

## CLINICAL IMAGE

# Stick to the ABC!

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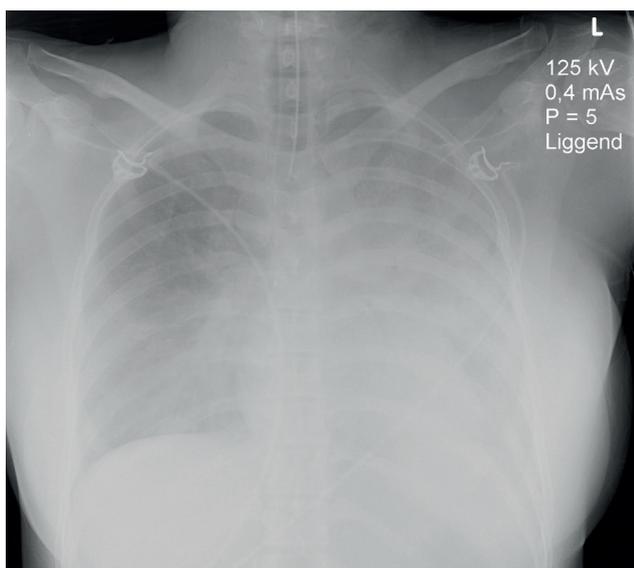
**Keywords** - Postobstructive pulmonary oedema, neurogenic pulmonary oedema, mayo tube, ABC method**Case history**

A 29-year-old woman was admitted to our hospital with multiple seizures. Her medical history denoted status epilepticus 6 years ago; after starting antiepileptic medication she had been free of epileptic seizures for the past 3 years. Other than this she had been completely healthy. On the neurology ward the woman developed respiratory instability during one of her seizures. We saw a postictal woman with stridor, who had already been given a Mayo tube to ensure the upper airway. Breathing frequency was 25/min with visible use of the accessory muscles. Even with supportive noninvasive bag ventilation the oxygen saturation remained low. In combination with the ongoing respiratory problems the patient remained unconscious; therefore, an intubation procedure was started to ensure the airway. During the intubation procedure the cause of the inspiratory stridor

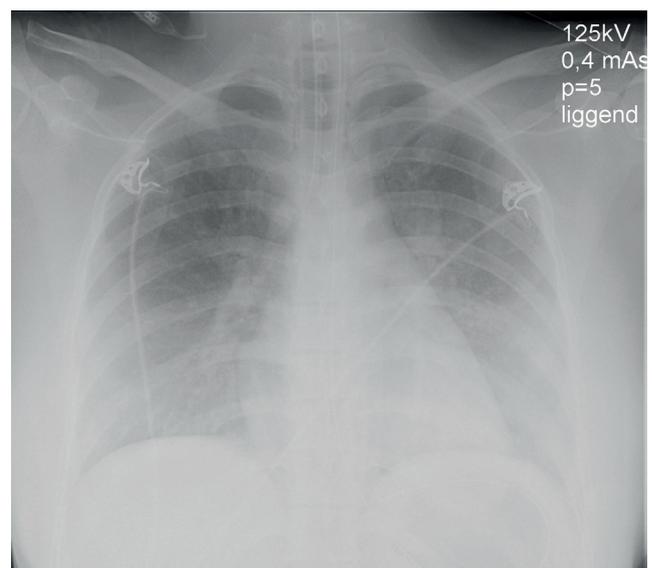
was revealed: the Mayo tube was positioned upside down, with the pharyngeal part aiming in the opposite direction to the palate. An uncomplicated intubation followed. Evaluation of the malplacement of the Mayo tube was not conclusive. The chest X-ray after intubation showed correct positioning of the endotracheal tube and massive bilateral interstitial and alveolar oedema (*figure 1*). The oedema was partly reabsorbed after two days (*figure 2*) and the patient was successfully extubated.

The aetiology of acute pulmonary oedema is diverse. In this case a cardiogenic cause seemed unlikely because of her medical history and normal electrocardiogram. The leucocytosis and increased C-reactive protein level resolved spontaneously making an infectious cause unlikely. Neurogenic pulmonary oedema (NPE) is a rare complication in patients with epilepsy,

**Figure 1.** Chest X-ray directly after intubation showing diffuse alveolar consolidations



**Figure 2.** Chest X-ray 2 days after intubation showing partial resolution of the alveolar consolidations



but the incidence seems to be related to the severity of the seizures. As in this case, it can develop within minutes and will resolve in a few days.<sup>1</sup> We assume that a combination of NPE and negative pulmonary pressure oedema (NPPE) secondary to the iatrogenic upper airway obstruction were the cause of the pulmonary oedema. Although the precise pathophysiology of NPPE is unclear, it appears to be related to an upper airway obstruction leading to high negative intrathoracic pressures which in turn results in a pressure gradient that drives fluid out of the capillaries into the interstitium and alveolar space. Hypoxaemia secondary to the lowered diffusing capacity causes vasoconstriction in the pulmonary vasculature which promotes the negative pressure gradient even more. A second mechanism suggested breaks in the alveolar epithelium and vascular membranes with leaking of protein-rich fluid leading to a rise in osmotic pressure gradient.<sup>2</sup> The majority of case reports and reviews describe patients in a post-anaesthetic setting, often with evident laryngospasm. Risk factors are: obstructive sleep apnoea, obesity, upper aerodigestive tract surgery or being a young and athletic male.<sup>3</sup> Treatment includes ensuring the airway; intubation is not always needed. Oxygen supplementation in combination with positive end-expiratory pressure or noninvasive positive pressure ventilation can be used, there is no evidence for using diuretics. If treated early, the clinical course usually rapidly resolves in 12-48 hours. The key lesson learned is that during an unsuccessful stabilisation using the ABC algorithm, checks should be done to ensure that earlier interventions were performed correctly.

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## References

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