Anesthetic consideration in patients with Obstructive Sleep Apnoea (OSA) for Modified Radical Mastectomy: Report of two cases for publication as a case report

A Kulkarni, U Sharma

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Abstract

To describe anaesthesia management of two patients with obstructive sleep apnea (OSA) for modified radical mastectomy (MRM). OSA is a sleep disorder characterized by periodic, partial or complete obstruction of the upper airway during sleep. Anaesthetic implications include the presence of comorbidities involving cardiovascular, respiratory and cerebrovascular system, morbid obesity, difficult airway and increased susceptibility to potential pulmonary complications in the postoperative period. The first patient was diagnosed case of OSA, using nasal CPAP at night for 6 years, Awake fibreoptic bronchoscopic intubation was performed as patient had difficult airway followed by conventional GA. Postoperative after extubation Nasal CPAP was used. The second patient had a history suggestive of OSA, BMI 43.7 kg/m2 and neck circumference 17 inches, she was treated as moderate OSA, Unilateral paravertebral block, followed by conventional GA was given. To conclude anaesthesia management of patients with OSA requires preoperative high index of suspicion, airway assessment, cautious use of narcotics, use of regional anaesthesia if feasible, postoperative oxygenation monitoring and availability of CPAP.

CASE REPORT

A 66 year old female, weight 69 kg, BMI 30.66 kg/m2 diagnosed as carcinoma breast was posted for right modified radical mastectomy MRM. On preoperative evaluation she gave history of snoring and excessive daytime somnolence six years back. Patient underwent Polysomnography, which revealed Apnoea / hypopnoea Index of 68.3 suggestive of severe obstructive sleep apnoea syndrome. During the sleep study her average minimum oxygen saturation was 77.1% and lowest recorded was 61%. She was advised nasal CPAP at night, which she was regularly using for last 6 years and was, relieved of her OSA symptoms. The patient had hypertension for 8 years controlled with tablet Enarapil maleate 5mg OD. On General Examination her heart rate was 88/min. Blood pressure was 130\70 mmHg. Airway examination revealed Mallampatti classification III, neck circumference was 15.6 inches. Pulmonary function Test was suggestive of moderate airway obstruction, room air Arterial Blood Gas analysis revealed PH -7.46 PaO2-59.9mmHg, PaCO2- 38.3 mmHg, O2 Sat 92.4%.

The patient was premedicated with Tablet ranitidine 150mg and Tablet Ondensetrone 16mg 2 hours prior to surgery.

Awake nasotracheal fiberoptic bronchoscopic intubation was done after achieving topical anaesthesia of the airway by nebulising her airway with 5ml 4% lignocaine. After placement, of endotracheal tube anesthesia was induced with IV Morphine 0.5mg/kg $^{-1}$, fentanyl 1µg/kg $^{-1}$, propofol 1 m/kg $^{-1}$, atracurium 0.5 mg/kg $^{-1}$. Anaesthesia was maintained with O2/N2O 33/77% sevoflurane and intermittent doses of fentanyl and atracurium with controlled IPPV. Monitoring included 5 lead ECG, SPO2, NIBP, ETCO2, and Spirometery. During surgery heart rate and blood pressure were within acceptable limits. At the end of the procedure surgical incision site was infiltrated with 20 ml 0.25% bupivacaine. Postoperative analgesia was managed with IV morphine 3 mg as per requirement and Diclophenac sodium 75 mg 8 hourly. The patient was oxygenated through T piece after the surgery, and her trachea was extubated 4 hours after the end of surgery, when the patient was conscious, communicative and breathing spontaneously adequate tidal volume. ABG analysis was within normal limits. She received Nasal CPAP after extubation, and had an uneventful recovery.

Second patient was a 44-year-old female, 105 kg (BMI 43.7

Kg\m ^{2}), posted for left MRM. She had history of snoring and excessive daytime somnolence. Sleep studies were not available. Patient was hypertensive for 5 years receiving Tablet Stamlobeta 50mg OD. She was investigated for weight gain 4 years back and diagnosed with hypothyroidism, for which she was receiving Tablet Eltroxin 50 µg OD. On preoperative evaluation, her heart rate was 76/minute, BP was 140\90 mmhg. Airway evaluation revealed Mallampatti classification II, Neck circumference was 17 .0 inches. Thyroid function tests were within normal limits. Echocardiography revealed LVEF 60%, Normal study. Preoperative ABG analysis breathing room air showed normal Pa CO2 tension.

The patient received Tablet Ranitidine 150mg and Tablet Ondensetron 16 mg 2 hours prior to surgery as premedication. In the OT the patient had a heart rate 82/minute, BP 114/66 mmHg. Left Paravertebral block was given at T3-T4 space by 22G spinal needle with 18ml 0.5% bupivacaine ,in sitting positon. T1-T6 dermatome block was confirmed before induction of anaesthesia. Sheets were kept below upper chest to achieve optimum position for intubation, patient was preoxygenated with 100% Oxygen for 3 minutes, orotracheal intubation was performed with cuff portex ETT (7.0 ID) in first attempt. On confirming tracheal placement of the ETT with capnography anesthesia was induced with IV midazolam 1 mg, fentanyl 0.75µg/kg $^{-1}$, propofol 0.5 mg /kg $^{-1}$ and atracurium 0.4mg/kg-1. Monitoring included 5 lead ECG, SPO2, NIBP, ETCO2, Spirometery. Anaesthesia was maintained with O2/N2O 33/77%, sevoflurane, intermittent doses of fentanyl and atracurium with controlled ventilation. During surgery one episode of decrease in systolic blood pressure (SBP) to 70 mmhg was noted and treated effectively with IV ephedrine 6mg. Trachea was extubated 50 minutes after the completion of surgery in the post operative ward, when the patient was awake, breathing adequate spontaneous tidal volume and maintaining oxygenation. Post operative pain was managed by the paravertebral block and NSAIDS, narcotics were not required. Oxygen saturation was monitored at night and Nasal CPAP was kept available. The patient had an uneventful recovery.

DISCUSSION

Obstructive sleep apnoea is defined as the absence of airflow for more than ten seconds despite continuing ventilatory efforts, five or more times per hour of sleep with associated decrease in arterial oxygen saturation (Sao2) of more than 4% [1].Obstructive Sleep Apnoea Syndrome (OSA Syndrome) is defined as recurrent apnoea or hypopnoea that are associated with clinical impairment as manifested by increased sleepiness or altered cardiopulmonary function.[1] Young et al in a community-based study of workers estimated 2% of women and 4% of men had symptoms sufficient to meet the criteria for OSA Syndrome. The prevalence of clinically diagnosed OSA in middle aged adults showed that at least 80% of moderate to severe OSA remain undiagnosed. Obesity (BMI >28 Kg/m2) is present in 60-90% of OSA patients evaluated in sleep clinics.[2]

A narrow floopy upper airway provides the pathophysiological basis for OSA. Abnormal anatomy of the pharynx, increased collapsibility of the upper airway and defective airway reflexes interact to produce OSA. Obesity is usually associated with OSA, the deposition of fat in the lateral pharyngeal wall results in decreased potency of the pharynx. This increases the risk of collapse particularly during sleep, when there is relaxation of the pharyngeal dilator muscles. Decreased upper airway activity with sleep (REM) leads to pharyngeal narrowing, first the structures tend to vibrate as turbulent flow patterns are produced resulting in snoring. Secondly the pharynx will tend to collapse due to Bernoulli effect with partial or complete obstruction. Hypercapnia and hypoxia follow which leads to arousal and pharyngeal opening due to increase in upper airway muscle activity. The improved airflow decreases carbon dioxide tension and improves oxygen saturation .The cycle repeats with sleep onset.[1,3]

Patients with OSA when subjected to sedation and anaesthesia are at risk of developing respiratory and cardiovascular complications postoperatively. Due to morphological alteration of upper airway, securing and maintaining a patent airway is difficult.[4] Preoperative evaluation should focus on history, signs and symptoms suggestive of OSA, especially in patients who are not diagnosed as OSA. Patients with diagnosed OSA who are being treated with CPAP should be informed to take their equipment to the hospital for postoperative airway management.[3] we had informed our first patient about the same, as she was a diagnosed case of severe OSA, treated with Nasal CPAP at night for six year.

Second case was suspected to have OSA, as she had history of heavy snoring, day time excessive somnolence, the physical characteristics suggestive of OSA were BMI 35kg/m2, Neck circumference > 16 inches . Sleep studies

were not available, but in view of more than two signs and symptoms as suggested by ASA Practice guidelines such patients should be treated as though they have moderate OSA. The ASA practice guidelines proposes the scoring system, which may be used to estimate whether a patient is at increased perioperative risk of complications from OSA .The scoring system depends on severity of sleep apnoea based on sleep study, invasiveness of surgery and anaesthesia, requirement of postoperative opioids and estimation of perioperative risk. Patients with a score of > 5 are at significantly increased perioperative risk from OSA.[5]. We measured ABG breathing room air in both the cases, If resting PaCO2 >50mmHg, one point should be added to the scoring system. Three month of treatment with CPAP may reverse the OSA induced cardiovascular and metabolic syndrome. CPAP when used consistently prior to surgery, one point should be subtracted .[5]

Preoperative assessment of risk that tracheal intubation may be difficult should be done, depending on Mallampati Staging system, increased neck circumference (> 17 inches for male and > 16 inches in case of female),and thyromental distance.[4] Benzodiazipines were avoided as premedication as they have relaxing effect on the upper airway musculature, causing an appreciable reduction of the pharyngeal space and consequently a higher risk of preoperative hypopnoea and hypoxia. The patients received oral ranitidine and Ondensetron to neutralise gastric acidity and prevent vomiting as OSA patients who are morbidly obese are at increased risk for aspiration of gastric fluid during induction of anesthesia.[6]

Anaesthetic management of the patient with OSA.The choice of anaesthetic technique sways in favour of Regional anaesthesia (RA) when possible as RA enables the patient to retain conscious control of respiratory function. A combined regional /General anaesthesia has also been suggested.[1] The second patient, with Moderate OSA received unilateral Para vertebral block followed by conventional General anaesthesia with orotracheal intubation and controlled ventilation. Optimisation of intubation position and preoxygenation with 100% Oxygen was done as obese patients with decreased FRC and increased oxygen consumption are at increased risk of hypoxia at induction. McCoy blade laryngoscope and gum elastic boogie were kept ready. Patient was intubated without any difficulty. Para vertebral block decreased the amount of narcotic requirement during surgery and also provided good postoperative analgesia; hence narcotics were not required for postoperative pain relief. The ASA practice guidelines for the postoperative pain management recommends regional analgesia as it decreases the adverse pulmonary outcomes compared to systemic opiods. The first patient was a diagnosed case of severe OSA, Mallampati grade III and Neck circumference 15.6 inches, in view of anticipated difficult intubation awake nasal fibre optic bronchoscopic intubation was performed followed by conventional general anaesthesia with controlled ventilation. For both patients anaesthesia was maintained with short acting agents fentanyl, sevoflurane and atracurium, to ensure prompt recovery of consciousness and upper airway integrity at the conclusion of surgery. Patients were monitored in the Post Anaesthesia Care Unit (PACU) and extubated only when fully awake, breathing spontaneously adequate tidal volume and maintaining oxygenation. The first patient received Nasal CPAP after extubation .It is recommended that patient who use CPAP preoperatively should use CPAP postoperatively as it may reduce the risk of airway obstruction and respiratory depression . N-CPAP resumed immediately after extubation allows free use of sedative and analgesic, without major respiratory complications.[7] Postoperative pain was managed with NSAIDS and IV Morphine as per requirement.

CONCLUSION

Patients with obstructive sleep apnoea are at risk of developing postoperative cardio respiratory complications when having surgery under general anaesthesia. Anaesthetists should be aware of the fact that undiagnosed OSA is common. In the case of a medical history suggestive of OSA,particularly in obese patients with a short bulky neck and a large tongue ,full night polysomnography should ideally be done before surgery takes place. Preoperative evaluation should also focus on airway assessment and if possibility of difficult intubations exists awake fibreoptic bronchscopic intubation should be considered. Nasal CPAP should be used after extubation in diagnosed patients of OSA using Nasal CPAP preoperatively. Regional analgesia, NSAIDS are preferred over narcotics for postoperative pain management. Postoperatively patients should be monitored in the postoperative care unit for oxygenation. CPAP should be kept available .

CORRESPONDENCE TO

Dr Anita Kulkarni, M.D. Consultant Anaesthesia - Rajiv Gandhi Cancer Institute and Research Centre, Sector V ,

Rohini , Delhi –110085(India) Address: C-701, KM Apartments, Plot No12, Sector 12, Dwarka Delhi-110078 (India) Email: anitackulkarni@gmail.com Phone no (Mobile) 09891064785

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Author Information

Anita Kulkarni, M.D.
Consultant Anaesthesia, Rajiv Gandhi Cancer Institute and Research Centre

Utpal Sharma, D.A.
Senior Resident, Anaesthesia, Rajiv Gandhi Cancer Institute and Research Centre