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Obese Patient with Undiagnosed Obstructive Sleep Apnea – A Cause of Post Operative Co2 Retention

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Abstract: *Morbidly obese patient pose a real challenge for anaesthesiologist as there is increased incidence of intra operative and postoperative complications in these patients. We present a case of 60 years old female weighing 110 kg (BMI-41) with carcinoma of breast posted for left modified radical mastectomy. In post operative period she retained co2. Her intraoperative and post operative management and possible causes of co2 retention are discussed. Many factors can contribute to this postoperative co2 retention. Undiagnosed sleep apnea should also be kept in differentials of causes of postoperative co2 retention especially in obese patients.*

Keywords: *obese, sleep apnea, co2 retention*.

I. Introduction

The prevalence of obesity continues to increase despite preventive strategies. Morbidly obese patients pose a real threat for anesthesiologist as there is increased incidence of intraoperative and postoperative complications in these patients. Obstructive sleep apnea (OSA) is frequently associated with obesity. OSA is defined as the occurrence of at least five apnea/ hypopea episodes in one hour. Symptoms typically associated with OSA include snoring, excessive day somnolence and restless sleep[1]. OSA frequently coexists with hypertension, congestive heart failure, myocardial infarction, arrhythmias and stroke[2]. It carries increased risk of difficult intubation, post operative respiratory depression and airway collapse leading to hypoxia and hypercarbia[3]. But around 80 % of these patients remain undiagnosed.[4] It is crucial for anesthetist to screen every operative patient carefully for this disorder and to have a strategy for perioperative care.

Case Report

Sixty years old female weighing 110 kg with a height of 162 cm (BMI-41) was scheduled to undergo left modified radical mastectomy for carcinoma of breast. Preoperative evaluation revealed that she had paraparesis since last 10 years. So, exercise tolerance could not be assessed. She had no history of diabetes mellitus, asthma, hypertension, IHD and seizures. She did not give any history suggestive of obstructive sleep apnea. On examination, her vitals were within normal limits. She had adequate mouth opening and class three mallampati score. But she had short and thick neck. Her all preoperative investigations including ECG and X ray chest were within normal limits. She was premedicated with tab ranitidine 150 mg orally night before surgery and 2 hrs prior to surgery and kept fasting for 8 hrs prior to surgery. On the day of surgery, all equipments for management of difficult airway were kept ready. After attaching standard monitors, 18 G peripheral canula was placed on her left hand. Patient was oxygenated with 100% O_2 in head up position for 3mins and induced with inj. tramadol 100 mg iv and inj propofol 2mg/kg iv. Inj. Succinyl choline 2mg/kg iv was given after checking adequacy of mask ventilation and airway secured with cuffed 7.5mm ID endotracheal tube. Anaesthesia was maintained with N_20 66% in o2 and 1-1.5% sevoflurane and ventilation was controlled to maintain normocapnia. Continuation monitoring of ECG, O2 saturation ETCO2 and NIBP was done. The surgery lasted for 2-.5 hrs. Patient was hemodynamicaly stable during intraoperative period. At the end of surgery, residual neuromuscular blockade was reversed with inj.atropine 0.02mg/kg and inj.neostigmine 0.05mg/kg IV. Patient was responding to verbal commands, had good respiratory efforts with adequate tidal volume and was maintaining her vitals in the normal range. On observing these findings, we extubated the patient and put on mask ventilation. But after few minutes of extubation, patient consciousness got depressed and Spo_2 started falling. Patient was given inj. Propofol 80mg iv and reintubated. Serial BGA showed increased PCo_2 and decreased Pao_2. Patient was shifted to Intensive Care unit (ICU). In ICU, patient was ventilated with IPPV mode with 10 cm H_2O Positive end expiratory pressure (PEEP). On Blood gas analysis, patient PCO_2 got normalized gradually. She was extubated on 2nd post op day and was given Bilevel positive airway pressure

(BIPAP) ventilation till third post operative day. She was transferred back to ward on the 4th postoperative day after she had recovered spontaneous breathing and sufficient muscle strength. Further post operative period was uneventful and she went home on 7th postoperative day.

II. Discussion

Obesity is an important health problem with constantly increasing incidence. It leads to coronary artery disease, hypertension, dyslipidemia, diabetes mellitus, degenerative joint disease and obstructive sleep apnea (OSA).[5] About 60-90% of patients with OSA has BMI>30 kg\m2.[6] But nearly 80-90% of OSA patients are undiagnosed[6,7.] The diagnosis can be elusive as the symptomatology of sleep apnea may be difficult to distinguish from normal variations in sleep behavior. Clinical examination carries a diagnostic sensitivity and specificity of only 50-60% for sleep apnea, even when performed by experienced sleep physicians.[8] Polysomnography remains the gold standard for diagnosing OSA. However restricted access and practical application may limit its utility in the preoperative setting. At present the optimal preoperative screening tool to assess for the presence and severity of OSA is not known.[9] Undiagnosed OSA patients may be associated with increased morbidity and mortality in perioperative period. These patients have a higher incidence of difficult intubation, post operative respiratory depression, airway collapse, increased admission to ICU and longer hospital stay.

In our case also, patient did not give any history suggestive of OSA preoperatively despite having morbid obesity. We screened our patient for suspected OSA by using stop-bang questionnaire[10,] but our patient was found at low risk for OSA (<2 on stop, <3 on stop- bang screening). As stated above, all screening tests have limited ability to detect undiagnosed sleep apnea. So some patients may be missed in preoperative screening like ours. In these patients sleep apnea may first be diagnosed intraoperatively if patient have problems with maintenance of the airway, proves difficult to intubate or observed post operatively to be snoring and having obstruction[9], as was found in our patient. An airway obstruction that is out of proportion to the apparent degree of sedation can suggest undiagnosed sleep apnea.[11] The inherent collapsibility of the pharynx predisposes to impaired respiration when regulation of pharyngeal muscle is depressed or impaired during sleep and anaesthesia.[12] Similarly in our patient, post op residual effect of anesthetic agents aggravated the underlying sleep apnea and led to post op airway obstruction, CO_2 retention and hypoxia. We reintubated the patient and ventilated with assist control mode with 10 cm H_2O (PEEP) and gradually weaned her from ventilator. Then kept her on non invasive ventilation for one day under continues monitoring and when serial BGA monitoring showed normal PaO_2 and PCO_2, she was shifted to ward.

To conclude, routine screening may miss obstructive sleep apnea patients. But in obese patients we should have higher suspicion of OSA even with normal screening results. In cases with suspected sleep apnea, limit use of sedative and narcotics in perioperative period. For pain control use of alternative forms of analgesia, such as NSAIDS, nerve blocks or local anesthetics should be considered. These patients should be monitored carefully in perioperative period for any hypoxia or hypercapnea. Diagnosed cases may be given Continuous positive airway pressure (CPAP) in perioperative period depending upon risk and ASA guidelines should be followed for further management.

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