

Delayed Awakening from Anesthesia Following Electrolyte and Acid-Base Disorders, Two Cases

Majid Razavi¹ (MD); Alireza Bameshki² (MD); Mehryar Taghavi Gilani^{3*} (MD)

¹ Assistant professor of Anesthesiology, Cardiac Anesthesia Research Center, Mashhad University of Medical Sciences, Mashhad, Iran.

² Associate Professor of Anesthesiology, Cardiac Anesthesia Research Center, Mashhad University of Medical Sciences, Mashhad, Iran.

³ Assistant professor of Anesthesiology, Research Center for Patients' Safety, Mashhad University of Medical Sciences, Mashhad, Iran.

ARTICLE INFO	ABSTRACT
<p>Article type: Case Report</p> <hr/> <p>Article history: Received: 2- Oct-2013 Accepted: 19- Dec-2013</p> <hr/> <p>Keywords: Alkalosis Delayed emergence General anesthesia Hypokalemia Impaired consciousness</p>	<p>Introduction: Delayed awakening from anesthesia remains one of the biggest challenges that involve anesthesiologists. Most commonly, delayed awakening is due to drugs effects persistence. Metabolic (like hypo-hyperglycemia), electrolyte, acid-base disorders and hypothermia may cause delayed emergence from anesthesia. Structural disorders of cerebral nervous system (like increase in intracranial pressure, brain ischemia) and psychological disorders can be regarded as other uncommon causes of this condition. Hypokalemia is induced by low potassium intake, excessive excretion from gastrointestinal (GI) and kidneys (like using diuretics) or a shift from extracellular space into intracellular space.</p> <p>Cases: In these two reported cases, although proper measures had been taken to reverse the effects of anesthetic drugs, the patients did not regain their consciousness as fast as expected. The only significant finding in postoperative tests, along with respiratory alkalosis, was low serum potassium level (K=2.5 and K=2.9 in the first and the second patients, respectively). Correction of serum potassium (to K=3.3 and K=3.2 in the first and the second, respectively) improved patients level of consciousness, and they were discharged from intensive care unit (ICU) with good general condition.</p> <p>Conclusion: During surgery; pain, stress, sympathetic increase, catecholamine release and the consequent β-stimulation, certain drugs, and respiratory alkalosis due to hyperventilation may cause acute shift of potassium into the cells, which will be intensified in the patients with preoperative hypokalemia. Hypokalemia induces consciousness impairment and increases muscle relaxation, both of which affect patient awakening. Serum potassium evaluation is recommended in cases of delayed emergence from anesthesia.</p>

► **Please cite this paper as:**

Razavi M, Bameshki AR, Taghavi Gilani M. Delayed Awakening from Anesthesia Following Electrolyte and Acid-Base Disorders, Two Cases. Patient Saf Qual Improv. 2014; 2(1): 65-68.

Introduction

Waking up from anesthesia is one of the most important concerns of anesthesiologists, patients and their families (1, 2). Although delay or failure in

patient's awakening is rare, it remains a very challenging subject for anesthesiologists.

In these cases every anesthesiologist tries to find out the underlying causes and to have them rectified. In this study, we introduce two patients

who had undergone emergency surgery. Patients were hyperventilated during operation. Consequent delay in awakening was observed which could probably be due to hypokalemia. We will discuss the causes and treatment method.

Case 1

Our first case was a 54-year-old woman (wt=75kg). She was hospitalized in ENT department for neck fasciitis. The patient did not have any history of previous diseases and was treated with clindamycin, vancomycin and ceftriaxon. After 24 hours, she was scheduled for emergent incision of the cervical abscess. The only noteworthy finding in her preoperative tests was a lower limit normal serum potassium level (K=3.5). Due to some difficulties in tracheal intubation, anesthesia induction was performed with 100 mg ketamine and 75mg succinylcholine. After respiration recovery, fentanyl (2 μ /kg), propofol (3-4mg/kg/hour) and atracurium (20mg) were administered for anesthesia maintenance. The operation duration was 105 minutes, patient vital signs were stable during operation and capnography was preserved around 30-32 Etc₂. At the end of operation after discontinuing of anesthetic drugs and respiratory recovery, neostigmine and atropine was administered to reverse anesthesia. Furthermore, the patient received naloxane because of the delay in waking up. Finally, after a 2 hour interval in recovery room, patient was delivered to ICU in deep coma, with stable vital signs and spontaneous breathing through tracheal tube. In ICU, the patient received respiratory support by CPAP and her blood tests were as follow: PH=7.52, PCO₂=27.1, HCO₃=21.8, PaO₂=133, BS=202, BUN=35, K=2.5, Ca=8.4, Mg=2.2mg/dl, WBC=10200, Hb=11. Having considered blood test results, 40meq KCL was added to each liter of patient intravenous infusion.

In our assessment 24 hours later (during the time in which the patient had received an approximate 120 meq kcl), she was in confused state and received only haloperidol. Blood tests showed following results: PH=7.47, PaCO₂=40, HCO₃=28.5, PaO₂=100, BS=140, BUN=15, Cr=0.4, K=2.9.

After spending 48 hours in ICU, our patient fully regained consciousness, respiratory stability, and hemodynamic stability. In this stage she was extubated. Blood test results in this phase were as follow: PH=7.45, HCO₃=26, PaCO₂=35, K=3.3, Na=135. On the third day our patient was transferred from ICU to hospital ward. (Her EKG is shown in Figure 1(admission to ICU) and Figure 2 (in ward)).



Figure1: ECG after ICU transfer



Figure2: ECG before ward transfer

Case 2

Our second patient was a 7-year-old boy (wt=30kg) who had been transferred for esophagoscopy due to foreign body ingestion.

Patient's preoperative CBC results were normal and he did not have any history of previous diseases.

Anesthesia was induced by 150 mg thiopentanol, 30 mg Succinylcholine and 100 μ g fentanyl. For anesthesia maintenance, 1.5% isoflurane and 60% N₂O was administered, and the patient received 10 mg atracurium after respiratory recovery. Surgery took 20 minutes long, and patient's respiration recovered after 1 hour. He was extubated when airway reflexes returned, but his GCS was 8-9 in this stage. Patient's body temperature was maintained at 36-37, and Etc₂≈30 during operation.

The blood tests in OR showed the following results: PH=7.45, PCO₂=25, HCO₃=20, BS=123.

Patient spent four hours in recovery room and he was delivered to ICU afterwards with following test results: PH=7.37, PaO₂=70, PCO₂=25, HCO₃=14.4, SaO₂=92%, BUN=10, Cr=0.6, Ca=10.5, Na=135, K=2.19, BS=120, Mg=1.8.

In ICU, patient received KCl and respiratory support with facial mask. After 12 hours, he started to recover consciousness (GCS=10-11), and his blood tests showed the following results: PH=7.40, PaCO₂= 34, PO₂= 101, SaO₂=99%, Na=133, K=3.2, BS= 149. Patient was discharged from ICU after 24 hours with full consciousness, stable vital signs and, optimal respiration.

Discussion

Recovering consciousness from anesthesia and awakening depends on various factors related to patient, anesthesia type, and duration of surgery.

When surgery is prolonged, careful evaluation of various factors which might affect consciousness would be necessary. The most common reason for prolonged anesthesia and patients' unconsciousness after anesthesia is persistent effects of anesthetic drugs and sedatives (1, 2). Opiates and benzodiazepines are the most common drugs considered in this group. Furthermore, neuromuscular block is a cause of patient's unresponsiveness to stimulations. After evaluating anesthetic drugs, metabolic and endocrine disorders like hypothyroidism (3), severe hypohyperglycemia, other disorders like hypothermia, acid and base disorders and electrolytes imbalance must be taken into consideration in the case of prolonged anesthesia (3). General anesthesia influences the level of serum electrolytes. Moreover, both propofol and ketamine (which have been used in one of our cases) may affect the intracellular Ca levels through NMDA receptors or voltage-dependent Ca channels. Calcium is known to induce neuronal excitability and to increase anesthesia depth (1). Hypocalcemia manifestations are more commonly observed in cases with respiratory alkalosis versus metabolic alkalosis (4). In our patients Ca and Mg were in normal limits and there were no clinical signs of calcium deficiency (Chevostek's sign or Trousseau's sign). Decrease in serum sodium level (due to low-tonicity liquids during surgery, or inappropriate secretion of anti diuretic hormone (SIADH) following surgery stress), decrease in serum potassium level, and respiratory acidosis or alkalosis (2) are examples of electrolytes, acid or base disorders. Finally, some rarer conditions such as central anticholinergic syndrome (5), structural disorders of nervous system (e.g. increase in intracranial pressure, bleeding, and

ischemia) (6), psychiatric disorders like hysteria (2, 7), and the consequent hypernatremia after hepatic hydatid cyst removal (8) may also hinder the process of recovery from anesthesia.

In both cases of this study, anesthesia was induced by succinylcholine, which may cause delayed recovery in patient with cholinesterase deficiency. Considering patients' spontaneous respiratory recovery at the end of surgery this diagnosis had been ruled out. In evaluating the possible causes of prolonged anesthesia in patients who had received short-acting drugs with intraoperative and postoperative hemodynamic stability and an acceptable glycemic and temperature control with no history of previous comorbidities (like hypothyroidism, etc), the physician's attention should be drawn to electrolyte, acid and base disorders.

In both patients, ETCO₂ was maintained around 30 mmHg. Hyperventilation due to anxiety, stress, and iatrogenic (intraoperative) causes leads to hypocapnia and its consequences associated with systemic manifestation. Respiratory alkalosis with the carbon dioxide pressure less than 36 mmHg results in reduced intracellular proton concentration and is in turn, draws potassium ion into cells. This leads to serum potassium level (K⁺) decrease which is correlated to the reduction in carbon dioxide partial pressure (reduction of 0.5meq/L of potassium per each 10-mmHg reduction of PaCO₂)(9). Surgery stimulation, sympathetic system stimulation, and releasing catecholamines, and also diuretics, beta-stimulators, insulin administration and induced hypothermia lead to increase potassium transport into cells, reducing serum potassium after anesthesia (9, 10).

The potassium shift and/or excretion due to the alkalotic state requires a considerable period of time to produce cardiac arrhythmias. Mild preoperative hypokalemia without any clinical features could, rapidly deteriorate after iatrogenic hyperventilation or surgery stimulation during and after anesthesia (4). As such, to prevent further complications, even in mild hypokalemic state, monitoring potassium levels and faster correction seems a wise choice.

Usually, serum potassium levels below 3.5meq/L are considered as hypokalemia. The common signs of hypokalemia are confusion, decreased level of consciousness, muscle weakness (more in lower extremity), constipation, nausea, vomiting, polyuria, depression, decrease in cardiac contractility, and change in cardiac rhythm. Hypokalemia causes ECG changes, including flat or inverted T waves; large U wave (greater than 1 millimeter and greater than T waves) could not indicate hypokalemia severity.

Hypokalemia intensifies the effects of non-depolarizing muscle relaxants (11).

At the time of transfer to ICU, our first patient had a preoperative serum potassium=3.3 meq/L, and her blood tests showed respiratory alkalosis and hypokalemia (K=2.5). Preoperative serum potassium level was not controlled for the second patient who had a rather simple surgery, but at the time of transfer to ICU, signs of mild respiratory alkalosis and hypokalemia were observed. Both patients received potassium and their consciousness improved with gradual correction of serum potassium, and finally they were transferred to hospital ward. One limitation of this study was the lack of bispectral index (BIS) monitoring that provides details of the level of anesthesia, though not an essential component of routine monitoring.

Conclusion

Various factors are involved in patients awakening after anesthesia. These factors have to be considered in evaluating patients with delayed awakening problem based on their occurrence. Our two patients did not respond to the routine therapeutic measures. The cases were kept in ICU and more detailed laboratory assessments have been preformed. We found that intensified hypokalemia due to hyperventilation or bidirectional effects of general anesthesia and electrolyte levels may be at play and its persistence after surgery was the cause of the delay in our patients' recovering their consciousness. Serum potassium evaluation is recommended in cases of delayed awakening from anesthesia.

Acknowledgement

The authors are committed to the satisfaction of the patients presented in this article.

References

- 1- Miller RD. *Millers, Anesthesia*. 7th Edition, United States of America, Elsevier Churchill, 2010. P 2722-2723.
- 2- Saranagi S. Delayed Awakening from Anaesthesia. *Internet J Anesthesiol*. 2009; 19(1). DOI: 10.5580/914.
- 3- Kumar VV, Kaimar P. Subclinical hypothyroidism: A cause for delayed recovery from anaesthesia? *Indian J Anaesth*. 2011; 55(4): 433-4.
- 4- Moon HS, Lee SK, Chung JH, In CB. Hypocalcemia and hypokalemia due to hyperventilation syndrome in spinal anesthesia -A case report. *Korean J Anesthesiol*. 2011; 61(6):519-23.
- 5- Brown DV, Heller F, Barkin R. Anticholinergic syndrome after anesthesia: a case report and review. *Am J Ther*. 2004; 11(2):144-53.
- 6- Deuri A, Goswami D, Samplay M, Das J. Nonawakening following general anaesthesia after ventriculo-peritoneal shunt surgery: An acute presentation of intracerebral haemorrhage. *Indian J Anaesth*. 2010; 54(6):569-71.
- 7- Albrecht RF, Wagner SR, Leicht CH, Lanier WL. Factitious disorder as a cause of failure to awaken after general anesthesia. *Anesthesiology*. 1995; 83(1):201-4.
- 8- Grati L, Toumi S, Gahbiche M. Failure to recover after anaesthesia for surgery of a liver hydatid cyst assigned to hypernatraemia. *Ann Fr Anesth Reanim*. 2009; 28(3):261-2.
- 9- Edwards R, Winnie AP, Ramamurthy S. Acute hypocapnic hypokalemia: an iatrogenic anesthetic complication. *Anesth Analg*. 1977; 56(6):786-92.
- 10- Brown MJ. Hypokalemia from beta 2-receptor stimulation by circulating epinephrine. *Am J Cardiol*. 1985; 30; 56(6):3D-9D.
- 11- Miller RD, Roderick LL. Diuretic-induced hypokalaemia, pancuronium neuromuscular blockade and its antagonism by neostigmine. *Br J Anaesth*. 1978; 50(6):541-4.