

**DELAYED EMERGENCE FROM ANESTHESIA ASSOCIATED WITH UNDIAGNOSED  
CENTRAL SLEEP APNEA SYNDROME****Isil DAVARCI\*, Celalettin DAGLI\*\*, Murat KARCIOGLU\*, Kasım TUZCU\*, Selim  
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Anestezi sonrası derlenmede gecikme; anestezi ilaçlarının rezidüel etkisi, narkotik ve sedatiflere karşı aşırı reaksiyon, rezidüel nöromusküler blokaj ve cerrahi süreye bağlı olarak multifaktöriyeldir. Hiperkapnik santral uyku apne sendromu, sıklıkla solunum merkezi disfonksiyonuna yol açan diğer hastalıklara ikincil olarak gelişen santral hipoventilasyon sendromlarında sık görülür. Burada, yedi yıl önce pons kavernomundan opere olan, 36 yaşındaki hastada pilonoidal sinus cerrahisi sonrası gözlenen geç derlenme ve genel anesteziyi izleyen beklenmeyen respiratuar depresyon olgusu sunuldu. Bu durumun daha önce tanı almamış hastada santral uyku apnesine bağlı olduğu düşünüldü. Hasta ayrıntılı tanı için uyku laboratuvarına yönlendirildi ve santral uyku apne sendromu tanısı konarak ev tipi CPAP cihazı tavsiye edildi. Sonuçta, beyin sapı cerrahisi geçirme hikayesi olan hastalarda santral uyku apne sendromu olabileceği akılda bulundurulmalı ve cerrahi öncesi dikkatli muayene yapılmalıdır.

**ABSTRACT**

Delayed awakening from anesthesia is often multifactorial including residual anesthetic drugs, excessive reaction to narcotics and sedatives, residual neuromuscular blockade and duration of surgery. Hypercapnic central sleep apnea syndrome is often encountered at central hypoventilation syndromes which may be secondary to other diseases that cause respiratory centre dysfunction. In this case, 36 years, which had operated from pontine cavernomas seven years ago, delayed awakening and unanticipated respiratory complications following general anesthesia after pilonidal sinus surgery was reported. This disorder may be due to central sleep apnea syndrome in a patient without previous diagnosis. He was referred to the sleep laboratory in aspect of the differential diagnosis. The patient was diagnosed as central sleep apnea syndrome and home device for CPAP was recommended. The patient who had a history of brainstem surgery should be taken into consideration for the candidate of central sleep apnea syndrome, and a meticulous evaluation should be done before surgery.

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## INTRODUCTION

Delayed awakening from anesthesia is often multifactorial including residual anesthetic drugs, excessive reaction to narcotics and sedatives, residual neuromuscular blockade and duration of surgery. In some cases, this disorder may be due to undiagnosed metabolic or neurological disorders (1).

Hypercapnic Central Sleep Apnea Syndrome (CSAS) is often encountered at central hypoventilation syndromes which may be secondary to other diseases that cause respiratory centre dysfunction. This disorder may be due to an undiagnosed neurological disorder and it can present with delayed awakening (1, 2).

In the present case, delayed awakening and unanticipated respiratory complications following general anesthesia after pilonidal sinus surgery was reported. This disorder may be due to CSAS in a patient without previous diagnosis. To our knowledge, this is the first case in terms of delayed awakening due to undiagnosed CSAS.

## CASE

A 36-year-old male patient was planned to have surgical operation due to pilonidal sinus. In the patient past history, he had panic disorder and a history of brain stem surgery due to pons cavernous malformation seven years ago. There was no abnormal finding in aspect of physical examination. The laboratory tests, electrocardiography and respiratory function tests were in normal limits. His body mass index was  $23.9 \text{ kg/m}^2$ , blood pressure, heart rate and SpO<sub>2</sub> were 110/70 mmHg, 65bpm, and 99% by monitoring, respectively. Propofol (160mg) and fentanyl (100 $\mu$ g) were given as an anesthetic agent, and the intubation was performed after giving succinylcholine (100 mg) for the muscle relaxation. Anaesthesia was maintained with sevoflurane and nitrous oxide in oxygen. After the succinylcholine administered, rocuronium (20 mg) was used for the maintaining muscle relaxation. The patient's vital signs were normal during the peroperative period.

At the end of the surgery, the residual neuromuscular blockade was reversed with atropine (0.02 mg /kg) and neostigmine (0.06 mg/kg), and then the patient was extubated. But, suddenly after extubation, the patient became apneic and needed reintubation in the operating room. The patient was observed with mechanically ventilated in the recovery room

approximately one hour. Neostigmine (1mg) and atropine (0, 5 mg) were repeated when he had problems regarding the tracheal tube and then the patient was extubated. But again he had insufficient spontaneous breathing, tendency for sleeping and apnea needed excitation for respiration and it is suggested that this might be due to the prolonged effect of opioid. Therefore, 20 µg doses of naloxone therapy were given with 5-minute intervals as a possibility for delayed awakening. Arterial blood gas analysis at this time showed severe respiratory acidosis despite adequate oxygenation: pH 7.22, PaO<sub>2</sub>: 90 mmHg, PaCO<sub>2</sub>: 88 mmHg, SpO<sub>2</sub>: 94%. But, the patient's situation did not change and again required tracheal intubation and he was hospitalized at the intensive care unit (ICU), connected the mechanical ventilator in continuous positive airway pressure (CPAP) mode.

The patient was comfortable and tolerated intubation without sedation and interestingly observed that he could breathe only with stimulation. The case was extubated due to lack of apnea periods during 4 hours. He had detailed consultations for the etiology of apnea. Neurologic and chest examination by the consultants were normal.

A further evaluation by cranial magnetic resonance imaging (MRI) was done for the differential diagnosis of possible disorder. It displayed that there were vascular malformation in the posterior pons, chronic hematoma, cerebellary atrophy, ischemia and infarct in the right mediolateral medulla oblongata due to previous surgery. The patient had no problem during the daytime while he was in the ICU. However, the confusion of the patient increased, and he became somnolent resulting in PaCO<sub>2</sub> rise in the morning of the first postoperative day. The patient's arterial blood gas analysis showed pH 7.151; pO<sub>2</sub> 205 mmHg; pCO<sub>2</sub> 167.1 mmHg; SpO<sub>2</sub> 92% and HCO<sub>3</sub> 57.1 mmol/l. Therefore, he had CPAP therapy. CPAP was applied intermittently when he stayed at the ICU. Later the patient was investigated for his sleeping habits. According to his wife, he was an intolerable snorer, also had apneic spells during his sleep, difficulty for waking up in the morning and was suffering from excessive daytime sleepiness. The patient's wife also mentioned that he had a history of brain stem surgery due to pons cavernous malformation seven years ago.

The patient was admitted to the clinic seven days later after the operation. Again, the similar situation in terms of apnea attacks was observed in the clinic especially in the early morning. He was referred to the sleep laboratory in aspect of the differential diagnosis and a sleep study was performed.

Polygraphic somnography showed 49 central apnea with an average time for 19.9 s and 31 mixed apnea with duration of 32.9 s. Mixed and central apneas occurred especially in non-REM sleep. The Apnea Hypopnea Index was 41.9 (there were 284 episodes of arousal, for an arousal index of 46.6 per hour, 119 obstructive hypopneas with an average duration of 33.2 s; 56 obstructive apnea with an average duration of 26.4 s; the average lowest desaturation was 69%. REM stage was %4 and REM latency was 86 minutes. the presence of obstructive apneas it is suggested that CSA may also induce OSA (3). In titration with Bilevel Positive Airway Pressure (BİBAP) night patient's AHI decreased to 17.7 per hour; the total number of obstructive hypopneas decreased but central apneas were persistent (totally 49, central AHI:8.0 per hour). After the tests, the patient was diagnosed as central sleep apnea syndrome and home device for CPAP was recommended.

## DISCUSSION

Delayed or prolonged to emergence from general anesthesia is a comparatively common and often multifactorial event in the postoperative period. This variable condition depends on several factors related to the patient, such as the type of anesthetic given, the length of surgery, residual drug effects, respiratory failure and neuromuscular deficiency. As well as, less commonly reasons are metabolic or electrolyte derangements for instance hyperglycemia, hypoglycemia, hypotension, hypoxia, hypothermia or intracerebral events (1).

Such a situation is encountered, initially; should attempt to exclude immediately the more common of these etiologies. Usually, the most common causes of delayed emergence from anesthesia are related to residual anesthetic effects for medication and/or previously used drugs (4, 5).

Incomplete neuromuscular recovery is a cause of prolonged recuperation after operation. Even small degrees of residual muscle weakness caused by inadequate breathing after anesthesia may be reason of hypercapnia, which may induce deep sedation or unconsciousness. In our case, thinking due to recurarization and possible to inadequate neuromuscular recovery, neostigmine and atropine were repeated. But there was no alteration in the situation of the patient. Metabolic disorders such as hypothermia, acidosis, hypokalemia, hypermagnesemia, hypoglycemia, and hyperglycemia may also cause delayed

recovery from muscle relaxants (6, 7). However, in our patient, metabolic disorder was not detected.

The opioids decrease sensitivity of the brainstem chemoreceptor to carbon dioxide; therefore respiratory depression and hypercapnia were caused by opioids. This may influence clarity of anesthetic drugs and carbon dioxide (7).

Despite naloxone had been given just in case prolonged opioid effect, but the patient's consciousness did not change. Moreover, the amount of fentanyl applied was as little as 100 µg. So, it was excluded from the residual opioid effects in the case.

There is no preoperative history of snoring or sleep apnea given by the patient and patient's family and not being aware of sleep apnea syndrome. It was very difficult to diagnose, why the patient had delayed emergence and persistent hypoventilation (1). Afterward, the case and his wife were questioned about the sleeping habits after operation. It is suggested that after the excluding the possible causes of delayed emergency, our patient could be the possibility of CSAS.

CSAS is a disease described by recurrent apneic period during sleep with no ventilatory effort. CSAS is grouped by hypercapnic or nonhypercapnic. Hypercapnic CSAS is usually characterized by central hypoventilation syndromes, which may be idiopathic or secondary to other disorders that cause damage to the respiratory center in central nervous system (2). In previous studies, CSAS is more common in patients with bilateral lesions of the medulla oblongata. Unilateral medullary lesions may also cause CSA in some cases. In addition, infarction of cerebellum and unilateral dorsolateral medulla because of stenosis of the unilateral vertebral artery are the other reasons of CSAS (8). In present case, MRI findings may depend on operation of brain stem surgery because of pons cavernous malformation. This operation may be effected to respiratory center in medulla oblongata.

Loudy snoring, excessive daytime somnolence, fatigue, dyspnea, headache and apneic spells during sleep are usual features of the syndrome. Commonly, arterial blood gas analysis shows hypercapnia, and the respiratory response to hypoxemia and hypercarby is usually found to be ineffective in these patients. CSAS is more frequent in patients with normal weight and airway (9, 10). In our patient, weight and airway were normal.

Although the presence of the some evidences all those supported to be CSAS including the history of the brain stem surgery, normal pulmonary and mediastinal anatomy, and central

apneic episodes characterized by progressive hypoxemi and hypercarbia, but the definitive diagnosis was set up by polysomnography.

In conclusion, it should keep in mind that the patient, who had prolonged recovery from anesthesia and postoperative respiratory depression, may have a surgical history regarding the brainstem or upper spinal cord. Therefore, the anesthetists ought to question on the patient's sleeping habits such as high volume snoring, apneic spells and over sleepiness in daytime. Eventually, the patient who had a history of brainstem surgery should be taken into consideration for the candidate of CSAS, and a meticulous evaluation should be done before surgery.

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