

Correspondence

Negative pressure pulmonary oedema

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EDITOR:

Negative pressure pulmonary oedema is a medical emergency that usually arises from attempted ventilations against an acutely obstructed upper airway, such as the one that occurs during laryngospasm, resulting in transudation of fluid from pulmonary capillaries to the interstitium. Frequently, this occurs in the perioperative period when general anaesthesia is used. Clinicians should promptly recognize this complication and commence appropriate management as early as possible [1–4].

Case

A 65-yr-old male was encountered at home by paramedics in respiratory distress. The paramedics immediately administered dexamethasone 12 mg intravenously and xylometazoline nebuliser to treat a suspected upper airway obstruction. He was transported to the emergency room of our hospital. His medical history revealed hypertension, hypercholesterolaemia and an acute myocardial infarction in 1999. He complained of a sore throat and was dyspnoeic. Physical examination revealed an inspiratory stridor and a temperature of 38.7°C. Infection parameters were white blood cells (WBC) $15.7 \times 10^3 \text{ g L}^{-1}$ (4–11) and C-reactive protein (CRP) 95 mg L^{-1} (0–5). Arterial blood gas showed pH 7.43 (7.35–7.45), PCO_2 4.3 kPa (4.5–6.0), PO_2 16.1 kPa (9.5–13.0) and SaO_2 99% (92–99) with oxygen mask. A chest X-ray showed no infiltrations.

In order to inspect the oral cavity and throat, the patient was put into the supine position. Suddenly the patient lost consciousness due to a complete upper airway obstruction. Despite respiratory efforts, no effective breathing was possible.

An emergency cricothyrotomy was performed. The trachea was cannulated and positive-pressure ventilation was started. A chest-X-ray, 2 h after the first X-ray, showed typical negative pressure pulmonary oedema (Fig. 1). The patient was sedated and ventilated overnight. The next morning, a percutaneous tracheostomy was inserted. A chest X-ray showed resolving oedema. A computed tomography scan of the head and chest showed an oedematous epiglottitis.

Discussion

This case illustrates the rapid onset of negative pressure pulmonary oedema and its resolution with appropriate interventions. Laryngospasm during intubation or after anaesthesia is the most common cause of upper airway obstruction leading to negative pressure pulmonary oedema, but there are several other causes such as epiglottitis, foreign-body aspiration, thyroid goitre, croup, obstructive sleep apnoea and upper airway tumour. Negative pressure pulmonary oedema is a rare entity with a high degree of morbidity; its rarity may lead to the failure of early recognition or to misdiagnosis. The



Figure 1.
Negative pressure pulmonary oedema after upper airway obstruction.

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cornerstones of management comprises early diagnosis with re-establishment of the airway, adequate oxygenation and application of positive airway pressure [1–4].

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Symptomatic atlantoaxial dislocation in Marfan's syndrome: anaesthetic considerations

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EDITOR:

Marfan's syndrome is a relatively uncommon familial disorder with an incidence of about 1:10 000 in most racial and ethnic groups [1]. It is characterized by widespread abnormalities of the skeletal, cardiovascular and ocular symptoms. Although cervical spine abnormalities including atlantoaxial dislocation are common in these patients, clinical problems associated with them are rare [2]. Clinically symptomatic cases of atlantoaxial dislocation in Marfan's syndrome are rarely reported in literature [3,4]. We describe a patient with Marfan's syndrome undergoing corrective surgery for atlantoaxial dislocation under general anaesthesia.

A 21-yr-old 174 cm male weighing 44 kg presented with a 4-yr history of progressive weakness of all four limbs following the fall of an object on his neck. Gradually, he developed difficulty in getting up from a squatting position without support. His past medical history was unremarkable. On examination, this tall patient revealed typical features of Marfan's syndrome: arachnodactyly, high arched palate, winging of scapula, ulnar deviation of metacarpophalangeal joints and bilateral congenital talipes equinovarus. There were no significant abnormalities on examination of the cardiovascular system. However, routine transthoracic echocardiography showed aortic root dilatation (42 mm) with

normal left ventricular function. The patient had diminished motor power in all four limbs (4/5). His gag reflex was impaired indicating involvement of lower cranial nerves. A magnetic resonance imaging of the cervical spine showed atlantoaxial dislocation with basilar invagination by an abnormally curved odontoid peg with significant cervical cord compression at the level of the atlas. The patient was diagnosed to have a craniovertebral junction anomaly associated with Marfan's syndrome. Surgical correction was planned, which included trans-oral odontoidectomy and posterior fixation. Two days before surgery, Gardner–Wells tong traction was attached under local anaesthesia to reduce the dislocation. The plan of awake fiberoptic intubation was discussed with the patient. On the day of surgery, he was premedicated with glycopyrrolate 0.2 mg intramuscularly, 1 h before induction. Awake intubation was performed after anaesthetizing the upper airway with lidocaine 4% viscous, bilateral superior laryngeal nerve blocks and a trans-tracheal injection of lidocaine 4%. Standard anaesthetic induction was followed using fentanyl, thiopental and rocuronium. Anaesthesia was maintained with O₂, N₂O (1:2) and isoflurane with intermittent bolus of rocuronium and fentanyl. Mean blood pressure (BP) was maintained at about 70–80 mmHg by titrating the inspired concentration of isoflurane along with boluses of fentanyl. Care was taken to prevent hypertensive episodes during various steps of anaesthesia and surgery with the use of an intravenous nitroglycerin infusion. Monitoring parameters included ECG, SpO₂, ETCO₂ and continuous arterial BP. The surgical procedure

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