Negative pressure pulmonary oedema with haemorrhage after 5-minute avalanche burial

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In March, 2016, three ski freeriders were surprised by an avalanche at 2500 m above sea level. Two escaped, but a 31-year-old man with no medical history of note was buried under snow. His hand was left poking out of the snow so he was found easily and excavated after about 3–5 min. According to his fellow skiers, his head had been buried about 0·5 m. The patient reported never having lost consciousness and remembering his friends excavating him, but being unable to breathe because compacted snow had blocked his airways. When his face was uncovered he was cyanotic but conscious and able to clear his airway himself.

The helicopter emergency team arrived 12 min after the accident. We found a fully oriented patient with both peripheral and central cyanosis, tachypnoea, and frank haemoptysis. He did not report having hit any rocks or objects during his burial under the snow. An Advanced Trauma Life Support body check showed no other injuries, and he reported no pain on thoracic compression. Oral inspection showed no bleeding wounds. Pulmonary auscultation revealed bilateral rales and crackles. We immediately started 100% oxygen flow through a mask, and took the patient by helicopter to the emergency department of a local alpine hospital in Samedan, Switzerland (1720 m above sea level).

On admission, his oxygen saturation was 93% on 100% oxygen via the mask. Heart rate and blood pressure were normal. A CT scan showed no signs of trauma but impressive bilateral diffuse pulmonary oedema (figure). Routine laboratory results, including haemoglobin, B-type natriuretic peptide, and troponin concentrations were normal.

Because we suspected negative pressure pulmonary oedema with pulmonary haemorrhage, the patient was transferred to the intensive care unit for intermittent therapy with non-invasive ventilation support. His haemoptysis stopped on day 1 after admission, when haemoglobin had dropped from 168 g/L on admission to 135 g/L. The patient recovered rapidly and was discharged home on day 4. Pulmonary function tests and an echocardiography 4 weeks after the accident were normal. 5 weeks later, he successfully climbed Chimborazo, a 6267 m high Ecuadorian volcano.

Negative pressure pulmonary oedema, also described as postobstructive pulmonary oedema, is well known among anaesthesiologists, often reported as a complication of laryngospasm after extubation. Other causes include strangulation, epiglottitis, foreign body aspiration, hypothyroidism, inspissated tracheal secretions, hiccups, croup, thyroid goitre, upper airway and mediastinal tumours, obesity, Ludwig angina, obstructive sleep apnoea, and biting of the endotracheal tube or laryngeal mask.[1] Oswalt described negative pressure pulmonary oedema in adult patients as a clinical complication of acute airway obstruction in 1977.[2] In 1999, Schwartz described a rare haemorrhagic form, which was assumed to be a complicating disruption of pulmonary capillaries.[3]

Forceful inspiration against glottic obstruction can result in intrathoracic pressures as low as –13 kPa, leading to an increased venous return to the right heart and a concomitant decrease in the flow from the left heart due to increased afterload. Both the increased pulmonary blood volume and the raised pulmonary venous pressure cause increased hydrostatic pressures and thus oedema. Additionally, the negative intrapleural pressure is transmitted to the lung tissue, contributing to a hydrostatic gradient that favours transudation of fluid from the pulmonary capillaries into the alveolar space.[4]

This increased negative pressure explains why healthy young men, who are able to generate great negative intrapleural pressures, have a higher incidence of negative pressure pulmonary oedema.[4] Other risk factors include short neck, acromegaly, and vocal cord paralysis.[4] Additionally, hypoxia can increase pulmonary vascular resistance and pulmonary capillary pressure, precipitating a hyperadrenergic state which is thought to redistribute blood from the systemic veins to the pulmonary circuit and increase pulmonary vascular resistance.[4] Although the precise cause of bleeding is uncertain, physical disruption of the alveolocapillary membrane might play a part.[3]

Negative pressure pulmonary oedema requires immediate oxygen supply as a first therapeutic measure. Negative airway pressure, the reason for the pathology, is mitigated by positive pressure through non-invasive

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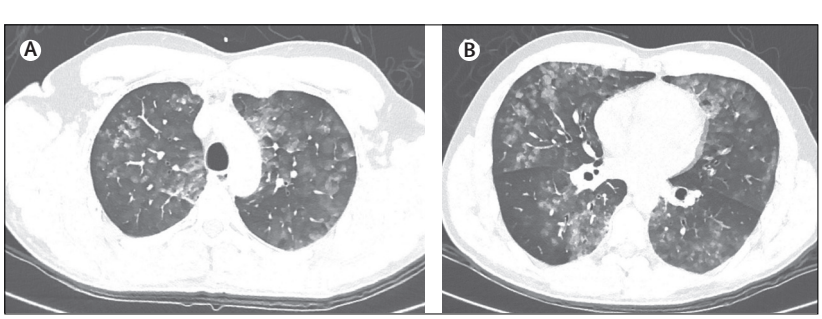

Figure: **Negative pressure pulmonary oedema with haemorrhage**
Axial CT scans done on admission to the emergency department show extensive macular alveolar infiltrations throughout both lungs. (A) Apical infiltrations. (B) Basal infiltrations.

ventilation. The therapeutic effect of diuretics in negative pressure pulmonary oedema with haemorrhage remains unclear. In the absence of persistent pathogenic stress, rapid recovery within a few hours can be expected; intubation and mechanical ventilation are only occasionally needed.[4] Severe hypoxaemia caused by the pulmonary shunting can necessitate protective ventilation (6 mL/kg tidal volume).[1]

Negative pressure pulmonary oedema with haemorrhage is a diagnosis by exclusion. In our case, aspiration, pulmonary contusion, and neurogenic and cardiac pulmonary oedema were differential diagnoses, but excluded due to the presence of haemorrhage, the rapid recovery, the absence of airway trauma or thoracic trauma, and the conscious patient. In patients with full recall of obstructed airways, prognosis is excellent if diagnosis and treatment are timely.[5]

Contributors
PG and JR cared for the patient. All authors wrote the report. Written consent to publication was obtained.

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Case Report Comment
The last gasp

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An avalanche victim who keeps his wits desperately scrambles in the tumbling snow attempting to swim to the surface of the tumultuous white cascade. He fails.

When someone is encased within an icy tomb, survival depends on having a patent airway adjacent to an air pocket, because asphyxia is the leading cause of death from snow avalanche.[1] In *The Lancet*, Paolo Glisenti and colleagues[2] describe a patient buried only for 5 min, and introduce the finding of harmful negative pressure pulmonary oedema with pulmonary haemorrhage. A man who remarkably remained awake throughout the episode forcefully inhaled unsuccessfully against an occluded mouth and nose. We will never know precisely the duration or inspiratory force exerted by the survivor, but we can easily imagine that it was desperate and convulsive, and know that it was sufficient to acutely damage his lungs.

Given the number of avalanche victims worldwide and paucity of similar reports, including from autopsies, there was something unique about this episode. My hypothesis is that the exquisite timing of the rescue showcased this pathophysiological phenomenon. By enabling the victim to survive and manifest the sequelae of markedly negative intrathoracic pressure (namely, increased central venous return and transcapillary pressure gradient, pulmonary arterial hypertension, and pulmonary capillary leak), his rescuers allowed him to reach a stage of illness that most avalanche victims do not.[3,4] His survival allowed time for pulmonary fluid, including blood, to redistribute and cause oedema.

This case offers guidance for survivors and rescuers. The suggestion to someone buried in an avalanche might be to make every effort to control emotions and try to breathe slowly, if it can be done without losing consciousness. This is easy in theory, but perhaps impractical in reality. A suggestion for rescuers is that negative pressure pulmonary oedema should be anticipated in anyone who survives burial in snow, or any other substance (eg, mud, sand, grain) that occludes the airway. In the field, supplemental oxygen should therefore always be administered, and in-hospital positive pressure ventilation considered early. For the hospital clinician, the duration between the sudden increase in intrathoracic pressure and onset of clinical negative pressure pulmonary oedema is not known, so cautious observation for a few hours seems prudent. It is left to the doctor's discretion to determine if a predisposing cause, such as cardiomyopathy, should be sought. If the avalanche occurred soon after arrival at high altitude and acclimatisation is not yet complete, one must be vigilant for high altitude pulmonary oedema, which is attributed in part to raised pulmonary arterial pressure.

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