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Undiagnosed atypical absence seizure that accidentally found during staying at post-anesthesia care unit

Seokhoon Kim, Minki Jung, Kye-Min Kim, Mun Cheol Kim, Sangseok Lee
Department of Anesthesiology and Pain Medicine, Sanggye Paik Hospital, Inje University, Dongil-Ro 1342, Nowon-Gu, Seoul, Republic of Korea, 01757

***Corresponding author**
Sangseok Lee
Email: s2248@paik.ac.kr

Abstract: A 49-year-old man without a specific past medical history underwent tympanoplasty under general anesthesia due to chronic otitis media. After uneventfully awakening from general anesthesia, the patient was delivered to post-anesthesia care unit (PACU). Ten minutes after arrival at the PACU, he became unresponsive to stimulation. Neurological examination revealed lack of awareness, verbal output, and pain response. After 20 minutes, the patient suddenly responded to pain and recovered consciousness. No neurological deficits were found. He was clinically diagnosed with an atypical absence seizure. Unlike grand-mal seizures, it is generally difficult to detect an absence seizure without a careful observation. Through this case report, we want to highlight the importance of careful monitoring of patients in the PACU. If symptoms develop, the patient should be protected from secondary damage due to external impact, treated to maintain spontaneous breathing, and consulted to a neurologist for further evaluations.
Keywords: Anesthetics, propofol, sevoflurane; Epilepsy, Absence; Post-anesthesia care.

INTRODUCTION

Seizures are a common occurrence in the general population, with an 8 to 10% lifetime risk of a single seizure and a 3% chance of a persistent seizure disorder [1]. Nissen *et al.* [2] reported the incidence of perioperative seizure in patients with a history of a seizure disorder. They retrospectively reviewed the medical records of all patients with a documented history of a seizure disorder who received an anesthetic. Twenty-two out of 641 patients experienced perioperative seizure activity with an overall frequency of 3.4% (95% CI, 2.2 to 5.2%). They showed that perioperative seizure was significantly related to the frequency of preoperative seizures ($P < 0.001$) and to the timing of the most recent seizure ($P < 0.001$). In addition, the frequency of perioperative seizures was significantly related to increased number of antiepileptic medications. The frequency of perioperative seizures in this patient population was affected neither by the type of surgery nor the type of anesthesia (general anesthesia, regional anesthesia, or monitored anesthesia care).

Absence seizures, one of several kinds of seizures, are known as ‘petit mal seizures’ (from the French for "little illness", a term dating from the late 18th century). They are characterized by a brief loss and return of consciousness, generally not followed by a period of lethargy (without a notable postictal state). We report a case of atypical absence seizure diagnosed by close monitoring of the patient in the post-anesthesia care unit (PACU); the seizure was not identified preoperatively and might have been misdiagnosed as delayed awakening from general anesthesia.

CASE REPORT

A 49-year-old man (weight, 82 kg and height, 175 cm) was admitted to the hospital with hearing difficulties due to chronic otitis media requiring an elective tympanoplasty. During the pre-anesthetic evaluation, he denied a history of medical illness, drug allergies, neurological disease, or seizure disorder, and he was not taking any medications. The preoperative laboratory tests showed no abnormalities.

He received glycopyrrolate 0.2 mg intramuscularly as a premedication. Arriving at the operating room, the patient showed alert mentality and fully cooperative state. General anesthesia was induced intravenously using propofol (1% Fresofol, Fresenius Kabi, Germany) 120 mg and alfentanil 0.5 mg. When the patient became unconscious, rocuronium (Esmeron®, Organon, Germany) 0.6 mg/kg was administered intravenously to facilitate tracheal intubation. Neither rigidity nor myoclonus was noted during induction of anesthesia. Anesthesia was maintained by administration of 4.5¬–6 vol% desflurane (Suprane, BAXTER, USA) and fresh gas flow at 4 L/min of 50% oxygen and 50% nitrous oxide mixture. The patient was ventilated with tidal volume of 8 ml/kg and respiratory rate of 10 breaths/min during surgery, maintaining an end-tidal carbon dioxide concentration of 30–35 mmHg. Body temperature was monitored with esophageal temperature probe

(Esophageal Stethoscope, DeRoyal Inc., Powell, TN, USA) and maintained at 35.5–35.7℃. After 45 minutes and 115 minutes after the intubation, additional dose of rocuronium 10 mg were administered intravenously. The last dose of rocuronium was administered at 80 minutes before extubation. Fentanyl (fentanyl citrate®, Hana pharmacy, Korea) was administered 50 μg at 22 minutes after the induction (175 minutes before extubation). After 165 minutes of surgical procedure, pyridostigmine 15 mg with glycopyrrolate 0.4 mg were administered intravenously for antagonizing the neuromuscular blocking effect of rocuronium. The patient recovered uneventfully and was fully awake within 15 minutes after discontinuation of anesthesia, and transferred to the PACU. Total anesthesia time was 210 minutes.

The patient was alert and oriented when he arrived at the PACU. However, after 10 minutes, he showed no response to any stimulation (including verbal and painful stimulations and shaking). Initially, we assumed that the state of the patient was related to a delayed awakening from general anesthesia due to recurarization of neuromuscular blocking drug or sedative effect of opioid for postoperative analgesia. He still showed stable vital signs and within-normal values of arterial blood gas analysis (ABGA) and serum glucose concentration. We confirmed the full recovery (TOF ratio = 1.0) from neuromuscular blockade by neuromuscular monitoring using TOF-Watch® (Organon, Oss, the Netherlands). After 20 minutes of unresponsive period at the PACU, the patient suddenly regained consciousness and was capable of communication. He revealed no focal neurologic deficit.

During the stay at the PACU, he was hemodynamically stable, with a spontaneous respiratory rate of 14 – 16 rate/min and SpO2 100% under 5 L/min oxygen facemask with reservoir. Laboratory test at the PACU showed normal values, including those of oxygen, carbon dioxide, glucose, and electrolytes. Finally, we suspected that the patient might have an epileptic condition and consulted a neurologist for a neurologic examination and differential diagnosis. At the time of examination, he was opening his eyes but could not keep eye contact. Pupils were isocoric, and light reflex was intact. There was no eyeball deviation. He did not respond to the stimulus and pain. There was no drooling and tonic posture. Vestibulo-ocular reflex and deep tendon reflex were normal. There was no pathologic reflex. Absence seizure was diagnosed by neurologic examination. The symptoms lasted for 20 minutes. After that, the patient recovered consciousness and showed normal response. There was no neurologic deficit. Radiologic study and electroencephalogram test to rule out organic lesion of the brain was scheduled through outpatient visits. Because further seizures had not occurred, the patient was transferred to the general ward. Time to discharge from admission at the PACU was about 100 minutes.

Postoperatively, we interviewed the patient and his family again to determine any previous history of seizure-like symptoms. It was revealed that he had experienced these symptoms infrequently (about once a month, maximum 3 times a month) for the past 2 years. The symptoms lasted for 40 to 60 seconds. The patient did not remember the duration of the symptoms exactly. His wife made a statement about it. Because the patient and his family did not recognize the seriousness of the symptoms at that time, they did not report his symptoms to the medical team before surgery. After this, we referred him to the neurology department, where he was evaluated for epilepsy through EEG, which showed a normal pattern. The patient was examined using digital electroencephalography (20 channel special EEG with EKG monitoring) during sleep and drowsiness states through the neurology outpatient follow up. The results showed that the posterior dominant rhythm was well regulated at 9 Hz activity, which was reactive to eye opening, and it was normal reaction. No definite interictal epileptiform discharges or focal slowings was found. Photic stimulation and hyperventilation did not induce unusual responses. Currently, he is in outpatient follow-up by the neurology department and has normal daily activities.

DISCUSSION

Many factors can evoke a post-anesthesia seizure. Nissen *et al.* [2] reported that most of the perioperative seizures were related to the patient's underlying condition and that the type of anesthesia or procedure does not affect their frequency. In this case, we assumed that the anesthesia drug (propofol) and previous history of epilepsy, which was not reported preoperatively, may be the cause of seizure attack in the PACU.

Many of the reported complications of propofol involve seizures, opisthotonus, or unusual muscle activity during induction or emergence from anesthesia, or in the recovery room [3]. No clear explanation for the excitatory effects of propofol has been provided. Seizure activity has rarely been identified on surface EEG recording, although this is, of course, usually done after the episode [4]. On the other hand, Modica *et al.*[5] reported, in their review article, about pro- and anti-convulsant effects of anesthetics in which no EEG or motor evidence of seizure activity was noted during desflurane administration. A recent report showed that propofol and desflurane have reliable anticonvulsant effects [6], whereas remifentanil in larger doses and sevoflurane appear to support epileptiform activity, although the clinical significance of these observations is unclear. Therefore, we can only assume that they can be one of the causative factors. However, it cannot be concluded.

The features of an absence seizure are as follows: it shows a gradual onset and offset (over seconds) and the duration of seizure is usually 5-30 seconds. It usually starts with impairment of awareness and responsiveness; however, there is no postictal period and patients are alert and attentive immediately. EEG shows "slow spike-wave" complexes (i.e., < 2.5 Hz). Contrary to these characteristics, this case has different features. The onset of the patient's symptom was sudden, after which it disappeared; there was no response to stimulations after loss of consciousness, and the symptoms lasted for about 20 minutes. Therefore, we presumed this as a case of atypical type absence seizure. The absence seizure has usually good prognosis and response to the treatment. When symptoms occur, it is sufficient to protect the patient from external shocks and maintain spontaneous breathing. However, if an absence seizure occurs immediately after awakening from general anesthesia, the duration of loss of consciousness after seizure may be prolonged due to the effects of opioids, muscle relaxants, and inhalation anesthetics remaining in the body. Therefore, we should carefully watch the occurrence of absence seizure in the PACU.

Anticipating seizure in patients without a particular disease and medical history is very difficult. In the PACU, the medical team should check the patient's blood pressure, heart rate, oxygen saturation, electrocardiogram, and general condition. Clonic movements and changes in heart rate and blood pressure caused by seizure can be easily identified. However, as our case demonstrates, there are only few seemingly obvious symptoms revealed during an absence seizure. The patient with atypical absence seizure stares (as they would in any absence seizure) but is often somewhat responsive. Eye blinking or slight jerking movements of the lips may occur. This behavior can be hard to distinguish from the person's usual behavior, especially in those with cognitive impairment. Unlike other absence seizures, rapid breathing usually cannot produce these seizures. Seizures usually last five to 30 seconds (commonly more than 10 seconds), with a gradual beginning and ending.

Because of these characteristics of absence seizure, we did not know initially that the patient had a seizure and thought it was only a delayed recovery from anesthesia. Therefore, we preferentially tried to identify possible causes of delayed recovery from anesthesia. As we did not carefully observe the patient from the beginning, we made the wrong diagnosis that he was having a delayed recovery from general anesthesia.

If delayed awakening after general anesthesia is clinically suspected, several etiologic factors should be considered, including the patient´s clinical factors (hypothermia, especially < 33℃ or marked metabolic disturbance, respiratory dysfunction), anesthetic factors (residual effects of anesthetics, sedatives, analgesics and neuromuscular blockers), duration of surgery, degree of pain/stimulation, and non-pharmacological factors (perioperative stroke, postoperative delirium, seizure, etc.)[7].

In the case of recurarization, we carried out neuromuscular monitoring using TOF-Watch and confirmed full recovery from neuromuscular blockade with a TOF ratio of 1.0. Opioids (alfentanil [6 μg/kg] and fentanyl [0.6 μg/kg]) were intravenously administered 175 minutes before the end of surgery. There were no residual opioid symptoms such as miosis or respiratory depression. An arterial blood gas analysis showed no hypoglycemia, hypercarbia, or hypoxia. During surgery, there was no hypotension, which can cause cerebral hypoxia or hypoperfusion. For the differential diagnosis of central anticholinergic syndrome, which can be caused by anticholinergics administered with antagonists, we did not use additional anticholinergics. Body temperature (esophageal) in the operating room was measured to be 35.5 to 35.7℃.

CONCLUSION

We report a case of atypical absence seizure that was not diagnosed preoperatively and was observed in the PACU. Generally, it would be very difficult to detect atypical absence seizure in the PACU without careful observation. It is important that careful observation and monitoring be performed not only during anesthesia but also in the PACU. Moreover, a detailed medical history taking should be conducted preoperatively. If epileptic seizure is suspected, neurologic consultation and careful evaluations are additionally required.

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Author Disclosure

The author declares that there is no conflict of interest regarding the publication of this paper.

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