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Prolonged postictal apnea after general anesthesia

Ahed Zeidan and Nazih Nahlé
Department of Anesthesiology, Sahel General Hospital, Lebanese University, Airport Avenue, PO Box 99/25, Ghobeiry, Beirut, Lebanon

To the editor: Tonic-clonic seizures that occur during emergence from general anesthesia can be associated with significantly prolonged apnea, persisting postictally. We present just such a case.

A 24-year-old 70-kg primigravida at 39 weeks' gestation with breech presentation was admitted to our department for emergency cesarean section. Her medical history was positive only for hypertension (150/95 mmHg) during the previous few days with a suspicion of preeclampsia but without medical treatment. Upon arrival, the patient had mild edema of the lower extremities. Her complete blood count and coagulation profile were within normal limits. Blood urea nitrogen and creatinine were also normal. Urinary protein was 1+. A rapid-sequence induction was performed using thiopental ($4\,mg\cdot kg^{-1}$) and succinylcholine ($1.5\,mg\cdot kg^{-1}$) followed by rocuronium ($0.4\,mg\cdot kg^{-1}$). After delivery, fentanyl 350 µg and oxytocin 30 IU were given. Anesthesia was maintained using 50% N_2O in oxygen. The surgery lasted 50 min and was uneventful. The patient started to breathe spontaneously with 100% O_2 and responded well to verbal commands by opening her eyes and protruding her tongue: A mixture of neostigmine $0.05\,mg\cdot kg^{-1}$ and atropine $0.02\,mg\cdot kg^{-1}$ was then injected intravenously. Vital signs showed her blood pressure (BP) to be 159/105 mmHg, heart rate (HR) 119 bpm, and respiratory rate 15 breaths per minute. $P_{ET_{CO_2}}$ was 34 mmHg. A few minutes later and before extubation, the patient suddenly lost consciousness and started to develop tonic-colonic movements suggestive of seizure. Spontaneous respiration ceased, the $S_{p_{O_2}}$ dropped to <90%, and the $P_{ET_{CO_2}}$ was 0 mmHg. Positive ventilation with 100% O_2 was initiated. Vital signs (BP and HR) showed no significant changes during manual ventilation, and the $P_{ET_{CO_2}}$ reached 30 mmHg. Apnea, which lasted about 15 min and persisted despite termination of the seizure (which lasted less than 2 min), was rapidly abolished by intravenous administration of 3 mg midazolam, after which ventilation improved and consciousness resumed. Forty minutes later the patient was extubated, and her postoperative course was uneventful.

Common causes of postoperative apnea include intraoperative hyperventilation, inadequate reversal of muscle relaxant, inadequate reversal of opioid, and hypothermia. Impaired ventilation, for a short period, is observed during generalized tonic-clonic seizures. Furthermore, seizure may be associated with a mechanical airway obstruction and can lead to cyanosis during this event.

Seizures may produce postoperative apnea, which may be the only recognized sign [1]. In our case, the possibility that the patient was preeclamptic and developed an eclamptic seizure during her emergence from anesthesia cannot be excluded. We note that the patient seized after reversal administration. Atropine, a tertiary amine, can cross the blood-brain barrier and has been associated with mild postoperative memory deficit; toxic doses are usually associated with an excitatory reaction. Seizure has never been reported with atropine administration; and neostigmine is a quaternary ammonium cholinesterase inhibitor that does not cross the blood-brain barrier.

Whatever the cause of the seizure, in our opinion, anesthesiologists should expect prolonged apnea and delayed recovery when tonic-clonic seizures occur during the immediate postoperative period. Persistent apnea postictally can be attributed to a residual depressant effect of anesthetics on respiration or an exaggerated effect of seizure-triggering anesthetic agents used during general anesthesia. Early administration of midazolam may be recommended to abolish seizures and subsequent apnea.

Reference

1. Hubbert CH (1977) Covert seizures as a cause of postoperative apnea. Anesth Analg 57:136–138

Address correspondence to: A.M. Zeidan
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