Case report of seizure-like movements after tonsillectomy under general anesthesia

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Abstract

Seizure-like movements after general anesthesia is not common. Abnormal movements following general anesthesia may be difficult to differentiate because it is difficult to find out the exact cause of these seizure-like movements. These cases can occur at any stage This is a rare case of seizure-like movement after tonsillectomy.

Key clinical message

Abnormal movements following general anesthesia may be difficult to differentiate and can occur in patients without a significant neurologic history. Anesthesiologists should be aware of the pathophysiology, risk factors, and management of this.

INTRODUCTION

Although uncommon, seizure-like movements after general anesthesia can occur. The causes of such abnormal movements include adverse drug reactions, emotional responses, emergence delirium, and shivering (1). Because many anesthetic drugs are used during general anesthesia, it is difficult to precisely determine the causative agents of seizure-like movements. There have been case reports in which anesthetic drugs induced myoclonic movements or seizures (2-4). Here, we present a case of a 24-year-old woman with seizure-like movements after tonsillectomy under general anesthesia.

CASE DESCRIPTION

A 25-year-old, 49-kg female patient with American Society of Anesthesiologists physical status I was admitted to our hospital for elective tonsillectomy under general anesthesia. She had previously undergone closed reduction of nasal bone fractures under general anesthesia at the age of 4 years, during which there were no adverse events. The drug used at that time was ketamine (50 mg). Otherwise, the patient had no underlying diseases and no past medical history. The preoperative laboratory tests, electrocardiogram (ECG), and chest X-ray were unremarkable. The patient entered the operating room without premedication. General anesthesia was induced with 40 mg of lidocaine, 100 mg of propofol, 40 mg of rocuronium, and 50 mcg of fentanyl. Tracheal intubation was performed without difficulty. Anesthesia was then maintained with 6 vol % of desflurane (40% oxygen and 60% air). The patient was monitored via non-invasive blood pressure (NIBP) measurements, ECG (lead II), pulse oximetry, and capnography. No issues emerged during tonsillectomy. At the end of the operation, the neuromuscular blockade was reversed by 200 mg sugammadex. The operation took 30 minutes, and the duration of anesthesia was 1 hour. On arrival in the post-anesthesia care unit (PACU), the patient had a mild macular rash on the whole body and complained of pruritus, mild dyspnea, and chills; she also developed shivering. The patient remained cardiovascularly stable. Oxygen was supplied at a flow rate of 5 L/min via face mask. After intravenous injection of 5 mg of dexamethasone and 4 mg of pheniramine, her symptoms and signs resolved. At 150 minutes after the end of anesthesia, she was taken

to the general ward, and periodic movements that lasted less than a minute and occurred at intervals of 2–3 minutes appeared; these took the form of uncontrolled generalized tonic clonic seizure-like movements of the whole body. Her head was tilted laterally and backward, and her body became rigid. Lorazepam (2 mg) was administered and her symptoms abated. At 2 hours after the first attack, she complained of rigidity in all extremities and was aware of the seizure-like movements; however, she remained cardiovascularly stable. A neurologist examined the patient and presumed that the abnormal movements were caused by drugs. The next morning, she trembled for 3 seconds on two occasions but had no recollection of any symptoms. Therefore, she underwent diffusion weighted magnetic resonance imaging (MRI). The MRI results were unremarkable. Subsequently, while waiting for electroencephalography, she shook her head for 10 seconds with her eyes closed. Keppra (levetiracetam) was continuously infused. Later that evening, the patient exhibited generalized tonic clonic seizure-like movements for 7 seconds. ; similar events occurred thereafter, but with shorter duration and longer intervals. The EEG revealed no epileptiform discharge; therefore, Keppra was discontinued and clonazepam (0.5 mg, twice a day) was given instead. At 3 days after surgery, there were no abnormal seizure-like movements, so clonazepam was also discontinued. Abnormal movements did not recur during hospitalization and the patient was discharged on day 7 after the operation. At 7 days after discharge, she did not report any abnormal movements.

This report is of a rare case of seizure-like movements after tonsillectomy under general anesthesia. This case study was approved by our Ethics Review Committee and Institutional Review Board (OC19ZESE0060). Written informed consent was obtained from the patient for the publication of this case report.

DISCUSSION

Extrapyramidal side effects after a single injection of propofol are uncommon. Several cases of propofol-related extrapyramidal symptoms have been reported (5-9). Acute extrapyramidal symptoms include dystonia (continuous spasms and muscle contractions), akathisia (motor restlessness), parkinsonism (characteristic symptoms including rigidity), bradykinesia (slowness of movement), tremor, and tardive dyskinesia (irregular, jerky movements) (10). Extrapyramidal symptoms are drug-induced movement disorders that include acute and tardive symptoms. These symptoms are often reported as side effects of antipsychotics but are sometimes related to other drugs, such as anesthetics (5,11). They may also occur as neurological side effects of general anesthesia in patients without a significant neurologic history. Our patient showed intermittent bouts of sustained contractions of the muscles in the face and extremities, which lasted for 4 days. In the PACU, we thought she was having an allergic reaction to sugammadex because her symptoms included chills, difficulty with breathing, shivering, and redness of the face, neck, arms, and upper chest. After injection of dexamethasone and pheniramine, her symptoms abated. Although there have been case reports of relief of extrapyramidal symptoms after injection of diphenhydramine, our patient did not respond to such treatment (12). After injection of lorazepam, the abnormal movements stopped.

According to the literature, propofol, fentanyl, ramosetron, and nefopam, as well as inhaled anesthetics, can cause perioperative seizure-like movements (2,4,13). In our case, we used a small dose of fentanyl (50 mcg) for induction of anesthesia. Although there have been a few reports of seizures after low-dose fentanyl administration (14,15), seizure activity is usually related to high doses of fentanyl (200 or 500 mcg/kg) (14). Fentanyl can be the cause of seizure-like movements, but it is unlikely that fentanyl and desflurane can be the cause in our case. The epileptogenic potential of volatile agents may involve destabilization of the cortex and delayed onset of inhibitory signaling (16). Desflurane use is associated with fewer cortical spikes compared to other inhaled anesthetics (e.g., enflurane and sevoflurane); accordingly, desflurane was used as the inhaled anesthetic agent in our case and it is unlikely that fentanyl and desflurane were the causative agents of seizure-like movements. Although the neurologist presumed that the cause of seizure might occur by one of drugs, there was specific signs like allergic reaction before the seizure-like movements in this case. As there are some case reports about the relationship between allergic reaction and epilepsy (17), seizure-like movements in our case might be related to the allergic reaction in PACU. If sugammadex that we used might cause allergic reaction, it triggers seizure-like movements but there was no case report that sugammadex caused seizure-like movements.

Recently, the relationship between propofol use and perioperative neurologic complications has been studied (18,19). Although the mechanisms underlying seizure-like movements and neuroexcitatory after propofol administration are not well understood (20), it is hypothesized that the extrapyramidal effects may be due to activation of excitatory pathways in the subcortical region, extended refractory periods in inhibitory pathways in the brainstem and spinal cord, or a combination of both (21). Neuroexcitation is a rare but well-known side effect of propofol anesthesia and sedation. These abnormal movements usually occur during induction of, and emergence from, anesthesia. Therefore, a rapid change in the propofol concentration in the brain appears to be responsible for seizures (19). Propofol has a long terminal elimination half-life, (21) which may be responsible for the slow return of propofol from poorly perfused peripheral compartments back into the central compartment. Hence, the concentrations of propofol can remain high for a long time within the nervous system, producing effects that depend on the balance between inhibitory and excitatory neurons. However, additional studies are needed to precisely determine the biochemical and biophysical origins of neuroexcitation related to propofol.

There have been many reports of abnormal movements associated with propofol use (9,20,22-25) but no definitive treatment has been developed.

Delayed neuroexcitatory symptoms after uneventful anesthesia are uncommon. There is a study that seizure like movements were observed during induction of anesthesia or sedation (24 patients; 34%), during emergence(28; 40%), or delayed (16; 23%) (24). Some cases occur in patients with underlying disease (e.g. renal failure, syncope, and leukemia) (6,26), history of convulsion (27), or use of propofol/nitrous oxide (28). Some cases occur in patients administered in continuous propofol infusion (29). However, our case occurred with only one injection of propofol and abnormal movements were observed during delayed.

Abnormal movements following general anesthesia may be difficult to differentiate and can occur in patients without a significant neurologic history. Anesthesiologists should be aware of the pathophysiology, risk factors, and management of perioperative abnormal movements.

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DISCLOSURE

All authors have no potential conflicts of interest to disclose.

AUTHOR CONTRIBUTIONS

Conceptualization: Joo CH. Investigation: Joo CH, Cho EJ. Writing - original draft preparation: Joo CH. Writing - review and editing: Joo CH and Cho EJ. Approval of final manuscript: all authors.

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References

1. Ananthanarayan C, Blight K. Dystonic reaction after anesthesia. *Can J Anaesth* 2001; 48: 101.

2. Zambelli A, Sagrada P, Pavesi L. Seizure associated with palonosetron. *Support Care Cancer* 2009; 17: 217.

3. Park PG, Shin HY, Kang H, Jung YH, Woo YC, Kim JY, Koo GH, Park SG, Baek CW. Seizure developed after palonosetron intravenous injection during recovery from general anesthesia -A case report. *Korean J Anesthesiol* 2012; 63: 173-6.

4. Durrieu G, Olivier P, Bagheri H, Montastruc JL, French Network of Pharmacovigilance C. Overview of adverse reactions to nefopam: an analysis of the French Pharmacovigilance database. *Fundam Clin Pharmacol* 2007; 21: 555-8.

5. Ries CR, Scoates PJ, Puil E. Opisthotonos following propofol: a nonepileptic perspective and treatment strategy. *Can J Anaesth*1994; 41: 414-9.

6. Islander G, Vinge E. Severe neuroexcitatory symptoms after anaesthesia–with focus on propofol anaesthesia. *Acta Anaesthesiol Scand* 2000; 44: 144-9.

7. Diltoer MW, Rosseneu S, Ramet J, De Wolf D, Spapen HD, De Turck BJ, Huyghens LP. Anticholinergic treatment for choreoathetosis in a child after induction with propofol. *Anesth Analg* 1996; 82: 670.

8. Chan A, Nickoll E, Thornton C, Dore C, Newton DE. Spontaneous movement after injection of propofol. *Anaesthesia* 1996; 51: 663-6.

9. Schramm BM, Orser BA. Dystonic reaction to propofol attenuated by benztropine (cogentin). *Anesth Analg* 2002; 94: 1237-40, table of contents.

10. Pierre JM. Extrapyramidal symptoms with atypical antipsychotics : incidence, prevention and management. *Drug Saf* 2005; 28: 191-208.

11. Tolan MM, Fuhrman TM, Tsueda K, Lippmann SB. Perioperative extrapyramidal reactions associated with ondansetron.*Anesthesiology* 1999; 90: 340-1.

12. Sherer J, Salazar T, Schesing KB, McPartland S, Kornitzer J. Diphenhydramine for Acute Extrapyramidal Symptoms After Propofol Administration. *Pediatrics* 2017; 139.

13. Jeon HW, Kang JH, Kim HS, Jo HY, Kim SH. A case of propofol-induced delayed-onset refractory myoclonic seizures. *J Clin Neurol* 2007; 3: 154-7.

14. Safwat AM, Daniel D. Grand mal seizure after fentanyl administration. *Anesthesiology* 1983; 59: 78.

15. Hsieh XX, Hsu YC, Cherng CH, Lin CC, Huang GS, Lin SL, Wu ZF, Yeh CC. Grand mal seizure induced by low-dose fentanyl and lidocaine in a young child. *Acta Anaesthesiol Taiwan* 2015; 53: 105-8.

16. Voss LJ, Sleigh JW, Barnard JP, Kirsch HE. The howling cortex: seizures and general anesthetic drugs. *Anesth Analg* 2008; 107: 1689-703.

17. Silverberg JI, Joks R, Durkin HG. Allergic disease is associated with epilepsy in childhood: a US population-based study. *Allergy*2014; 69: 95-103.

18. Bevan JC. Propofol-related convulsions. *Can J Anaesth* 1993; 40: 805-9.

19. Saunders PR, Harris MN. Opisthotonus and other unusual neurological sequelae after outpatient anaesthesia. *Anaesthesia* 1990; 45: 552-7.

20. Borgeat A, Dessibourg C, Popovic V, Meier D, Blanchard M, Schwander D. Propofol and spontaneous movements: an EEG study.*Anesthesiology* 1991; 74: 24-7.

21. Campbell GA, Morgan DJ, Kumar K, Crankshaw DP. Extended blood collection period required to define distribution and elimination kinetics of propofol. *Br J Clin Pharmacol* 1988; 26: 187-90.

22. Makela JP, Iivanainen M, Pieninkeroinen IP, Waltimo O, Lahdensuu M. Seizures associated with propofol anesthesia. *Epilepsia* 1993; 34: 832-5.

23. Anderson BJ, Marks PV, Futter ME. Propofol–contrasting effects in movement disorders. *Br J Neurosurg* 1994; 8: 387-8.

24. Walder B, Tramer MR, Seeck M. Seizure-like phenomena and propofol: a systematic review. *Neurology* 2002; 58: 1327-32.

25. Orser B. Propofol-induced neuroexcitation and receptor desensitization. *Can J Anaesth* 1994; 41: 366-71.

26. Finley GA, MacManus B, Sampson SE, Fernandez CV, Retallick R. Delayed seizures following sedation with propofol. *Can J Anaesth*1993; 40: 863-5.

27. Thomas JS, Boheimer NO. An isolated grand mal seizure 5 days after propofol anaesthesia. *Anaesthesia* 1991; 46: 508.

28. Reynolds LM, Koh JL. Prolonged spontaneous movement following emergence from propofol/nitrous oxide anesthesia. *Anesth Analg*1993; 76: 192-3.

29. Bendiksen A, Larsen LM. Convulsions, ataxia and hallucinations following propofol. *Acta Anaesthesiol Scand* 1998; 42: 739-41.