Singapore Med J 1999; Vol 40(3):174-175

Negative Pressure Pulmonary Oedema Caused by Biting and Endotracheal Tube Occlusion – A Case for Oropharyngeal Airways

E H C Liu, P S W Yih

ABSTRACT

A patient had general anaesthesia for laparoscopic surgery. She bit on and occluded her endotracheal tube during recovery from anaesthesia. Strong inspiratory efforts during airway obstruction caused negative pressure pulmonary oedema. The pulmonary oedema resolved within 24 hours. Use of an oropharyngeal airway as a bite block could have prevented this complication.

Keywords: negative pressure pulmonary oedema, oropharyngeal airways

CASE REPORT

A 26-year-old woman presented with sudden severe lower abdominal pain. She had mild asthma, had not needed her salbutamol inhaler for over 6 months and was in good health prior to this admission. She weighed 63 kg and preoperative cardiovascular and chest examination was normal. Abdominal ultrasound showed a complex mass in the right ovary, thought to be a haemorrhagic corpus luteum. Ectopic pregnancy could not be excluded even though a urinary HCG test was negative. She was prepared for urgent laparoscopic surgery.

Anaesthesia was induced with fentanyl 100 μg, propofol 150 mg and suxamethonium 100 mg. Cricoid pressure was applied until confirmation of correct placement of the endotracheal tube. Anaesthesia was maintained with isoflurane (end tidal 0.8%) in nitrous oxide 66% and oxygen, pethidine 50 mg and muscle relaxation with atracurium. Laparoscopic surgery was uncomplicated and lasted one hour. A total of 1250 mL of Hartmans solution was infused. Oxygen saturation, airway pressure, end tidal capnography, pulse rate and blood pressure were within normal limits throughout surgery. Neuromuscular blockade was reversed with neostigmine 2.5 mg and atropine 1.2 mg and the patient started breathing. The patient was placed in a left lateral position, breathing 100% oxygen, in preparation for extubation when awake.

During awakening, the patient bit on the endotracheal tube, completely obstructing it. She made strong inspiratory efforts but there was no gas exchange. Her oxygen saturation did not fall initially. Suxamethonium was prepared as her oxygen saturation fell to 70% but was not given as she stopped biting. An oropharyngeal airway was inserted to function as a bite block. Ventilation was assisted on 100% oxygen and oxygen saturation recovered to 100%. Pink frothy secretions appeared in the endotracheal tube and moderate amounts were removed with a suction catheter. She was then extubated awake. On examination, she had fine crepitations in the mid and lower zones of both lungs, but no wheezes and no stridor. Her respiratory rate was 20 breaths per minute. She was able to deep breathe and cough up the pink frothy secretions. Her oxygen saturation was 100% on 50% oxygen via Ventimask and 90% on room air. Her chest X-ray showed bilateral diffuse pulmonary infiltrates, worst in the lower zones. A 12-lead ECG was normal. We made a diagnosis of negative pressure pulmonary oedema caused by airway obstruction.

Her oxygen saturation improved to 95% on room air after 4 hours of recovery room care. She was discharged to the gynaecology ward breathing 30% oxygen via a Venti mask. Twenty-four hours after surgery, she still had a few fine crepitations in both lower zones, but her oxygen saturation on room air was 99%. Her only complaint was of muscle ache in both shoulders. This was most probably due to suxamethonium given at induction. She was discharged 3 days after surgery.

DISCUSSION

Acute pulmonary oedema in our patient was due to strong inspiratory efforts producing excessive negative intrathoracic pressure while biting and obstructing the endotracheal tube. The speed of onset, the appearance of pink frothy secretions after relief of obstruction and the relatively fast recovery are characteristic of negative pressure pulmonary oedema (NPPE)[1]. After extubation, she had no difficulty breathing, and no signs of asthma, airway obstruction or heart failure.

The pathophysiology of NPPE is multifactorial. The major factor is marked negative intrapleural pressure produced by strong inspiratory efforts while the airway is obstructed. In young healthy patients,

Department of Anaesthesia
National University Hospital
5 Lower Kent Ridge Road
Singapore 119074

E H C Liu, MBChB, FRCA
Registrar

P S W Yih, MBBS,
MMed (Anaes)
Consultant

Correspondence to:
Dr P S W Yih

this negative pressure may be as great as – 50 cm water. This increases venous return, right ventricular filling and pulmonary capillary blood volume. Interventricular dependence leads to increased left ventricular end diastolic and left atrial pressures, reducing pulmonary venous return and further increasing pulmonary capillary blood volume. The increased pulmonary blood volume, adrenergic mediated pulmonary vasoconstriction and hypoxic pulmonary vasoconstriction increase pulmonary hydrostatic pressure, while the negative intrapleural pressure also decreases the interstitial hydrostatic pressure[1,2]. Direct injury to the alveolar capillary membrane increases permeability[3]. All these increase the tendency for fluid to move out of the pulmonary capillaries, resulting in interstitial and alveolar oedema.

The presentation is usually within minutes of airway obstruction but may not be evident until after relief of obstruction, hence it is also called post obstructive pulmonary oedema. Bhavani-Shankar reported airway bleeding and haemoptysis as a distinct complication, which may coexist with and/or be misdiagnosed as pulmonary oedema[4]. Our patient did not have haemoptysis though she had clinical and radiological signs of pulmonary oedema.

Emergence laryngospasm is the most common reported anaesthetic cause of airway obstruction and negative pressure pulmonary oedema[1]. Laryngospasm during extubation can be prevented by extubating patients either deeply anaesthetised or fully awake, but not when in between. However, inappropriate biting tends to occur when patients are half awake and still intubated. We could only find one report of biting and endotracheal tube occlusion causing NPPE[5]. However, there were 3 critical incident reports of this cause and complication in our hospital in the last 12 months. Could this be as common a complication as laryngospasm induced pulmonary oedema? Mild NPPE may be missed.

We could have immediately administered suxamethonium to stop the patient from biting and occluding the endotracheal tube, and to stop strong inspiratory efforts during airway obstruction. This could have minimised or prevented NPPE. However, the use of suxamethonium raises some concerns. Firstly, most patients stop biting quickly without the need for suxamethonium. Secondly, the duration of action of suxamethonium, after neostigmine has been administered, is uncertain. A third concern is awareness in a patient reparalysed at the end of general anaesthesia.

Most episodes of NPPE resolve within a few hours and oxygen therapy is usually all that is required[1]. A short period of mechanical ventilation and positive end expiratory pressure may speed up recovery. The role of loop diuretics is not certain[6].

Perhaps an oropharyngeal airway with a reinforced 'bite block' segment should be routinely inserted after endotracheal intubation. It should be removed only after extubation, particularly when patients are extubated awake. The oropharyngeal airway can prevent a patient from biting, damaging and completely occluding the endotracheal tube, and prevent severe NPPE and hypoxia. Bite blocks such as those used in gastro-duodenal endoscopy, are even more effective in preventing occlusion, but are less commonly available in the anaesthetic setting. The oropharyngeal airway will also facilitate oropharyngeal suction and maintenance of a patent upper airway after extubation if patients are extubated 'deep'.

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