CASE STUDY

Negative Pressure Pulmonary Oedema After Septoplasty

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Abstract Negative pressure pulmonary oedema (NPPO) is an anaesthetic complication due to acute obstruction of the upper airway, whose main cause is laryngospasm. The pathophysiology involves a strong negative intrapleural pressure during inspiration against a closed glottis, which triggers excessive pressure in the pulmonary microvasculature. Although its diagnosis can be difficult, its recognition helps to minimise morbidity and mortality. This article presents a case of NPPO due to postextubation laryngospasm.

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PALABRAS CLAVE
Edema pulmonar; Presión negativa; Laringoespasmo

Resumen El edema pulmonar por presión negativa (NPPE) es una complicación anestésica por obstrucción aguda de la vía aérea superior, su principal causa es el laringoespasmo. La fisiopatología radica en una marcada presión negativa intrapleural durante una inspiración contra glotis cerrada, la cual desencadena una presión excesiva en la microvasculatura pulmonar. El diagnóstico puede ser difícil, su reconocimiento ayuda a minimizar la morbimortalidad. En este artículo se presenta un caso de NPPE por laringoespasmo postextubación.

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Introduction

Negative pressure pulmonary oedema (NPPO) occurs in 0.1% of postsurgical patients and the percentage rises to 11% if there is a degree of upper airway obstruction (UAO). In paediatrics the frequency reaches 9.4–9.6%.1–3

It usually occurs in patients who are young, healthy and of middle age with an American Society of Anaesthesiology (ASA) classification of I–II. However, it is also observed in children and the elderly.

There are predisposing factors such as sleep apnoea, macroglossia, tracheal stenosis or nasopharyngeal abnormalities.

The strong negative intrapleural pressure secondary to the inspiratory effort generates sufficient transmural gradient in the pericapillary pulmonary interstitium to produce

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oedema. Hypoxaemia, hypercapnia, acidosis and adrenergic hypersecretion are added to the picture, determining the developmental, radiological, clinical and physiopathological characteristics of this condition.  

Clinical presentation varies from mild forms with desaturation up to severe symptoms of alveolar haemorrhage and marked hypoxia.  

Symptoms can appear from the first minutes of the obstruction up to several hours afterwards. Treatment ranges from oxygen therapy and furosemide in mild cases, to mechanical ventilation with intermittent positive pressure and renal function support in the most severe cases.

Clinical Case

A male patient, 28 years old, weighing 80 kg, was scheduled for septoplasty. He was given premedication of i.v. midazolam+fentanyl, preoxygenated and induced with propofol, remifentanil and rocuronium (all at the recommended doses).

Following induction and orotracheal intubation, the patient was connected to a Dräger Primus® ventilator, with a tidal volume of 650 ml and a respiratory rate of 11 min.

Pulmonary auscultation showed good ventilation, without additional noises. He was kept with sevoflurane at a minimum alveolar concentration (MAC) of 1, reducing the endogenous drugs by 50%. Once the surgery was finished, the patient was extubated when he began to reject the endotracheal tube (BIS 70). The patient, upon noticing the nasal plugging produced by the intervention, began to make a strong inspiratory effort against a closed glottis and contracting his abdominal muscles. After various attempts at forced inspirations, it was possible to clear the UAO and free the obstruction. Manual ventilation was given using a face mask and oxygen at 100% with positive pressure, with stridor at the beginning.

The clinical picture was considered postextubation laryngospasm. The patient recovered spontaneous ventilation and consciousness, so he was transferred to the postanaesthesia recovery unit (PARU). Saturation remained below 90% using pulse oxygen; he was tachypneic with forced inspiration and cough as if to clear the pharyngeal area, without any positive results. Oxygen values increased only with O2 at 100% using a face mask with a reservoir bag (92%). Crackles were heard, mostly bilateral medial basal. Blood gas analysis and chest X-ray were performed, comparing the X-ray with the preoperative one (Figs. 1 and 2). Differential diagnoses were considered: blood gas analysis (pH: 7.24; PaCO2: 57; PaO2: 82; bicarbonate: 21; base excess: −8; and O2 Sat: 91%) and chest X-ray (bilateral perihilar alveolar pattern with a slight right dominance, lacking pleural effusion and cardiovascular shadow changes). The patient improved with oxygen therapy at 100% using face mask with a reservoir bag and a diuretic (i.v. furosemide 20 mg in the PARU).

Discussion

There should be a differentiation between NPPO and other post-surgical manifestations that include pulmonary oedema. The association between UA and pulmonary oedema was described for the first time in 1927 in animals,  

in humans in a child in 1973  

and the first adult case was published in 1977.  

Its true incidence is unknown; some authors indicate that it is produced in 11% of patients who require an active intervention of the airway for acute obstruction.  

In our review, we found 34 cases reported, mostly male (26/34 or 76%).  

Average age was 31 years (range: 14–67 years). The most frequent cause of UA obstruction was laryngospasm (30/34 or 88%).  

What happened most often was that the pulmonary oedema was recognised in minutes after the obstruction. Resolution was rapid, within 48 h, except for 3 patients that needed more time, recovering within the first week.

Treatment consisted of endotracheal intubation plus mechanical ventilation (18/34 or 53%), with positive end expiratory pressure (PEEP) (8/18 or 44% of the intubated patients) and use of mask with continuous positive airway pressure (CPAP) in 3/34 patients (9%).  

Seven patients received corticoids, 10 received diuretics and another 10
did not require any type of treatment (29%). Associated risk factors were identified in the minority of the cases (12/34 or 35%).\textsuperscript{12,13}

Clinical presentation is dyspnoea, expectoration, cyanosis, tachypnoea and tachycardia; the risk factors are obesity, sleep apnoeas, a short neck and excessive development of muscle mass.\textsuperscript{13}

A respiratory clinical exam during the immediate postoperative period will identify the presence of signs of lung oedema, as long as the presence of stridor, pulmonary cracking and respiratory insufficiency, assessed using pulse oxygen analysis, are taken into account.\textsuperscript{4,10,12}

Spontaneous resolution of the entity in mild cases and resolution with few therapeutic measures contribute to the under diagnosis of the problem. It should be emphasised that it is important to keep this condition in mind following any type of surgical intervention in healthy, young patients who present hypoxaemia not explained by other causes and establish the therapeutic measures needed to avoid an unfavourable patient evolution.\textsuperscript{14}

In this specific case, we believe that the evolution, although brief, would have been better if CPAP had been added to the treatment in the PARU.

Due to the bilaterality of the condition, the following were ruled out: aspiration pneumonitis (of late appearance and usually compromising the lower right lobe), pulmonary oedema from fluid overload (small supply of 750 ml during the surgery), anaphylaxis (no known atopy and there was no typical skin reaction), neurogenic cause (usually in neurosurgical patients or those with encephalic lesions), respiratory distress or acute pulmonary lesion (no haemorrhage or hemoptysis), unknown underlying cardiac cause (no electrocardiograph alterations or previous valve pathology) and postoperative residual curarization (reversal with sugammadex at 2 mg/kg).

**Conflict of Interest**

The authors declare no conflict of interest.

**References**