 6 (E1V2M3), respiratory rate of 32 breaths·min^{-1}, body temperature of 38.2°C, and oxygen saturation of 68% on room air. Lung auscultation demonstrated bilateral crackles with reduced chest expansion. Blood pressure was 102/52 mmHg, and heart rate was 98 beats·min^{-1}. Despite oxygen administration via a non-rebreathing mask at 15 L·min^{-1}, oxygen saturation increased only to 90–94%.

Arterial blood gas analysis showed SpO_2/FiO_2 ratio of 80, consistent with ARDS. The patient underwent early endotracheal intubation and mechanical ventilation, then was transferred to the intensive care unit. Ventilation was continued in the intensive care unit using a lung-protective strategy with volume-controlled synchronized intermittent mandatory ventilation (VC-SIMV), tidal volume 6 mL·kg^{-1} predicted body weight, PEEP 5–6 cmH_2O, and FiO_2 gradually reduced from 90% to 40%. Within five hours, repeat arterial blood gas analysis demonstrated marked improvement in oxygenation, with the PaO_2/FiO_2 ratio increasing to 252.

During the early course of intensive care management, the patient was febrile on admission, with body temperature gradually normalizing thereafter. No clinically significant electrolyte abnormalities were observed during the intensive care unit (ICU) stay. Empirical antibiotic therapy was administered during the initial phase of treatment, and no microbiologically confirmed pneumonia was identified. Clinical improvement was observed by day two, characterized by a reduction of crackles on auscultation. Serial chest radiographs showed progressive resolution of bilateral alveolar opacities, and the patient was successfully weaned from mechanical ventilation and extubated on day four (Figure 1). Overall, the clinical and radiological findings were consistent with moderate ARDS following seawater drowning.

Serial clinical and ventilatory parameters are summarized in Table 1. Hemodynamic status remained stable throughout the ICU stay, while ventilatory requirements progressively decreased within the first 24 hours. Serial arterial blood gas analyses (Table 2) demonstrated rapid correction of hypoxemia, with PaO_2/FiO_2 ratio improving from 142 on admission to greater than 250 within five hours of lung-protective mechanical ventilation. This rapid improvement was accompanied by progressive radiological resolution and allowed early weaning and successful extubation on day four.

Case 2 – Freshwater Drowning

A 23-year-old male with a predicted body weight of 95 kg was admitted

approximately two hours after freshwater drowning in a swimming pool, with an estimated submersion time of 2–3 minutes. On presentation at the emergency room, he was agitated with decreased consciousness and tachypnea.

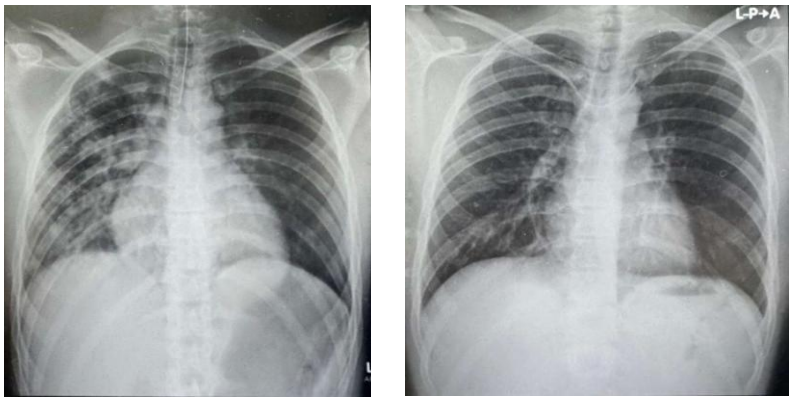


Figure 1. Chest X-ray on admission revealed bilateral diffuse alveolar infiltrates consistent with moderate ARDS following drowning. Follow-up imaging after extubation (day 4) demonstrated significant radiological resolution, paralleling clinical recovery.

Table 1. Clinical course and ventilatory parameters

Parameter	Day 0 (Admission)	Day 1	Day 2	Day 4 (Pre-extubation)
Blood pressure (mmHg)	95–116 / 44–70	112–130 / 47–88	101–120 / 49–76	104–115 / 58–69
Heart rate (beats/min)	76–90	65–80	60–75	48–58
Oxygen saturation (%)	99–100	99–100	95–100	100
Temperature (°C)	37.5–38.2	36.5–37.7	36.4–37.0	36.8–37.0
Ventilation mode	VC-SIMV	VC-SIMV	CPAP	Room air
Tidal volume (mL)	480	480	PS 10	–
Respiratory rate	20	20	–	–
FiO_2 (%)	90 → 40	40	40	–
PEEP (cmH_2O)	6–5	5	5	–

Table 2. Serial arterial blood gas analysis

Parameter	Admission	5 hours	Day 2	Pre-extubation
pH	7.26	7.34	7.38	7.42
PaO_2 (mmHg)	128	232	201	94
$PaCO_2$ (mmHg)	50	43.9	42	40.2
FiO_2	0.9	0.4	0.4	0.4
PaO_2/FiO_2 ratio	142	>250	>250	–
Base excess	6.6	2.6	0.2	0.5
HCO_3^- (mmol/L)	29.9	23.1	24.8	25
SaO_2 (%)	98.9	100	100	97.9

Vital signs revealed a respiratory rate of 30–34 breaths·min^{-1}, oxygen saturation of 75% on room air; axillary temperature of 35.6°C, and delayed capillary refill more than two seconds. Lung examination demonstrated bilateral rales and wheezing. Oxygen administration via a non- rebreathing mask at 15 L·min^{-1} improved oxygen saturation only to 88%, prompting admission to the intensive care unit.

Arterial blood gas analysis demonstrated severe hypoxemia, with a SpO_2/FiO_2 ratio of 104, consistent with ARDS. The patient was subsequently intubated and mechanically ventilated using volume-controlled ventilation with a tidal volume of 6 mL·kg^{-1} predicted body weight, PEEP 8 cmH_2O, and FiO_2 100%, which was later titrated according to oxygenation. Within five hours of initiating mechanical ventilation, oxygenation improved markedly, with the PaO_2/FiO_2 ratio increasing to 252.

On admission, the patient was hypothermic, which resolved following active rewarming measures. During the ICU stay, transient hypokalaemia was observed and corrected with potassium supplementation without associated cardiac arrhythmias. Empirical antibiotic therapy was initiated early; however, no microbiologically confirmed pneumonia was identified. Gradual clinical improvement was observed, with decreasing crackles and improved lung compliance. Serial chest radiographs demonstrated resolution of bilateral pulmonary infiltrates, and the patient underwent stepwise ventilator weaning, with successful extubation on day six (Figure 2). Hemodynamic status remained stable throughout the ICU stay.

The clinical course and ventilatory parameters of Case 2 are summarized in Table 3. Despite presenting with severe hypoxemia and meeting criteria for ARDS on admission and later confirmed to be severe ARDS based on PaO_2/FiO_2 ratio after tracheal intubation and blood gas analysis, the patient demonstrated rapid improvement in oxygenation following early initiation of lung-protective mechanical ventilation. Serial arterial blood gas analyses (Table 4) showed a marked increase in the PaO_2/FiO_2 ratio from 82 at admission to greater than 250 within five hours, with further normalization over subsequent days. This improvement was accompanied by gradual radiological resolution and allowed stepwise ventilator weaning, culminating in successful extubation on day six.

DISCUSSION

Drowning remains a major cause of accidental injury worldwide and is frequently complicated by acute respiratory failure in survivors who reach hospital care. The predominant mechanism of morbidity following drowning is hypoxemia caused by aspiration-induced lung injury, which may rapidly evolve into ARDS. Although the pathophysiology of drowning has been described for decades, the clinical relevance of distinctions between freshwater and seawater drowning remains debated, particularly in the context of modern intensive care and lung-protective ventilation strategies.

In this case series, we report two young adult patients who developed ARDS diagnosed based on the Berlin criteria following freshwater and seawater drowning, respectively.[6,7]

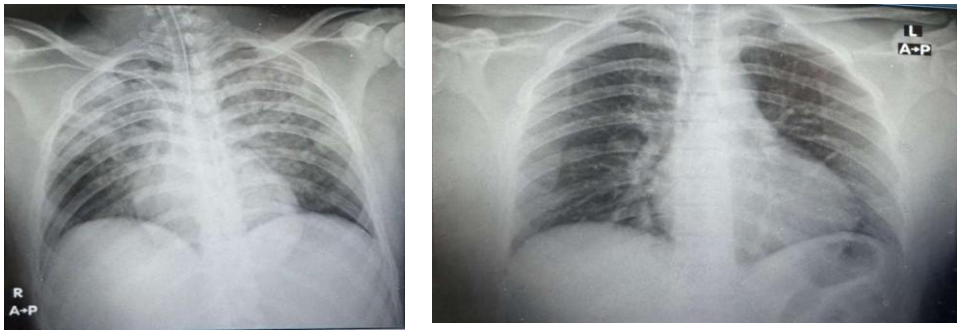


Figure 2 - Chest X-ray on admission showed bilateral diffuse alveolar infiltrates consistent with severe ARDS following freshwater drowning. Follow-up imaging prior to extubation demonstrated substantial radiological resolution, in parallel with clinical recovery.

Table 3. Clinical Course and Ventilatory Parameters (Case 2)

Parameter	Day 0	Day 1	Day 2	Day 4	Day 6 (Pre-extubation)
Blood pressure (mmHg)	102–110 / 61–70	98–135 / 60–78	107–157 / 57–61	135–165 / 62–70	145–174 / 77–79
Heart rate (beats/min)	82–90	88–115	90–113	46–58	77–103
Oxygen saturation (%)	75–88	98–99	98–100	99–100	99
Temperature (°C)	35.8–36.4	35.6–37.4	37.8–37.3	36.3–37.5	36.0–36.2

Parameter	Day 0	Day 1	Day 2	Day 4	Day 6 (Pre-extubation)
Ventilation Mode	NRBM - VCV	VCV	VCV	VCV	VC SIMV → CPAP
Tidal volume (mL)	- / 600	600	600	600	600 - PS 12
Respiratory rate	34 / 20	20	20	20	20
FiO_2 (%)	- / 100	50	50	50	40%
PEEP (cmH_2O)	- / 8	8	8	8	5

Table 4. Serial Arterial Blood Gas Analysis (Case 2)

Parameter	Admission	5 hours	Day 2	Day 4	Pre-extubation
pH	7.20	7.24	7.36	7.41	7.47
PaO_2 (mmHg)	82.1	252	188	204	102
$PaCO_2$ (mmHg)	44.7	41.6	36	32.2	30.5
FiO_2	1.0	1.0	0.5	0.5	0.4
PaO_2/FiO_2 ratio	82	252	376	408	–
Base excess	−7.1	−7.9	−2.5	−0.1	−0.8
HCO_3^- (mmol/L)	19.8	18.9	22.8	22.9	22.0
SaO_2 (%)	95.4	99.6	97.7	99.9	99.9

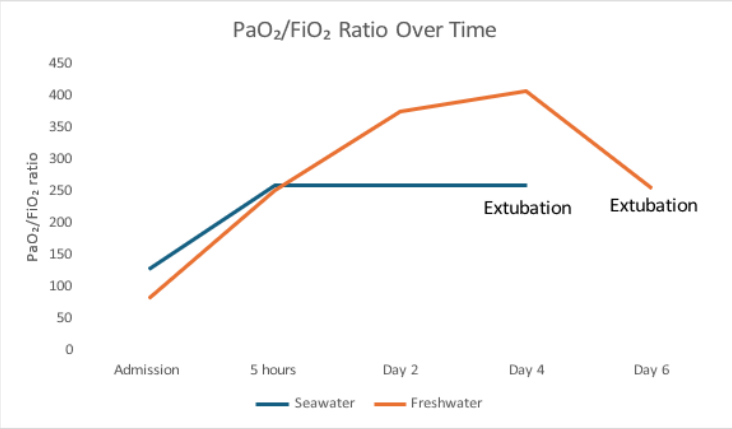


Figure 3 - PaO_2/FiO_2 ratio over time in seawater and freshwater drowning–associated ARDS, demonstrating rapid improvement in oxygenation following early lung-protective mechanical ventilation.

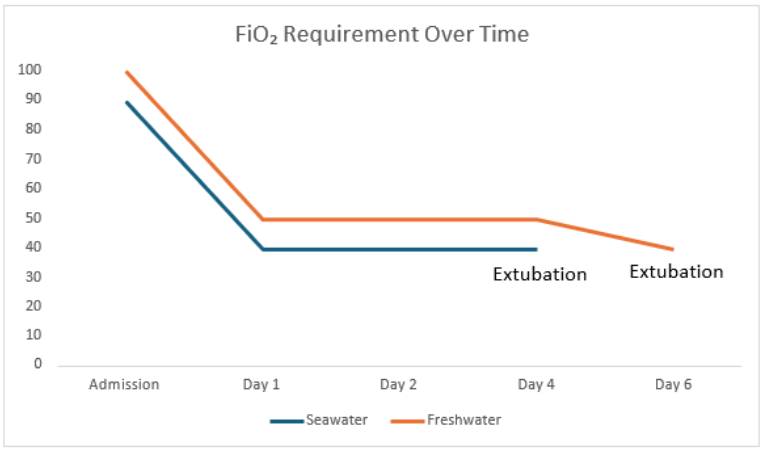


Figure 4 - Progressive reduction in FiO_2 requirement over time in both cases, reflecting successful ventilator weaning following early improvement in oxygenation.

Despite differences in the type of aspirated water and initial severity of hypoxemia, both patients demonstrated rapid improvement in oxygenation, radiological resolution, and favourable clinical outcomes following early initiation of lung-protective mechanical ventilation. These cases provide important insights into the pathophysiology, reversibility, and optimal management of drowning-associated ARDS, with particular relevance to anaesthetic and critical care practice.

The primary insult in drowning is respiratory impairment caused by immersion or submersion of the upper airway in liquid, resulting in aspiration and hypoxemia. Once water enters the lower respiratory tract, it disrupts normal alveolar function through several mechanisms. Aspiration leads to dilution, washout, and inactivation of pulmonary surfactant, increasing alveolar surface tension and promoting widespread alveolar collapse. This process reduces lung compliance, increases intrapulmonary shunting, and causes severe ventilation– perfusion mismatch, ultimately manifesting as hypoxaemic respiratory failure and ARDS (Figure 5). [2,8,9]

The clinical severity of drowning-related lung injury is primarily determined by the volume of aspirated fluid rather than its composition. Aspiration of as little as 1–3 $mL \cdot kg^{-1}$ of either freshwater or seawater is sufficient to disrupt pulmonary surfactant through dilution and washout, leading to alveolar instability and diffuse alveolar damage. Although freshwater and seawater differ in osmolarity, both produce comparable degrees of lung injury, as osmotic gradients across the delicate alveolar–capillary membrane compromise membrane integrity and increase permeability, promoting transudation of fluid, plasma, and electrolytes into the alveolar space. At the molecular level, surfactant phospholipids and proteins, particularlydipalmitoylphosphatidylcholine (DPPC) are highly sensitive to dilution and displacement, resulting in loss of surfactant function, formation of protein-rich non-cardiogenic pulmonary oedema (often haemorrhagic), and impaired gas exchange. The combined effects of alveolar flooding, surfactant loss, and increased capillary–alveolar permeability culminate in severe ventilation–perfusion mismatch, intrapulmonary shunting, atelectasis, bronchospasm, and the rapid onset of profound hypoxemia with bilateral alveolar opacities observed at presentation. [10,11]

In addition to surfactant dysfunction, aspiration alters alveolar–capillary membrane permeability. Both hypo-osmolar (freshwater) and hyperosmolar (seawater) fluids can damage the alveolar epithelium and pulmonary endothelium, facilitating plasma leakage into the alveolar space. This non-cardiogenic pulmonary oedema further compromises oxygen diffusion and lung compliance.[10,12]Stress-induced catecholamine surges during hypoxia may exacerbate endothelial injury, amplifying pulmonary oedema and inflammatory responses.[10]

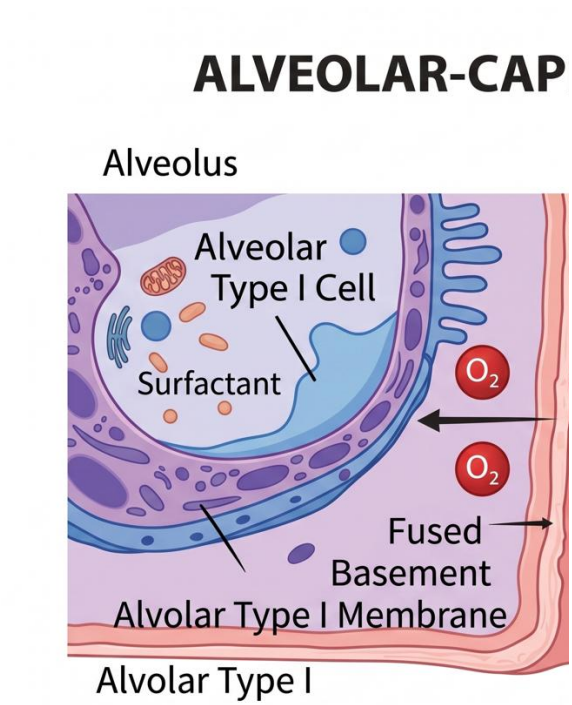


Figure 5. Alveolar–capillary membrane structure and gas exchange. Schematic illustration of the normal alveolar–capillary membrane showing the close apposition of the alveolar epithelium, fused basement membrane, and capillary endothelium, which facilitates efficient oxygen (O_2) and carbon dioxide (CO_2) exchange. In drowning-associated lung injury, aspiration-induced surfactant dysfunction and disruption of the alveolar–capillary membrane increase permeability, leading to alveolar flooding, impaired gas exchange, and development of acute respiratory distress syndrome (ARDS).

Traditional teaching has emphasised physiological differences between freshwater and seawater drowning, largely based on osmolarity. Freshwater aspiration was historically thought to cause rapid absorption of fluid into the circulation, haemodilution, haemolysis, and electrolyte disturbances, whereas seawater aspiration was believed to cause hypertonic alveolar oedema and intravascular fluid shifts.[3] However, contemporary clinical evidence challenges the relevance of these distinctions in living patients.

Modern observational studies and systematic reviews demonstrate that the volume of aspirated water in survivors is typically insufficient to cause clinically significant electrolyte or haematological abnormalities.[9,10,13] Instead, pulmonary injury dominates the clinical picture, and outcomes appear to be driven by the severity of hypoxemia, the duration of submersion, and the timeliness of rescue and respiratory support, rather than by the composition of the aspirated fluid.

Large cohort studies comparing freshwater and seawater drowning have shown similar rates of ARDS, mechanical ventilation, pneumonia, and mortality. In the multicentre cohort study by Reizine et al., freshwater drowning was associated with worse outcomes; however, this difference was attributed to confounding factors such as circumstances of drowning, psychiatric comorbidities, and severity at presentation rather than intrinsic

differences in water composition. [8,14] Our cases support this interpretation because both cases exhibited comparable recovery trajectories once lung-protective ventilation was applied, although the freshwater drowning patient initially presented with more severe hypoxemia than the seawater case. Caution is needed as only two cases were reported but this opens up the opportunity for further research with more cases.

The clinical trajectory observed in our two patients is broadly consistent with the limited published literature on drowning-associated lung injury, although differences in patient demographics, illness severity, and rescue requirements warrant cautious interpretation. In a retrospective cohort of 43 adult near-drowning victims, Gregorakos et al. reported that lung injury resolved rapidly in most survivors, with normalisation of oxygenation by day four in approximately three-quarters of patients and radiographic improvement in around two-thirds, alongside short hospital stays (mean ≈ 5 days) and low mortality.[15] Although that cohort comprised predominantly elderly patients, the pattern of rapid, day-four oxygenation recovery closely parallels the course of our seawater case, who was extubated on day four, and is in keeping with the view that promptly managed drowning-related lung injury is frequently and rapidly reversible. Multicentre cohort data similarly indicate that the duration of mechanical ventilation does not differ substantially according to the initial respiratory support strategy, and that outcomes are governed more by submersion circumstances and the severity of hypoxemia than by water composition.[13]

With respect to water salinity, a large French multicentre cohort of 270 critically ill drowning patients found freshwater drowning to be independently associated with 28-day mortality; however, this association was attributed largely to the circumstances of freshwater events, including a higher incidence of cardiac arrest and psychiatric comorbidity, rather than to an intrinsic effect of water tonicity, and pooled data demonstrate comparable rates of drowning-associated pneumonia after freshwater and seawater aspiration.[14,16]

This observation is reflected in the present series. Although the freshwater drowning patient initially presented with more severe hypoxemia, fulfilling criteria for severe ARDS, he experienced a favourable clinical course with successful extubation by day six. This outcome likely reflects factors such as young age, a short estimated submersion time, a witnessed event, and timely advanced resuscitative and critical care interventions, rather than the salinity of the aspirated water itself.

The principal difference between our cases and several previously published reports lies in the intensity of respiratory support required. In contrast to recent case reports and small series in which drowning-associated ARDS progressed to refractory hypoxemia requiring veno-venous extracorporeal membrane oxygenation (ECMO), frequently in the context of cardiac arrest, barotrauma, or persistent PaO_2/FiO_2 ratios below 80 despite optimised lung-protective ventilation, neither of our patients required rescue therapy, even though one met criteria for severe ARDS.[17, 18] This divergence likely reflects differences in submersion duration, the absence of refractory hypoxemia or air-leak

complications, and timely escalation to invasive ventilation, and it underscores that drowning-associated ARDS spans a wide severity spectrum from rapidly reversible disease manageable with conventional lung-protective ventilation, as in the present cases, to fulminant respiratory failure requiring extracorporeal support. Because these comparisons draw on heterogeneous study designs, age groups, and treatment eras, and because the present report comprises only two patients, they should be regarded as hypothesis-generating rather than confirmatory.

A central feature of both cases was the early initiation of lung-protective mechanical ventilation, which likely contributed to the favourable outcomes observed. Both patients were ventilated using low tidal volumes (~6 $mL \cdot kg^{-1}$ predicted body weight), moderate levels of PEEP, and progressive reduction of inspired oxygen fraction, in accordance with established ARDS management principles.

From an anaesthetic and critical care perspective, drowning-associated ARDS presents a unique challenge in airway management. While non-invasive respiratory support may be considered in selected patients, persistent hypoxemia, high work of breathing, and altered mental status increase the risk of delayed intubation and patient self-inflicted lung injury (P- SILI). Excessive spontaneous inspiratory effort can exacerbate alveolar injury through high transpulmonary pressures, worsening pulmonary oedema and inflammation. [10,11]

In both cases, early intubation was performed based on physiological deterioration rather than prolonged trials of non-invasive ventilation. Oxygenation improved rapidly following invasive ventilation, allowing early reduction of FiO_2 and avoidance of prolonged exposure to hyperoxia. This observation supports current recommendations favouring timely escalation to invasive ventilation in severe hypoxaemic respiratory failure, particularly when airway protection is compromised.

In the freshwater drowning case, transient hypokalaemia was observed during intensive care management, without associated cardiac arrhythmias. This electrolyte disturbance was likely multifactorial, related to stress response, catecholamine surge, intracellular potassium shift, and diuretic therapy rather than direct effects of freshwater aspiration, as clinically significant electrolyte changes due to drowning are uncommon.[9] Electrolyte imbalance was corrected with supplementation and did not affect clinical outcome.

Differences in initial body temperature were also noted between the two cases. The seawater drowning patient presented with early febrile response, likely reflecting aspiration- related inflammatory lung injury, whereas the freshwater drowning patient was hypothermic on admission, consistent with prolonged exposure in a swimming pool environment. Both temperature abnormalities resolved with supportive care and did not influence respiratory recovery.

Although both patients received empirical antibiotics, no microbiologically confirmed drowning-associated pneumonia was identified. Current evidence does not support routine prophylactic antibiotic use following drowning; however, early antimicrobial therapy is frequently initiated in severe cases based on clinical judgment.[13]

One of the most striking features of drowning-associated ARDS is the potential for rapid reversibility. In both patients, PaO_2/FiO_2 ratios improved markedly within hours of initiating lung-protective ventilation, and chest radiographs demonstrated progressive resolution of bilateral alveolar opacities over a few days. This pattern contrasts with ARDS secondary to sepsis or pneumonia, where inflammatory lung injury and fibrosis may prolong mechanical ventilation and delay recovery.

Radiological improvement in drowning-associated ARDS likely reflects the clearance of alveolar fluid, restoration of surfactant function, and recruitment of previously collapsed alveoli once hypoxemia and shunt are corrected.[13] The rapid radiographic resolution observed in these cases aligns with previous reports describing short ventilation durations and favourable pulmonary outcomes in drowning survivors managed promptly.[8]

Several prognostic factors have been consistently associated with outcomes after drowning, including duration of submersion, presence of pre-hospital cardiac arrest, water temperature, and timeliness of rescue and resuscitation. [9] Both patients in this series were young, had relatively short estimated submersion times, and received early advanced care, which likely contributed to favourable neurological and respiratory outcomes.

From a broader anaesthetic and critical care perspective, these cases reinforce the importance of early recognition of drowning-associated ARDS and adherence to standard ARDS management principles. They also highlight that distinctions between freshwater and seawater drowning, while relevant in forensic and post-mortem contexts, have limited utility in guiding acute ventilatory management in living patients.[15]

CONCLUSION

This report describes two patients with drowning-associated ARDS who experienced favourable respiratory recovery following early airway management and lung-protective mechanical ventilation. Given the limited sample size, single-centre nature of the report, and the presence of favourable prognostic factors in both patients, these findings should not be generalised to all drowning-associated ARDS cases. Further prospective studies are needed to clarify the reproducibility of these observations and the optimal ventilatory management of this patient population.

~~In conclusion, Drowning-associated ARDS, regardless of freshwater or seawater aspiration, appears to be a highly reversible form of acute lung injury when managed with early airway management and lung-protective mechanical ventilation, with outcomes driven more by timely adherence to established ARDS principles than by water salinity.~~

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