

CASE REPORT

Hyper-acute neurogenic pulmonary oedema in a prehospital setting: a case report

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(Lucija Galič)**Abstract**

Introduction: Neurogenic pulmonary oedema (NPO) is a form of pulmonary oedema which can develop after an acute central nervous system (CNS) lesion, most often a subarachnoid haemorrhage (SAH). Its pathogenesis is associated with a surge of catecholamines which are released into the systemic circulation following an abrupt increase in intracranial pressure or a strategic CNS lesion. NPO typically presents with dyspnoea, tachypnoea, hypoxia, pink frothy sputum, bilateral crackles on auscultation, and bilateral infiltrates on chest X-ray in a few hours or days following the event.

Case report: The following case report describes a case of hyper-acute NPO following a SAH, which presented as acute respiratory failure in the minutes after the event and required fast and aggressive treatment in the prehospital setting.

Conclusion: The management of NPO is primarily focused on prompt diagnosis and treatment of a CNS event and supportive care.

Keywords

Pulmonary oedema; Prehospital emergency care; Neurology

1. Introduction

Neurogenic pulmonary oedema (NPO) is defined as hypoxic respiratory failure shortly after a catastrophic central nervous system (CNS) event or strategic CNS lesion [1]. It follows a subarachnoid haemorrhage (SAH) in up to 25% of cases [2], and is also seen with intracerebral and subdural haemorrhage, stroke in the insula or brainstem, traumatic brain or spinal cord injury, epilepsy, and meningitis [1, 3–5]. NPO usually presents with dyspnoea, tachypnoea, hypoxia, pink frothy sputum, bilateral crackles on auscultation, and bilateral infiltrates on chest X-ray, and usually resolves within 24 to 72 hours with appropriate management [1, 3].

The pathophysiology of NPO is poorly understood, but there are several proposed mechanisms [1, 3]. An abrupt increase in intracranial pressure or a strategic CNS lesion (such as in the insula or dorsolateral medulla) can cause a sudden systemic surge of catecholamines [3–5]. This can lead to reversible myocyte injury and segmental wall motion abnormalities, impaired heart pumping function, elevated cardiac enzymes, ECG changes, and arrhythmias, albeit with no clinical evidence of an acute myocardial infarction [1, 3, 6–8]. A catecholamine-induced abrupt increase in systemic arterial pressure increases the left ventricle afterload, which can lead to pulmonary congestion and hydrostatic transudative pulmonary oedema [1, 3]. Increased pulmonary capillary pressure can damage the pulmonary endothelium, resulting in vascular leak and exudative pulmonary oedema [1, 3]. A CNS injury is also related to a systemic inflammatory response, with release

of pro-inflammatory cytokines that can further aggravate lung injury and oedema [1, 2, 7]. Autonomic nervous system activation in CNS events is related to atrial tachyarrhythmias such as atrial fibrillation (AF), which can impair the cardiac pumping function and further contribute to the development of NPO [8].

NPO can lead to acute cardiopulmonary failure with global hypoperfusion and hypoxia leading to severe secondary ischemic brain damage or death [6, 7, 9, 10]. For this reason, NPO usually warrants emergent intensive care unit (ICU) management of pulmonary oedema and treatment of the underlying CNS event [1, 7].

In this article we report a case of hyper-acute NPO due to an aneurysmal subarachnoid haemorrhage, which required prompt and aggressive treatment beginning in a prehospital setting.

2. Case report

The patient was a 59-year old nurse with a history of arterial hypertension and Hashimoto's thyroiditis. She had undergone a cardiac catheter ablation due to atypical atrioventricular nodal re-entrant tachycardia three years before, and was taking 4 mg of perindopril daily.

The patient developed a headache with general malaise, tachycardia and palpitations while in her workplace, and a few hours later she suddenly lost consciousness. The prehospital emergency medicine team (PEMT), consisting of an emergency physician and three paramedics, responded to the

TABLE 1. The patient’s vital signs at time of arrival of PEMT, immediately after intubation, and on arrival at hospital.

	0 min	4 min	43 min
	On arrival	After RSI	In hospital
Respiratory rate (breaths per minute)	5	15	10
Heart rate (beats per minute)	180	170	115
Blood pressure (mmHg)	Unmeasurable	132/76	138/91
Oxygen saturation (%)	Unmeasurable	82	89
Consciousness	Coma	Sedated	Sedated
End tidal CO ₂ (mmHg)	/	26 (hyperventilated with BVM)	32

PEMT, prehospital emergency medicine team; RSI, rapid sequence intubation; BVM, bag valve mask.

emergency call and arrived at the scene in eight minutes. Upon arrival, the patient was unresponsive to any stimuli, and had constricted, unreactive pupils. There was pink frothy sputum in her mouth, and diffuse bilateral crackles on lung auscultation. The patient was breathing agonally with a rate of 5 breaths per minute, and had a palpable pulse over the carotid artery. Arterial blood pressure and oxygen saturation were unmeasurable. The 3-lead ECG monitor showed AF with a rate of 180 beats per minute.

The PEMT performed a rapid sequence intubation (RSI) with fentanyl 100 mcg IV, etomidate 20 mg IV and suxamethonium 100 mg IV, and the patient was initially ventilated in IPPV mode with 100% oxygen with a frequency of 10 breaths per minute and a minute ventilation of 6 litres per minute. Because of the clinical signs of pulmonary oedema the patient was administered furosemide 40 mg IV. She received minimal fluid resuscitation of approximately 250 mL of crystalloid before admission to hospital. At admission her haemoglobin (Hb) was 155 g/L. The patient’s vital signs are shown in Table 1.

Once stable, the patient was transported to a Division of Neurology 43 minutes after the first contact with the PEMT. On admission, computed tomography (CT) of the head showed an extensive subarachnoid haemorrhage at the base of the brain, and CT angiography of the cerebral arteries showed a ruptured aneurysm of the anterior communicating artery (Fig. 1). A chest X-ray showed signs of moderate to severe pulmonary oedema with bilateral alveolar and interstitial infiltrates (Fig. 2).

The patient underwent immediate endovascular coiling of the aneurysm (Fig. 3) and was transferred to neurological ICU, where she remained under treatment for 35 days. The patient was ventilated with IPPV mode, PEEP 8 cm H₂O, a frequency of 8 breaths per minute and tidal volume of 560 mL. Oxygen supplement was reduced from 100% to 30% the second day. Diuresis was spontaneous and transiently increased due to furosemide and mannitol given for pulmonary and cerebral oedema. Fluid balance was maintained. With supportive care, the pulmonary oedema had regressed almost completely on day 2 after the event (Fig. 2). In the first 14 days she required high doses of vasoactive support with noradrenaline and argipressin, and inotropic support with dobutamine. An ECG showed sinus rhythm with frequent paroxysmal AF. She had a cardiac index (CI) of 3.5 L/min/m² and elevated levels of troponin I up to 14,000 ng/L (positive at >40 ng/L), which be-

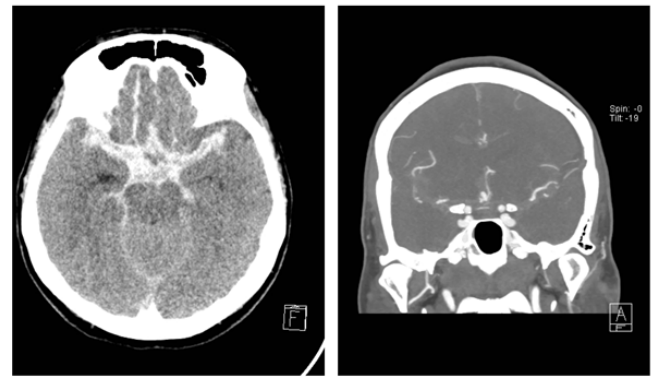


FIGURE 1. Head CT (left) and CTA (right) at time of admission to a Division of Neurology (CT—Computed Tomography, CTA—CT Angiography).

gan to decrease on the second day. Echocardiography showed no signs of segmental wall motion abnormalities. After three weeks she was gradually taken off analgo-sedation; she was breathing spontaneously with a percutaneous tracheostomy, and began to open her eyes spontaneously and to verbal stimuli, without any other meaningful contact.

3. Discussion

This case demonstrates that, unlike the usual presentation in hours to days, NPO can develop within minutes and can cause hyper-acute respiratory failure. On-field differential diagnoses included both cardiogenic and neurogenic causes of the patient’s sudden loss of consciousness, so the prehospital treatment was symptomatic. Nevertheless, respiratory failure was successfully managed by early RSI and on-field mechanical ventilation in an attempt to reverse the pulmonary oedema and hypoxia.

The patient received intravenous loop diuretic furosemide as a symptomatic treatment of pulmonary oedema. Diuretic therapy is often used in SAH patients with pulmonary oedema, even though the oedema is not of cardiac origin [7]. However acute impairment of cardiac function was probably present in our patient, as demonstrated subsequently by reduced CI, elevated troponin levels and the need for inotropic and vasoactive support in hospital. Additionally, the patient was in AF which could be triggered by the SAH and could contribute to the development of NPO. Therefore our case could most

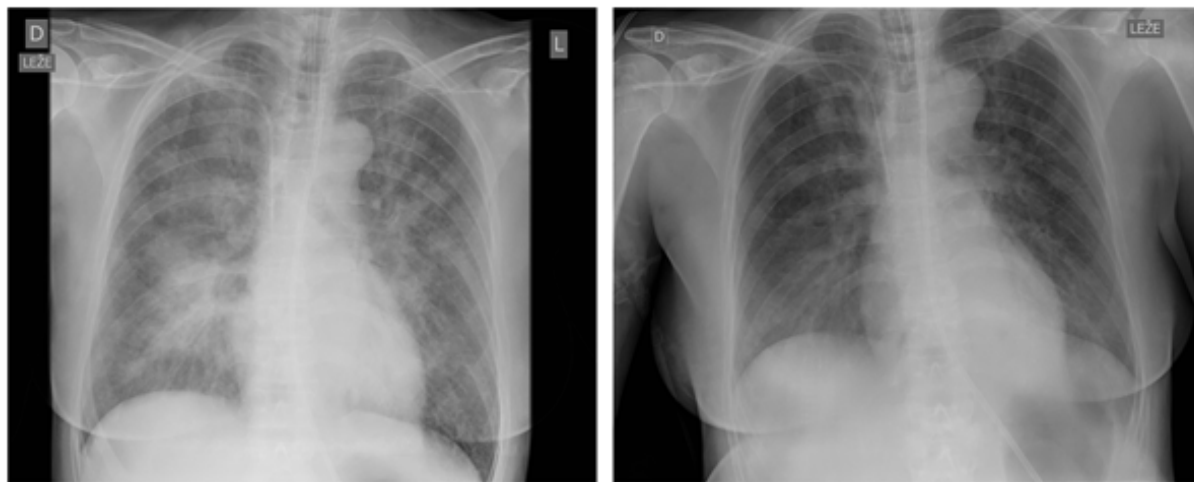


FIGURE 2. Chest X-ray at time of admission to a Division of Neurology (left) and the next day (right).



FIGURE 3. DSA after endovascular coiling of the aneurysm (DSA—Digital Subtraction Angiography).

comprehensively be described as a cardiogenic neurogenic oedema, as it was a consequence of an unfortunate cascade of events, beginning with a neurological condition which had a detrimental effect on the patient's cardiac function. It should be noted that some authors suggest urgent α -(phentolamine) and β -adrenergic (propranolol) blockade, as this could decrease catecholamine-related myocardial injury [7]. On the other hand, it is interesting that β -stimulating catecholamines, especially dobutamine, also improve cardiac function in NPO [7, 10].

It should also be noted that diuretics can aggravate hypovolemia and add a hypovolemic contribution to the cardiogenic shock. In an emergency setting, therefore, it is a challenge to estimate the need for fluid resuscitation. Due to the unknown volume status and clinical signs of pulmonary oedema, the use of emergency diuretics was a reasonable choice. Since the

patient's Hb was slightly elevated upon arrival at hospital, it points towards a justified use of a diuretic, since increased Hb matches increased extravascular lung water content. In such cases the use of point-of-care ultrasonography could help towards an optimal fluid resuscitation in the field.

The ratio of the dead space ventilation (V_d) to tidal volume (V_t) (V_d/V_t) by expired CO_2 (volumetric capnometry) was not available prior to admission to the ICU. This would contribute to a more precise estimation of ventilation-perfusion disturbances. The severe ventilation-perfusion disturbance was demonstrated by severe hypoxemia and acted upon in the field.

4. Conclusions

In some cases, NPO can take a hyper-acute course, leading to respiratory failure and secondary ischemic brain injury. This case encourages emergency physicians to consider NPO in patients with diminished consciousness and respiratory failure without an obvious cardiac or pulmonary condition. Early analgesia, sedation, relaxation, endotracheal intubation and proper ventilation are crucial in treating any pulmonary oedema in a prehospital setting, but prompt diagnosis and treatment of a CNS event can further improve the patient's management and outcome.

AUTHOR CONTRIBUTIONS

LG and VH conceived of the presented manuscript. LG wrote the manuscript with support from VH and AF. AF contributed mostly with the hospital course of diagnosis and treatment. All authors contributed to the final version of the manuscript. VH supervised the project.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Research was approved by National Medical Ethics Committee of Republic of Slovenia. Reference number of the approval: 0120-533/2020-7.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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