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Case Report

A Treatment Option for Post-Injection Sciatic Neuropathy: Transsacral Block with Methylprednisolone

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Background: Accidental intraneural injection induced nerve injury is an iatrogenic tragedy and intramuscular injection (IM) is the most common injury mechanism affecting the sciatic nerve. The most frequent presentation of sciatic nerve injury includes radicular pain and paresthesia with almost immediate onset of variable motor and sensory deficit.

Objectives: Intraneural injection is a common injury mechanism of the sciatic nerve and generates neuropathic pain with inflammatory neuritis. Steroids inhibit the production of inflammatory mediators and reduce ectopic discharges on damaged neural membranes. The results of transsacral steroid injection on neuropathic pain in 5 patients with accidental sciatic nerve injury due to intraneural injection were presented in this report.

Design: Report of 5 cases.

Description of Cases: Five patients, 32, 34, 45, 54 and 70 years old respectively, complaining of severe neuropathic pain, paresthesia and progressive weakness of the lower extremity with difficulty in walking secondary to gluteal injection were admitted to the clinic. The symptoms were resistant to drug therapies. Electromyography disclosed axonal damage of the sciatic nerve. The initial examination of the patients revealed a Numeric Rating Scale (NRS) of 10, 10, 9, 9, and 10 respectively.

Results: Diagnostic block was performed through the unilateral S1-S2-S3 sacral foramina with 22-G spinal needle by 5 mL 1% lidocaine into each foramen. NRS scores decreased to 1, 2, 2, 2 and 1, respectively. One week later, the patients were administered 80 mg methylprednisolone with 1% lidocaine in 15 mL solution shared equally in each foramen. The patients were checked one month after therapeutic block and a full recovery was achieved in all patients.

Conclusion: The neuropathic pain due to accidental intraneural injection of the sciatic nerve would be an acceptable indication for transsacral nerve block with corticosteroids in the treatment of sciatic neuropathic pain symptoms.

Key words: Sciatic neuropathy, transsacral block, methylprednisolone

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Accidental intraneural injection induced nerve injury is an iatrogenic tragedy and intramuscular injection (IM) is the most common injury mechanism affecting the sciatic nerve. The most frequent presentation of sciatic nerve injury includes radicular pain and paresthesia with almost immediate onset of variable motor and sensory deficit. The most common scenario for sciatic nerve injection injury occurs when the site of the needle insertion is located more medial and/or inferior to the recommended site on the upper and outer quadrant of the buttock (1). Additionally, the thickness of subcutaneous tissue and depth of gluteus musculature in children, and poor gluteal covering in elderly patients are predispositions to this type of nerve injury (2).

In review of reported cases, sciatic neuropathy occurs with significant neurological deficits that range from minor to severe transient sensory disturbance to paralysis with poor recovery (3). The accidental peripheral nerve injuries associated with direct needle trau-

ma results in widespread axonal and myelin degeneration and precipitates sensitization (4). Direct trauma to the nerve also activates the secretion of inflammatory mediators, pro-inflammatory cytokines, and initiates the inflammatory cascade. Consequently, the ectopic discharges on damaged neural membranes contribute to the development of peripheral neuropathy and neuropathic pain with inflammatory neuritis (5-7).

The recommended treatment following traumatic nerve injuries ranges from the conservative approach to immediate operative exposure and neurolysis or resection and anastomosis (3). The underlying mechanism complicates the management progressively and severe, traumatic or refractory neuropathy that does not show improvement over time requires being re-evaluated. In this situation, the optimum treatment strategy can only be achieved by identifying the mechanism for patients suffering from neuropathic pain due to accidental intraneural injection.

Since inflammatory mechanisms play such an important role in the development of peripheral neuropathy following nerve damage, management should likewise be based on these underlying inflammatory mechanisms. Therefore, anti-inflammatory treatment with corticosteroids is indicated to improve neuropathic pain due to nerve injury by inhibiting production of inflammatory mediators, reducing prostaglandin synthesis, and suppressing ectopic neural discharges from the injured fibers (6,8-28). Based on this knowledge, we performed transsacral corticosteroid injections in the management of sciatic neuropathy and present the successful results of 5 patients following accidental intraneural injection.

Case Reports

We conducted a case series with sciatic nerve injury following IM injection referred to our Pain Clinic in Adana, Turkey from January 2008 to February 2009. Five patients complaining of severe neuropathic pain, paresthesia and progressive weakness of the lower extremity with difficulty in walking secondary to gluteal injection were admitted to the clinic. Case 1 was a young nurse who used a mirror to IM inject herself with diclofenec sodium to treat menstrual pain. Case 2 was a female patient with chronic renal insufficiency whose sciatic nerve injury occurred following monthly IM injections of vitamin B12. Case 3 was a female patient who suffered from a migraine headache and was referred to emergency service for management of an attack with meperidine injection. Case 4 and 5 were male patients with renal colic; hyoscine-N-butyl bromide had been injected intramuscularly for pain treatment by experienced staff nurses. The demographic data, the duration of symptoms and previous therapies are listed in Table 1.

Immediately after the injections, all patients reported an intense burning pain at the site of injection and began to experience pain and paresthesia extending from the hip region down the back of the thigh and the leg into the toes. Associated with the sensory disturbance, the patients also noticed progressive weakness of the lower extremity with difficulty in walking and foot drop that improved over several weeks. The patients had an average 8-month history of persistent pain in the buttock region radiating to the leg, calf, and toes. The patients also noted worse pain while lying down, flexing and walking.

Table 1. *The characteristics of the patients and background of neuropathic pain.*

	Age (year)	Gender	Weight (lbs)	Height (inches)	Cause of Neuropathic Pain	Duration of Symptoms	Previous Therapies
Case 1	32	Female	132	63	IM diclofenac injection	6 months	NSAID, Myorelaxants
Case 2	34	Female	99	59	IM vitamin B12 injection	4 months	Myorelaxants Tramadol drop
Case 3	45	Female	143	64	IM meperidine injection	8 months	NSAID, Tramadol oral
Case 4	54	Male	154	67	IM hyoscine-N-butyl bromide injection	1 year	NSAID, Physical therapy
Case 5	70	Male	176	71	IM hyoscine-N-butyl bromide injection	1 year	NSAID, Physical therapy

IM, Intra-muscular; NSAID, Non-steroidal anti-inflammatory drugs

The initial examination of the patients revealed a Numeric Rating Scale (NRS) scores of 10, 10, 9, 9, and 10, respectively. The disabling problem with persistent sensory deficit in all patients was the unpleasant paresthesia sensation along the sciatic nerve distribution which also disturbed the nocturnal sleep of all. The symptoms were resistant to drug therapy with non-steroidal anti-inflammatory drugs, myorelaxants, and opioids. Case 4 and 5 also received physiotherapy; however, no recovery was achieved. Neuropathic symptoms included paresthesia, burning sensation, numbness, unpleasant sensation in the skin like pricking, tingling, sensitivity to touch when wearing tight clothes, and sudden pain-like electric shocks were questioned separately. The sensory test for allodynia and hyperalgesia was performed and motor functions were also evaluated. The details of the characteristics of the neuropathic pain as well as the initial and recovered neuropathic symptoms were listed in Table 2. After a routine assessment and physical examination of the patients, needle electromyography (EMG) was analyzed and axonal damage of the sciatic nerve was disclosed in all patients.

Following the diagnosis of sciatic neuropathy due to intraneural injection, a sacral nerve block without fluoroscopy was performed to gain access to the proximal site of the nerve injury via the transsacral approach in the prone position with a pillow under the pelvis. After preparation of a wide area of the skin with antiseptic solution, the sacral hiatus was palpated and S4 sacral foramen was identified at a half-inch superior and lateral to the sacral cornu. Then the S3 foramen was identified a half-inch superior and a half-inch lateral to the S4 foramen and this maneuver was repeated in an analogous manner for both the S1 and S2 foramina. A 22-gauge spinal needle was inserted into each foramen and advanced slowly perpendicular to the skin. If bony contact was made, the needle was withdrawn into the subcutaneous tissue and re-advanced in a slightly more superior and lateral trajectory. This maneuver was repeated until the needle was walked off the posterior sacrum. Diagnostic block was performed through the unilateral S1-S2-S3 foramina by 5 mL 1% lidocaine into each foramen (Fig. 1)(29).

NRS scores were recorded as 5, 6, 6, 7 and 5 one week after the diagnostic nerve block. Then the patients were administered 80 mg methylprednisolone with 1% lidocaine in 15 mL solution injected equally in each foramen. Two patients experienced leg weakness following the transsacral block with 5 mL of 1% lidocaine at each site and were observed in the recovery room and discharged after resolution of the motor block. At one month follow-up, severity of initial neuropathic symptoms, sensorial and motor tests were re-evaluated. Nearly complete recovery from initial symptoms including motor weakness were recorded for all patients and NRS scores were reported as 1, 2, 2, 2 and 1, respectively. Numbness was the only remaining neuropathic symptom with 10 to 20 percent of initial values in the first 3 patients as demonstrated in Table 2. The patients were also checked after 3 months and no motor and sensory deficit was noted.

Discussion

Sciatic nerve injury and dysfunction is a common cause of lower extremity symptoms in clinical prac-

Table 2. *Pre- therapy, post-diagnostic, post- therapy and 3 months after post-therapy numbering rating scores, initial and unimproved symptoms with remaining percentages*

	Pre-therapy NRS	Post-diagnostic NRS	Post-therapy NRS	3 mos. after post-therapy NRS	Initial symptoms	Unimproved Symptoms with Remaining Percentages
Case 1	10	5	1	1	Paresthesia, burning sensation, numbness, allodynia, hyperalgesia, motor weakness	Numbness (10%)
Case 2	10	6	2	2	Paresthesia,burning sensation, numbness, pricking, tingling, allodynia, hyperalgesia, motor weakness	Numbness (20%)
Case 3	9	6	2	2	Paresthesia, burning sensation, numbness, allodynia, hyperalgesia, motor weakness	Numbness (10%)
Case 4	9	7	2	2	Paresthesia,burning sensation, numbness, stabbing pain, allodynia, hyperalgesia, motor weakness	None
Case 5	10	5	1	1	Paresthesia, burning sensation, numbness, shock-like pain, allodynia, hyperalgesia, motor weakness	None

NRS: Numeric rating scores.

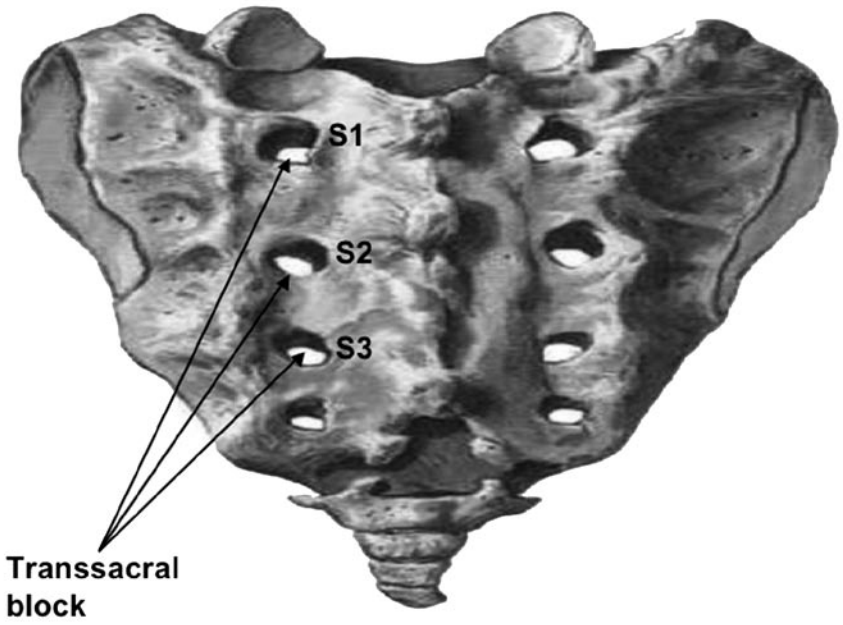

Fig. 1. *The arrows point out the S1-S2-S3 foramina for transsacral block.*

tice. Etiologies of sciatic neuropathy include traumatic, compressive, ischemic, neoplastic or idiopathic causes. Although femur fracture, hip dislocation, fracture, laceration, gunshot wound, and posterior thigh compartment syndrome may cause traumatic injury in the sciatic nerve, intragluteal injection is on the top of the list of etiological factors in traumatic sciatic neuropathy cases with a frequency of 28% (30).

Sciatic neuropathy with motor and sensory deficits appears with radiculopathies in the lower extremity. A complete sciatic nerve lesion can present with pain, paresthesia, and sensory loss through the sciatic distribution, difficulty in knee flexion, and a drop foot with loss of dorsiflexion, plantar flexion, invertors and evertors. The findings of the physical examination might corroborate with S1 radiculopathies but the onset of symptoms according to the clinical history and electrodiagnostic evaluation of sciatic neuropathy may help to differentiate the diagnosis (31).

Treatment options for sciatic nerve injuries due to intraneural injections are also challenging. The common suggestion is to perform the preferred conservative or surgical therapy earlier to provide a better chance of recovery and to minimize long-term sequelae. As presented in a case report, the chance of recovery is small with performing physiotherapy 12 months after the nerve injury (32). In another report, overall results were poor in performing surgical interventions between 3 and 8 months after the occurrence of injection injury (1).

The patients complaining of severe neuropathic pain secondary to injections might also have difficulty in reporting with this unexpected clinical situation. Therefore, the treatment for sciatic neuropathy should have an aggressive recovery effect on the mechanism and the response should be independent from the interval until the treatment. Adjuvant analgesics like antidepressants and anticonvulsants, are generally involved in medical therapies for neuropathic pain and an adequate response for neuropathic pain symptoms might also be expected in traumatic sciatic neuropathy. However, targeting a rapid response to the therapy and existing accompanying symptoms of motor deficit prevents the clinician from the primary preference of adjuvant analgesics in sciatic neuropathy.

All these assessments demonstrate that underlying mechanisms have to be the basis when selecting treatment approaches. The trauma to the sciatic nerve either from needle damage, the pressure effect of an injected agent, or the toxic effects on nerve fibers precipitates perineural tissue inflammation. Pro-inflammatory cyto-

kines including interleukin (IL)-1β, IL-6β, IL-10 and tumor necrosis factor-α (TNF-α) are produced at the site of nerve injury and play a significant role in the inflammatory response, neurogenic extravasation and edema formation. The secretion of inflammatory mediators and perineural response to injury generates the development of neuropathic pain. The ectopic discharge activity from the injured site up-regulates and sensitizes the nociceptors and contributes to the development of central sensitization. The occurrence of spontaneous ectopic neuronal firing then manifests at the behavioral level as allodynia and hyperalgesia (5,6,8,9,33-35).

The effective therapy for neuropathic pain due to traumatic sciatic nerve injury can be achieved by reducing production of inflammatory mediators at the site of nerve injury (5,6,8,9,33-35). Corticosteroids have been postulated to inhibit pro-inflammatory cytokines and nuclear factor κB, reduce prostaglandin synthesis, silence neural firing, and reverse their input to central neurons. They also change pain behaviors with their membrane stabilizing and analgesic effects when topically applied. Corticosteroids also inhibit neurogenic extravasation and perineural edema formation probably by reducing the expression and release of substance P. Several animal studies have investigated the effect of corticosteroids on neuropathic pain behaviors and up-regulation of inflammatory receptors. A decrease in sensitivity has been considered to be the mechanism of corticosteroid treatments in neuropathic pain (6,9,12,13,36,37).

Based upon the results of animal studies, we preferred to treat the post-injection sciatic neuropathy in our patients by applying corticosteroids locally at the proximal site of the nerve injury. Transsacral block was selected to be performed through the unilateral S1-S2-S3 sacral foramina to prevent probable nerve damage to sciatic nerve. Diagnostic block was primarily performed to prove the effect of transsacral block in resolving pain arising from the sciatic nerve. These diagnostic blocks utilizing 5 mL 1% lidocaine for each foramen demonstrated immediate recovery from severe pain, and even though neuropathic pain symptoms didn't improve completely until after the therapeutic block, the NRS scores remained lower than their initial values.

A complete clinical response to diagnostic block with 1% lidocaine was not expected actually and was depended on the membrane stabilizing and the analgesic effects of lidocaine in selected concentration and volume. The therapeutic block was performed with 80 mg methylprednisolone and 1% lidocaine in 15 mL solution and achieved a complete recovery for neuropathic pain symptoms, sensorial and motor functions during 3-month follow-up. Patients reported on-going recovery from the symptoms at regular visits with 3-month intervals. While this report was being written, all patients were contacted regarding their final conditions and all confirmed a complete recovery.

Conclusion

This is the first presentation in the literature of transsacral block with 80 mg methylprednisolone and 1% lidocaine in 15 mL solution through the S1-S2-S3 sacral foramina for the treatment of persistent neuropathic pain due to sciatic neuropathy. We recommend that the deficit in the sciatic nerve following accidental intraneural injection with no evidence of recovery over time might be an acceptable indication for transsacral nerve block with corticosteroids to provide relief of neuropathic pain symptoms, sensorial, and motor dysfunction by reducing the causative inflammatory mediators around the nerve.

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