

Obstructive Atelectasis Caused by an Endobronchial Blood Clot Following Negative-Pressure Airway Injury in Non-Fatal Drowning: A Case Report

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Background: Atelectasis is most commonly obstructive in origin. An endobronchial blood clot is an uncommon but fully reversible cause that is easily overlooked, especially when the preceding hemoptysis is limited to a single episode and has resolved by presentation. Drowning has not previously been described as a precipitant. **Case presentation:** A 26-year-old previously healthy woman choked on water while swimming at night and experienced a single episode of hemoptysis the following day. She subsequently developed progressive dyspnoea and presented with an oxygen saturation of 85–90% on a non-rebreathing mask at 10 L/min. Chest radiography demonstrated right upper-zone opacification accompanied by ipsilateral tracheal deviation and elevation of the right hemidiaphragm — findings that indicate volume loss rather than alveolar filling. High-flow nasal cannula oxygen and chest physiotherapy did not improve the patient, and saturation fell to 81%. Serial radiographs over the following days showed progression to near-complete opacification of the right hemithorax with worsening mediastinal shift. Bronchoscopy with bronchoalveolar lavage revealed and retrieved an organized endobronchial blood clot. Oxygen saturation rose to 100% immediately after the procedure, arterial blood gases normalized, and the radiograph showed full re-expansion with return of the trachea and hemidiaphragm to the midline. **Conclusions:** Forceful inspiratory effort against a closed or obstructed glottis during drowning can disrupt tracheobronchial vessels and produce airway bleeding — negative-pressure airway injury — a mechanism that is distinct from negative-pressure pulmonary edema and that has different radiographic and therapeutic implications. The resulting clot may obstruct a lobar bronchus and cause resorptive atelectasis that is refractory to supplemental

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oxygen and physiotherapy. Radiographic volume loss is the key sign distinguishing this entity from swimming-induced and negative-pressure pulmonary edema, in which lung volume is preserved or increased. Early bronchoscopy is both diagnostic and definitive.

Keywords: Atelectasis; Bronchoscopy; Case report; Drowning; Endobronchial blood clot; Negative-pressure airway injury

1. Introduction

Atelectasis is conventionally divided into obstructive and non-obstructive forms. In the obstructive variety, occlusion of a conducting airway isolates the distal lung; the trapped gas is progressively absorbed into the pulmonary capillary blood, and the unventilated segment collapses. The extent of collapse depends on the level of the occlusion and may involve a single segment, a lobe or an entire lung [1,2]. Because the volume of the affected region falls, the mediastinum, trachea and ipsilateral hemidiaphragm are drawn towards the collapsed lung — a radiographic signature that distinguishes obstructive atelectasis from processes that fill the alveoli without reducing lung volume [3].

Endobronchial obstruction is usually chronic or subacute: bronchogenic tumor, mucus plugging complicating asthma or cystic fibrosis, an aspirated foreign body, or a malpositioned tracheal tube [2,3]. An obstructing blood clot is a less familiar but well-recognized cause, first documented by Wilson in 1929 and subsequently reported after pulmonary infarction, thoracic trauma, pulmonary artery injury, bronchoscopic biopsy and mechanical ventilation [4–6]. Because a clot is radiolucent and the antecedent hemoptysis may be trivial or forgotten, the diagnosis is frequently delayed until the collapse fails to respond to conventional measures [5,7].

Drowning is defined as respiratory impairment from submersion or immersion in liquid [8]. During submersion, the victim initially breath-holds and then makes increasingly forceful inspiratory efforts against a closed or reflexly obstructed glottis [9,10]. The very large

subatmospheric intrathoracic pressures generated in this way are the same forces that, in the peri-anesthetic setting, produce negative-pressure pulmonary edema [11,12]. Less widely appreciated is that these pressures may instead — or additionally — rupture tracheobronchial vessels and cause frank airway bleeding, an entity Bhavani-Shankar and colleagues termed negative-pressure airway injury and explicitly distinguished from negative-pressure pulmonary edema [13].

To our knowledge, obstructive atelectasis caused by an endobronchial clot arising from negative-pressure airway injury during drowning has not previously been reported. We describe such a case, in which a young woman with progressive and oxygen-refractory hypoxemia was found at bronchoscopy to have an obstructing clot whose removal produced immediate and complete resolution. The report is presented in accordance with the CARE guidelines [14].

2. Methodology

2.1 Patient information

A 26-year-old woman presented with progressive shortness of breath. Two nights earlier she had choked on water while swimming at night. On the following day she coughed up blood on a single occasion, after which the hemoptysis did not recur. Dyspnoea began thereafter and worsened steadily. She had no previous history of hemoptysis, smoking or chronic lung disease.

2.2 Clinical findings

On arrival at the referring hospital, general physical examination was unremarkable. Vital signs, however, were abnormal: peripheral

oxygen saturation was only 85–90% despite a non-rebreathing mask delivering 10 L/min.

2.3 Timeline

Summarises the sequence of events.

Table 1. Timeline of clinical events.

Time point	Event	Oxygenation and imaging
Day –2 (night)	Choking episode while swimming at night	—
Day –1	Single episode of fresh haemoptysis; no recurrence thereafter	—
Day 0 (21 Nov 2023)	Progressive dyspnoea; presentation to referring hospital	SpO ₂ 85–90% on non-rebreathing mask 10 L/min; chest radiograph: right upper-zone opacity with ipsilateral tracheal deviation and elevated right hemidiaphragm (Fig. 1A)
Day 0–1	High-flow nasal cannula (flow 50 L/min, FiO ₂ 0.9) and chest physiotherapy; no clinical improvement	Progressive desaturation
Day 1 (22 Nov 2023)	Referred to the secondary hospital with a working diagnosis of atelectasis	SpO ₂ 81% on non-rebreathing mask 10 L/min; chest radiograph: increasing opacification (Fig. 1B); arterial blood gas: hypoxaemia with respiratory alkalosis
Day 3 (24 Nov 2023)	Continued deterioration despite supportive measures	Chest radiograph: near-complete opacification of the right hemithorax with worsening mediastinal shift (Fig. 1C)
Day 3–4	Bronchoscopy with bronchoalveolar lavage; organized endobronchial blood clot identified and retrieved (Fig. 2)	—
Immediately after procedure	Marked symptomatic improvement; hemodynamically stable	SpO ₂ 100% on high-flow nasal cannula; arterial blood gas normalized; chest radiograph: full re-expansion with trachea and hemidiaphragm returned to the midline (Fig. 1D)
Subsequent course	Dyspnoea resolved; no adverse effect of the procedure observed	—

SpO₂, peripheral oxygen saturation; FiO₂, fraction of inspired oxygen.

2.4 Diagnostic assessment

The initial chest radiograph (Fig. 1A) showed opacification of the right upper zone, with tracheal deviation to the right and elevation of the right hemidiaphragm. This combination indicates loss of volume in the right lung and therefore points to an obstructive rather than an alveolar-filling process; the absence of bilateral infiltrates and of an enlarged cardiac silhouette argued against both cardiogenic and non-cardiogenic pulmonary edema.

Serial radiographs documented progression rather than resolution: the opacity extended over the following days until almost the whole right hemithorax was opaque, with increasing mediastinal displacement towards the affected side (Fig. 1B, 1C). Arterial blood gas analysis demonstrated hypoxemia with respiratory alkalosis, a pattern consistent with a large intrapulmonary shunt and compensatory hyperventilation.

Laboratory investigations, including a full blood count, showed no leucocytosis or other evidence of infection.

The working diagnosis was resorptive atelectasis of the right lung. At the time, alternative obstructive causes considered were infection,

mucus plugging, asthma, and cystic fibrosis. Given the radiographic volume loss, the absence of any evidence of infection, the failure to respond to oxygen and physiotherapy, and the antecedent haemoptysis, an obstructing endobronchial lesion was regarded as the most likely explanation, and bronchoscopy with bronchoalveolar lavage was undertaken.

2.5 Therapeutic intervention

Bronchoscopy with bronchoalveolar lavage was performed. The procedure identified an organized, dark endobronchial blood clot within the right-sided airway (Fig. 2A) and retrieved it; the specimen is shown in Fig. 2B. The procedure was completed without adverse effects.

2.6 Follow-up and outcomes

The response was immediate. Vital signs stabilized, oxygen saturation rose to 100% on high-flow nasal cannula (flow 50 L/min, FiO₂ 0.9), and repeat arterial blood gas analysis showed a normal pH, PaCO₂, and PaO₂. The post-procedural chest radiograph showed complete re-expansion of the right lung, with the trachea and right hemidiaphragm returned to normal positions (Fig. 1D). The patient no longer had dyspnoea.

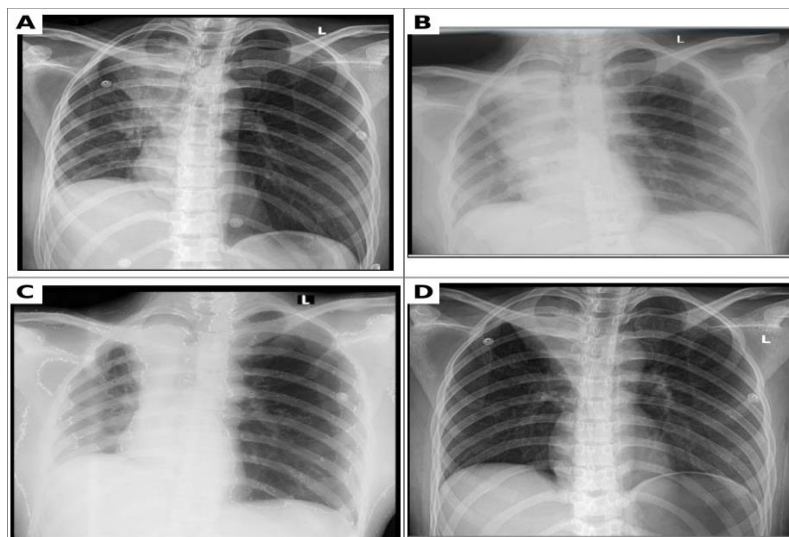


Figure 1. Serial chest radiographs. (A) At presentation (21 November 2023): opacification of the right upper zone with tracheal deviation to the right and elevation of the right hemidiaphragm, indicating volume loss. (B) 22 November 2023 and (C) 24 November 2023: progressive opacification of the right hemithorax with increasing ipsilateral mediastinal shift despite high-flow oxygen and chest physiotherapy.

(D) After bronchoscopic removal of the endobronchial clot: complete re-expansion of the right lung with the trachea and hemidiaphragm restored to the midline.

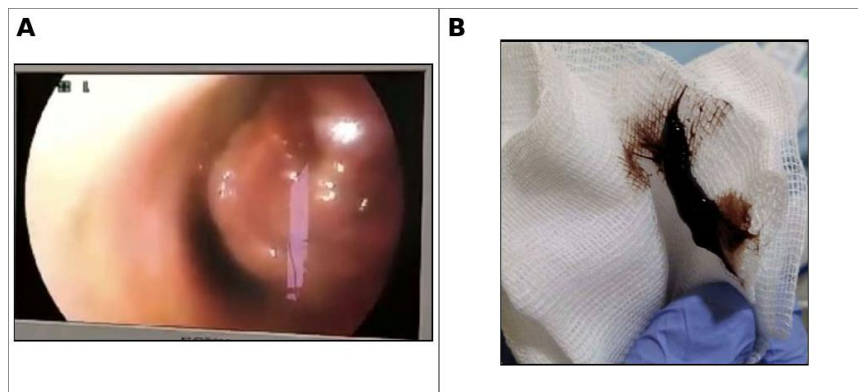


Figure 2. Bronchoscopic findings. (A) Endoscopic view of the right-sided airway at bronchoscopy. (B) The organised endobronchial blood clot retrieved during the procedure.

. Discussions

This case illustrates a mechanistic sequence that, to our knowledge, has not previously been assembled in a single patient: a drowning event generating large subatmospheric airway pressures, rupture of tracheobronchial vessels with airway bleeding, formation of an obstructing endobronchial clot, and progressive resorptive atelectasis that was refractory to oxygen therapy but resolved completely within minutes of bronchoscopic clot removal. Each link in this chain is individually documented in the literature; their combination explains a presentation that might otherwise be misattributed to immersion-related pulmonary oedema or to aspiration pneumonitis.

3.1 From drowning to endobronchial clot

During submersion the victim initially holds the breath. As hypercapnia and hypoxaemia progress, the breakpoint is reached and involuntary inspiratory efforts begin against a glottis that is closed voluntarily, obstructed by laryngospasm, or occluded by aspirated liquid [8–10]. Bronchospasm provoked by fluid entering the lower airways adds a further site of obstruction [10]. The resulting Mueller-type manoeuvre can generate intrathoracic pressures as low as -50 to -100 cmH₂O, and it is these pressures, rather than the aspirated water itself, that mediate much of the early pulmonary injury [11,12].

Two distinct consequences may follow. The better known is negative-pressure pulmonary oedema, in which the steep transvascular pressure gradient drives protein-poor fluid from the pulmonary capillaries into the interstitial and alveolar spaces [11,12,15]. The second, described by Bhavani-Shankar and colleagues in a patient who developed haemoptysis rather than frothy oedema fluid after severe laryngospasm, is mechanical disruption of the tracheobronchial microvasculature with bleeding directly into the airway lumen [13]. The same investigators emphasised that this negative-pressure airway injury should be distinguished from negative-pressure pulmonary oedema, a distinction that is not merely semantic: oedema fluid is cleared by intact alveolar transport mechanisms and responds to positive-pressure ventilation, whereas intraluminal blood organises into a clot that positive pressure cannot remove and that may in fact be driven more distally [15,16].

Once formed, an endobronchial clot behaves as any other intraluminal foreign body. Gas distal to the occlusion is absorbed into capillary blood, the segment collapses, and the resulting shunt produces hypoxaemia that is characteristically refractory to increases in inspired oxygen — precisely the pattern observed in our patient, in whom escalation from a non-rebreathing mask to high-flow nasal cannula at an inspired oxygen fraction of 0.9 failed to improve saturation. Persistent lobar collapse after an episode of

airway bleeding should raise immediate suspicion of an obstructing clot [5,6].

3.2 Why the mechanism is not pulmonary barotrauma

It is worth stating explicitly that the injury described here is not barotrauma. Pulmonary barotrauma results from the expansion of trapped intrapulmonary gas as ambient pressure falls during ascent, in accordance with Boyle's law, and is a hazard of compressed-gas diving; its manifestations are alveolar rupture with pneumothorax, pneumomediastinum, subcutaneous emphysema and arterial gas embolism [17,18]. Our patient was swimming at the surface, was never exposed to an increased ambient pressure and had none of these findings. Conflating barotrauma with negative-pressure injury is a common error, but the two differ in their physics, their radiographic manifestations and their management, and only the latter is applicable to surface drowning.

3.3 Differential diagnosis and the value of radiographic volume loss

Three immersion-related diagnoses required consideration. Swimming-induced pulmonary oedema is an increasingly recognised form of non-cardiogenic oedema affecting healthy swimmers, and its features overlap strikingly with this presentation: acute dyspnoea and cough with haemoptysis in up to two-thirds of cases, hypoxaemia, and onset during or shortly after swimming in cold open water, with a recognised predilection for women [19–21]. Negative-pressure pulmonary oedema shares a similar clinical picture but follows obstructed inspiration [11,12]. Aspiration pneumonitis after submersion is a third possibility [9,10].

The chest radiograph discriminated between these possibilities more effectively than any other single investigation. Both forms of pulmonary oedema fill the alveoli without reducing lung volume; the expected pattern is bilateral perihilar or diffuse alveolar opacification with a normally positioned mediastinum and diaphragm, and the process typically resolves within 24 to 48 hours with supportive care [19,21,22]. Our patient

instead showed unilateral opacification accompanied by ipsilateral tracheal deviation and hemidiaphragmatic elevation — unequivocal signs of volume loss — and the abnormality progressed rather than resolved over four days. Volume loss on the radiograph therefore constitutes the single most useful bedside discriminator between an obstructing endobronchial lesion and immersion-related pulmonary oedema, and its recognition should prompt early bronchoscopy rather than continued escalation of oxygen therapy.

The absence of fever, leucocytosis and a raised inflammatory response argued against aspiration pneumonitis and infective consolidation, while the patient's age, the lack of any preceding respiratory symptoms and the normal cardiac silhouette made bronchogenic malignancy and cardiogenic oedema improbable. The definitive discriminator, however, was direct visualisation: bronchoscopy converted a probabilistic differential diagnosis into a certain one and simultaneously provided the cure.

3.4 Management of an obstructing endobronchial clot

When a patient is stable and oxygenating adequately, a small endobronchial clot may be managed expectantly, since spontaneous lysis and expectoration commonly occur within a few days [5]. Where hypoxaemia is progressive or refractory, as here, active removal is indicated. Flexible bronchoscopy with saline lavage, suction and forceps extraction is the appropriate first-line approach and was sufficient in the present case [5,6,23]. When these measures fail — typically with large, firm or organised clots — the reported alternatives include bronchoscopic cryoextraction, which freezes the water content of the clot and permits its removal en bloc and has been proposed as the technique of choice for massive endobronchial clot [24,25]; rigid bronchoscopy with optical forceps, which affords superior airway control and suction capacity [23,26]; endobronchial instillation of a fibrinolytic agent [27]; and, in intractable cases with a persistent bleeding source, bronchial artery embolisation or surgical resection [23].

Two practical points deserve emphasis. First, chest physiotherapy and high-flow oxygen — both appropriate for mucus-related collapse — are ineffective against an organised clot and their continued use delays definitive treatment; in this patient they were pursued for several days while the collapse progressed. Second, because a clot is radiolucent, its presence cannot be confirmed or excluded radiographically; computed tomography may show an intraluminal filling defect and abrupt bronchial cut-off, but bronchoscopy remains the reference standard and should not be deferred while further imaging is obtained.

4. Conclusion

Obstructive atelectasis caused by an endobronchial blood clot is a rare but readily reversible complication of non-fatal drowning. The mechanism is negative-pressure airway injury — disruption of tracheobronchial vessels by the

large subatmospheric pressures generated during inspiratory effort against an obstructed glottis — and it is distinct from both negative-pressure pulmonary oedema and pulmonary barotrauma. Radiographic volume loss distinguishes it from immersion-related pulmonary oedema, and hypoxaemia refractory to supplemental oxygen should prompt early bronchoscopy, which is simultaneously diagnostic and curative.

5. Ethics approval and consent to participate

All procedures performed were in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. The requirement for individual informed consent was waived by the Ethics Committee / Institutional Review Board of the Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.

6. References

- Peroni DG, Boner AL. Atelectasis: mechanisms, diagnosis and management. *Paediatr Respir Rev*. 2000;1(3):274–8.
- Grott K, Chauhan S, Dunlap JD, Bollu PC. Atelectasis. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2024.
- Woodring JH, Reed JC. Types and mechanisms of pulmonary atelectasis. *J Thorac Imaging*. 1996;11(2):92–108.
- Wilson LB. A case of bronchial obstruction by blood clot with massive collapse of the lung. *Am J Med Sci*. 1929;178:496–500.
- Arney KL, Judson MA, Sahn SA. Airway obstruction arising from blood clot: three reports and a review of the literature. *Chest*. 1999;115(1):293–6.
- Kim SH, Kim JW, Lee JH, Park HK, Choi SH. Management of a case of endobronchial blood clot in the postoperative period. *Ann Card Anaesth*. 2010;13(2):161–3.
- Manchanda S, Kalra N, Sharma A. Organized blood clot masquerading as endobronchial tumor: a review of management and recent advances. *Respir Med Case Rep*. 2018;25:57–60.
- Idris AH, Berg RA, Bierens J, Bossaert L, Branche CM, Gabrielli A, et al. Recommended guidelines for uniform reporting of data from drowning: the “Utstein style”. *Circulation*. 2003;108(20):2565–74.
- Szpilman D, Bierens JJ, Handley AJ, Orlowski JP. Drowning. *N Engl J Med*. 2012;366(22):2102–10.
- Carter E, Sinclair R. Drowning. *Contin Educ Anaesth Crit Care Pain*. 2011;11(6):210–3.
- Bhattacharya M, Kallet RH, Ware LB, Matthay MA. Negative-pressure pulmonary edema. *Chest*. 2016;150(4):927–33.
- Bhaskar B, Fraser JF. Negative pressure pulmonary edema revisited: pathophysiology and review of management. *Saudi J Anaesth*. 2011;5(3):308–13.
- Bhavani-Shankar K, Hart NS, Mushlin PS. Negative pressure induced airway and pulmonary injury. *Can J Anaesth*. 1997;44(1):78–81.

- Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D; CARE Group. The CARE guidelines: consensus-based clinical case report guideline development. *J Clin Epidemiol*. 2014;67(1):46–51.
- Koch SM, Abramson DC, Ford M, Peterson D, Katz J. Bronchoscopic findings in post-obstructive pulmonary oedema. *Can J Anaesth*. 1996;43(1):73–6.
- Kim HJ, Kim JS, Kim KH, Lee JH. Negative pressure pulmonary hemorrhage after laryngospasm during the postoperative period. *Acute Crit Care*. 2018;33(3):191–5.
- Dawn KL, Sharma T. Pulmonary barotrauma. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
- Casadesús JM, Aguirre F, Carrera A, Boadas-Vaello P, Serrando MT, Reina F. Diagnosis of arterial gas embolism in SCUBA diving: modification suggestion of autopsy techniques and experience in eight cases. *Forensic Sci Med Pathol*. 2018;14(1):18–25.
- Grünig H, Nikolaidis PT, Moon RE, Knechtle B. Diagnosis of swimming induced pulmonary edema — a review. *Front Physiol*. 2017;8:652.
- Hårdstedt M, Seiler C, Kristiansson L, Lundeqvist D, Klingberg C, Braman Eriksson A. Swimming-induced pulmonary edema: diagnostic criteria validated by lung ultrasound. *Chest*. 2020;158(4):1586–95.
- Miller CC, Calder-Becker K, Modave F. Swimming-induced pulmonary edema in triathletes. *Am J Emerg Med*. 2010;28(8):941–6.
- Peachner DF, Martina SD, Otteni CE, Wester TE, Potter JF, Moon RE. Immersion pulmonary edema and comorbidities: case series and updated review. *Med Sci Sports Exerc*. 2015;47(6):1128–34.
- Sehgal IS, Dhooria S, Agarwal R, Behera D. Use of a flexible cryoprobe for removal of tracheobronchial blood clots. *Respir Care*. 2015;60(7):e128–31.
- Lee H, Leem CS, Lee JH, Lee CT, Cho YJ. Successful removal of endobronchial blood clots using bronchoscopic cryotherapy at bedside in the intensive care unit. *Tuberc Respir Dis (Seoul)*. 2014;77(4):193–6.
- Bhat A, Vaidya M, Modi M, Sethi S. Bronchoscopic cryoextraction: a novel approach for the removal of massive endobronchial blood clots causing acute airway obstruction. *Southwest J Pulm Crit Care*. 2013;7(3):173–7.
- Ernst A, Feller-Kopman D, Becker HD, Mehta AC. Central airway obstruction. *Am J Respir Crit Care Med*. 2004;169(12):1278–97.
- Tsukamoto T, Sasaki H, Nakamura H. Treatment of hemoptysis patients by thrombin and fibrinogen–thrombin infusion therapy using a fiberoptic bronchoscope. *Chest*. 1989;96(3):473–6.

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8. Author Declaration

The requirement for individual consent for publication was waived by the institutional review board. No personally identifiable information is presented: the patient is not named, no identifying features appear in any image, and the radiographic and bronchoscopic images reproduced here contain no data by which the patient could be recognised.