Case Report

Transient Neurologic Syndrome After Spinal Anesthesia With Epidural Steroid Treatment

L. Pirbudak Cöcelli, MD; Ibrahim Erkutlu, MD; Gunhan Karakurum, MD; Neslihan Avci, MD; Rauf Gül, MD; and Ünsal Öner, MD

Department of Anesthesiology, Gaziantep University, Gaziantep, Turkey; Department of Neurosurgery, Gaziantep University, Gaziantep, Turkey; and Department of Orthopedics, Gaziantep University, Gaziantep, Turkey

ABSTRACT

BACKGROUND: Transient neurologic syndrome (TNS) is a rare complication of spinal and epidural anesthesia. It is defined as paradoxic postoperative back pain radiating to the lower extremities with no neurologic deficits. Because it is a self-limited disease, the treatment is usually symptomatic and consists of NSAIDs and injections of a neuromuscular-blocking drug at the trigger points. The syndrome may be resistant to this treatment regimen and may last for several months, resulting in a long convalescence.

CASE SUMMARY: A 63-year-old Turkish woman (height, 165 cm; weight, 71 kg) underwent hemorrhoidectomy in the jackknife position using spinal anesthesia. No adverse events occurred during puncture or surgery or in the immediate postoperative recovery period. Recovery from the sensory and motor block was normal. Twenty-four hours after surgery, lower limb and plantar pain developed with no sensory or motor deficit. Neurologic examination revealed normal motor and sensory function. Electroneuromyography showed partial denervation potential of muscles innervated by the left sciatic nerve. The symptoms were suggestive of TNS. Combination oral NSAID treatment with amitriptyline (25 mg/d) and gabapentin (1200 mg/d) was initiated. Because the pain still persisted 6 weeks after surgery, epidural steroid injection with triamcinolone acetate (80 mg) with isotonic saline was administered, resulting in definite pain relief (visual analog scale score = 0).

CONCLUSIONS: Epidural steroid treatment was effective in this patient with TNS resistant to treatment with NSAIDs, amitriptyline, and gabapentin. Future studies are needed to evaluate this treatment. (Curr Ther Res Clin Exp. 2009;70:316–322) © 2009 Excerpta Medica Inc.

KEY WORDS: anesthesia, spinal, analgesia, epidural, postoperative complications, neurologic.

Accepted for publication April 8, 2009.

© 2009 Excerpta Medica Inc. All rights reserved.

doi:10.1016/j.currtheres.2009.08.002

0011-393X/$ - see front matter
INTRODUCTION

Spinal anesthesia is associated with multiple advantages compared with general anesthesia, including reduced morbidity and mortality, improved postoperative analgesia, and enhanced cost-effectiveness. Although rare, neurologic injury is a possible complication.

First described in 1993, transient neurologic syndrome (TNS) is characterized by back pain radiating to the legs, without sensory or motor deficit, that occurs after a spinal block. TNS usually resolves spontaneously within several days. It is most commonly associated with hyperbaric lidocaine (incidence up to 11.9%) but has also been reported with bupivacaine (1.3%). There are also case reports of TNS after epidural anesthesia. This syndrome has been most commonly reported among outpatients after surgery performed in the lithotomy position and is thought to be associated with early ambulation. TNS appears to be lowest among inpatients after surgery performed in positions other than the lithotomy position. Few cases of TNS have been reported after the administration of spinal lidocaine for cesarean section. The pathogenesis of TNS is unclear and controversy exists as to whether it represents neurotoxicity or myofascial pain resulting from musculoskeletal strain.

Intrathecal administration of local anesthetics is reported to increase glutamate concentration in cerebrospinal fluid and histopathologic changes of motor neurons in the lumbar spinal cord, suggesting damage to dorsal and ventral roots. After peripheral nerve injury, inflammatory cells play an important role in the development of pain. With inflammatory cell activation, vasodilatation and extravasation of plasma proteins are accompanied by the release of chemical mediators, including serotonin, bradykinin, substance P, and products from the cyclooxygenase and lipoxygenase pathways of arachidonic acid metabolism. The result is a chemical sensitization of high-threshold nociceptors, which start to transmit low-intensity painful stimuli. Corticosteroids are potent anti-inflammatory agents that act via inhibition of phospholipase A2. Additionally, a direct analgesic effect on C fibers has been reported.

TNS is managed symptomatically with NSAIDs and injections of neuromuscular-blocking drugs at trigger points. A report of the treatment of these patients with systemic and/or epidural corticosteroids was not identified in the English literature in an unrestricted search of Google and MEDLINE using the terms: anesthesia, spinal, analgesia, epidural, postoperative complications, neurologic, methylprednisolone, gabapentin, spinal anesthesia, transient neurologic symptoms, spinal cord injury, sciatic nerve injury, neurotoxicity, and epidural corticosteroid injection. Herein, we report a case of treatment with an epidural corticosteroid in a patient who developed TNS after a surgical procedure performed in the jackknife position with spinal anesthesia.

CASE REPORT

A 63-year-old Turkish woman (height, 165 cm; weight, 71 kg) was admitted to the Gaziantep University Outpatient Pain Clinic, Gaziantep, Turkey, with a complaint of pain originating from the left knee and extending through the leg and plantar portion of the left foot. The pain was described as “a streak of lightning” or “pin pricks” mainly in the plantar aspect of the foot. The patient had a history of hemorrhoidectomy.
Data on the surgical history of the patient were collected by evaluating the patient's file (e.g., surgical notes written by the surgeon, the anesthesia report by the anesthesiologist, ward progress notes, consultation notes) and by personal communication with the involved staff.

The pain started 24 hours after surgery and did not respond to analgesic treatment. The visual analog scale (VAS) scores (0–10; 0 = no pain, 10 = worst possible pain) had increased from 3 and 4 to 6 and 7 in 6 weeks after surgery and prior to clinic admission. Spinal anesthesia was performed using 4 mL of hyperbaric bupivacaine administered via a 25-G spinal needle. There were no complications during (e.g., advanced penetration to bone, paresthesia, bloody cerebrospinal fluid flow) or after (severe hypotension) the procedure. Surgery lasted ~1 hour and was completed successfully. No gluteal intramuscular injection was performed before or after the surgery.

The patient stated that massage and exercise relieved the pain and cold aggravated it. The findings on sensory examination were normal, and motor examination of the left lower limb revealed normal deep tendon reflexes with 4/5 strength in ankle plantar flexion and dorsiflexion. Electroneuromyography (ENMG) performed 10 days after the patient’s referral showed a partial lesion of the left sciatic nerve. Fifteen days later, the findings on a repeat control ENMG were completely normal. Combination oral NSAID treatment with amitriptyline (25 mg/d) and gabapentin (1200 mg/d) was initiated. The drug doses were doubled within 1 week; however, adverse effects (AEs) (orthostatic hypotension, nausea, dizziness) necessitated a return to the initial doses. At week 6 of treatment, the patient was referred to our clinic because of ongoing pain. The patient was otherwise healthy; specifically, she did not have diabetes, hypertension, or peripheral artery disease, nor was she using any drugs chronically. Laboratory findings and results of plain radiographic studies and magnetic resonance imaging (MRI) were compiled, and specialists were consulted to rule out vertebral infection, disc herniation, trauma, or any other occult disease that might be responsible for the symptoms.

We planned pain management with epidural analgesia and supplementation with oral gabapentin 1200 mg/d and tramadol 150 mg/d to alleviate depression. After written informed consent was obtained, epidural injection was administered via the median approach while the patient was being monitored (electrocardiograph, blood pressure, pulse oximetry). We reached the epidural space (L4–L5) using the resistance-loss technique with an 18-G Touhey needle (Perifix 401, B. Braun Medical Inc., Bethlehem, Pennsylvania). When the needle was in the subarachnoid space, 5 mL of bupivacaine 0.25% with fentanyl 25 µg was administered via an epidural catheter, resulting in a slight increase in the patient’s pain (VAS = 8). After 30 minutes, triamcinolone acetate* 80 mg with isotonic saline 10 mL was administered into the epidural space. The patient laid in the left lateral decubitus position for 20 minutes after injection. The pain disappeared 30 minutes after the steroid was administered. The patient was observed for 2 hours after the injection for possible complications. Tramadol hydrochloride 150 mg/d, gabapentin 1200 mg/d, and ascorbic acid 1 g/d were prescribed for the next 6 months. VAS scores were recorded before injection, 30 minutes

*Trademark: Kenakort® A (Bristol-Myers Squibb, Maslak, Istanbul).
after injection, and 2, 4, and 6 weeks after the injection (8, 0, 0, 0, and 0, respectively). At the last follow-up visit, the patient was free of pain and neurologic examination and ENMG were normal.

DISCUSSION

TNS is characterized by pain in the buttocks, legs, and feet that is not associated with any neurologic deficit after spinal anesthesia. Symptoms develop within 24 hours after the effect of neuroaxial anesthesia disappears, often 2 to 4 hours after the patient regains mobility. Symptoms usually decrease within a few hours to a week. In rare instances, the symptoms continue for as long as 1 year. Philip et al reported that symptoms occurred within 24 hours, resolved within 48 hours without any intervention, and were not associated with sensory or motor deficits or functional impairment. If spinal anesthesia is used and a neurologic complication is noted postoperatively, a neurology consultation should be obtained promptly.

In the current case, radiologic and laboratory evaluations did not indicate that other conditions should be considered in the differential diagnosis. The erythrocyte sedimentation rate and C-reactive protein concentration were not elevated, and MRI was negative for vertebral infection and disc herniation. Moreover, the consulting physicians did not suggest alternative diagnoses, including any intra-abdominal or intrapelvic pathology, that might cause back pain.

A traumatic cause for the patient’s condition was also considered. In the literature, 5 cases of sciatic nerve injury were identified after cesarean section under regional anesthesia. Silva et al reported 2 cases of right-sided sciatic nerve injury secondary to positional nerve compression. The suggested etiology in 1 case was nerve compression due to remaining in the same sitting position for a prolonged period. Full recovery occurred in 5 months. The other case was attributed to nerve compression in the presence of severe hypotension. The patient’s motor deficit resolved in 3 months, although sensory deficit and mild pain persisted for 1 year. In another case, Umo-Etuk and Yentis suggested that left lateral tilt during surgery caused the injury. The patient recovered completely in 6 weeks. Roy et al and Postaci et al described 2 cases similar to that of Umo-Etuk and Yentis. They also suggested that compression of the sciatic nerve in the left lateral tilt position was the mechanism of injury. In addition, Postaci et al reported that during spinal needle insertion through the L3–L4 vertebral interspace, the patient complained of paresthesia radiating throughout her back and down her left leg. These cases suggest that patient positioning in the setting of profound motor blockade might have a role in the pathogenesis of these neurologic symptoms. Because this was the first case to be reported in the jackknife position, the possible patterns and mechanisms of nerve compression, if any, are yet to be determined.

If our patient’s symptoms were secondary to a traumatic spinal intervention or nerve compression, longer, more substantial alterations in the initial and control ENMGs would have been likely and the neurologic examination would probably have been more representative of the injury. Although the initial ENMG revealed a partial nerve lesion, it was performed early in the evaluation and may have been inaccurate.
The ENMG changes associated with denervation due to neurologic injury require 14 to 21 days to appear in the lower extremities. The findings on the control ENMG study that was performed after 3 weeks appeared completely normal, excluding any true nerve injury.

Permanent neuronal injury has been reported after damage to nerve cell membranes due to a high concentration of local anesthetic. Glutamate neurotoxicity occurred in the exotoxic and oxidative pathways. In the exotoxic pathway, toxically high concentrations of calcium ion penetrated the intracellular region. The increase in neuronal calcium induced activity of numerous enzymes (eg, adenosine triphosphatase, phospholipase, protease, endonuclease) that resulted in irreversible neuronal damage. In the oxidative pathway, excess production of reactive oxygen compounds was thought to cause the neurotoxicity. For this reason, we included vitamin C, a potent antioxidant, as part of this patient’s therapy.

Extracellular calcium concentration decreases within minutes after spinal cord injury. This is possibly directly related to the liberation of neurotransmitters and may in turn activate membrane-related phospholipases. High-dose methylprednisolone treatment interferes with many of these mechanisms. Administered in high doses, methylprednisolone blocks lipoperoxidation. The drug may also restore the calcium balance of the cell membranes and block phospholipase activity. Therefore, we used corticosteroid therapy because of its anti-inflammatory and antioxidative effects. Our literature search identified no other reports of the treatment of the patients with systemic or epidural corticosteroids.

Although systemic steroid treatment can affect blood-glucose and serum-electrolyte concentrations, serum lipid profile, and systemic blood pressure in healthy subjects, steroids administered by epidural injection are not associated with these effects. Moreover, epidural steroid injection is not associated with adrenocortical suppression, which is an important AE of systemic steroids. Triamcinolone acetate is less water soluble and has more depot effects compared with betamethasone dipropionate. As a result, it is absorbed slowly from the epidural space and may cause longer local effects. Therefore, triamcinolone acetate was selected in the present case.

Pharmacotherapy is the cornerstone of systemic pain management. Commonly suggested pharmacologic treatments for neuropathic pain include tricyclic antidepressants, anticonvulsants, opioids, and nonopioid analgesics. Although a positive effect was reported, even with small daily doses (100–1200 mg/d), questions remain about the potential for treatment failure with low-dose gabapentin. Rapid dose escalation might be responsible for an increase in the incidence of AEs. Studies suggest that if the drug is ineffective at low doses, a time-limited high-dose course (≤3600 mg/d) may be attempted before the drug is abandoned and that slower titration may reduce central nervous system AEs. However, studies have found that both tramadol and gabapentin are useful in mitigating neuropathic pain. In addition, a recent trial suggested that a combination of continuous brachial plexus blockade with memantine may be useful in reducing acute and persistent neuropathic pain. Because our patient failed to respond favorably to NSAIDs or amitriptyline and gabapentin, and because higher doses of these drugs resulted in an increased incidence of AEs, we prescribed...
a 6-month regimen of tramadol 150 mg/d and ascorbic acid 1 g/d, keeping the gabapentin dosage fixed at 1200 mg/d. Slow titration (ie, doubling the drug doses within 1 week) seemed time-consuming, so it was not preferred. Before this treatment, the patient received epidural analgesia with bupivacaine and fentanyl during which she experienced a paradoxic increase in pain that was possibly associated with the bupivacaine administration. Corticosteroid administration, however, permanently relieved the TNS pain both in the early term and at follow-up. Future studies are needed to evaluate this treatment option.

CONCLUSION
Epidural steroid treatment was effective in this patient with TNS resistant to treatment with NSAIDs, amitriptyline, and gabapentin.

ACKNOWLEDGMENT
The authors have indicated that they have no conflicts of interest regarding the content of this article.

REFERENCES

ADDRESS CORRESPONDENCE TO: L. Pirbudak Cöcelli, MD, Universite PTT Şubesi, PK 43, 27310, Gaziantep, Turkey. E-mail: lutfiyep@hotmail.com