Neurological Deficit Post Transforaminal Epidural Steroid Injection in a Patient with Degenerative Lumbar Scoliosis and Spinal Stenosis

Dejeneratif Lomber Skolyoz ve Spinal Darlık Olan Bir Hastada Nörolojik Açıklık Transforaminal Epidural Steroid Enjeksiyonu Sonrası

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ABSTRACT

Transforaminal epidural steroid injection (TFESI) is one of the techniques used to treat pain due to spinal stenosis and disc herniation. Major complications like neurological deficit after TFESI can be a devastating event for both patient and doctors. We are reporting a case who developed unexpected neurology deficit post TFESI. This is a case of 79yo gentlemen with degenerative lumbar scoliosis and severe spinal stenosis presented for lower back pain with radicular pain on both lower limbs. He was scheduled for right L4 and left L3 TFESI in view of failed pharmacotherapy. Post TFESI patient's condition was complicated with permanent bilateral lower limb paresis and paralysis. MRI did not show any hematoma or cord edema to support the clinical findings. The exact pathophysiology of this complication is unclear. We are reporting this case as part of literature that demonstrates potential risk of TFESI in patient with lumbar scoliosis and severe spinal canal stenosis.

Keywords: Spinal stenosis, transforaminal, epidural, steroid, paralysis, complication, paresis, scoliosis

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ÖZET

Transforaminal epidural steroid enjeksiyonu (TFESI), spinal stenoz ve disk herniasyonuna bağlı ağrıları tedavi etmek için kullanılan tekniklerden biridir. TFESI sonrası nörolojik defisit gibi büyük komplikasyonlar hem hasta hem de doktorlar için yıkıcı bir olay olabilir. TFESI sonrası beklenmeyen nöroloji defisiti gelişen bir olguyu bildiriyoruz. Bu, her iki alt ekstremitede radiküler ağrı ile birlikte bel ağrısı için sunulan dejeneratif lomber skolyoz ve şiddetli spinal stenozlu 79 yaşındaki bir beyefendi vakasıdır. Başarısız farmakoterapi nedeniyle sağ L4 ve sol L3 TFESI planlandı. TFESI sonrası hastanın durumu, kalıcı bilateral alt ekstremite parezi ve felç ile komplike hale geldi. MRG klinik bulguları destekleyecek herhangi bir hematom veya kord ödemi göstermedi. Bu komplikasyonun kesin patofizyolojisi belirsizdir. Bu olguyu lomber skolyozlu ve ciddi spinal kanal stenozu olan hastalarda TFESI'nin potansiyel riskini gösteren literatürün bir parçası olarak sunuyoruz.

Anahtar Sözcükler: Spinal stenoz, transforaminal, epidural, steroid, felç, komplikasyon, parezi, skolyoz

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INTRODUCTION

Transforaminal epidural steroid injection (TFESI) is one of the techniques used to treat pain due to spinal stenosis and disc herniation. Evidence showed that TFESI can be used as surgery sparing intervention, and is superior to caudal and interlaminar epidural approach for radicular pain treatment (1). A review article by Chang mentioned TFESI as a safe procedure as most of the complications seen are minor. At the same time, few cases are reported to develop major complications such as epidural abscess, hematoma, spinal cord infarction, dural and subdural puncture (2). We are reporting a case of unexpected neurological deficit post TFESI in a patient with degenerative lumbar scoliosis combined with spinal stenosis.

CASE REPORT

A 79 years old gentlemen, presented to our pain clinic with a history of lower back pain with radicular pain for a 1-year duration. He is a known case of well controlled hypertension, diabetic mellitus and ischemic heart disease. He was able to walk with the aid of walking stick. His muscle power was good at both lower limbs with only reduced sensation over the right L5 dermatome. His lumbar magnetic resonance imaging (MRI) showed mild scoliosis of lumbar vertebrae with convexity to the left, multilevel degenerative disc disease, with severe spinal canal stenosis at the level L3/L4 and L4/L5 level. Also noted to have impingement of bilateral L3 and L4 exiting nerve root as well as L4 and L5 traversing nerve roots. (Figure 1).

His clinical findings were correlating with the MRI. In view of failed pharmacotherapy, patient was scheduled for TFESI at Right L4 and Left L3 level.

On the day of the procedure, the patient was put under monitoring and was placed in a prone position. Procedure was done under strict aseptic technique under fluoroscopy guidance. Area of interest was identified and confirmed with contrast. After a negative aspirate, prepared drugs (10mg triamcinolone +3.75 mg Ropivacaine: total volume 2ml) were given at left L3 level using 22G (100mm) spinal needle. Using the same technique, right L4 level was identified and epidural space was confirmed with contrast. After injection 1ml of drugs noted blood stain in the needle hub, immediately injection stopped and procedure was abandoned. Within a minute, patient claimed unable to move both lower limbs and lost sensation below the knees. Immediately patient was positioned supine and transferred to recovery for observation. Hemodynamically, patient was stable and denied any other symptoms. Review of patient after two hours showed loss of bilateral L2-S1 motor power with reduced sensation L4-S2 dermatome level and areflexia. Patient also had urinary incontinence but anal tone and bulbocavernosus reflex were intact. Proceeded with urgent MRI, showed no epidural hematoma or cord edema (Figure 2).

Subsequently, patient was referred to neurosurgical team and started on systemic steroids. Repeat whole spine MRI was done after 48 hours to look for evidence of spinal cord infarct which usually appears later. There was no evidence of any spinal cord infarct seen. To our surprise, we noted evidence of syringomyelia at T6-T12 level (Figure 3) as previously only lumbar MRI was done. Review after three months did not show much neurological improvement. Patient was only able to move his toes and reduced sensation below knee level of both lower limbs with urinary incontinence.

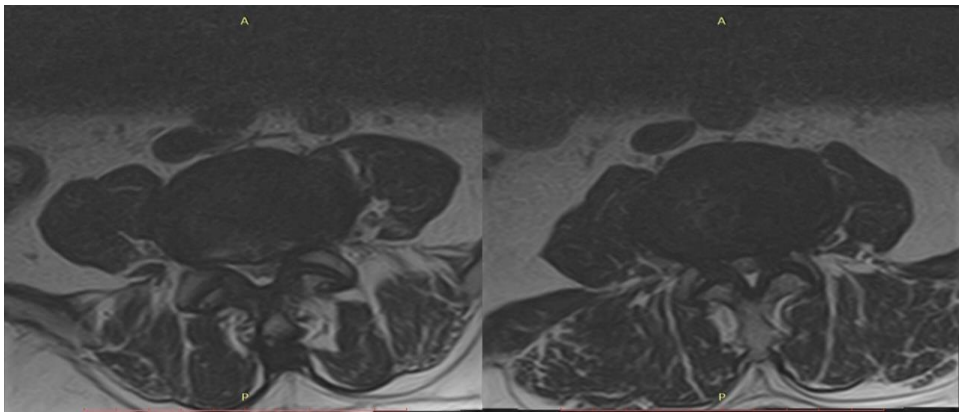

Figure 1: T2 weighted axial view at L4/L5 and L3/L4 showing circumferential disc bulge indenting on to the thecal sac causing severe spinal canal stenosis (AP diameter 0.5cm). Bilateral exiting foramina and lateral recesses are narrowed impinging bilateral exiting and traversing nerve roots (Right > Left). Bilateral ligamentum flavum hypertrophy.

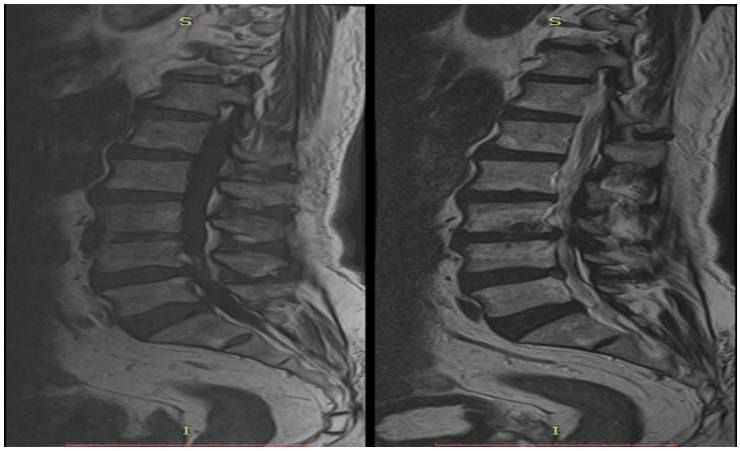

Figure 2: T1/T2 weighted sagittal image shows multilevel posterior disc bulge from L1/L2 till L5/S1. Schmorl's node at inferior end plate of L3. No epidural hematoma noted.

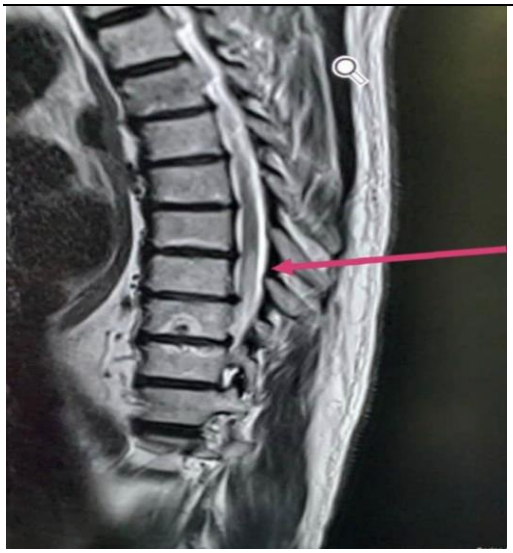

Figure 3: T2 weighted sagittal view shows hyperintense area (syringomyelia) at centre of cord T7 till T12 (arrow). No hematoma or infarct noted.

DISCUSSION

The rate of TFESI as a therapeutic spinal procedure for patients with spinal stenosis and radicular pain is rising rapidly over the years. Major complications post TFESI can be devastating for both patient and treating doctor (3).

Up to date, only few cases were reported to have permanent neurological deficit post TFESI. Houten reported 3 cases which developed lower limb paresis and paralysis post TFESI. They postulated that all these complications were due to vascular injury to the abnormally low dominant radiculomedullary artery. The thoracolumbar MRI in all three cases demonstrated an increased signal in the low thoracic spinal cord region on T2 weighted imaging consistent with cord edema as evident of spinal cord infarct. In our case, even though we had vascular injury events, the repeated thoracolumbar after 48 hours post intervention did not show any evidence of spinal cord infarct (4).

Another study reported similar complication due to direct particulate drug injection to the artery, causing embolization and spinal cord infarct (5). We used particulate steroid (triamcinolone) as well for our patient. In view of back flow of blood into hub after given small volume of drug, accidental arterial injection cannot be excluded. Both the reported cases again showed evidence of cord edema at lower thoracic region in the repeat MRI after 48hours, which is unlikely in our case.

Kim et al. reported a similar case where patient with spinal stenosis develop neurological deficit after right L2-L3 level TFESI. MRI showed evidence of epidural hematoma which required surgical evacuation and improved neurology after that (6).

Patients with severe spinal stenosis may have more fragile blood vessels due to the stenotic compression to arterial and venous system. This increases the risk of vascular injury and epidural hematoma.

hally et al. reported a case of patient with severe spinal stenosis and nerve impingement undergoing right L5 level TFESI. Post procedure patient developed right lower limb paralysis with foot drop and urinary incontinence. Similar to our study, follow up MRI was non-contributory but neurological improvement was seen immediately after laminectomy and discectomy surgery (7).

The pathophysiology of neurological deficit post TFESI could not be explained in our patient since there are no changes in the follow up MRI as seen in previous reported cases. There is always a possibility of disease progression which lead to the neurological deficit. We are reporting this case as part of a new and growing literature that demonstrates potential risk of TFESI in patient with degenerative lumbar scoliosis and severe spinal canal stenosis. Patients should be given clear explanation about such complications even if it's rare.

Conflict of interest

No conflict of interest was declared by the authors.

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