A Case of Seizure in a Patient Following Percutaneous Endoscopic Lumbar Discectomy

Sung Hoon Kim, Geun Sung Song, Soon Ki Sung, Dong Wuk Son

Department of Neurosurgery, Pusan National University College of Medicine, Yangsan, Korea

Seizure following percutaneous endoscopic lumbar discectomy (PELD) is extremely rare. We report that generalized seizure occurred in a patient with radiating right leg pain after PELD under sevoflurane anesthesia. Cerebrospinal fluid (CSF) was detected from a dura tear in the operative field. On emergence from anesthesia, generalized tonic-clonic activity continued for approximately 2 minutes and the level of consciousness was decreased to a stuporous state. Under sedation, a pneumocephalus which was thought to be caused by the dura tear was evaluated with a brain computed tomography (CT) and a continuous slow wave was found on electroencephalography (EEG) without any epileptiform discharges. Eight hours postoperatively, the decreased level of consciousness recovered, and after 2 weeks, the patient was discharged without any neurologic sequelae. Clinicians should recognize the epileptogenic potential of sevoflurane and limit the maximum dose with avoidance of hypocapnia by hyperventilation. If an intracerebral lesion is accompanied, it may increase the possibility of the occurrence of seizure.

Key Words: Seizure ㆍDiscectomy ㆍPercutaneous ㆍPneumocephalus ㆍSevoflurane

INTRODUCTION

Whereas there are many reports in which post-operative seizure have been detected after brain surgery, there has been relatively little described regarding generalized seizure following spinal surgery. During induction or recovery with sevoflurane anesthesia in pediatric patients, the incidence of generalized seizure has been reported to be approximately 6%4). However, in adult patients, generalized seizure is relatively rare. Hilty et al. reported that an intracerebral lesion could be a predisposing factor for the occurrence of seizures on emergence from anesthesia4). There is a report that pneumocephalus can also induce seizures9). We report that generalized seizure occurred in an elderly patient following PELD.

CASE REPORT

A 67-year-old female with low back pain and radiating right leg pain for 2 months was admitted to the clinic. According to the medical records, she had taken medications for hypertension and hyperlipidemia for 5 years. On laboratory findings, no abnormal findings were noted. On the neurologic exam, low back pain and radiating right leg pain were demonstrated, but no weakness was evident in the lower extremities. On the radiologic exam, there was right extrusion of the L3/4 disc with right lateral canal stenosis, diffuse bulging of the L4/5 disc with a faint high intensity zone (HIZ) and bilateral intervertebral foraminal narrowing, and central bulging and left foraminal bulging of the L5/S1 disc with a high signal area, to the medical records, she had taken medications for hypertension and hyperlipidemia for 5 years. On laboratory findings, no abnormal findings were noted. On the neurologic exam, low back pain and radiating right leg pain were demonstrated, but no weakness was evident in the lower extremities. On the radiologic exam, there was right extrusion of the L3/4 disc with right lateral canal stenosis, diffuse bulging of the L4/5 disc with a faint high intensity zone (HIZ) and bilateral intervertebral foraminal narrowing, and central bulging and left foraminal bulging of the L5/S1 disc with a high signal area,
suggesting a vascular lake rather than HIZ (Fig. 1). Due to the patient's severe pain and anxiety, general anesthesia was administered on request. Anesthesia was maintained with nitrous oxide and sevoflurane. PELD in the right L3/4 disc space was performed in the prone position. The extruded indigo carmine-stained disc was removed using forceps. In the operative field, a dura tear occurred from which CSF leaked. The endoscope was removed and a 1-point suture was placed with a dressing. Upon emergence from anesthesia, sudden jerking movements began in the legs and spread to the body. The oxygen saturation (SpO₂) measured by pulse oximetry was decreased. Generalized tonic-clonic activity continued for approximately 2 minutes. The level of consciousness was decreased to a stuporous state. Sedation was achieved immediately by administration of intravenous thiopental and rocuronium with effective ventilation. The SpO₂ returned rapidly to normal and the seizure ceased with spontaneous ventilation. A brain CT was done and the patient was transferred to the intensive care unit (ICU) with an endotracheal tube in place. On the brain CT, a pneumocephalus was noted in the middle cranial fossa, circle of Willis, and left Sylvian fissure without any abnormal findings (Fig. 2). On the neurologic exam, the level of consciousness was still stuporous and the ocular light reflex were sluggish bilaterally. Seizure recurred and phenytoin was administered immediately. After cessation of the seizure, a general evaluation including an EKG, echocardiography, and laboratory testing were done. However, no abnormal findings were

**Fig. 2.** Brain computed tomography. The pneumocephalus was noted in the middle cranial fossa, circle of Willis, and left Sylvian fissure without any abnormal findings (Fig. 2). On the neurologic exam, the level of consciousness was still stuporous and the ocular light reflex were sluggish bilaterally. Seizure recurred and phenytoin was administered immediately. After cessation of the seizure, a general evaluation including an EKG, echocardiography, and laboratory testing were done. However, no abnormal findings were

**Fig. 3.** Electroencephalography. Continuous slow wave suggestive of mild diffuse cerebral dysfunction was checked and no epileptiform discharges were noted.
detected. Continuous slow waves in the EEG was suggestive of mild diffuse cerebral dysfunction with no epileptiform discharges noted (Fig. 3). A brain magnetic resonance image (MRI) including a diffusion image was done and showed no abnormal findings other than the pneumocephalus. There was no personal or family history of seizures. After 8 hours of the surgery, the patient awoke with non-verbal cooperation and the endotracheal tube was removed. On the 2nd postoperative day, the level of consciousness had recovered to the alert state and both ocular light reflexes to light were intact. The radiating right leg pain was relieved and low back pain was decreased. After 2 weeks of the surgery, the patient was discharged without any neurologic complications.

**DISCUSSION**

Even though there are many reports about post-operative seizures after a craniotomy, there are little reports of seizure following spinal surgery. Manaka et al. reported that the causes of post-operative seizures after craniotomy may involve the topical application of hemoglobin onto the brain leading to the massive generation of free radicals and the disturbance of ion balance caused by cerebral ischemia or hypoxia. In our case, there was no hemorrhage onto the brain or cerebral ischemia. Also, there was no personal or family history of seizures and the patient did not use any stimulant-type drugs. On emergence from general anesthesia, a sudden jerking movement began in the legs and spread to the body for approximately 2 minutes. With respect to the cause of the generalized seizure, three points should be considered.

First, on the brain CT after the seizure, except for the pneumocephalus in the middle cranial fossa, circle of Willis, and left Sylvian fissure, no abnormal findings were noted including no hemorrhage. With the operative field, a dura tear with CSF leakage occurred during the PELD. The pneumocephalus might have been caused by the dura tear in the operative field. Roderick suggested that because nitrous oxide is dissolved in blood and enters the air-containing space more rapidly than nitrogen exits, if a patient is allowed to inhale nitrous oxide, it may increase the intracranial pressure (ICP) by expanding the volume of the pneumocephalus. In our case, the patient was maintained with nitrous oxide under general anesthesia, which may have increased the ICP by increasing the volume of the pneumocephalus. Markham reported that seizures occur in patients with a pneumocephalus. Even though there is no literature about the mechanism of seizures by a pneumocephalus, it could have served as the nidus of seizures initiated by increasing the ICP.

Second, procedure itself could be the risk factor of seizure. Choi et al. reported that seizure during PELD can happen owing to the increased speed of saline irrigation or longer operative time. They may increase the cervical epidural pressure, resulting in the increased ICP. Neck pain is the most common prodromal symptoms. In our case, the patient did not complain of neck pain during PELD. However, owing to the unwanted dura tear, increased speed of saline irrigation and longer operative time to prevent bleeding by dura tear may aggravate the cervical epidural pressure, resulting in the more increased ICP.

Third, sevoflurane anesthesia was used in our patient. Even though tens of millions of patients have been given sevoflurane during anesthesia, there have been several reports of seizure in patients under sevoflurane anesthesia. There are a few reports that the incidence of seizures under sevoflurane anesthesia is approximately about 5%. The mechanism of the epileptogenic effect of sevoflurane is thus far unknown. In the early 1990s, it was first described that abnormal EEG findings under sevoflurane anesthesia were detected in children. Constant et al. reported that the incidence of epileptiform EEG changes correlate with the increasing dose of sevoflurane. Using a maximum of 1.5 minimum alveolar anesthetic concentration (MAC) sevoflurane will decrease the possibility of epileptogenic activity. In our case, a continuous slow wave pattern suggestive of mild diffuse cerebral dysfunction on EEG after the seizure was noted and no epileptiform discharges were detected. Because we could not perform an intra-operative EEG, we could not identify whether epileptiform EEG changes occurred during anesthesia. Until the level of consciousness of the patient recovered, the continuous slow wave pattern on EEG monitoring continued. Limiting the depth of anesthesia by sevoflurane is essential. In addition, during sevoflurane anesthesia, hypocapnia induced by assisted ventilation may amplify greater EEG changes, most notably in very young and very old patients. Thus, these authors recommended that for the prevention of post-operative seizures, the dose of sevoflurane should be limited to <1.5 minimum alveolar anesthetic concentration (MAC) and hypocapnia should be avoided.

In our case, the pneumocephalus may have aggravated the epileptogenic condition under sevoflurane anesthesia. In the view of the enormous amount of sevoflurane anesthesia given world-wide, concern about the epileptogenic potential of sevoflurane may be minimal. However, accompanying intracranial lesions may increase the possibility of the occurrence of seizure during sevoflurane anesthesia.

**CONCLUSION**

We managed a rare case of generalized seizure followed by PELD. It should be considered that accompanying intracra-
nial lesions like pneumocephalus by dura tear may increase the possibility of the occurrence of seizure. By avoiding unnecessary bleeding during PELD, speed of saline irrigation and duration of operative time should be minimized.

With the recognition of the epileptogenic potential of sevoflurane, clinicians should limit the maximum dose of sevoflurane and avoid hypocapnia by hyperventilation.

**ACKNOWLEDGEMENTS**

This work was supported for two years by Pusan National University Research Grant

**REFERENCES**