

Myoclonic Seizure in the Postanesthesia Care Unit After Thoracic Laminectomy

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Neurological sequelae after anesthesia and surgery are becoming an increasing medicolegal problem. Seizure is a rare complication after general anesthesia for thoracic laminectomy. Here we report a case of generalized seizure in the postanesthesia care unit (PACU) after thoracic laminectomy because of an unrecognized dura tear. The possible mechanisms and risk factors are discussed.

Case Report

A 37-yr-old man with acute spinal cord compression at T9-10 because of pseudoarthrosis, which was diagnosed by computed tomography (CT) and magnetic resonance imaging, of the spine was scheduled for decompression laminectomy. The patient had no previous surgery and no significant medical history except mild hypertension but no seizure disorder. Diclofenac sodium 50 mg tid and acetaminophen 500 mg tid were being taken for pain, both of which were discontinued on the day of surgery. His preoperative laboratory data were within normal limits. Anesthesia was induced with thiopental 250 mg IV (patient's body weight was 62 kg) followed by rocuronium 40 mg IV to facilitate endotracheal intubation. Anesthesia was maintained with isoflurane at 1.5–2.0 minimum alveolar anesthetic concentration in oxygen with an intermittent dose of rocuronium given for muscle relaxation. An indwelling arterial line was inserted into the left radial artery, and a central venous pressure line was placed in the right internal jugular vein. The patient was turned to the prone position on the operating table and underwent a T7-L1 thoracic laminectomy with internal instrumentation. Vital signs remained stable throughout the 8-h surgery, and 2 U of packed red blood cells were transfused. At the end of surgery after the reversal of the residual muscle relaxant effect, the patient resumed spontaneous ventilation with eyes open on command. Because the body temperature registered 35°C (by rectal probe) and the patient tolerated the oroendotracheal tube well

without coughing, the endotracheal tube remained in place, and the patient was transported to the PACU.

Upon arrival to the PACU, the vital signs were: body temperature of 35.1°C, blood pressure of 130/90 mm Hg, heart rate of 100 bpm, respiratory rate of 20 breaths/min (spontaneous ventilation with oroendotracheal tube in place attached to T-piece on O₂ 4 L/min), and an SpO₂ of 98%. At 30 min after the arrival to the PACU, the patient suddenly developed generalized seizure, which was aborted by the IV administration of midazolam 5 mg. Vital signs showed a blood pressure of 155/95 mm Hg, heart rate of 105 bpm, and respiratory rate of 24 breaths/min. Serum electrolytes, ionized calcium, and magnesium levels were taken and were within normal limits. Neurological examination showed the patient unresponsive (either because of postictal state or the effect of midazolam) without focal neurological signs. However, bouts of generalized seizure lasting approximately 15 s occurred spontaneously every 10–15 min. For seizure control, intermittent doses of midazolam 2.5 mg up to a total dose of 10 mg were given. An initial dose of phenytoin 1 g IV was given and then a 100-mg IV infusion every 8 h. Mechanical ventilation was instituted. No further seizure was observed. Emergency CT of the brain was performed, which showed meningeal dura enhancement with intracranial hypotension or meningeal reaction but no signs of cerebral edema or intracranial hemorrhage (Fig. 1). The patient was admitted to the intensive care unit for continued care. On admission to the intensive care unit, 75 mL of serosanguinous fluid was noted in the Hemovac[®] drain that was placed in the fascial layer next to the implanted Harrington rods and externally attached to an elastic bulb without suction.

On the next day, the patient became fully awake without any seizure activity; therefore, extubation was done. After extubation, on questioning, the patient complained of severe headache even in supine position when 300 mL of cerebrospinal fluid (CSF) was collected in the Hemovac drain from the surgical wound. Dural tear after laminectomy was diagnosed, and on postoperative Day 3, an exploratory thoracic laminotomy was performed, during which a 3-mm dura tear was found at the T8 region that was repaired. The patient was kept on straight bed rest with IV hydration and analgesic drugs. Two days after the second surgery, the patient reported improvement of his headache, and phenytoin was stopped. On postoperative Day 5, the patient was able to sit up without headache or seizure and tolerated oral feeding. Electroencephalography performed on postoperative Day 12

Accepted for publication May 13, 2002.

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DOI: 10.1213/01.ANE.0000023663.68007.3B



Figure 1. Brain computed tomography (CT) showed meningeal dura enhancement with intracranial hypotension or meningeal reaction (arrow) intracranial hypotension (decreased distance between brain and dura).

showed normal tracing, and the patient was discharged home on postoperative Day 14 without any anticonvulsant drug. A 6-mo follow-up showed that the patient had returned to normal life without either a seizure or headache problem.

Discussion

Seizure occurring after surgery may have various causes related to anesthesia or surgery. The anesthetic course in this patient was uneventful and without hypotensive or hypoxic insults, and none of the anesthetics used was known to be seizurogenic in this case. The patient's seizure occurred shortly after arrival in the PACU, and there was no inciting medication given at that time. Also, no antibiotic solution was used to irrigate the wound before wound closure. Therefore, it would be reasonable to exclude anesthesia or the related care in the immediate postanesthesia period as the cause of this patient's seizure.

A number of complications have been reported with laminectomy surgery, with the most common being hematoma or nerve injury (1-3). Postdural headache is also associated with dura tear during laminectomy with the resultant CSF leakage (4,5). Barbaccia (6) reported a case of uncontrollable myoclonus of the lower extremities in a patient in the PACU after lumbar laminectomy and L3-4 discectomy, and on close examination, it was found that 10 mL of 60% meglumine diatrizoate had been given inadvertently for the myelogram during surgery.

Seizures induced by the intrathecal administration of various contrast materials have been reported by different authors (7-10). However, there was no myelography performed or contrast material given in our case. Crofts et al. (11) recently reported a case of tonic-clonic seizure in an elderly patient on the ninth postoperative day after continuous spinal analgesia for postoperative pain control after hemicolectomy, and a CT scan of the brain revealed acute bilateral frontal intracerebral hemorrhage. Subdural or intracerebral hemorrhage results from lumbar puncture (12-15). The mechanism of these morbidities seems to be the result of CSF leakage causing low CSF pressure and traction on the cerebral structures, including the meningeal vessels. In our patient, even though there was no intracerebral hemorrhage demonstrated by CT scan or neurological examination, intracerebral hypotension with meningeal reaction was observed in the CT, and it would be reasonable to assume that an acute rapid CSF loss from a sizable dura tear (3 mm) would cause an acute decrease in CSF pressure resulting in such a meningeal irritation with a manifest seizure. The fact that this patient's seizure abated completely and has not recurred after the repair of the dura tear seems to support this postulation. In searching the literature, we have found no previous case in which seizure disorder was observed with CSF leak without intracerebral hematoma.

In summary, postoperative seizure after laminectomy may occur as a result of unrecognized dura tear during surgery, resulting in acute CSF loss and the drastic decreasing of the CSF pressure either with or without intracerebral hemorrhage. Supportive care and expeditious diagnostic evaluation must be instituted immediately to avoid any untoward sequelae.

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*Journal of the International Anesthesia Research Society, the Society of Cardiovascular Anesthesiologists,
the Society for Pediatric Anesthesia, the Society for Ambulatory Anesthesia,
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