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Effect of High Spinal Unmasking Undiagnosed OSA

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ABSTRACT *Obstructive sleep apnea (OSA) is the most common type of sleep apnea and is caused by obstruction of the upper airway. It is characterized by repetitive pauses in breathing during sleep, despite the effort to breathe, and is usually associated with a reduction in blood oxygen saturation. These pauses in breathing, called "apneas".1*

Obstructive sleep apnea is an often underdiagnosed condition where a patient's upper airway obstructs during sleep at night.2,3 Not only can resultant chronic hypoxia make patients susceptible to various illnesses but their quality of life may be severely impaired.3,4 Our case report of a 60 yr old male posted for open reduction and internal fixation of fractured left neck of femur serves to illustrate how a patient's previously undiagnosed obstructive sleep apnea was uncovered during high spinal blockade.

Case report

A 60yr old male, weight 105 kg and height 172 cm (body mass index 35.4 kg·m–2), posted for open reduction and internal fixation of fractured left neck of femur. Past H/O k/c of HTN controlled on T. Amlodipin 5mg OD since past 6-8 months. The patient had a history of fall which resulted in a fracture NOF. After a thorough pre-anaesthetic evaluvation patient was planned for spinal and epidural anaesthesia. Electrocardiogram, pulse oximeter, and non-invasive blood pressure monitors were attached. The epidural catheter was placed at the L3–L4 inter-vertebral space, catheter was easily inserted and no blood, cerebrospinal fluid, or paresthesia was noted. Spinal anaesthesia was given at L4-L5 inter-vertebral space with 0.5% of 3.4ml hyperbaric bupivacaine + 30micrograms clonidine. Initially T1 sensory level was achieved, which receded to a T4 sensory level at approximately one hour and 45 min into the surgery. Capnography was used to monitor the patient's pattern of breathing throughout the case. During the course of the surgery, 4-5 apneic episodes were noted, often associated with respiratory efforts. The events occurred on a regular basis and would be preceded by the patient drifting asleep and closing his eyes. However, during each apneic episode, the patient remained easily arousable and would continue to breathe normally until the next apneic event. When aroused, the patient would be alert, oriented, and responsive. The frequency of apneic spells declined as the block receded.

Throughout the surgery vital remained stable, with a heart rate between 64 to 90 beats·min–1, blood pressure varied from 80/50 to 136/84, and oxygen saturation between 97 to 98% on 5-6 L·min–1 oxygen by mask. Mephentermine 3 mg iv was administered on three occasions to treat mild hypotension which recovered immediately. The entire surgical procedure lasted 2 hours and 20 min without any intraoperative complications. The patient was then transferred to the PACU in stable condition. Epidural bupivacaine 0.125% was administered at a rate of 5 mL·hr–1 for pain control. No opioids were given. During the 2hr PACU stay, an apneic episode was observed, resulting in a desaturation to 92%. Oxygen saturation on the ward ranged from 96% when the patient was experiencing pain to 93% when the patient was pain free.

A postoperative respirology consultation was advised in view of a high suspicion of obstructive sleep apnea, also on further queries it was revealed that patient snores, had difficulty in staying awake during the day, choking at night, awakening from sleep multiple times during the night, morning fatigue, and memory loss.

The sleep study performed was consistent with severe obstructive sleep apnea with a 71% sleep efficiency and an apneahypopnea index of 62.6. The oxygen saturation nadir reached 86%. There was no improvement with changes in position. All stages of sleep were entered. The patient was started on 10 cm H2O nasal continuous positive airway pressure resulting in considerable symptomatic improvement, and was eventually titrated up to14 cm H2O in gradual increments.

Discussion

The report showed that how high neuraxial block can flare up the undiagnosed obstructive sleep apnea which makes it necessary to uderstand the relation between obstructive sleep apnea and neuraxial anesthesia.

Patients with obstructive sleep apnea have a tendency to obstruct their airway during sleep and somnolent states. The pathophysiologic mechanism includes several factors such as relaxation of the pharyngeal muscles in combination with a variety of upper airway abnormalities.[1,3]

In addition, a large percentage of obese patients, as was the case with this patient, have underlying obstructive sleep apnea.[1,2,3] In this setting, the peripheral stimulation required for the patient to maintain an awake state was severed by the spinal anesthetic. The result of this deafferentation caused him to enter a plane of somnolence, therefore allowing his obstructive sleep apnea to manifest as it does during sleep.

There are evidence that spinal anesthesia in itself produces sedation. Pollock et al.5 demonstrated that in unpremedicated patients receiving spinal lidocaine, both the Assessment of Alertness / Sedation Scale and a self sedation scale diminished in comparison with patients receiving a spinal placebo. A proposed mechanism for this effect is de-afferentation from proprioceptive and sensory stimuli to the central nervous system caused by spinal blockade; these inputs are likely involved in maintaining patients in the awake state. This may have explained the tendency of this patient to experience repeated apneas during the course of his spinal anesthetic.

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Gentili et al.6 studied groups of patients receiving hyperbaric bupivacaine and found an increasing degree of sedation as the level of the spinal block became higher. A second group of patients, in whom midazolam 1 mg iv increments were added, revealed an additive effect on sedation.

Epidural analgesia has also been associated with decreased minimum alveolar concentration requirements when combined with general anesthesia,[7] again supporting the deafferentation phenomenon. If one extrapolates this finding to the postoperative period where local anesthetic infusions are used for analgesia, this may partially explain the observation that this patient's saturation decreased on the ward when pain was well controlled (reduced afferent signals).

There are reports in the literature[8] regarding the use of neuraxial opioids in patients with known or suspected obstructive sleep apnea. Perioperative opioids in this patient population have been associated with adverse events, including respiratory and circulatory arrest. This effect is not isolated to the immediate postoperative period. Opioids were not included as part of the anesthetic management in this case, either intraoperatively or postoperatively. Despite this, the patient still experienced apneic events, thereby eliminating opioid use as a cause of sedation. The avoidance of opioid administration may, in fact, explain why the patient did not experience severe apnea to the point of desaturation, and he remained easily arousable. Other causes for increased somnolence were excluded. The patient did not receive appreciable doses of sedative medications, the hemodynamics remained stable ensuring adequate brain perfusion, arterial oxygen saturation remained unchanged, and an intraoperative glucose measurement was normal. This case serves to underline the importance of considering additional intraoperative monitoring. Evaluation of respiration for regional anesthesia is required, capnography for regional anesthesia is not mandatory. Observing the chest can be difficult, especially when full-body drapes are applied and the chest is not directly visible. Capnography to assess the pattern of breathing is easy to apply, and provides the additional benefit of an apnea alarm. Our experience leads us to suggest that all patients who have received neuraxial anesthesia should be monitored with capnography, regardless of their past medical history. This is particularly true if sedation is required, as it has been shown that patients with spinal anesthetics exhibit increased sensitivity to sedating agents.[5,9]

Conclusion : Obstructive sleep apnea is a disease whose primary pathology manifests when the patient is either asleep or in a state of altered arousal, where the upper airway can obstruct. This situation can be replicated with a high spinal anesthetic, where deafferentation of peripheral stimuli may result in an altered level of arousal. This is an important consideration, as the disease may manifest itself for the first time during the course of anesthesia. We suggest complete respiratory monitoring should be applied for cases involving neuraxial regionalanaesthesia.

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