An adult non-obese male, a case of obstructive sleep apnoea posted for tonsillectomy and septoplasty – challenges faced

INTRODUCTION

Obstructive sleep apnoea (OSA) is a disorder characterised by repeated disruption of breathing during sleep. Sleep apnoea is more common in males, obese,[1,2] those with large neck, smaller airway in the nose, mouth or throat. Children may have sleep apnoea due to enlarged tonsils or adenoids.[3] However, non-obese adults too may have enlarged tonsils causing obstruction, though rarely so.

This condition represents complex challenges like difficulty in mask ventilation, laryngoscopy and intubation, accelerated arterial desaturation, postoperative respiratory obstruction and problems regarding choice of analgesia.

CASE REPORT

A 30-year-old male patient with history suggestive of OSA was posted for tonsillectomy and septoplasty under general anaesthesia. He had history of recurrent attacks of rhinitis on and off over a year. Besides, he gave history of snoring severe enough to disturb the person next door, episodes of airway obstruction, probable hypopnoea during sleep and daytime somnolence. He was thus investigated and polysomnography done. The study showed minimum SpO2 values of 51% lasting up to 10 seconds, seven episodes of SpO2 <88%, total snoring episodes of 205 and mean duration of snoring episodes of 6 seconds. The average minimum O_2 saturation was 83.9%, maximum heart rate 141/min with an average of 96.4/min and desaturation index of 81.1. The AHI (Apnoea hypopnoea index) was significantly high at 80.0/hour. He had enlarged tonsils and left deviated nasal septum. On admission, a thorough preoperative evaluation was done. General and systemic examination did not suggest any gross abnormality. The blood pressure was 130/80 mmHg. The calculated BMI (body mass index) was 26.1 kg/m^2 and neck circumference 41cm. He had a high arched palate, large tongue [Figure 1] and an MPC (Mallampati classification) grading of 3. The thyromental distance was 6cm and sternomental distance was 11.7 cm. The routine investigations including the PCV (Packed cell volume), ECG (Electrocardiogram), CXR (Chest X-ray), thyroid function and coagulation studies were normal. After a thorough preoperative assessment, confirmation of NBM (Nil by mouth) status and written informed consent, the patient was taken up for tonsillectomy under general anaesthesia. He was premedicated with inj. atropine 0.6mg IM and inj. diclofenac sodium 75 mg IM half an hour prior to surgery. After securing the intravenous line, inj. ranitidine 50mg. and inj. ondensetron 4 mg were given. The patient was preoxygenated with 100% oxygen for five minutes and induced with gradually increasing concentration of sevoflurane up to 8% with 100% oxygen. Obstruction during mask ventilation was noted and an oral airway was thus inserted. After good jaw relaxation, decrease in muscle tone and depth of respiration, laryngoscopy was attempted. Laryngoscope was difficult to negotiate due to large tongue and only the epiglottis was visualised (Cormack and Lehane grade III), suggesting difficult intubation. Blind nasal intubation was done using a no. 7.5 cuffed endotracheal tube. The tube was secured and throat pack inserted. Maintenance of anaesthesia was done using o_2, sevoflurane and vecuronium as muscle relaxant. Enlarged tonsils were removed. After achieving haemostasis, checking adequacy of reflexes, tone, power and awake status, extubation was done after thorough suctioning under vision and pack removal. Inj. paracetamol 1gm IV was given postoperatively every 6 hourly over 24 hours. Intraoperatively, pulse rate, non-invasive blood pressure, SpO2 (oxygen saturation) and $EtCO_2$ (end tidal carbon dioxide) were monitored. Postoperatively, the patient was kept in right lateral position with oxygen by ventimask and nasopharyngeal airway *in situ*.

Figure 1: Large tongue, short neck

We monitored the pulse rate, blood pressure and oxygen saturation throughout the first postoperative night, off O_2 mask. Clinically, we found that the number of hypoapnoeic episodes had dropped, the minimum drop in O_2 saturation was 80% and the daytime somnolence was considerably lower the next day.

Septoplasty was deferred for the present as it would involve plastering the nose and further jeopardize respiration.

DISCUSSION

OSA has been recognized as a major contributor of mortality and morbidity in the developed countries. In India, studies show that in patients between 35-65 years of age, the prevalence is 19.7% in males and 7.4% in females.[4,5] A concern for increased perioperative risk caused the American Society of Anaesthesiologists (ASA) to recommend that patients be screened for risk of OSA preoperatively. One useful screening tool, the STOP-BANG model,[6] consists of 8 items. S- Snore loudly, enough to be heard through closed doors? T- Feel Tired or fatigued

during the daytime almost every day? O- Observed that you stop breathing during sleep? P- History of high blood Pressure, with or without treatment? B- (BMI) >35 kg/m^2 A- Age >50 years, N- Neck circumference >40 cm. G - Male Gender. Patients are considered to be high risk of having OSA if they have three or more of the STOP-BANG criteria. Our patient was a male, did snore loudly and observed to have almost stopped breathing during sleep. He complained of daytime fatigue and had a neck circumference of 41cm. He was therefore a high risk.[6,7]

The total number of apnoeas and hypopnoea per hour is called the apnea-hypopnea index (AHI). Values of 6-20, 21-50 and >50 indicate mild, moderate and severe OSA, respectively. Our patient had an index of 80/hour (severe category).

Perioperative optimization with CPAP (Continuous positive airway pressure) or BiPAP (Bi-level positive airway pressure) should be considered, if OSA is severe.[8,9] However as our patient was not on home CPAP and was posted for tonsillar resection as a mode of correction, we kept it as an option postoperatively. An airway surgery under general anaesthesia falls into the high-risk category.[10]

The patients with OSA may be exquisitely sensitive to all central nervous system depressant drugs,[10] with a potential for upper airway obstruction or apnoea with minimal doses. Hence, preoperative benzodiazepines or opioids should be avoided. Here, we gave non-sedative analgesic intramuscular diclofenac sodium preoperatively and intravenous paracetamol postoperatively. Intubation can be done with the patient awake or under anaesthesia. It is important to fully preoxygenate the patient because they desaturate rapidly. In our patient, mask ventilation was difficult due to presence of large tongue and slight receding mandible. Inserting an oral airway helped.

Propofol may be used for induction of anaesthesia with its fast onset, short duration and good relaxation. However, it will reduce the pharyngeal muscle tone causing obstruction and may cause apnoea.[10] Intraoperative monitoring including SpO_2, $EtCO_2$ and ECG is important due to increased chances of hypoxia, hypercarbia and arrhythmias. Beside death from airway obstruction, another great danger is rapid development of negative pressure pulmonary oedema. Our patient was extubated in the operating room when fully awake with a nasopharyngeal airway *in situ*.

CONCLUSION

Thus a case of severe OSA was managed successfully after thorough understanding of the possible perioperative problems and taking care of all the challenges faced therein.

Pradnya M Bhalerao, Sunita M Khedkar, Vijay H Patil, Bhausaheb R Kawade

Deartment of Anaesthesiology and Critical Care, B. J. Medical College and Sassoon General Hospitals, Pune, Maharashtra, India

Address for correspondence:
Dr. Pradnya Bhalerao,
D-15/10-11, Saritanagari-2, Off, Sinhagad Road,
Pune - 411 030, Maharashtra, India
E-mail: dr.pradnyabhalerao@gmail.com

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