

A Rare Cause of Pneumoperitoneum after Drowning: Gastric Laceration Following Seawater Submersion

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Abstract

Drowning is a public health problem responsible for substantial morbidity and mortality worldwide, particularly among adolescents. We report the case of a 15-year-old male admitted to our trauma center after a witnessed seawater submersion lasting approximately 10 minutes. On admission, he was obtunded and hypothermic, with respiratory instability and marked abdominal distension. Rapid neurological and respiratory deterioration required emergency orotracheal intubation. Whole-body computed tomography showed bilateral ground-glass opacities consistent with aspiration-related lung injury, peri-esophageal pneumomediastinum, and a large-volume compressive pneumoperitoneum. Emergency exploratory laparotomy identified a 4 cm full-thickness laceration of the lesser gastric curvature, which was repaired primarily with omentoplasty and subhepatic drainage. The post-operative course was favorable, with successful extubation and complete neurological recovery. The mechanism of the gastric injury could not be established with certainty; acute gastric overdistension is the most plausible explanation, since marked abdominal distension was already present before mechanical ventilation was instituted. This case illustrates a rare but life-threatening abdominal complication of seawater drowning and the importance of distinguishing surgical from non-surgical pneumoperitoneum in this setting.

Keywords

Seawater Drowning, Pneumoperitoneum, Gastric Perforation, ARDS, Pneumomediastinum, Intensive Care

1. Introduction

According to the World Health Organization, drowning accounts for approximately 360,000 deaths annually, representing the third leading cause of unintentional injury death worldwide [1]. Seawater drowning induces specific pathophysiological mechanisms: aspiration of hypertonic fluid disrupts alveolar surfactant, promotes osmotic fluid shifts into the alveolar space, and precipitates severe hypoxemia and acidosis [2] [3]. Acute respiratory distress syndrome (ARDS) is the most feared pulmonary complication and remains the primary driver of morbidity and mortality in hospitalized drowning victims [4].

Pneumoperitoneum as a complication of drowning is exceptional and has rarely been described in the literature. Its etiology in this context is complex: it may arise from barotrauma secondary to positive-pressure ventilation, from aerophagia during the drowning event, or—as in our case—from a traumatic visceral injury sustained during submersion [5] [6]. We present this case given its rarity, the diagnostic challenge it posed, and the surgical management required.

2. Case Report

2.1. Clinical Presentation and Initial Management

A 15-year-old male (60 kg, 165 cm) with no relevant past medical history was brought to our emergency department at approximately 13:00, one hour after a witnessed seawater submersion lasting an estimated 10 minutes. He was retrieved from the water conscious but confused. No bystander cardiopulmonary resuscitation and no mouth-to-mouth or bag-mask ventilation were performed at the scene.

On arrival, the Glasgow Coma Scale (GCS) score was 14/15 with obtundation and agitation. Pupils were symmetrical and reactive. Heart rate was 100 bpm and blood pressure 110/70 mmHg, with peripheral mottling. Core temperature was 35.2°C. The abdomen was markedly distended on inspection and palpation.

E-FAST showed no pericardial, pleural or intraperitoneal free fluid and no pneumothorax. Diffuse bilateral B-lines were noted on lung ultrasound, consistent with aspiration-related acute lung injury.

Initial resuscitation was promptly instituted: supplemental oxygen via non-rebreather mask, intravenous access and active external rewarming. Ten minutes after admission, neurological status deteriorated with worsening respiratory distress, prompting emergency orotracheal intubation and mechanical ventilation. A nasogastric tube was placed (atraumatic), and gastric aspiration retrieved sandy sediment, consistent with seawater and sand ingestion.

Initial ventilatory settings: VT = 330 ml, RR = 12 c/min, PEEP = 5, and FiO₂ = 100%

Of note, marked abdominal distension was documented on arrival, before mechanical ventilation was instituted.

Furosemide 20 mg and methylprednisolone 1 mg/kg (60 mg) were administered as adjunctive therapies; their rationale and the current evidence are ad-

dressed in the Discussion.

(Furosemide helps achieve a negative fluid balance to improve oxygenation and reduce ventilation duration, especially in cases of fluid overload. Methylprednisolone is administered based on the timing and severity, aiming to modulate inflammation in early ARDS. Both treatments are individualized measures to optimize respiratory and hemodynamic parameters, not specifically proven therapies for seawater drowning-induced ARDS.)

A consultation with the surgery team was requested, and a CT scan was ordered after stabilization to rule out any other trauma, and the patient went to the OR approximately 30 minutes later. CT was used because the patient could be stabilized sufficiently for imaging; this case does not support routine CT imaging in all drowning victims, but rather imaging directed by clinical findings and the patient's stability.

2.2. Initial Investigations

Arterial blood gas was performed before intubation under high flow oxygen mask and the analysis demonstrated a mixed acidosis (pH 7.19, PaCO₂ 49 mmHg, HCO₃⁻ 18.6 mEq/L) with moderate hyperlactatemia (3.2 mmol/L), hypernatremia (Na⁺ 148 mEq/L), hyperchloremia (Cl⁻ 114 mEq/L), and significant hyperglycemia (26.4 mmol/L). PaO₂ was 157 mmHg under high-flow oxygen. Hemoglobin was 13.67 g/dL.

The PaO₂ was 157 mmHg under FiO₂ 0.8, giving a PaO₂/FiO₂ ratio of approximately 196.

Full blood count showed hemoglobin 13.8 g/dL, hematocrit 43%, white blood cells 6300/mm³, and platelets 262,000/mm³. Biochemistry was notable for preserved renal function (creatinine 8.6 mg/L, urea 0.32 g/L), mild hepatic enzyme elevation (AST 36, ALT 20 IU/L), elevated LDH (391 IU/L), elevated CPK (706 IU/L), mild CRP elevation (4.2 mg/L), and a troponin level of 70.1 ng/L. Electrolytes were Na⁺ 146 mEq/L and K⁺ 3.8 mEq/L.

2.3. Imaging

The CT scan revealed:

- Pulmonary: bilateral diffuse ground-glass opacities with areas of condensation, more pronounced in the upper lobes, consistent with aspiration-induced ARDS.
- Mediastinal: foci of pneumomediastinum in the peri-esophageal region (**Figure 1**).
- Abdominal: large-volume compressive pneumoperitoneum displacing intra-peritoneal organs (**Figure 2**). The distal tip of the nasogastric tube appeared to abut the greater curvature without definitive evidence of parietal discontinuity on initial review.

These findings raised immediate concern for gastric perforation with tension pneumoperitoneum physiology.

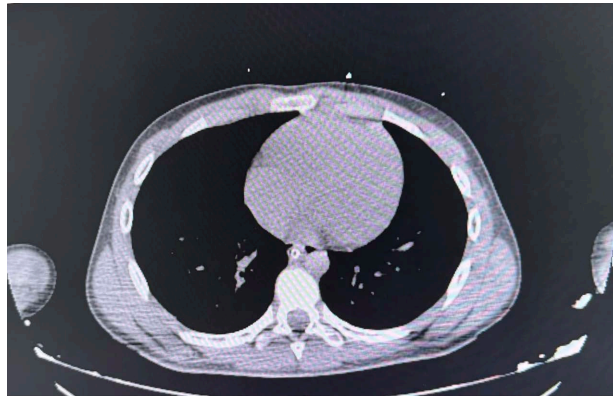


Figure 1. Axial contrast-enhanced chest computed tomography at the level of the heart, showing foci of pneumomediastinum in the peri-oesophageal region, together with bilateral parenchymal opacities consistent with aspiration-related acute lung injury.

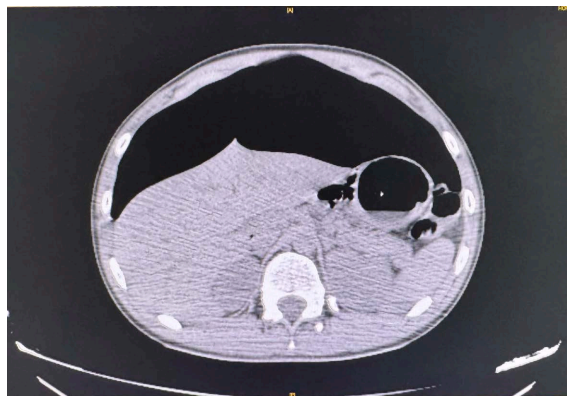


Figure 2. Axial abdominal computed tomography showing a large-volume pneumoperitoneum, with a compressive anterior collection of free intraperitoneal air displacing the liver posteriorly.

2.4. Surgical Management and Anesthesia

The patient was transferred emergently to the OR. General anesthesia was maintained with inhalational and intravenous anesthetic agents, supplemented with opioid analgesia and neuromuscular blockade as required. Mechanical ventilation was adjusted to maintain adequate oxygenation and normocapnia, and fluid therapy was guided by the patient's hemodynamic status. Hemodynamic stability was maintained throughout the procedure, estimated blood loss was 1000cc. One unit of packed red blood cells was transfused, and no vasopressor support was required throughout the procedure. The transfusion was therefore performed in the context of operative blood loss and perioperative hemoglobin decline rather than documented preoperative shock.

A 4 cm full-thickness lesser-curvature gastric laceration was confirmed at laparotomy (**Figure 3**). The gastric laceration was a confirmed operative finding; its precise mechanism could not be established. There was no reported external trauma, fall, collision, or abdominal compression, and the nasogastric tube had been inserted atraumatically. The lesion may therefore have resulted from acute gastric

overdistension or another internal mechanical mechanism, but direct traumatic injury cannot be completely excluded. Instrumentation-related injury is considered less likely because the nasogastric insertion was atraumatic and no other instrumentation-related lesion was identified.

Primary surgical repair was performed, complemented by subhepatic drainage and omentoplasty (**Figure 4**). No other intra-abdominal injuries were identified. The procedure was completed without complications.

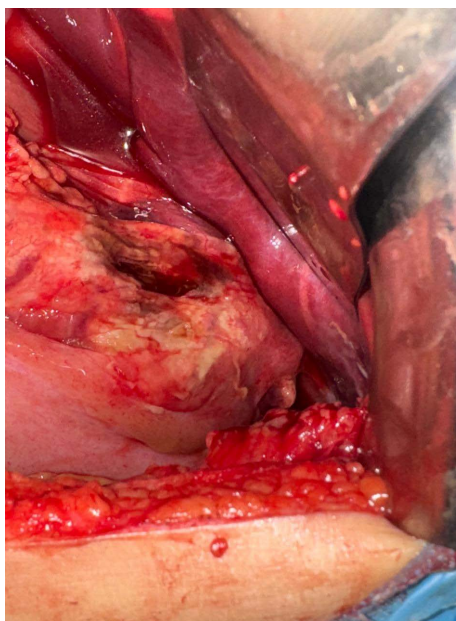


Figure 3. Intraoperative view at exploratory laparotomy showing the 4 cm full-thickness laceration of the lesser curvature of the stomach.

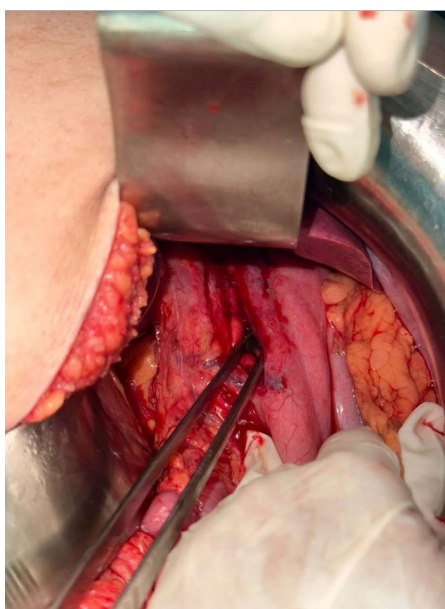


Figure 4. Alternative view at exploratory laparotomy showing the 4 cm full-thickness laceration of the lesser curvature of the stomach.

2.5. Postoperative Course

The patient returned to the intensive care unit intubated and mechanically ventilated. Abdominal distension progressively resolved, respiratory function improved, and mechanical ventilation was gradually weaned. He was successfully extubated and remained neurologically intact. The ICU stay lasted four days. At the end of the documented ICU course, he was fully conscious (GCS 15/15), hemodynamically and respiratory stable, with complete neurological recovery.

3. Discussion

Seawater drowning produces pathophysiological effects distinct from those of freshwater drowning. Aspiration of hypertonic saline (osmolarity ≈ 1000 mOsm/L) draws fluid from the pulmonary vasculature into the alveolar space, disrupts surfactant and causes severe hypoxaemia [2] [7]. The expected profile combines intravascular volume depletion with hypernatraemia. Our patient was hypernatraemic and showed peripheral mottling, but he was normotensive on admission and never required vasopressor support, so overt hypovolaemic shock was not documented. The mixed acidosis, hyperlactataemia and hyperglycaemia at presentation reflect the combined effects of tissue hypoperfusion, anaerobic metabolism and the acute stress response.

ARDS is the principal pulmonary complication of drowning. The CT findings of bilateral ground-glass opacities and upper-lobe consolidation were consistent with early ARDS from aspiration-induced lung injury, as previously described [4] [8], and the $\text{PaO}_2/\text{FiO}_2$ ratio of 196 mmHg under PEEP 5 cm H_2O corresponded to moderate ARDS. The elevations in troponin and creatine kinase are consistent with the hypoxia-related myocardial and skeletal muscle stress documented in severe drowning episodes.

Furosemide and methylprednisolone were administered in this case for suspected drowning-related pulmonary oedema according to the treatment practice used at the time. However, contemporary drowning guidance does not support routine corticosteroid administration and does not recommend routine diuretic therapy for drowning-related pulmonary oedema [3] [9] [10]. Their use should therefore be interpreted as part of the initial historical management of this patient rather than as evidence-based standard therapy.

The most distinctive feature of this case was the large compressive pneumoperitoneum arising from a gastric laceration—a rare and potentially fatal complication of drowning. Three pathophysiological mechanisms are proposed in the literature [5] [6] [11] [12]:

- 1) Barotraumatic pneumoperitoneum (Macklin effect): alveolar rupture under positive-pressure ventilation drives air along broncho vascular fascial sheaths into the mediastinum and sub-diaphragmatic space.
- 2) Aerophagia-related gastric overdistension: panic-driven aerophagia or bag-mask ventilation during resuscitation rapidly inflates the stomach, potentially causing hydrostatic rupture.

3) Traumatic visceral injury: direct mechanical force during submersion causes full-thickness gastric or intestinal laceration.

Three published observations are informative. Petri *et al.* described a 37-year-old diver who sustained a 4 cm rupture of the lesser gastric curvature after a rapid ascent from 37 metres, attributed to expansion of swallowed air; the size and location of the lesion were identical to those in our patient [13]. Ayub *et al.* reported a 14-year-old boy with a complete gastro-oesophageal junction avulsion after near-drowning, in whom CT showed pneumomediastinum, pneumoperitoneum and free fluid concentrated at the oesophageal hiatus [14]. A third report described a 7 cm laceration extending from the abdominal oesophagus to the lesser curvature in a drowning victim after approximately two minutes of bystander cardiopulmonary resuscitation including mouth-to-mouth ventilation [15]. Together these reports show that gastric injury can occur in the absence of any obvious external abdominal trauma.

In the present patient, the operative finding of a 4 cm full-thickness lesser-curvature laceration establishes the injury but not its mechanism. The chronology is nevertheless informative: marked abdominal distension was documented on arrival, before intubation and mechanical ventilation, which argues against ventilator-associated barotrauma as the primary cause. No bystander resuscitation was performed, which distinguishes this case from the CPR-related reports and removes chest-compression and mouth-to-mouth hyperinflation from the differential. Acute gastric overdistension by swallowed seawater and air during the submersion is therefore the most plausible mechanism, although direct traumatic injury cannot be entirely excluded. Instrumentation-related injury is unlikely, given the atraumatic nasogastric insertion and the absence of any other lesion. The peri-oesophageal distribution of the pneumomediastinum may reflect a Macklin-type thoracic air dissection [6] [16], but may equally represent air tracking upward from the gastric injury through the oesophageal hiatus, as illustrated by the case of Ayub *et al.* [14].

The distinction between surgical and non-surgical pneumoperitoneum is clinically important. Mechanical ventilation, cardiopulmonary resuscitation, pneumothorax, and other thoracic mechanisms can produce non-surgical pneumoperitoneum [12]. Conversely, free intraperitoneal air accompanied by marked abdominal distension, visceral displacement/compression, clinical deterioration, or evidence of gastrointestinal injury may indicate a surgical emergency. In this patient, the combination of marked abdominal distension and large-volume compressive pneumoperitoneum on CT prompted emergency laparotomy, which confirmed a gastric laceration. CT therefore contributed to the decision-making process but should be performed selectively according to clinical suspicion and patient stability rather than systematically in every drowning victim with abdominal distension.

This case exemplifies the complexity of managing simultaneously an acute respiratory emergency and an acute abdominal surgical emergency in the ICU set-

ting. Close coordination between the intensivist and surgical team, combined with the Trauma Center infrastructure, was essential to the favorable outcome.

4. Conclusion

We report a rare and life-threatening case of seawater drowning complicated by aspiration-related acute lung injury, pneumomediastinum, and a large-volume pneumoperitoneum caused by a surgically confirmed gastric laceration. The operative lesion was confirmed, whereas its precise mechanism remains uncertain in the absence of external trauma. In drowning patients with marked abdominal distension, clinicians should consider both surgical and non-surgical causes of pneumoperitoneum. CT may be useful when clinical findings raise suspicion for an intra-abdominal complication and the patient's condition permits imaging. Early multidisciplinary management involving intensive care, anesthesia, radiology, and surgery was central to the favorable outcome.

Ethics and Consent

Written informed consent for publication of the case and associated clinical information was obtained from the patient's parent. Institutional ethics review requirements were fulfilled according to the applicable institutional policy, with the case report covered by the institution's approval.

Author Contributions

Saile, A., Mouhaoui, B. and Aggoug, B. were responsible for the intensive care management and anesthesia management of the patient and they conceived the case report. Saile, A. performed the literature search and drafted the manuscript. Aggoug, B. supervised the work and critically revised the manuscript. All authors critically revised the manuscript for important intellectual content, read and approved the final version, and agree to be accountable for all aspects of the work.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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