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Foot Drop of Contralateral Limb after Deformity Correction in a Polio Patient: A Case Report

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Postpoliomyelitis syndrome is a common neurological disorder that occurs in patients who have experienced paralytic poliomyelitis. Recently, as a result of vaccination against poliovirus, incidence of poliomyelitis is exceedingly low. However, many patients with postpolio syndrome may encounter anesthesia when undergoing surgery, such as for correction of foot deformity and other operations. We report on a 45-year-old woman who experienced paralysis of her contralateral limb after operation on the left foot under spinal anesthesia. Postoperative electromyography/nerve conduction study (EMG/NCS) was performed in order to determine the cause of paralysis. Motor power of the sequelae involved leg showed improvement with time and recovered fully to the preoperative level at six months after the index operation. A precise evaluation, including a physical examination and EMG/NCS, should be performed preoperatively when spinal anesthesia is planned for postpolio syndrome patients.

Key Words: Poliomyelitis, Spinal anesthesia, Paralysis, Postpoliomyelitis syndrome

Postpoliomyelitis syndrome (PPS) is a late neurological disorder that occurs in patients who have recovered from paralytic poliomyelitis.[1,2] Although the incidence of paralytic poliomyelitis is extremely low in civilized countries after vaccination against poliovirus, surgeons may encounter foot deformities in patients with PPS, which need to be corrected operatively. Therefore, the safety issues after anesthesia in patients with PPS are still of great concern[3] and Hodgson et al.[4] had reported the neurotoxicity of a spinal anesthetic drug in an experimental animal study. However, there has been no report on the aggravation of leg paralysis related with spinal anesthesia. We report the case of a 45-year-old woman who experienced paralysis of her contralateral leg after surgery on the left foot under spinal anesthesia.

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CASE REPORT

A 45-year-old woman (160.7 cm, 59.1 kg) came to the outpatient department complaining weakness of the left lower extremity after a fall from a bicycle two months earlier. She had a history of poliomyelitis on the left lower extremity. She presented with combined equinus deformity and tenderness of the anterior tibialis insertion site on her medial cuneiform bone. The motor power of the anterior tibialis on her left side was grade 0, and the power of the extensor digitorum longus was grade 2. The power of all muscles on her right side was grade 5. The patient underwent an ultrasound, and on the impression of anterior tibial tendon rupture, the patient underwent surgery.

The operation was performed under spinal anesthesia. In the left lateral decubitus position, spinal anesthesia was utilized via a midline approach at the L5-S1 level with a 25-gauge spinal needle. The patient did not complain of any electrical pain during needle insertion. After confirming clear cerebrospinal fluid leakage, 14 mg of heavy bupivacaine 0.5% was injected intrathecally for anesthesia. No epidural catheter was inserted. The position was changed to the supine position after intrathecal injection. The level of spinal

anesthesia was T6 dermatome according to an alcohol swab. She received perioperative antibiotics including flomoxef 500 mg intravenously and midazolam 1 mg via intravenously for preoperative sedation.

The operation was performed in the supine position, and a tourniquet was applied to the left mid-thigh area with 270 mmHg of pressure. On the right lower extremity, sufficient padding was applied as usual. The surgery took approximately 2 hours and went without any special events. A calcaneal osteotomy with medial displacement and dorsiflexion osteotomy of the first metatarsal were performed for the correction of the cavovarus deformity (Figs. 1, 2). Operations and postoperative recovery from anesthesia were carefully monitored by an anesthesiologist, and there were no specific events.

Ten hours after surgery, when the effect of the anesthetic had worn off, the orthopedic resident was notified by the nurse that the right lower extremity power was decreased. Upon a physical examination by the orthopedic resident, the extensor hallucis longus power was decreased to grade 1 and the ankle dorsiflexion power was decreased also to grade 1 on the right side. Sensory feeling on the dorsum of foot had decreased to 20%, as described by the patient. Upon suspicion of a hematoma of the spinal cord, a magnetic resonance imaging of the L-spine was taken immediately. There were no lesions showing evidence of a significant hematoma (Fig. 3).

The symptoms of the patient did not improve two days after the surgery. An electromyography (EMG) and nerve conduction study (NCS) was performed, revealing sequelae of the anterior horn cell lesion of the right lower extremity, most likely due to residual poliomyelitis. Ankle range of motion exercise and stretching were consistently done via rehabilitation with ankle-foot orthosis. Three weeks after the surgery, ankle dorsiflexion and extensor hallucis longus showed slight improvements, but these conditions were still below grade 2. Upon postoperative day at three weeks, EMG/NCS suggested both possibility of the presence of right peroneal neuropathy around the fibular head level, with a moderate partial axonotmesis state and sequelae of anterior horn cell disease. Six months after the surgery, the ankle dorsiflexion and extensor hallucis longus power were improved to the preoperative level. EMG/NCS was also comparable with clinical manifestation (sequelae of anterior horn cell disease, bilateral L4 and below, stationary state, and a markedly improved state of the right neuropathy, with no definite evidence of ongoing disease activity).

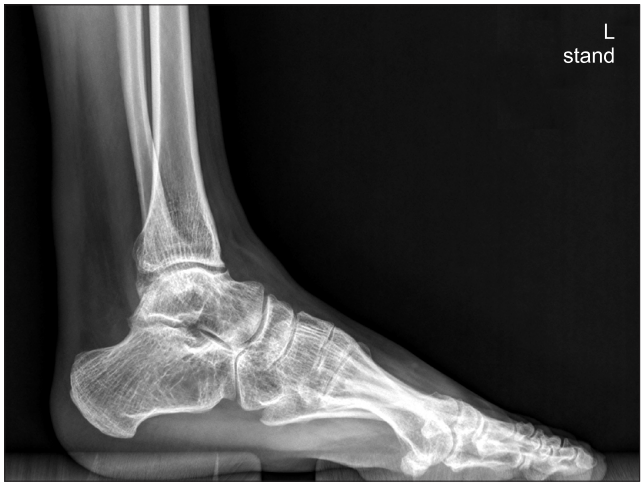

Figure 1. Preoperative standing lateral radiograph of the foot and ankle.

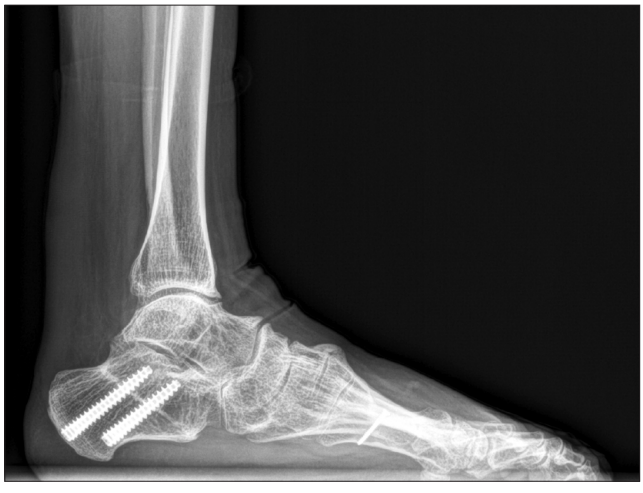

Figure 2. Postoperative standing lateral radiograph of the foot and ankle.

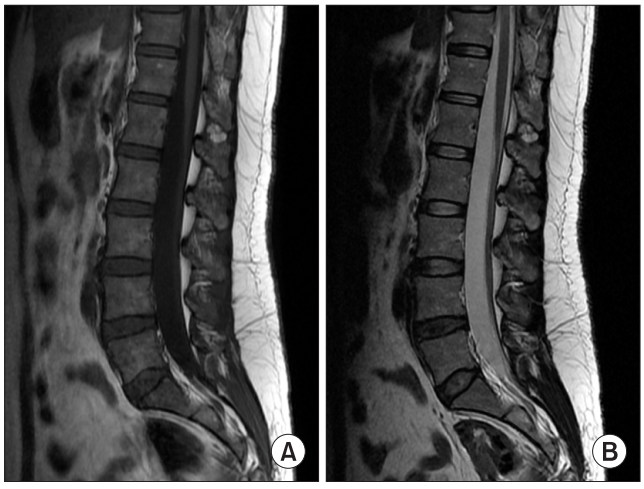

Figure 3. Postoperative L-spine magnetic resonance imaging. There were no lesions showing evidence of hematoma. (A) T1 sagittal image, (B) T2 sagittal image.

DISCUSSION

Spinal polio, the most common form of paralytic poliomyelitis, results from a viral invasion of the motor neurons of the anterior horn cells or the ventral gray matter section in the spinal column, areas responsible for the movement of the muscles, including those of the trunk, limbs and the intercostal muscles.[5] PPS is a common neurological disorder which occurs in individuals who have recovered from paralytic poliomyelitis.[6] The cause of PPS (new weakness or muscular fatigue) remains unclear, although the most widely accepted hypothesis, proposed by Wiechers and Hubbell,[7] attributes these symptoms to the distal degeneration of axons in the greatly enlarged motor units that develop during recovery following acute paralytic poliomyelitis. After recovery from acute paralytic poliomyelitis, motor units can become seven or eight times larger than normal[8] through terminal axonal sprouting and the reinnervation of denervated muscle fibers. Many of these denervated muscle fibers are reinnervated by sprouts from neighboring axons, but eventually this balance is lost and permanent denervation occurs, with the resultant increased weakness.[1] The enlarged and weakened motor units are much more susceptible to damages compared to those in normal patients.

Spinal anesthesia of a patient is done by the injection of a local anesthetic near the spine, resulting in anesthesia of both pain-conducting nerves and motor neurons. Residual polio patients are very sensitive to anesthetics reacting on their already damaged anterior horn cells. Many anesthesiologists tend to avoid the use of regional anesthetics in poliomyelitis patients due to complications that exacerbate disease or cause other complications. Although there are no case reports of side effects after spinal anesthesia in PPS patients, Hodgson et al.[4] reported neurotoxicity of drugs given intrathecally in animal studies.

In our case, the patient did not complain of weakness of the right lower extremity and patient was considered to have near normal level on preoperative physical examinations. After operation, we thought that the patient had some damage to the anterior horn cells of the right lower extremity or peroneal nerve palsy, which was suggested on the postoperative EMG/NCS study revealing. Because peroneal nerve palsy was not appeared in EMG/NCS until postdamage 2 or 3 weeks, the results of postoperative 2 days was only revealing sequelae of the anterior horn cell lesion of the right lower extremity. In a foot three-dimensional multi-segment gait analysis, the dorsiflexion motion of the right ankle decreased suggesting the weakness of ankle dorsiflexor muscles.

The exact cause of leg paralysis in our case is not clear yet. Both the damage by the spinal anesthetic drug on the weakened anterior horn cells and/or subtle compression of peroneal nerve during perioperative period might contribute to the development of leg paralysis. In both situations, previously damaged nerve is thought to be a reason of leg paralysis caused by minor injuries on peripheral nerve systems. In our case, motor power of paralyzed leg recovered to preoperative level in six months after surgery with conservative treatment of ankle-foot orthosis.

In patients with PPS, previous articles only focused on the effects of general anesthesia.[9] However, foot drop after spinal anesthesia was reported in some studies,[10] although those patients were not PPS patients. We present the case of poliomyelitis that reveals that spinal anesthesia, along with general anesthesia, should be conducted with great caution on patients with residual poliomyelitis. A preoperative discussion with an anesthetics specialist and special care not to provoke peripheral nerve damage, especially on peroneal nerve, should be mandatory to avoid any side effects of the anesthesia. In conclusion, a precise preoperative evaluation, including a physical examination and EMG/NCS, should be mandatory when the operative treatment under spinal anesthesia is planned for PPS patients. We think that anesthesia options other than spinal anesthesia should be considered in patients with PPS if possible.

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