Post-injection sciatic neuropathy (PISN) from an inadvertent intramuscular (IM) injection in the gluteal region is a type of iatrogenic sciatic nerve injury. The possible mechanisms of injury include direct mechanical damage of the needle, secondary ischemic damage, and neurotoxic effects of the injected agent. In addition to children, individuals over 65 years of age and underweight individuals having a thin gluteal fat layer and low muscle mass are at an increased risk for PISN. In about 90% of patients, symptoms start immediately after the injection. Paresthesia, numbness, radicular pain, and motor weakness of various severities along the sciatic nerve distribution are frequent symptoms. Common peroneal division of the sciatic nerve is more frequently affected than the tibial division. In some cases, even if there is no motor deficit, there may be an excruciating and incapacitating pain which is resistant to analgesia and may cause a devastating disability.

Post-injection sciatic neuropathy is mainly diagnosed based on clinical evaluation, magnetic resonance imaging (MRI), and electrophysiologic studies (EPSs). Specific MRI and EPS findings in PISN are helpful for the differential diagnosis of PISN from spinal and non-spinal causes of sciatica. The EPSs also seem to play a key role in localizing the site of injury and planning the management or prediction of prognosis.

The treatment of PISN involves conservative and/or surgical interventions based on the severity and the duration of the injury. The use of appropriate devices for early mobilization, and occupational therapy, as well. For patients with severe or complete motor deficit who showed no evidence of recovery overtime or those with severe, persistent pain unresponsive to medications are suitable candidates for surgical exploration.

To date, several interventional procedures for the management of pain have been attempted with various medications at the site of lesion level. However, the
effect of the proximity of the injection to the site of nerve injury still remains unknown and may lead to further nerve injury. Despite therapeutic purposes, a repeat injection from the area where the nerve damage occurs can also generate anxiety and disadvantage in these patients. Unlike these procedures, Eker et al.\[10\] blindly underwent transsacral steroid injections of 80 mg methylprednisolone and 1% lidocaine through the S1-S2-S3 sacral foramina in five patients with intractable neuropathic pain due to PISN who were resistant to medications and reported almost complete improvement at three months of follow-up.

Imaging-guided techniques, with either fluoroscopy or computed tomography (CT), provide confirmation of localization, flow, and no vascular flow in the sacral region, while using particulate steroid. Thus, they increase the accuracy of spinal procedures and reduce complication rates, at the expense of radiation exposure.\[11\] Herein, we report three adult male cases with neuropathic pain due to PISN who were resistant to conservative management and treated with fluoroscopy-guided transsacral block.

**CASE REPORT**

Consecutive three patients were referred to our algology outpatient clinic for neuropathic pain due to PISN who were resistant to conventional pharmacotherapy and physiotherapy (Table 1):

1. A 61-year-old man (Patient 1) who was administered an IM non-steroidal anti-inflammatory drug (NSAID) (diclofenac sodium 75 mg/3 mL) injection 14 months ago in the emergency service. He presented with burning and sharp back pain, numbness, and paresthesia started in his left leg just after the IM injection.

2. A 75-year-old man (Patient 2) felt sudden burning pain, numbness, and paresthesia in his left leg after an improper IM injection of NSAID (diclofenac sodium 75 mg/3 mL), which was applied for toothache 10 months ago by an individual who was not a healthcare professional.

3. A 56-year-old man (Patient 3) felt sudden-onset of numbness and tingling in his right leg after an IM analgesic (dexketoprofen trometamol 50 mg/2 mL) when he was admitted to the emergency service for cluster headache five months ago.

All three patients presented with severe neuropathic pain, paresthesia, and causalgia in the buttock level radiating to the back of thigh, calf, and toes on the affected side. They described the pain as burning, shooting, and throbbing or tightness. Patients 2 and 3 also noted disability in daily activities. On physical examination, there was a moderate weakness (Medical Research Council [MRC] 4/5) in Patient 1 and severe weakness (MRC 2/5) in Patient 3 at his left foot and toe dorsiflexion, but plantar flexion of the ankle was intact. Eversion of the ankle was much weaker than inversion for both of them (4/5 and 3/5,

### Table 1. Patient characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (year)/sex</th>
<th>BMI (kg/cm²)</th>
<th>Affected lower extremity</th>
<th>Duration (mo)</th>
<th>Initial signs/symptoms</th>
<th>Treatments</th>
<th>Baseline NRS/LANSS pain score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>61/male</td>
<td>23.8</td>
<td>Left</td>
<td>14</td>
<td>Neuropathic pain, paresthesia, burning, numbness, allodynia, hyperalgesia, motor weakness</td>
<td>NSAIDs, pregabalin, tramadol physiotherapy</td>
<td>5/17</td>
</tr>
<tr>
<td>2</td>
<td>75/male</td>
<td>28.9</td>
<td>Left</td>
<td>10</td>
<td>Neuropathic pain, paresthesia, burning, numbness, allodynia, hyperalgesia</td>
<td>NSAIDs, gabapentin, oxycodone hydrochloride physiotherapy</td>
<td>8/24</td>
</tr>
<tr>
<td>3</td>
<td>56/male</td>
<td>29.7</td>
<td>Right</td>
<td>5</td>
<td>Neuropathic pain, paresthesia, burning, numbness, allodynia, hyperalgesia, motor weakness</td>
<td>NSAIDs, gabapentin</td>
<td>10/17</td>
</tr>
</tbody>
</table>

BMI: Body mass index; NRS: Numerical Rating Scale for pain (minimum score= 0; maximum score= 10); LANSS: Leeds Assessment of Neuropathic Symptoms and Signs Pain Scale (minimum score= 0; maximum score= 24); NSAIDs: Non-steroidal anti-inflammatory drugs.
respectively). The ipsilateral ankle jerk was depressed in Patients 1 and 3. Proximal muscles of the thigh, hip, and hamstrings were normal in all patients. Patient 3 was able to mobilize with the aid of a foot drop splint. Sensory assessment revealed an obvious disturbance at the dorsum of the foot, first dorsal web space, lateral knee, and posterior calf on the affected extremity.

All patients were on combined medical treatment consisting of NSAIDs, anticonvulsants, and moderate-to-strong opioids for neuropathic pain along the sciatic nerve distribution. Patients 1 and 2 also received physiotherapy; however, no recovery was obtained (Table 1).

The neuropathic pain severity along the sciatic nerve distribution was evaluated by a 10-point Numeric Rating Scale (NRS) and confirmed with the Turkish version of the Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) pain scale. The pain intensity (NRS) scores of Patients 1, 2, and 3 were 5, 8, and 10 points, respectively and the LANSS scores were 17, 24, and 17 before the transsacral block, respectively.

All three patients were diagnosed with PISN according to the medical history, neurological, and EPSs findings. Lumbar radiculopathy and others causes of sciatica or polyneuropathy were considered in the differential diagnosis. Lumbar nerve root compression was excluded by lumbar MRI. Only one patient (Patient 3) had a hip MRI of the affected side with and without gadolinium, which demonstrated abnormal thickening and increased T2 signal intensity of sciatic nerve at the buttock level. The time interval between injection and EPS was 30 to 55 days on average, and EPS findings of all patients were consistent with isolated sciatic mononeuropathy, proximal to the hamstring innervation, characterized by acute partial axonal damage, which typically involved the peroneal branch predominantly.

We assessed the current clinical status of the patients and offered fluoroscopy-guided transsacral block owing to our experience in fluoroscopy-guided spinal injections. All patients were informed in detail about the transsacral block therapy, agreed with the procedure, and a written informed consents were obtained.

The patients were monitored with fluoroscopy in the prone position. The skin overlying the area of intervention was cleaned with povidone-iodine and covered with a sterile drape. The S1, S2, and S3 sacral foramina were imaged under fluoroscopy in the anteroposterior view. Local anesthesia was administered at each entry point. Under the fluoroscopic guidance, a 10-cm, 22G Stimuplex needle (B. Braun Medical Ltd., Melsungen, Germany) was inserted into the S1, S2, and S3 foramina, respectively (Figure 1a). Motor and sensory response of the corresponding root was obtained by electrical stimulation at 1.2 mVA. The contrast material was injected to confirm the absence of vascular access and spread of contrast into the foramen (Figure 1b). Then, 15 mL mixture of 80 mg methylprednisolone and 2% lidocaine (mixed with isotonic saline in an approximate 1:1 dilution) solution were injected equally in each foramen. No complications occurred during and after the procedure.

The NRS scores at the first hour after the procedure showed that the procedure provided a dramatic pain

**Figure 1.** Transsacral block under fluoroscopic guidance. (a) Lateral fluoroscopy of needles’ position near left S1, S2 and S3 sacral foramina. (b) On fluoroscopy in an anteroposterior view showing contrast spread in S1, S2 and S3 roots.

**Figure 2.** Trends in changes of NRS score in three cases with PISN undergoing transsacral block. NRS: Numerical Rating Scale for pain; PISN: Post-injection sciatic neuropathy.
relief. The NRS and LANSS pain scores decreased three weeks after the procedure, and the decline was persistent three months after the therapy in Patients 1 and 3; however, the pain was intensified three months after the block in Patient 2. The NRS and LANSS scores during the three months after the procedure are shown in Figures 2 and 3. At the end of the third month, the reduction rates in neuropathic pain and components were found to be ≥50% in Patients 1 and 3, while it was only 20% in Patient 2. In Patient 1, only opioid analgesic drug could be withdrawn from the medical treatment used for neuropathic pain. In Patient 3, full recovery was achieved and medications for neuropathic pain were tapered and finally discontinued. No increase in the strength of the weaker muscles or in the ankle jerks was observed after the treatment in Patients 1 and 3. However, there was no change in the current medical treatment of Patient 2, and the patient was referred to a tertiary hospital for further evaluation for spinal cord stimulation.

**DISCUSSION**

In this report, we present three adult patients with neuropathic pain associated with PISN. In our cases, therapeutic transsacral block was performed with 80 mg methylprednisolone and 1% lidocaine in 15 mL solution under the fluoroscopic guidance for neuropathic pain secondary to sciatic nerve injection injury. At three months of follow-up, neuropathic pain and symptoms reduced by more than 50% in two cases, while a significant improvement was unable to be achieved in the other case. However, none of the cases experienced complications during or after the procedure.

Current data regarding the pathological changes which occur in the proximal neuronal structures of the lesion subsequent to a nerve injury encourage us to prefer transsacral block for the treatment of persistent neuropathic pain for the presented cases. In an experimental study, sciatic nerve injury-induced rats were subjected to pulsed electrical stimulation in the epidural space between T10 and L3 for about two months. Following the electrical stimulation, an increase in the number of neurons in the dorsal root ganglion (DRG) and in the anterior horn was detected with increased myelinated fibers of the sciatic nerve.

As reported previously, the inflammation which is involved in the lesion area subsequent to peripheral nerve injury also affects and impairs ipsilateral DRG and dorsal horn through axons. It is believed that sciatic nerve injury causes macrophages and T-lymphocytes to migrate into the injury site, leading to a specific immune reaction through proinflammatory cytokines (i.e., tumor necrosis factor alpha [TNF-α]). On the other hand, it has been shown that sciatic nerve injury induces Type 1 collagen synthesis and extracellular matrix accumulation through fibroblasts around the ipsilateral DRG. These data suggest that these factors and mediators possibly contribute to neuropathic pain associated with sciatic nerve injury in DRG and spinal cord neurons. Transforaminal approach to deliver epidural steroids and local anesthetics probably interrupt the inflammatory cascade in the affected DRG, which is directly responsible for neuropathic pain and thus reduces neuronal damage.

In the literature, there is only one case series about transsacral block for sciatic nerve injury. In this case report, Eker et al. blindly performed a transsacral block of iatrogenic sciatic nerve injury in five patients and obtained nearly complete improvement in the neuropathic complaints at the end of three months of follow-up. In our study, we also performed the same doses of the mixture of steroids and local anesthetic agents as Eker et al., however, we did not perform a diagnostic block before the procedure. Although the duration of injection after the injury was similar in all cases, several reasons might cause the discrepancy in the post-injection efficacy. First, the individual comparison was insufficient, as the number of patients in both studies was very small. Another reason may be that the severity of sciatic nerve injury in injected patients might be different. Although selective lumbosacral nerve root block (lumbosacral transforaminal epidural block) is often delivered by ultrasound or in a fluoroscopy-controlled manner in clinical

![Figure 3. Trends in changes of LANSS scores in three patients with PISN undergoing transsacral block.](image-url)

LANSS: Leeds Assessment of Neuropathic Symptoms and Signs Pain Scale; PISN: Post-injection sciatic neuropathy.
trials, complications associated with unrecognized incorrect needle placement (vascular/neural trauma or intravascular injection) may be encountered.[18] In addition, a negative needle aspiration is not a reliable sign to exclude intravascular injections.[19] Therefore, even if no complication has been reported in the aforementioned study, the blind selective nerve block does not appear to be a reasonable risk for clinicians.

At the end of three months of follow-up, the question of whether the technique applied to the affected nerve roots was sufficient or not came to our mind, since the treatment results were different in three patients. We injected a mixture of steroid and local anesthetic solution divided equally between the three sacral foramina (S1, S2, S3) in accordance with the literature.[10] Iatrogenic sciatic nerve palsy involves the common peroneal division more severely and frequently than tibial division. However, the common peroneal nerve originates from postaxial branches of L4, L5, S1 and S2 as a part of the sciatic nerve, and the severity of injury and nerve roots involved varies from patient to patient.[20] Therefore, the rationale behind this therapeutic attempt may not be as strong as we once thought. In our opinion, detailed clinical evaluation including the pain diagram and, more importantly, EPS may provide significant information to guide the management before epidural injections for patients with sciatic nerve injury. Thus, additional epidural injections can be applied to other related nerve roots, when deemed necessary.

In conclusion, it seems that fluoroscopy-guided ipsilateral transsacral block can be considered as a safe and alternative treatment option for conservative treatment-refractory PISN, although this intervention may not be equally effective in all patients. This case series demonstrates that proximal areas to the lesion may be one of the target areas for the treatment of persistent neuropathic pain due to sciatic nerve injury.

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REFERENCES