



## Negative Pressure Pulmonary Oedema Following Adenoidectomy Under General Anaesthesia: A Case Series

*œdème pulmonaire par pression négative après adénoïdectomie sous anesthésie générale:  
Une série de cas*

B. O. Bolaji\*<sup>†</sup>, O. O. Oyedepo<sup>†</sup>, A. D. Dunmade<sup>‡</sup>, O. A. Afolabi<sup>‡</sup>

### ABSTRACT

**BACKGROUND:** Negative pressure pulmonary oedema (NPPE) is a potentially life-threatening complication of laryngospasm that occurs during or after general anaesthesia. It is a complication of poorly treated or unrecognized laryngospasm occurring at extubation or later in the postoperative period.

**OBJECTIVE:** To emphasize prompt recognition of laryngospasm during or after general anaesthesia and to prevent its progression to NPPE in a resource-challenged environment.

**METHODS:** Three children aged two to four years, one of whom was a known sickle cell anaemia patient presented with adenoid hypertrophy. Surgery was postponed on account of upper respiratory tract infection in each of them.

**RESULTS:** Following treatment of upper respiratory tract infection, they had adenoidectomy under general anaesthesia. They all developed severe laryngospasm at extubation. This progressed to NPPE which was diagnosed on clinical parameters. The children were subsequently admitted to the intensive care unit (ICU) for mechanical ventilation with high FiO<sub>2</sub> (0.7–1) and PEEP between 12–24 hours. While two of the children survived, the child with sickle cell anaemia died in the ICU.

**CONCLUSION:** Negative pressure pulmonary oedema is a self limiting complication of laryngospasm if it is well managed. However, its outcome may not be good in a patient with intercurrent medical illness such as sickle cell anaemia in which hypoxaemia is deleterious. *WAJM* 2011; 30(2): 121–124.

**Keywords:** Adenoidectomy, tracheal extubation, laryngospasm, negative pressure pulmonary

### RÉSUMÉ

**CONTEXTE:** L'œdème pulmonaire par pression négative (EPPN) est une complication potentiellement mortelle de laryngospasme qui se produit pendant ou après une anesthésie générale. Il est une complication de laryngospasme mal traitée ou non reconnue se produisant à l'extubation ou plus tard dans la période postopératoire.

**OBJECTIF:** Mettre l'accent sur la reconnaissance rapide de laryngospasme pendant ou après une anesthésie générale et pour empêcher sa progression vers l'EPPN dans un contexte où les ressources font défaut.

**METHODES:** Trois enfants âgés de deux à quatre ans, dont l'un était un patient connu drépanocytose présentait une hypertrophie adénoïde. La chirurgie a été reportée en raison d'une infection des voies respiratoires supérieures dans chacun de ces cas.

**RÉSULTATS:** Après le traitement des infections des voies respiratoires supérieures, ils avaient bénéficié d'une adénoïdectomie sous anesthésie générale. Ils ont tous développé un laryngospasme sévère à l'extubation. Cette progression à EPPN a été diagnostiquée sur des paramètres cliniques. Les enfants ont ensuite été admis à l'unité de soins intensifs (USI) pour la ventilation mécanique avec FiO<sub>2</sub> élevée (0.7-1) et PEEP entre 12-24 heures. Alors que deux des enfants ont survécu, l'enfant souffrant d'anémie falciforme est décédé à l'Unité de soins intensifs.

**CONCLUSION:** L'œdème pulmonaire à pression négative est une complication auto limitation de laryngospasme si elle est bien gérée. Toutefois, ses résultats ne peuvent pas être bonne chez un patient présentant une maladie intercurrente médicale telles que l'anémie falciforme dans lequel l'hypoxémie est délétère. *WAJM* 2011; 30(2): 121–124.

**Mots-Clés:** Adénoïdectomie, Extubation, Laryngospasme, Pression pulmonaire négative

Departments of <sup>†</sup>Anaesthesia and <sup>‡</sup>Otorhinolaryngology, College of Health Sciences, University of Ilorin, Ilorin, Nigeria.

\*Correspondence: Dr. B. O. Bolaji, P. O. Box 563, Ilorin, Nigeria. E-mail: olubolajidr@unilorin.edu.ng

Abbreviations: ENT, Ear, Nose, and Throat; ETT, Endotracheal Tube; FiO<sub>2</sub>, Fractional Inspired Oxygen Concentration; ICU, Intensive Care Unit; NPPE, Negative Pressure Pulmonary Oedema; PEEP, Positive End Expiratory Pressure; SpO<sub>2</sub>, Oxygen Saturation.

## INTRODUCTION

Negative pressure pulmonary oedema (NPPE) is a potentially life-threatening complication of poorly managed or unrecognized laryngospasm that occurs during or after general anaesthesia. It commonly occurs at extubation or later in the post-operative period. The overall incidence of laryngospasm in a large Scandinavian study was 0.78% and the risk was greater in certain subgroups such as children with asthma or airway infection.<sup>1</sup> Repeated airway infection occurs in children with hypertrophied adenoids, tonsils or both.

Negative pressure pulmonary oedema may cause serious morbidity and the patient may require intubation, ventilation and management in an intensive care setting.<sup>2</sup> Although the symptoms may resolve within 24 hours with appropriate management, they may progress to adult respiratory distress syndrome leading to death.<sup>3</sup> The three case reports are presented so that anaesthetists and surgeons practising in our environment with limited resources who do not have facilities such as mobile X-rays and arterial blood gas analyzers can recognize this complication and commence appropriate management promptly to obtain a successful outcome.

### Case 1

This was a two-year-old male child, who presented at the Ear, Nose and Throat (ENT) clinic at the age of eight months with a history of noisy breathing noticed a few weeks after birth. Other associated symptoms were recurrent mucopurulent bilateral nasal discharge, fever and snoring. Physical examination revealed a small for age child, with noisy respiration both awake and asleep with mild dyspnoea. The respiratory rate was 40/min. Ear, nose and throat examination revealed mouth breathing, moderately enlarged tonsils, purulent nasal discharge and loss of nasal patency. A diagnosis of adenoid hypertrophy with radiological confirmation was made. The packed cell volume was 36%, with a white cell count  $14.9 \times 10^9/l$ . The serum electrolytes and urea concentrations were normal. He was scheduled for adenoidectomy after failure of conservative management but

surgery was postponed six times on account of active recurrent upper airway infection.

### Case 2

A 4-year-old male child presented to the ENT clinic with a history of persistent mouth breathing and nasal discharge noticed a few weeks after birth. This was associated with nasal blockage alternating with recurrent mucopurulent and foul smelling nasal discharge, cough productive of yellowish sputum and symptoms of obstructive sleep apnoea. Examination revealed an ill looking boy, small for age with persistently opened mouth, mild fever and dyspnoea. He was not cyanosed. The respiratory rate was 35/min and auscultation of the chest revealed transmitted sounds with good air entry. The pulse rate was 100/min and the heart sounds were normal. Examination of the throat revealed moderately enlarged tonsils. A diagnosis of adenoid hypertrophy which was confirmed with extended neck X-ray.

His packed cell volume was 32%, with a white cell count of  $6.0 \times 10^9/l$ , and a platelet count of  $316 \times 10^9/l$ . The serum electrolytes and urea were within normal limits. He had adenoidectomy under general anaesthesia with endotracheal intubation after two postponements on account of active upper airway infection.

### Case 3

The third child, a male, was also four years old previously diagnosis of sickle cell anaemia, presented to the ENT clinic with a two-year history of recurrent noisy breathing and associated recurrent cough, nasal discharge alternating with nasal blockage, mouth breathing and symptoms of obstructive sleep apnoea. Physical examination revealed a small for age child, pale and slightly icteric with features of sickle cell anaemia. Nasal examination revealed mucopurulent nasal discharge, loss of nasal patency with persistent mouth breathing. Auscultation of the chest revealed transmitted sounds bilaterally. The heart sounds were normal. Examination of the throat revealed moderately enlarged tonsils. A diagnosis of adenoid hypertrophy was made.

The packed cell volume was 25% (steady state PCV 23–24%), with a white cell count of  $8.0 \times 10^9/l$ . The serum electrolytes and urea results were normal. X-ray of the post-nasal space was consistent with adenoid hypertrophy. He was scheduled for adenoidectomy but surgery was postponed three times because of upper airway obstruction

## Anaesthetic and Intensive Care Management

The three children had adenoidectomy under general anaesthesia with endotracheal intubation. Anaesthesia was induced with halothane in oxygen and the airway was secured with suxamethonium 1mg/kg. The lungs were ventilated with 66% nitrous oxide in oxygen in cases 1 and 2 but 50% nitrous oxide in oxygen in the child with sickle-cell anaemia, and 0.5% halothane while muscle paralysis was provided with pancuronium 0.1mg/kg. Analgesia was provided with fentanyl 2µg/kg. Residual neuromuscular paralysis was reversed with a combination of neostigmine 0.04mg/kg and atropine 0.02mg/kg.

Case 1 was extubated awake but he suddenly developed severe laryngeal spasm. Manual ventilation with a face mask was difficult and the SpO<sub>2</sub> dropped to less than 80%. Anaesthesia was deepened with halothane and his airway was re-secured with suxamethonium 1mg/kg. He developed pulmonary oedema evidenced by pink, frothy secretions in the endotracheal tube (ETT) and bilateral crepitations on chest auscultation. The patient was ventilated manually with 100% oxygen, and was given frusemide 1mg/kg. He was admitted to the Intensive Care Unit (ICU) on account of poor respiratory function. In the ICU he was mechanically ventilated for 24 hours with positive end expiratory pressure (PEEP) of 5 cm H<sub>2</sub>O and a high fractional inspired oxygen concentration (FiO<sub>2</sub>) of 0.7–1. The patient was extubated uneventfully the following day and was discharged to the ward on the third day.

Case 2 developed laryngeal spasm after extubation with markedly reduced air entry, an SpO<sub>2</sub> of less than 80% and difficulty with mask ventilation. Anaesthesia was deepened with halothane and his airway was promptly

**Table 1: Features of three children who developed Negative Pressure Pulmonary Oedema following Adenoidectomy under General Anaesthesia**

Feature	Case 1	Case 2	Case 3
Age at First Presentation	8 months	4 years	4 years
Duration of Symptoms	8 months	4 years	2 years
Age at Surgery	2 years	4 years	4 years
Associated Medical Disease	NIL	NIL	Sickle-cell anaemia
Packed Cell Volume (%)	36	32	25
White Cell Count /( $L \times 10^9$ )	14.9	6.0	8.0
No. of Times Surgery Postponed	6	2	3
Period of Ventilation (h)	24	18	<12
Length of Stay in ICU (h)	72	24	<12
Outcome	Alive	Alive	Died

re-secured with suxamethonium 1mg/kg. The patient was ventilated with 100% oxygen. A second attempt to extubate the trachea when he became fully awake on the operating table was unsuccessful as he still had recurrent laryngospasm. The trachea was re-intubated. A frothy pinkish secretions in the ETT and bilateral crepitations on chest auscultation suggested the development of pulmonary oedema for which he had i.v. frusemide 1mg/kg. He was admitted to the ICU. In the I.C.U. he was mechanically ventilated for 18 hours (PEEP 5cm H<sub>2</sub>O, high FiO<sub>2</sub> of 0.7–1), extubated uneventfully on the first post-operative day and was discharged to the ward in a stable condition on the same day.

Case 3 developed severe laryngeal spasm with bilateral reduced air entry, on chest auscultation. His airway was re-secured with suxamethonium and he was initially manually ventilated with 2% halothane in oxygen followed by 100% oxygen only. The ETT had frothy pinkish secretions. His spontaneous respiration was poor, with a respiratory rate of 45/min, while SpO<sub>2</sub> was less than 90% with FiO<sub>2</sub> of 1. He was admitted into the ICU, mechanically ventilated (PEEP 5cm H<sub>2</sub>O, high FiO<sub>2</sub> 0.7–1). However, his SpO<sub>2</sub> remained very low (between 60 and 80%) and he died on the same day.

## DISCUSSION

Negative pressure pulmonary oedema is a rare but life-threatening complication of upper airway obstruction. Its occurrence has been reported to

be between 0.05–0.1% of all anaesthetics.<sup>2</sup> The morbidity associated with the condition may be as high as 40%.<sup>4</sup> It is a complication of poorly treated or unrecognized laryngospasm occurring at extubation or later in the postoperative period. Risk factors include children with asthma, upper airway infections, those undergoing oesophagoscopy or hypospadias repair.<sup>1</sup>

Adenoid hypertrophy also known as the pharyngeal or Luschka's tonsil is a widespread condition seen in children 3–6 years of age.<sup>5</sup> It is associated with recurrent airway infections and may result in cancellations of surgery as seen in the reported cases. The dilemma is to choose between surgery or postponement of surgery but these children hardly have periods of freedom from upper airway infection. Postponement may be indefinite! A decision to operate is usually based on discussions between the anaesthetist and the surgeon, the experience of the anaesthetist and the preparedness to handle eventual laryngospasm.

Laryngospasm is a forceful, involuntary spasm of the laryngeal musculature caused by stimulation of the superior laryngeal nerve which is the sensory innervation of the larynx. The signs of laryngospasm include inability to ventilate the patient and rapid desaturation. These were seen in these patients. Prevention can be achieved by extubating the patient using a 'no touch' technique when the patient is awake,<sup>6</sup> or under deep anaesthesia (possibly after a

magnesium infusion).<sup>7</sup> Early intervention to break laryngospasm is aimed at preventing complications such as negative pressure pulmonary oedema or aspiration. It consists of applying a gentle jaw thrust and if it can not be relieved, increasing the depth of anaesthesia, giving intermittent positive pressure ventilation with 100% oxygen and a small dose of suxamethonium i.v. 0.25–0.5mg/kg or i.m. 4–6mg/kg. Propofol has been used to increase the depth of anaesthesia because of its rapidity of onset and predictability.<sup>8</sup> Halothane as was used in these three cases is suitable for children<sup>9</sup> but its delivery depends on alveolar ventilation which is reduced in the setting of severe laryngospasm.

Negative pressure pulmonary oedema (NPPE) has been reported to occur in up to 4% of cases of laryngospasm.<sup>10</sup> It is a rapidly reversible condition once prompt management is commenced. The diagnosis was based on the clinical setting of severe laryngospasm, frothy pinkish secretions seen in the ETT, bilateral crepitations on chest auscultation and arterial oxygen desaturation. Confirmation with chest radiograph was not possible as a mobile X-ray machine was not readily available in our centre. The management of NPPE consists of tracheal intubation, mechanical ventilation with PEEP along with diuretic therapy and haemodynamic monitoring in the ICU. The three reported cases had severe laryngospasm at extubation which was complicated by NPPE and were subsequently admitted to the ICU for respiratory support. As arterial blood gas analyzer was not available, SpO<sub>2</sub> monitoring was used as a guide for oxygenation although it is unreliable in haemoglobinopathy. While the management was successful in the first two cases, the third, a known sickle-cell anaemia patient died. In Nigeria, 25% of the population are carriers of the sickle cell gene while the prevalence of sickle cell anaemia is about 20 per 1000 live births which means that about 150,000 children are born annually with the disease.<sup>11</sup> Hypoxia is a potent stimulus for infarctive crises in sickle cell anaemia and this child could have developed acute chest syndrome leading to his death. For such patients, laryngospasm under general

anaesthesia and any other factor that results in hypoxic episodes must be avoided.

### Conclusion

Negative pressure pulmonary oedema is a self limiting complication of laryngospasm resolving within 24 hours once it is well managed even with the limited resources in our environment. It must however be avoided in all patients, especially those with intercurrent medical diseases such as sickle cell anaemia because of the possibility of a poor outcome.

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