CASE REPORT

Failed ventilation with LMA Proseal® in a patient with sleep apnea syndrome

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ABSTRACT

Patients with sleep apnea syndrome (SAS) have excessive adipose tissue in oropharynx which can obstruct the airway. A high prevalence of difficult intubation has been reported in these patients and an association between the severity of SAS and difficult intubation has been suggested. LMA Proseal* (LMA Company, USA) has been launched as a better alternative to LMA classic, as it provides good airway seal due to its modified cuff. The improved seal of LMA Proseal* (PLMA*) has an advantage in obese patients where higher airway pressures are required for positive pressure ventilation. We found that LMA Classic* provided better airway management than PLMA* in one of our obese patients with SAS.

Key Words: LMA Proseal* (PLMA*); LMA Classic*; Sleep apnea syndrome; Positive pressure ventilation; Supraglottic device

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INTRODUCTION

Obstructive sleep apnea is a syndrome, characterised by partial or complete obstruction of the upper airway during sleep due to inadequate pharyngeal muscle tone. These patients may have a compromised airway due to oropharyngeal narrowing as there is deposition of fat in collapsible segments of the airway. SAS severity is measured by the apnea hypoapnea index (AHI) which is defined as number of apnea / hypoapnea events per hour of sleep and the lowest oxygen saturation associated with an abnormal respiratory events during sleep. Theoretically, the larger the pharyngeal tissue, the higher the AHI will be. Patients with SAS have many implications for anesthesia. These patients usually pose a problem of difficult intubation as they have abnormal facial and upper airway morphology, e.g. retrognathia, short and thick neck and a large tongue.2 Hiremath et al found a high prevalence of SAS in patients with difficult intubation retrospectively.3 Sleep apnea results in fragmented sleep, hypoxemia, day time somnolence and altered cardiopulmonary functions.

PLMA® is a modification of classic® LMA. It has a larger and deeper bowl with no grille and cuff extends posteriorly to give a more effective seal around the glottis. It has a drainage tube running parallel to the airway tube which provides a bypass channel for regurgitated gastric contents and allows rapid diagnosis of mask misplacement. Since the airway tube of PLMA® is shorter than LMA Classic® and of similar calibre, airway resistance is 20% more than LMA Classic®. The PLMA® is superior to LMA Classic® for providing positive pressure ventilation and at a given cuff pressure, provides twice the seal pressure of the LMA Classic®. The improved seal is of advantage in obese patients where higher airway pressure are required for positive pressure ventilation.4

A patient of obstructive sleep apnea is reported where PLMA* failed to provide adequate airway approach as compared to conventional LMA Classic*.

CASE REPORT

A 51 year old male, weighing 90 kg, ASA grade II, with the diagnosis of gall stones and paraumbilical hernia was posted for cholecystectomy. He was a known case of SAS for 2 years. He also suffered from hypertension for two months and was on tab atenolol 50 mg and tab amlodipine 5 mg. His ECG showed ST segment flattening with T-wave inversion in V2-V5 leads. Chest x-ray revealed cardiomegaly. On general physical examination, he was found to have a short thick neck with a pulse rate of 80/min and B.P. 130/80 mmHg. Echocardiography revealed concentric left ventricular hypertrophy with ejection fraction of 54%. Symptoms of SAS were relieved with exercise and weight reduction. Pulmonary function tests revealed FEV1 3.5 L, FVC 4.04L, FEV1/FVC 86%, PEFR 297L/min and oxygen saturation 93% on room air. Polysomnographic studies revealed moderate apnea / hypoapnea episodes. A high risk informed consent was obtained. Antihypertensive drugs were ordered to be continued but no sedative drugs.

Standard monitoring e.g. electrocardiography, SpO, and non-invasive blood pressure, was applied. Preoxygenation was carried out with 100% oxygen for 6 minutes. Routine induction protocol was followed. Direct laryngoscopy revealed Cormack and Lehane grade 4 view. Intubation could not be achieved despite two attempts. Fibroptic bronchoscope was not available to help intubation. Further attempts on intubation were abandoned to avoid airway trauma and sympathetic stimulation leading to surges in blood pressure in this patient. PLMA® size 4 was inserted and correct placement was confirmed by gentle inflation. An orogastric tube could be passed through the drain tube and a drop of gel placed over the proximal end of drain tube ruled out mask malposition. Airway pressures and end tidal CO, were within normal limits. The surgery was started. Patient was ventilated by IPPV.

After about five minutes of placement, an increased resistance to ventilation was felt. Patient could not be ventilated adequately. EtCO₂ rose to 60-70 mmHg. Airway pressure increased to 40 cmH₂O. We considered it to be suboptimal positioning or an inappropriate size. An attempt at repositioning did not succeed in lowering the airway resistance. We replaced PLMA* size 4 with size 5. Gas leakage and resistance to ventilation was still noted. It was then replaced with LMA Classic* size 4. Fibroptic assessment could have been helpful, but it was not available. To our relief, the patient could now be ventilated adequately. Airway pressure dropped down and end tidal CO₂ also lowered

down to 40 mmHg. Rest of intraoperative period was uneventful. Surgery lasted for one hour. At the end of surgery, when patient was fully awake, LMA was taken out. Nasopharyngeal airway was placed for 24 hours postoperatively so as to avoid obstruction.

DISCUSSION

Controlled ventilation with tracheal intubation is the choice if general anesthesia with relaxation is the only available option. Nasal continuous positive airway pressure may be applied if airway obstruction persists, and it should be started before surgery and resumed immediately after extubation in cases of SAS.⁵

The cause of increased resistance to ventilation in our patient could be malpositioning, infolding of epiglottis or improper size. PLMA® provides more effective seal than LMA Classic® at same airway pressure⁴ due to the broader proximal cuff plugging the oropharynx more effectively and also the ventral cuff pressing the dorsal cuff firmly into periglottic tissues. 68

The difficulty in ventilation with PLMA® in our case could be explained due to epiglottis impinging in the lumen of the airway tube during insertion causing obstruction. This is not seen with LMA Classic® as it has aperture bars. Though downfolded epiglottis does not impede airflow with PLMA® due to presence of accessory vent, but in this case impediment could be significant due to excessive perilaryngeal tissues. In addition any supraglottic airway device (SGD) with a large inflatable hypopharyngeal component can cause mechanical airway obstruction by vocal cord closure secondary to glottic compression.8

Stacy et al have reported 20% incidence of airway obstruction with these airway management devices. They hypothesized epiglottic downfolding or mechanical cord closure.9 In its resting state, the hypopharynx is usually closed. Any device occupying the hypopharynx sufficiently to form a seal must open it and push the glottis anteriorly. This will inevitably cause glottic compression in patients with unfavourable anatomy. The incidence of mechanical cord closure is 0.4%.10 Over-enthusiastic insertion and inflation of the PLMA® cuff beyond its optimal position results in near complete airway obstruction, presumably because of forward displacement of the glottic inlet. Application of cricoid pressure with the LMA Classic® may also simulate the same condition. 11 Another cause of impaired ventilation could be infolding of the aryepiglottic folds. 12 We could not perform a fiberoptic assessment, though it would have provided important clues about the etiology of the obstruction. It is strongly

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recommended in the assessment of airway obstruction with SGD's whenever clinical circumstances allow. The best options in case of inadequate ventilation after insertion of a SGD are to remove and reinsert it, or to opt for a different size. We exercised both of these options but failed. However, the change of SGD (LMA Classic* for PLMA*) resulted in successful ventilation.

Sedation and narcotic based analgesia was avoided in postoperative period in our patient as it could exacerbate symptoms of sleep apnea. NSAID's and local infiltration at incision site is preferred.

CONCLUSION

PLMA* has specially developed to improve the seal and ensure effective ventilation, but in our patient with SAS, it failed in its stated purpose. The cause of its failure cannot be ascertained with certainty. In case of failure, either the size or type of the SGD should be changed.

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