Letter to the Editor

Prolonged spinal anesthesia in three brothers

To the Editor,

We write to share our observations of an extremely prolonged sensory and motor blockade after spinal anesthesia in three brothers. The literatures contain only a few case descriptions of this occurrence, and authors have speculated that the cause might be a very serious complication, such as epidural hematoma.\(^1\)\(^2\)\(^3\)\(^4\) After central neural blockade, unusually prolonged motor and sensory blockade may be the only sign of spinal epidural hematoma.\(^2\)\(^3\) Attempts to use magnetic resonance imaging or computed tomography to identify causes for this abnormally long blockade have been unsuccessful.\(^1\)\(^2\)\(^3\)\(^4\) No previous reports have indicated genetic factors as being potentially relevant;\(^2\)\(^3\) however, our experience suggests that genetic factors or disorders should be kept in mind.

A 24-year-old American Society of Anesthesiologists (ASA) I male patient (Case 1) with unremarkable medical history was scheduled for lumbar discectomy. Spinal anesthesia was induced with the patient in the sitting position. A midline approach at the L3–L4 interspace was used and 2.4 mL 0.5% hyperbaric bupivacaine was administered successfully via a 27-gauge Quincke needle on the first attempt. Maximum block height was T-8. Surgery was performed with the patient in the prone position and was uneventful; however, it was necessary to delay discharge until 36 hours postoperatively because of prolonged motor blockade of the patient’s lower extremities. There was no decreasing in the intensity of both motor and sensory blockade in the first 26 hours. Magnetic resonance imaging during the prolonged blockade was normal, and the patient was discharged in good health after the blockade resolved spontaneously. During the event, we received new historical information that the patient’s two brothers also experienced the same problem after spinal anesthesia.

Eighteen months before Case 1, another brother (20 years old, ASA I; Case 2) of Patient 1 had undergone knee arthroplasty at our hospital. We reviewed this patient’s medical record and verified details in a telephone conversation with him. Spinal anesthesia had been performed in the sitting position, with a midline approach at the L3–L4 interspace and with 2.4 mL 0.5% hyperbaric bupivacaine administered successfully via a 27-gauge Quincke needle on first attempt. There was no decrease in the intensity of both motor and sensory block in the first 26 hours. Maximum block height was T-8. Regression of pinprick, touch, and cold were also prolonged. The procedure was uneventful but discharge was delayed for 30 hours because of prolonged motor blockade of the patient’s lower extremities. Magnetic resonance imaging was normal and the patient was ultimately discharged without problem after the motor and sensory blockade resolved spontaneously.

Approximately 2 years before Case 1, the other brother of Patient 1 (a 21-year-old ASA I patient at the time; Case 3) had undergone knee arthroplasty at another center. We obtained information about this case from Patient 1 and in a telephone conversation with Patient 3. No specifics regarding the drugs or equipment used were available; however, this brother had also undergone spinal anesthesia and there were no unexpected events during the procedure. His discharge was delayed approximately 48 hours because of motor and sensory blockade of the lower extremities. We offered to perform magnetic resonance imaging, but the patient did not accept it.

We first speculated that prolonged motor and sensory blockade after spinal anesthesia in this family could be from complications of the procedure; however, the genetic factors may be involved in some way. In cases with prolonged block, the differential diagnosis should include hematoma formation secondary to spinal intervention, cauda equina syndrome, transient radicular irritation (TRI), and anterior spinal artery syndrome.\(^5\)\(^6\) There was no pain, paresthesia, or hemorrhage during the insertion of the spinal needle, and spinal puncture was successful at the first attempt in our case. Thus, development of hematoma or nerve damage because of needle insertion was unlikely. Moreover, the patient did not develop low blood pressure that would decrease blood flow, nor had been administered intravenous vasopressor drugs. Considering the height of the patient, the bupivacaine and opioid doses used were in accordance with the suggested doses in the literature. No other neurological symptoms or signs developed in conjunction with prolonged sensory and motor block in the postoperative period in our patient. Transient neurological symptoms (TNS) and TRI are terms used to describe bilateral pain in the lower back or buttocks and/or radiating down the lower extremities that develop after recovery from spinal anesthesia. They are not accompanied by any motor, sensory, or sphincter dysfunction or signs of meningeal irritation.\(^6\) Cauda equina syndrome and TRI are associated with accompanying vascular conditions, old age, epinephrine, or hyperbaric bupivacaine use, and the lithotomy position in patients undergoing spinal anesthesia. The causative mechanism is suggested to be nerve cell membrane damage and neuronal injury because of the high concentrations of local anesthetics.\(^7\) We did not detect any additional neurological symptom or sign in our case that could be associated with the aforementioned pathologies.

We believe that genetic factors deserve further consideration in such cases. Future studies and case reports are warranted to elucidate the etiology and to identify potential contributing factors.
References