Negative pressure pulmonary oedema following use of ProSeal LMA

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ABSTRACT

Negative pressure pulmonary oedema (NPPO) is a life threatening condition, manifested due to upper airway obstruction in a spontaneously breathing patient. Upper airway obstruction caused by classic laryngeal mask airway (cLMA) and ProSeal laryngeal mask airway (PLMA) has been reported, and NPPO has also been reported following the use of cLMA. Search of literature did not confirm NPPO following the use of PLMA. We encountered a female patient of NPPO scheduled for incision and drainage of an abscess who had signs of airway obstruction following PLMA insertion. Multiple attempts were made to get patent airway without success. PLMA was replaced with endotracheal tube following which pink frothy secretion appeared in breathing circuit. Patient was managed successfully with ICU care.

Key words: Airway obstruction, negative pressure, negative pressure pulmonary oedema, ProSeal laryngeal mask airway

INTRODUCTION

Negative pressure pulmonary oedema (NPPO) occurs in a patient breathing spontaneously against an obstructed upper airway.[1,2] Pulmonary oedema has been observed following upper airway obstruction caused by classic laryngeal mask airway (cLMA).[3,4] ProSeal laryngeal mask airway (PLMA) has also been reported to cause upper airway obstruction.[5] However, review of literature did not reveal any case of NPPO with the use of PLMA. We report a case of upper airway obstruction following PLMA insertion followed by pulmonary oedema in a spontaneously breathing patient.

CASE REPORT

A 10-year-old female patient weighing 25 kg presented with the complaints of painful swelling over lateral aspect of right chest wall and high-grade fever for past 4 days. She had tachycardia (PR 128/min) with normal blood pressure (114/71 mmHg). Her airway assessment revealed Mallampati class II, adequate mouth opening and short neck with limited head and neck movement. Local examination revealed a tense and tender swelling present over lateral aspect of right chest wall. On systemic examination, no abnormality was detected. Her investigations revealed anaemia (haemoglobin: 8.6 gm%) and leucocytosis (total leucocyte count: 19,500/mm³). A clinical diagnosis of an abscess (?pyogenic) over right chest wall was made and patient was scheduled for incision and drainage under general anaesthesia.

In the operating room, monitoring for electrocardiography, pulse oximetry and noninvasive blood pressure (by oscillometry method) was commenced by using COLIN-306 monitor. Anaesthesia was induced with fentanyl 2 µg/kg and propofol 2.5 mg/kg, and maintained with sevoflurane 1-2% in oxygen and nitrous oxide (1:1) with spontaneous ventilation on face mask through Magill’s circuit. PLMA size 2.5 was inserted without muscle relaxant and cuff was inflated with 14 ml air. The bite block was kept between the incisors. Following insertion of PLMA, difficulty in spontaneous ventilation was observed with chest retraction due to airway obstruction. Her head was almost in neutral position.
as sniffing position was not possible due to restricted movement of head and neck. Three attempts were made to correct the position of PLMA with restricted movement of head and neck to get an unobstructed clear airway, but no success was achieved. Resistance was felt in the bag when assisted ventilation was tried following each attempt. The value of partial pressure of end-tidal carbon dioxide (PETCO₂) was zero and tracing line was touching to the base of the capnograph. The whole event of correction of position and observation of respiration took about 2 min. Meanwhile, SpO₂ started dropping to a minimum of 92% on inspired fraction of oxygen (FiO₂) 0.5. PLMA was removed and no difficulty on mask ventilation was observed. Her trachea was then intubated with 5.5-mm-ID cuffed endotracheal tube under the effect of atracurium (0.5 mg/kg) and intermittent positive pressure ventilation (IPPV) was started. Following intubation, anaesthesia was maintained with oxygen and nitrous oxide (1:1) with halothane (0.5-1%) through Bain’s circuit. After tracheal intubation, PETCO₂ initially observed was 47 mmHg, which later on came down to 42 mmHg with normal waveform of capnograph. However, there was no improvement in SpO₂ even on IPPV with FiO₂ 0.5. Surgery was continued. On auscultation of chest, extensive coarse crepts were observed bilaterally. After few minutes, pink frothy secretions appeared in the endotracheal tube and breathing circuit with further fall in SpO₂ to a minimum value of 86% on FiO₂ 1. Clinical diagnosis of pulmonary oedema was made and managed with diuretic, narcotic and IPPV under atracurium.

After surgery, she was put on ventilator for IPPV with positive end expiratory pressure (PEEP) in ICU. She responded well to the therapy. She was weaned off from ventilator and her trachea was extubated after 11 h of mechanical ventilation. Her 12-lead electrocardiogram, arterial blood gas analysis (PaO₂ 111 mmHg, PaCO₂ 31 mmHg, pH 7.37) and X-ray chest were within normal limits. She was discharged from ICU after 24 h in a satisfactory condition.

**DISCUSSION**

NPPO following upper airway obstruction is well documented.[1,2] Multiple causes including airway devices, e.g. cLMA and PLMA have been reported to cause upper airway obstruction.[3-5]

The mechanism of pathogenesis of NPPO is multifactorial.[6] Creation of marked negative intrathoracic pressure by the forceful inspiratory efforts against an obstructed glottis or upper airway promotes increased blood flow to the pulmonary circulation, thereby increasing the pulmonary arterial pressure.[6] Marked negative pressure also causes a rise in transcapillary pressure gradient that favours shift of fluid from the capillaries into alveoli. Also, the hypoxaemia due to upper airway obstruction triggers intense sympathetic discharge which results in increased pulmonary arterial constriction and pressure, damaging pulmonary capillaries with increased permeability which further worsens pulmonary oedema[6] [Figure 1]. Management of NPPO depends on severity of pulmonary oedema and hypoxia. Effective airway management and immediate treatment with oxygen and diuretics is sufficient in most of the cases of NPPO.[6] Continuous positive airway pressure (CPAP) or PEEP is recommended in patients of NPPO requiring mechanical ventilatory support.[2,7]

Three mechanisms of upper airway obstruction caused by PLMA have been described: (1) PLMA tip (and drain tube) enters in the glottis, leading to obstruction; (2) sides of PLMA bowl fold inwards with glottic occlusion; and (3) PLMA tip behind the larynx compresses the posterior larynx causing arytenoid malfunction.[7] The above-mentioned problems also occur with cLMA, but are more frequent with PLMA due to its larger size and softer material.[6] Laryngospasm triggered by LMA and downfolding of epiglottis caused by LMA are also well-known causes of airway obstruction.[4,9]

Mechanical closure of the vocal cords with PLMA has also been observed in neutral position of head and neck despite easy and correct placement of PLMA that was relieved in sniffing position.[5] In our patient, proper sniffing position could not be achieved due to restricted neck movements and almost neutral position of the head might be the possible cause of upper airway obstruction with PLMA.

Stix et al. observed supraglottic or glottic obstruction caused by PLMA in 15 out of 317 patients and confirmed by fibreoptic examination that the mask was folded medially, a malposition that was also noted by Archie Brain using prototype masks of similar construction.[10,11] In our patient, fibreoptic examination could not be done due to desaturation. The cause of airway obstruction in our patient was supposed to be PLMA with restricted movement of
head and neck. The reason of restricted movement of head and neck was not clear.

On the basis of our observations, it may be concluded that airway obstruction caused by PLMA may lead to NPPO. Difficult sniffing position due to restricted head and neck movement may contribute to airway obstruction with PLMA.

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