Risk, benefits and complications of epidural steroid injections: A case report

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Acute low back pain, radiculopathy, and associated disabilities have a prevalence of 2% in the United States, which represents a significant health problem and a major cause of workplace absence. The condition primarily affects 25- to 45-year-old men and women. Epidural steroid injections (ESIs) are commonly administered to relieve this pain and improve mobility without surgery.

This case report presents issues related to the treatment of an epidural hematoma in an 87-year-old man receiving long-term anticoagulant therapy. It reviews the risks, benefits, side effects, complications, and contraindications to ESI and the American Society of Regional Anesthesia consensus recommendations for the performance of neuraxial procedures in patients receiving anticoagulant therapy. It is imperative that any provider who performs ESIs know the risks, benefits, complications, and contraindications for this procedure. Practitioners must also be able to recognize and manage or appropriately refer patients having immediate or delayed complications related to the placement of an ESI.

Key words: **Corticosteroids, epidural steroid, injection, pain.**

Acute lower back pain associated with lower extremity radicular neuropathy has a prevalence of approximately 2% in the United States.[1-3] The condition exhibits no gender or race differences but primarily affects persons 25 to 45 years old.[3] The pain often starts with a traumatic event, such as lifting a heavy object or falling in an awkward manner that may cause compression of one or several spinal nerve roots. This compression, coupled with the subsequent inflammatory process, causes radicular pain.[1,2,4] Most patients describe this pain as sharp, originating in the lower back and radiating to one or both lower extremities. Many of these injuries heal over time; however, the issue for most patients is controlling the pain and maintaining function until healing can occur.[4]

Epidural steroid injections (ESIs) are commonly administered to relieve pain and improve mobility without surgery. Medicare data files show that the number of ESIs performed in the United States increased from 444,000 in 1993 to 636,000 in 1998 but dropped to 482,000 in 1999.[5] Anesthesia providers performed at least 75% of these procedures during every year of analysis and 85% of the ESIs in 1999.[5]

The rationale for administration of ESIs is based on the assumption that inflammation of the spinal nerve root causes radicular pain and the epidural corticosteroids relieve this pain, allowing time for healing and physical therapy.[4,6] However, long-term administration of ESIs has not been shown effective, and such practice is discouraged.[6]

Although rare, a variety of side effects and technical complications are associated with ESIs. They include local discomfort, infection, steroidal side effects, dural puncture, postdural puncture headache (PDPH), epidural hematoma, and nerve injury.[6-10] It is imperative that anesthesia providers who performs ESIs as part of their practice be able to recognize and manage any immediate and delayed complications. Clinicians must understand the relative and absolute contraindications and the risk of potential complications and, before performing this invasive procedure, should educate each and every patient planning to have it about the risk/benefit ratio.[1,4]

Case summary

An 87-year-old man was admitted to the emergency department with an approximate 1-week history of bilateral lower extremity weakness, loss of sensation, and bowel and bladder incontinence. Magnetic resonance imaging revealed a thoracolumbar epidural hematoma extending from T10 to L2 with compression of the intrathecal sac from level T12 to L2. The patient was receiving long-term warfarin therapy for a history of multiple cerebral vascular accidents and had been receiving ESIs for chronic lower back pain. Per patient history, a dural puncture occurred during the most recent corticosteroid injection.

The neurosurgeon scheduled the patient for emergency evacuation of the hematoma and decompression of the spinal cord. Preoperative evaluation revealed a coagulation abnormality with an international normalized ratio of 2.3, a prothrombin time of 23 seconds, and a partial thromboplastin time of 31 seconds. These clotting alterations were treated with 8.4 mg of recombinant DNA factor VII (Novoseven) and 4 U of fresh frozen plasma. Additional preparation included a standard anesthesia preoperative

workup and obtaining consent for general anesthesia and surgery.

Approximately 45 minutes after the patient arrived in the preoperative holding area, repeated coagulation studies revealed normalized prothrombin time, partial thromboplastin time, and international normalized ratio. The neurosurgeon decided to proceed with surgery. Other laboratory test results included a white blood cell count of 14.2 x 10^9/L, hemoglobin concentration of 15.7 g/dL, hematocrit value of 46% (0.46), and a platelet count of 355 x 10^9/L. The patient's electrolyte concentrations were within normal limits. His vital signs were as follows: temperature, 36.5°C; Spo_2, 96% while breathing room air; pulse, 105 beats per minute; and blood pressure, 160/90 mm Hg. The airway examination revealed an edentulous patient with a Mallampati class II/IV, 4 finger breadths thyromental distance, 4 finger breadths mouth opening, and full range of motion of the neck. An 18-gauge intravenous catheter was in place from his admission to the emergency department. The patient was brought to the operating room, where a left radial arterial blood pressure monitoring line and an additional 18-gauge intravenous catheter were placed before induction.

Anesthesia was induced slowly with 100 µg of fentanyl, 14 mg of etomidate, 7 mg of vecuronium, and 1% isoflurane. Atraumatic endotracheal intubation was successful on the first attempt. Hemodynamic stability was maintained while the patient was placed in the prone position using laminectomy rolls, padding to all pressure points, and a facial rest. Endotracheal tube position was confirmed with positive end-tidal carbon dioxide measurement and equal bilateral breath sounds before and after prone positioning of the patient. General anesthesia was maintained throughout the case with 1.2% isoflurane, 60% oxygen/air, intermittent boluses of fentanyl, and vecuronium.

The thoracolumbar region was prepped and draped after radiographs were taken to confirm the appropriate level for incision before performing the laminectomies. Following local infiltration, a skin incision was made, and the paravertebral musculature and fascia were divided, exposing the vertebral laminae. After ensuring hemostasis, the surgeons continued with clot evacuation. They discovered a large, dark, gelatinous epidural hematoma, with compression and discoloration of the thecal sac from level T12 to L2. No active bleeding was identified. Once the hematoma was evacuated, intraoperative ultrasound was used to confirm pulsatility of the elements of the thecal sac and eliminate the concern of additional intrathecal hematoma. Hemostasis was obtained, the wound was closed, and a sterile dressing was applied. The estimated blood loss was 250 mL, and urine output was 220 mL. Intraoperative fluid administration included 1,550 mL of crystalloid, and 3 U of fresh frozen plasma. The patient remained hemodynamically stable throughout the case and was turned supine and extubated before transport to the surgical intensive care unit for postoperative recovery.

Postoperative progress

After an uncomplicated 48-hour stay in the surgical intensive care unit, the patient was transferred to the general surgery unit for recovery and rehabilitation. Despite rehabilitation efforts, there was no improvement in the preoperative neurological deficits. After 2 weeks on the general surgery unit, the patient was transferred to a secondary care facility for continued physical and occupational therapy.

Case discussion

• *Description of radicular pain.* Radicular pain is often the result of nerve root inflammation and irritation.[2] Isolated mechanical compression of the nerves causes only motor deficits and altered sensation, but not pain.[4] Inflammation at the epidural space and nerve roots provoked by a herniated disk is a significant factor in causing radicular pain.[4,6]

Evidence of nerve root inflammation has been demonstrated during surgery in patients with radicular pain from lumbar disk herniation.[4] Also, high levels of phospholipase A_2, an enzyme that controls the initial inflammatory cascade, have been demonstrated in herniated disk material from surgical samples in humans.[6] Research in dogs and rats revealed severe inflammation locally at the epidural space and nerve root after injection of autologous nuclear disk material into the epidural space.[1] Furthermore, animal models have demonstrated that injection of phospholipase A_2 into the epidural space induced demyelination of nerve roots with ectopic discharges, which is considered the primary pathophysiological mechanism of radicular pain.[1,11] Clinical practice and animal research suggest that lumbar radicular pain is the result of inflammation of the nerve root in the epidural space secondary to leakage of disk material or compression of the nerve root and its vasculature.[4,6]

• *Origin and description of ESIs.* In 1960, Brown[12] reported complete relief of the symptoms of radiculopathy in 4 patients treated with ESI. That same year, Goebert et al[13] gave 3 injections of procaine and methylprednisolone to 239 patients with radiculopathy and reported that 58% of patients had relief of greater than 60% of their pain and symptoms.

Since then, techniques and indications for ESIs have constantly evolved. Initially, large volumes of local anesthetics, corticosteroids, or a corticosteroid–local anesthetic combination were injected to ensure adequate coverage of the affected area due to the inability to locate the precise level of injury. Improved imaging techniques now allow more precise, fluoroscopically guided, ESI injections that use lower doses of glucocorticoids. A variety of glucocorticoids have been used with no conclusive evidence leading to the use of one or another[6]; however, methylprednisolone is most commonly injected.[4] There has been no conclusive evidence supporting the use of any specific corticosteroid, local anesthetic, or combination of agents.

Epidural steroid injections are not global treatments for all causes of low back pain.[1] Patients with clear signs of acute radiculopathy are most likely to benefit because the exact level of disease in the spine can be identified and corticosteroids can be precisely placed to reduce inflammation.[4,14] Other general indications for ESIs include failure to respond to rest, nonsteroidal anti-inflammatory drugs, or physical therapy; and examination findings of nerve root compression and inflammation.[1,4] Benefits of ESIs include relief of radicular pain, improved quality of life, reduction of analgesic consumption, improved maintenance of work status, and elimination of the need for surgery in many patients.[1,15]

Several theories may explain the beneficial effects of glucocorticoids in the treatment of low back pain and radiculopathy. Glucocorticoids directly or indirectly inhibit the synthesis or release of a number of inflammatory substances, including phospholipase A_2, arachidonic acid, interleukin 1 and prostaglandins.[1,4,6] Leukocytes, present due to the inflammatory process, seem to *rest* in the affected area. These resting leukocytes adhere to the endothelium where they penetrate vascular walls, increasing capillary permeability, which subsequently leads to tissue edema. It is theorized that glucocorticoids cause the endothelium to be less adherent to these resting leukocytes.[1,16] Thus, glucocorticoids, reduce acute inflammation and help prevent chronic inflammation, decreasing radiculopathy.[4]

• *Efficacy of ESIs.* The literature reporting the effectiveness of ESIs is inconclusive.[1,17] Many reliable studies suggest that ESIs provide significant benefits to patients with low back pain and radiculopathy.[1,18-20] Other studies dispute the efficacy of ESIs.[18,19,21] These varied results are most likely due to the lack of a large number of well-controlled, double-blind studies and to the variation in clinician technique. Renfrew et al[22] reported that, without fluoroscopy, even experienced clinicians placed the ESI at the incorrect level up to 40% of the time. Most studies that disputed the efficacy of ESIs did not use fluoroscopy to ensure that the injection was performed at the presumed level of disease, which is critical to the success of ESIs.[1,14] When ESIs were placed with fluoroscopy, 60% to 84% of patients reported some relief of symptoms.[14,15,18,19]

Fluoroscopically guided ESIs will likely have a higher rate of corticosteroid placement at the precise level of injury and, therefore, possibly higher reported rates of symptom relief. However, using fluoroscopy for all ESIs would significantly increase the procedure cost and, thus, reduce the cost-effectiveness of this method of treating low back pain and radiculopathy. It is important to note that many studies that did not use fluoroscopy also reported significant relief of patient symptoms.[18,19] Unfortunately, there is no conclusive evidence, particularly from clinical trials, to endorse the efficacy of ESIs placed with or without fluoroscopy.[17,23,24]

Aside from correct placement, response to the injection can be related to several other factors such as the type and quantity of corticosteroid preparation used, volume of material injected, underlying pathophysiology, and the duration of symptoms.[4,6] None of these factors were consistent among the studies noted.[14,15,18,19] One point all authors agreed on was that ESIs are most effective in combination with a well-designed spinal rehabilitation program.[1,4] Questions for future research include the following: Who are the appropriate candidates for ESIs? Does the precise level of injection under fluoroscopic guidance improve efficacy? What is the ideal material for injection and volume?[17,18] Until studies are completed, practitioners will need to perform ESIs using their best clinical judgment.

Clinicians should carefully consider the number of ESIs to be performed due to the risk/benefit ratio and the cost of treatment.[2] Studies have demonstrated that patients without a positive response to an initial injection experienced improvement after a second or third ESI.[1,6] Generally, up to 3 ESIs are performed if clinically indicated. Some clinicians schedule a series of 3 and proceed with all 3 regardless of the clinical response to the first or second injections.[1,4] Concrete evidence pertaining to the duration of the effect of ESIs has not been established, but several studies have shown that their effects decrease over time.[1,18,19]

Another important factor in the effectiveness of ESI is the timing of the injections. More than 75% of patients who have had symptoms for less than 3

months had a significant reductions in their symptoms.[15,18] When patients have radiculopathic symptoms for longer than 3 months, responses rates vary and decrease dramatically.[1,18] Patients with shorter durations of symptoms also received more sustained relief than those with chronic pain.[18]

• *Side effects, complications, and contraindications to ESI.* Absolute contraindications to ESI include systemic infection, local infection at the site of the planned injection, bleeding disorder or anticoagulant therapy, allergy to glucocorticoids, and patient refusal.[1] Few complications of ESIs have been reported in the literature, which includes more than 7,000 case studies.[1]

More complications are related to the invasiveness of the procedure than to the injection itself. Due to the invasive technique, it is important for clinicians to realize the risk/benefit ratio and any contraindications each individual patient may have when considering an ESI.[1,4] Side effects and technical complications associated with ESIs include infection, steroidal side effects, dural puncture, PDPH, epidural hematoma, and nerve injury.[6,8-10]

As with any spinal injection procedure, the risks of neuraxial infection are present. This risk is increased when improper aseptic technique is used or bacteremia is present.[8] Theoretically, the immunosuppressive effects of glucocorticoids could increase the risk of infection.[1,7,8] There have been a few reports of aseptic meningitis, epidural abscess, and bacterial meningitis after an ESI. In a meta-analysis of 64 studies that included nearly 7,000 cases, there was no report of complications due to infection.[1,20] Neuraxial infection in general is characterized by fever, nausea, headaches, and neurological signs, including burning pain in the lower extremities, seizures, and altered mental status.[1,8]

A rare complication, occurring in only 0.01% of ESIs, at normal glucocorticoid injection doses is suppression of the hypothalamic-pituitary-adrenal system.[1] The dose-dependent adrenal suppression and resultant Cushing syndrome may last 2 to 4 weeks.[1] The cushingoid symptoms of fluid retention, electrolyte imbalances, moon facies, abnormal fat distribution, and skin lesions can remain for several months.[8,25] The exact causal pathways by which ESIs affect the neuroendocrine system is unknown.[1,8] Because glucocorticoids increase blood glucose levels secondary to glycogenolysis and gluconeogenesis, ESIs may also increase difficulty controlling blood glucose levels in patients with diabetes.[25]

The most common technical complication of ESIs is inadvertent dural puncture, which has an incidence as high as 7% regardless of the purpose of the neuraxial injection (ie, epidural for regional anesthesia or ESI).[1,10] Fortunately, the probability of unintentional injection of glucocorticoid into the subarachnoid space is very low.[1] One study reported only 1 dural puncture in 5,334 patients receiving ESIs resulting in a small subdural hematoma, which resolved without permanent complications.[21] It is noteworthy that all ESIs in that study were placed under fluoroscopy with epidurography.[21] Dural puncture may directly result in PDPH, which occurs in 20% to 50% of all dural punctures.[1,8,24] It is estimated that the overall incidence of PDPH in all neuraxial injections (ESI, subarachnoid block, or epidural block) could be as high as 7%.[26]

The diagnosis of PDPH is based on clinical features and can occur immediately after dural puncture, although typically, the onset is several hours later.[27] The headache is usually frontal or occipital and often involves the neck and shoulders.[27] The pain is usually described as dull or throbbing and can range from mild to debilitating. The distinguishing feature of PDPH is the postural nature of the headache, which intensifies as the patient sits or stands and often completely disappears when the patient returns to the supine position.[24] Conservative management results in 90% of PDPHs resolving within 10 days.[24] Jankowski[24] presents a thorough review of treatment for PDPH.

Direct trauma from needle placement during ESI rarely results in significant neurological injury, especially if the injection is placed below the tip of the conus medullaris (usually located at L2).[24,28] Trauma to neural tissue by a needle is usually accompanied by pain or paresthesia. One should never advance a needle or inject solution if a patient complains of pain or shows signs of discomfort.[29]

Patients who experience paresthesias during needle placement could be at risk for long-term neurological complications. In 2 large studies examining spinal and epidural injections, 1 retrospective and 1 prospective, 67% and 63%, respectively, of persistent paresthesias were preceded by paresthesias during needle placement.[28,30] There are no data to guide a clinician's decision to continue with the block or move to a different interspace should paresthesia occur.[30] When paresthesia is encountered, careful postprocedural follow-up should be conducted.[29]

• *Epidural hematoma after an ESI.* An extremely rare complication, occurring in approximately 1:200,000 neuraxial injections, is the epidural hematoma.[31] An epidural hematoma can be caused by vascular trauma

Table. Recommended time interval between last anticoagulant dose and epidural corticosteroid injection or other neuraxial procedure[34]*

Anticoagulant medication	Recommended timing of safe administration of neuroaxial procedure
Fibrinolytic and thrombolytic medications	No recommendation (no data available)
Warfarin	Document normal INR; approximately 5-7 d after discontinuing therapy
LMWH	Twice daily dosing: LMWH 24 h after surgery; remove catheter 2 h before first dose Once daily dosing: May perform neuraxial block 10-12 h after administration; wait 4 h after needle or catheter placement before next dose
Intravenous heparin	May heparinize 1 h after neuraxial block; remove catheter 2-4 h after discontinuation of heparin
Heparin subcutaneous (mini dose)	None; platelet count should be checked for patient receiving heparin >4 d
NSAIDs (eg, ibuprofen)	No delay necessary with NSAIDs
Ticlopidine	14 d
Clopidogrel	7 d
Glycoprotein IIb/IIIa inhibitors	8-48 h before block placement

* INR indicates international normalized ratio; LMWH, low-molecular-weight heparin; NSAIDs, nonsteroidal anti-inflammatory drugs.

from a needle or placement of an epidural catheter in the subarachnoid or epidural space. Expansion of an epidural hematoma in the closed space of the spinal canal can cause compression of the spinal cord and result in nerve ischemia.[24] An epidural hematoma may go unnoticed until there is permanent neurological damage.[24] Early signs and symptoms vary depending on the level of occurrence and the size of the hematoma. They include new-onset numbness, weakness, bowel and bladder dysfunction; and, rarely, severe radicular back pain.[24] Epidural hematomas may develop in relation to vascular malformations, coagulopathies, and anticoagulant therapy or may occur spontaneously.[24] Anticoagulant therapy is associated with up to 24% of epidural hematomas.[32]

More patients than ever are receiving anticoagulant therapy (warfarin, heparin, thrombolytics, and low-molecular-weight heparin), which presents a significant challenge to clinicians considering ESIs or any neuraxial injection for their patients.[24] The risk of performing an ESI on a patient receiving warfarin, heparin, thrombolytics, or low-molecular-weight heparin must be weighed carefully against the benefits of the ESI.[33] It is important for clinicians and patients to discuss the risks and benefits, however small they may be, so that patients may make informed choices about receiving an ESI.[24] A detailed history could be the most effective and economical method of determining the risk.[24] Patients should be asked if they have experienced difficulties with hemostasis as evidenced by epistaxis, gingival bleeding, ecchymoses, hemoptysis, hematuria, abnormal menstrual bleeding, and hematemesis.[24] A complete list of the patient's medications, including recently stopped medication, should be obtained to determine anticoagulant therapy, herbal supplements, and other medications that might interfere with coagulation.[24]

Different types and classes of anticoagulants have different pharmacokinetic properties that affect the timing until neuraxial procedures can be safely performed.[33,34] Despite a number of observational and retrospective studies of the incidence of epidural and spinal hematoma associated with various anticoagulants and neuraxial techniques, there are no absolute contraindications regarding the safety of neuraxial anesthesia and anticoagulants.[34] The American Society of Regional Anesthesia and Pain Medicine has written a series of consensus statements based on the available literature for administration of neuraxial techniques in the presence of various anticoagulants.[34] Their recommendations are summarized in the Table. An updated version of the consensus statements on neuraxial anesthesia and anticoagulants can be found at http://www.asra.com, along with statements that address newer anticoagulants.

Conclusion

Currently, lower back pain, radiculopathy, and associated disabilities represent a significant health problem and a major cause of absence from work in the United States.[3] As back pain and radiculopathy persist, it is likely that patients may receive 1 or multiple ESIs as part of their treatment. ESIs have been used for nearly half a century to treat back pain and radiculopathy

and are currently implemented in many pain practices. As of 1999, 482,184 ESIs were being performed annually by anesthesia providers in the United States.[5] This case report reviewed the care of an 87-year-old man receiving anticoagulant therapy who had an epidural hematoma 1 week after an ESI. This very rare complication occurs in approximately 1:200,000 ESIs. It is imperative that all providers, anesthesia or otherwise, who perform ESIs as part of their practice know the contraindications and are able to recognize and appropriately manage any immediate or delayed complications related to the placement of an ESI.

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