Intraoperative Polyuria Following Sevoflurane Administration

Sevofluran Uygulamasının Ardından İntraoperatif Poliüri

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ABSTRACT

Sevoflurane has been used widespread due to its favorable pharmacokinetic profile. It is used as a volatile anaesthetic agent in the operating theatre (OT), and a sedative agent in the intensive care unit (ICU). However, the use of sevoflurane is not without side effects. The following is a case report detailing a patient who developed polyuria following intraoperative use of sevoflurane.

Key Words: Sevoflurane, polyuria, Total Intravenous Anaesthesia Agent

Received: 06.27.2020 **Accepted:** 08.01.2020

ÖZET

Sevofluran, olumlu farmakokinetik profili nedeniyle yaygın olarak kullanılmaktadır. Ameliyathanede (OT) uçucu anestezik ajan ve yoğun bakım ünitesinde (YBÜ) sedatif ajan olarak kullanılır. Bununla birlikte, sevofluran kullanımının yan etkileri yoktur. Aşağıda, intraoperatif sevofluran kullanımı sonrası poliüri gelişen bir hastayı ayrıntılı olarak anlatan bir vaka sunusu verilmiştir.

Anahtar Sözcükler: Sevofluran, poliüri, Total İntravenöz Anestezi Ajanı

Geliş Tarihi: 27.06.2020 **Kabul Tarihi:** 01.08.2020

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doi:http://dx.doi.org/10.12996/gmj.2021.34

INTRODUCTION

Since sevoflurane's introduction, its use has been widespread due to its favorable pharmacokinetic profile(1). In addition to its main function as a volatile anaesthetic agent, sevoflurane's role has expanded more recently into the intensive care units (ICU) where it also functions as a sedative agent(2-3).

Polyuria following administration of sevoflurane had been described in some case reports(4-7). The following is a recent case report detailing a patient who developed polyuria following intraoperative use of sevoflurane.

CASE REPORT

A healthy 22-year-old girl presented to the hospital following a motor vehicle accident. She complained of numbness from the neck downwards. A CT scan done showed a burst compression fracture of the lower cervical spine (C5 and C6) with traumatic spinal canal stenosis. She was planned for anterior cervical corpectomy and fusion (ACCF) of C4/C5/C6. In the operating room, the patient was placed in supine position and was on standard monitoring, including blood pressure, electrocardiogram and oxygen saturation monitor. The patient was induced with intravenous fentanyl, propofol and rocuronium. Anaesthesia was maintained with administration of sevoflurane (MAC 0.9-1.1).

From the third hour of surgery onwards, we noticed a significant increase in the patient's hourly urine output from an initial volume of 500mL/hour (12.5ml/kg/hour) to a peak of 1.7L/hour (42.5ml/kg/hour). Her baseline urine output prior to surgery was 20-50ml/hour (0.5-1.25 ml/kg/hour).

She was soon getting tachycardic, with an average heart rate ranging between 100-130 beats-per-minute, and blood pressure was ranging between systolic 85-160 mmHg and diastolic 45-92 mmHg. Inotropic agents were titrated up to maintain a Mean Arterial Pressure (MAP) of more than 65mmHg. A total of 7500mL of crystalloids, 1500mL of colloids, and 2 units of packed red blood cells were administered. The cumulative urine output was 7700mls, with a total estimated blood loss of 600mls.

Several hours into surgery, the mode of anaesthesia was changed from sevoflurane to Total Intravenous Anaesthesia Technique (TIVA) using propofol. After cessation of sevoflurane, hourly urine output showed a reducing trend. By the end of surgery, urine output was 500mls/hour (12.5 mls/kg/hour), and vital signs normalized. The surgery lasted nine hours. Around 6 hours after returning to the ICU, the polyuria resolved (urine output 1ml/kg/hr). After observing this improvement, we decided not to use Desmopressin.

Her blood and urine investigations pre-operatively, intra-operatively and post-operatively is presented in Table 1.

Table 1: Blood and urine investigation results

Parameters	Pre-op	Intra op	Post op (1hr)	Post op (12hr)
Hemoglobin (g/dL)	12.6	-	10.6	10.5
Hematocrit (%)	36	-	31	30
Sodium (mmol/L)	139	146	152	138
Potassium (mmol/L)	4.4	4.2	4.2	4.3
Calcium (corrected) (mmol/L)	2.18	-	2.30	2.36
Urea (mmol/L)	4.5	3.5	3.1	5.4
Creatinine (mmol/L)	35	28	31	29
Glucose (mmol/L)	-	7.4 - 10.3	-	-
Urine osmolality (mOsm/kg)	-	273	-	-
Serum Osmolallity (mOsm/kg)	-	315	-	-
Urine sodium (mmol/L)	-	125	-	-

DISCUSSION

Urine-concentrating mechanism of kidney is mainly regulated by Arginine Vasopressin (AVP), which is released from the posterior pituitary gland. Surgical stress can increase the secretion of AVP, among other factors such as decreased circulating volume, increased serum osmolality, release of various peptide and medications(8). Presence of AVP causes an increase in expression of Aquaporin-2 (AQP2), a water channel protein located at the principal cells of the renal collecting duct. This in turn leads to an increase in permeability of the collecting tubules to reabsorb water, thus concentrating the urine.

The common causes for perioperative polyuria include iatrogenic overhydration, diuretic therapy, uncontrolled hyperglycemia, Central Neural System (CNS) surgeries, and central/nephrogenic diabetes insipidus(9). Pertaining to our patient, she is a young lady without any previous medical illness, was not given any diuretic medication and had good sugar control throughout surgery. Other causes of nephrogenic diabetes insipidus such as lithium toxicity, hypercalcemia, hypokalaemia or demeclocycline has also been ruled out.

Hence, we suspected that her polyuria was due to diabetes insipidus secondary to an anaesthetic agent given intraoperatively. Among the list of drugs administered to her, only sevoflurane could be the most likely cause of polyuria. The subsequent change in mode of anaesthesia to TIVA led to normalizing trend in the urine output within 30 minutes of sevoflurane discontinuation.

The exact mechanism on how sevoflurane affects the urine-concentrating ability is still unknown. One of the main hypothesis by Morita(10) is that sevoflurane causes a transient disruption of the mobilization process of AQP2 to the apical membrane of the renal collecting duct cells. Less water is reabsorbed from the collecting ducts due to less AQP2, thus leading to polyuria.

CONCLUSION

Polyuria is a rare side effect of sevoflurane, which is potentially harmful should it go unnoticed. Since not all patients under general anaesthesia require an indwelling urinary catheter insertion, it is difficult to ascertain the prevalence of sevoflurane-induced polyuria.

The incidence could perhaps be higher than reported, and therefore should not be ignored. We would also suggest that TIVA with propofol is a good alternative to replace sevoflurane for the maintenance of anaesthesia in patients with high risk of developing polyuria.

Conflict of interest

No conflict of interest was declared by the authors.

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