

A Case of Pulmonary Haemorrhage Following Jet Ventilation for Vocal Cord Surgery

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Abstract

Introduction: This case report highlights haemoptysis occurring after post-extubation laryngospasm. **Clinical Picture:** General anaesthesia using Sanders jet ventilation with a Benjamin tube was administered for a patient undergoing vocal cord biopsy. He developed laryngospasm followed by significant pulmonary haemorrhage and widespread crepitations in the lung. **Treatment:** Laryngospasm was aborted with assisted ventilation with oxygen 100% via bag and mask. **Outcome:** Oxygenation was well maintained with nasal prongs only postoperatively and haemoptysis resolved after 2 days. **Conclusion:** It is imperative to prevent laryngospasm from occurring and swift action must be taken to avoid pulmonary haemorrhage.

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Key words: Haemoptysis, Jet ventilation, Negative pressure pulmonary injury

Introduction

Laryngospasm occurring at extubation is not an uncommon complication seen with use of endotracheal tubes and laryngeal mask airway. We report a case of laryngospasm on extubation of a Benjamin jet tube resulting in pulmonary haemorrhage.

Case Report

The patient was an ASA 1 (American Society of Anesthesiologists 1), 38-year-old male weighing 80 kg and 1.7 m tall. He was scheduled to undergo excision and biopsy of a left vocal cord granuloma after a course of steroids failed to alleviate his sore throat and cough. He was a non-smoker. Preoperative examination and investigations were normal.

General anaesthesia was planned with the use of a Benjamin jet tube for improved surgical access and Sanders jet ventilation. The jet ventilator was attached to an oxygen/air mixing device set at 40 pounds per square inch, with the anaesthetist timing the breaths in synchrony with surgical needs. After intubation with propofol and atracurium, the patient was maintained on propofol infusion and intermittent doses of atracurium. Surgery lasted 45 minutes and haemostasis was secured. Hitherto, anaesthesia was uneventful with haemodynamic parameters and oxygenation well maintained. Neostigmine with atropine

was given for reversal. The patient was breathing spontaneously with good respiratory effort whereupon it was decided to remove the Benjamin tube. Soon after, the patient experienced difficulty in ventilation, developed a crowing noise on inspiration and started to desaturate. Breathing was assisted by bagging with 100% oxygen and pulse oximetry reading was 95% to 97% thereafter. He became increasingly restless and agitated although he was able to obey commands and grip the anaesthetist's fingers tightly. Half the previous dose of reversal was added and the airway was quickly suctioned between bagging. Following this, the patient was able to draw several deep inspiratory breaths but suddenly, gave a cough and expectorated about 400 mL of fresh blood. Physical examination revealed crepitations bilaterally although he was able to maintain 98% saturation with a simple face mask. Two doses of frusemide 80 mg was given and although respiratory efforts were good, haemoptysis continued for 48 hours. A bronchoscopy performed by the surgeon immediately after surgery did not show any active bleeding from the operative site or the mucosal surfaces of the trachea. Lesions, such as polyps or tumours, were not seen but blood-stained secretions were noted arising from the lower airways. Blood investigations, like full blood count and coagulation studies to exclude bleeding tendencies, did not reveal any abnormalities. Chest X-rays

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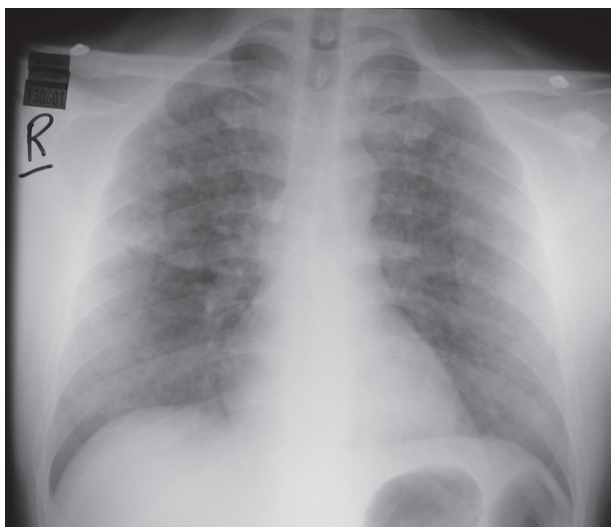


Fig. 1. Chest X-ray immediately postoperative.

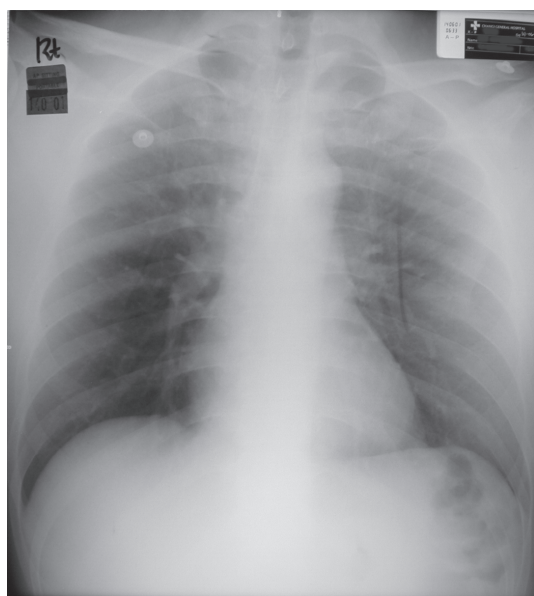


Fig. 2. Chest X-ray after 24 hours.

done immediately after surgery (Fig. 1) showed patchy air shadows in both lung fields which improved the next day (Fig. 2). The patient was weaned onto oxygen 3 L/min via nasal prongs overnight and discharged on the second post-operative day. No further complications were noted on his follow-up visit 1 week later.

Discussion

Pulmonary oedema occurring after relief of laryngospasm has been well reported. Inspiratory efforts against an obstructed upper airway result in development of negative intrathoracic and transpulmonary pressure (Mueller manoeuvre).¹ Neurogenic, cardiac and hydrostatic mechanisms have been cited as possible mechanisms for negative pressure pulmonary oedema.^{2,3}

Deepika et al,⁴ in 1997, performed a retrospective study on 30 affected patients and showed that this phenomenon was more frequent in healthy (ASA I & II), middle-aged male patients. Rezaiguia-Delcaux et al⁵ also demonstrated an impairment of alveolar fluid clearance by volatile anaesthetic agent in rats which may contribute to the pathogenesis of negative pressure pulmonary oedema.

Frank haemoptysis occurring as a consequence of laryngospasm has been less frequently reported. One possible explanation lies in deep inspiratory efforts made against a closed glottis which results in significant negative pleural pressure. Consequently, there is an increase in left ventricular preload and afterload. Venous return also increases due to reduced right atrial pressure and increased mean systemic pressure from venoconstriction brought about by anxiety, hypoxia and hypercarbia. This increased right ventricular volume then causes the interventricular septum to shift to the left, resulting in reduced left ventricular diastolic compliance. In addition, catecholamine-induced systemic vasoconstriction increases the afterload, thereby depressing left ventricular ejection. These cardiac changes together with the negative pleural pressure result in increased pulmonary trans-capillary pressure which then break the alveolar-capillary membrane. Red blood cells leak into the alveoli and, thus, frank haemorrhage is seen.⁶ However, findings from another report whereby diffuse punctate haemorrhages throughout the tracheobronchial tree were visualised fibrooptically⁷ suggest that pulmonary haemorrhage occurs because systemic bronchial vasculature is disrupted. Bhavani-Shanker et al⁸ believed that this could be the mechanism resulting in pulmonary haemorrhage in their reported case of negative pressure induced pulmonary injury with laryngeal mask insertion.

The incidence of post-obstructive pulmonary oedema varies between 0.05⁹ and 0.094%,⁴ but that for pulmonary haemorrhage is still uncertain. There has been an increasing awareness of this complication occurring especially in the relevant fields of anaesthesia, otolaryngology and respiratory medicine and we suspect that pulmonary haemorrhage is underreported. Hitherto, pulmonary haemorrhage has been seen in mainly cases that developed laryngospasm upon extubation of an endotracheal tube or laryngeal mask airway. However, there has recently been one report of pulmonary haemorrhage occurring in an intubated patient and the authors concur with previous case reports that this was the result of bronchial vessel damage.¹⁰

This is the first reported incident of pulmonary haemorrhage occurring after the use of a Benjamin jet tube with Sanders jet ventilation. The Benjamin jet tube (Fig. 3) consists of a narrow calibre tube with “petals” at the distal end to hold the tube in the central position of the trachea. It is advantageous in laryngeal surgery as it allows good



Fig. 3. Benjamin tube.

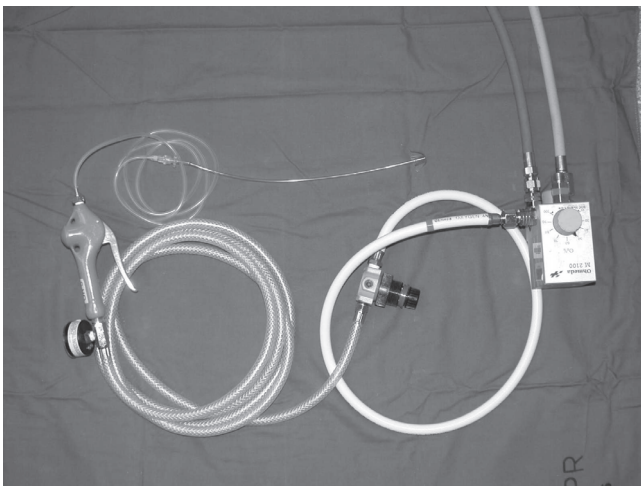


Fig. 4. Sanders Venturi Jet Ventilator.

surgical exposure. Oxygen-enriched air is delivered via a venturi jet ventilator (Fig. 4) set at 40 to 50 pounds per square inch. Complications from jet ventilation usually manifests as barotrauma with possibility of pneumothorax or pneumomediastinum, especially if the laryngeal papilloma was large enough to obstruct the glottis. Alveolar haemorrhage as a result of pulmonary barotrauma is very rare and, hitherto, there has been only 1 reported case.¹¹ With the onset of haemoptysis shortly after the patient drew several deep inspiratory breaths, this strongly supports our speculation that this complication was induced by laryngospasm and not trauma from jet ventilation. Moreover, bleeding was never noted during the procedure and the patient was able to maintain good arterial saturation until this point. Besides airway trauma, the differential diagnoses considered included aspiration pneumonitis, tumour, polyp and tuberculosis. Of these, aspiration pneumonitis was a possibility as the Benjamin jet tube was uncuffed and

therefore did not protect the lungs from gastric contents, blood or debris. However, since the patient's condition improved swiftly and hypoxaemia was not seen, this is unlikely.

Triggering factors for laryngospasm in this patient are varied. Laryngospasm could have been brought about by blood or debris in the upper airway. Having dysphonia and chronic cough may have predisposed him to a "spasm-ready" state¹² which was compounded by manipulation from the surgical site.

With the use of jet ventilation, the pressure is lowest at the glottis and this causes air passing through the vocal cords to draw them together, thus narrowing the laryngeal opening. An increased inspiratory effort in the presence of a "small" laryngeal opening which causes marked negative intrathoracic pressure causes severe "stress-failure" of the alveolar-capillary membrane. "Stress failure" is a term that has been used to describe the mechanical disruption of the alveolar-capillary membrane with subsequent impairment of barrier function brought about by increased pulmonary wall stress.¹³ However, in our patient, the negative intrathoracic pressure was generated by laryngospasm rather than jet ventilation but this must be borne in mind.

In view of this, it is of great concern to any clinician to either prevent laryngospasm or when faced with a case of laryngospasm, to abate it as soon as possible. There have been several suggested methods of preventing laryngospasm. Topical lignocaine 4% at 4 mg/kg was found to reduce the incidence of postoperative stridor and laryngospasm in a randomised, prospective study of 133 patients undergoing tonsillectomy and adenoidectomy.¹⁴ One should consider this routine prior to extubation for surgeries performed on the vocal cords. Especially so in anaesthesia where a significant number of these cases arise, awake extubation especially for "at risk" cases is prudent. Muscle paralysis should also be well reversed as highlighted by O'Connor to avoid airway obstruction.¹⁵

In the event that laryngospasm ensues, it is imperative that the process be corrected swiftly as it has been suggested that pulmonary haemorrhage comes about when there has been an extreme elevation in pulmonary capillary transmural pressure.¹⁶ A small dose of neuromuscular blocker has been useful in overcoming laryngospasm and should be used early. McConkey⁹ noted that in a series of 6 cases, the mildest case of post-obstructive pulmonary oedema occurred with the use of succinylcholine.

Intravenous nitroglycerin at 4 mg/kg has also been used effectively in treating laryngospasm by relaxing the tracheal muscles.¹⁷

Treatment modalities for post-obstructive pulmonary oedema in reported cases have ranged from nasal prongs, CPAP mask and intubation with positive pressure

ventilation. Pulmonary haemorrhage in all reported cases were short-lived and limited to 3 to 4 days with symptoms subsiding and chest X-rays clearing up during this period.

In conclusion, the consequences of negative pressure thoracic changes may culminate in pulmonary haemorrhage which is due to stress failure of the pulmonary capillary membrane or injured tracheo-bronchial vessels. This complication though short-lived and does not appear to be more life-threatening than pulmonary oedema causes more distress to the patient. It behooves the physician to prevent laryngospasm from occurring and to intervene immediately should it arise.

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