

Case Report

SPONTANEOUS RESOLUTION OF PRESUMED ACUTE EPIDURAL HEMATOMA FORMATION AFTER LUMBAR EPIDURAL STEROID INJECTION

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Epidural steroid injections are interventional pain procedures often used to treat lumbar radicular pain. The most serious complication of this procedure is the formation of a spinal epidural hematoma, which can result in profound permanent neurologic deficits if left untreated.

A 76-year-old woman with mild lumbar spinal stenosis (L4-L5, L5-S1) and lumbar dextroscoliosis, previously on 81mg of aspirin daily (discontinued at 14 days prior to procedure) and not on anticoagulation therapy, underwent a lumbar epidural steroid injection (T12-L1). Post-procedurally, she developed bilateral leg paralysis. A magnetic resonance imaging (MRI) study revealed a fluid collection concerning for hematoma. Neurosurgery was consulted, but at the time of evaluation, she had near resolution of her presenting symptoms and the decision was made to monitor her for 48 hours. Three months after discharge, MRI revealed no persistent symptoms or radiographic evidence of sequelae from epidural hematoma.

The frequency of spinal epidural hematomas after epidural steroid injections is unknown. This patient did not have traditional risk factors of severe spinal stenosis or the use of anticoagulant or antiplatelet agents. A radiographic fluid collection was seen, which may represent blood or persistent injectate. A formal surgical diagnosis was not obtained, as her symptoms spontaneously improved without further need for intervention.

We report the first case of presumed persistent injectate compression of the lumbar spinal cord, resulting in bilateral lower extremity weakness in a patient with dextroscoliosis, mimicking spinal epidural hematoma with spontaneous resolution without intervention.

Key words: Epidural steroid injection, spinal epidural hematoma, dextroscoliosis, lumbar radiculopathy, spinal stenosis, lower extremity paralysis

Epidural steroid injections are one of many interventional pain procedures performed to treat lumbar radiculopathy and lumbar spinal canal stenosis. Complications associated with this procedure include vascular uptake of medication, vasovagal events, hypertension, dizziness, increased pain, allergic reaction, headache, and spinal epidural hematoma formation, among others (1). One of the most cata-

strophic complications is the formation of a spinal epidural hematoma, leading to cord compression, resulting in potentially irreversible neurologic damage. Prior studies and case reports discuss spinal epidural hematomas in patients who have undergone epidural catheter placements, but the available literature for spinal epidural hematomas in interventional pain procedures is not as robust (2,3). From the epidural catheter literature, it seems that spinal epidural hematoma formation occurs most often in patients on anticoagulation therapy, antiplatelet therapy, or in those who have a traumatic or difficult placement (3).

With the assistance of fluoroscopy in interventional pain procedures, the most common risk factor would be patients who are on anticoagulation therapy for

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conditions such as atrial fibrillation or antiplatelet therapy for coronary artery disease and cerebral vascular disease. The timing of cessation of these therapies creates a challenging scenario, and guidelines have been provided by the American Society of Regional Anesthesia and Pain Medicine (4) and the American Society of Interventional Pain Physicians (5). However, despite following these guidelines, spinal epidural hematomas can still occur. Here we describe an episode of presumed epidural hematoma formation in a patient on daily low-dose aspirin (81 mg), discontinued 14 days prior to her procedure and not on current anticoagulation, with mild spinal stenosis below the level of injection and with dextroscoliosis of the lumbar spine.

Case Report

A 76-year-old woman with a body mass index of 39, a history of degenerative disc disease, lumbar dextroscoliosis, lumbar radiculopathy, and no prior spinal surgeries presents to the interventional pain clinic for diagnostic epidural steroid injection under fluoroscopy. Previously, she successfully had 4 other lumbar epidural steroid injections at the L1-2 level with persistent pain relief for greater than 8 weeks (Fig. 1). She was consistently taking 81 mg of aspirin daily during this period,

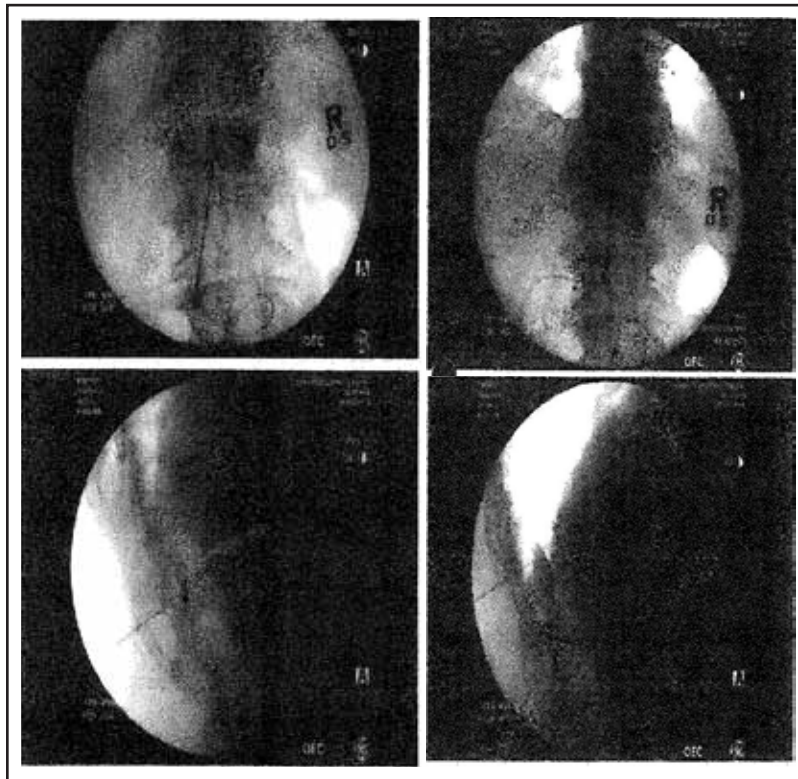


Fig. 1. Prior (left, L1, L2) and day of (right, T12, L1) intraoperative imaging.

which was always discontinued 14 days prior to every procedure. She was never on anticoagulants and never took nonsteroidal anti-inflammatory drugs due to the side effect of an upset stomach. Minimal sedation was performed with intravenous midazolam and fentanyl under monitored anesthesia care. The patient was prepped with betadine and sterilely draped. A local anesthetic of 1% lidocaine was injected subcutaneously over the T12-L1 level with a 25 gauge needle. Under fluoroscopic guidance, an 18 gauge Tuohy needle was advanced, with the loss of resistance technique, into the posterior epidural space. Negative aspiration of cerebrospinal fluid or blood was confirmed without signs of paresthesias. Contrast was injected, illustrating longitudinal spread on anteriorposterior and lateral views, without signs suggestive of loculations present near the targeted site of injection. Eighty milligrams of kenalog diluted into 8 mL total of preservative-free normal saline was injected. The patient tolerated the procedure well and was transported to the recovery unit at 10:42 AM.

Once in the recovery unit, she started developing right lower extremity weakness that progressed to left lower extremity weakness with numbness extending upward above the waist to the level of the umbilicus. Though she had previous difficulty with walking, often requiring a walker for assistance, she noted she had increased difficulty with movement compared to her baseline ambulatory state. Concerned for spinal epidural hematoma, she was urgently transferred to the nearby emergency department for



Fig. 2. Post-procedural MRI.

further evaluation and imaging. She arrived at 12:16 PM, where a physical exam revealed the absence of rectal tone or rectal sensation in addition to the weakness and numbness noted above. Labs were obtained, revealing a hematocrit of 42.5%, platelet of 220 K/uL, sodium of 140 mmol/L, potassium of 4 mmol/L, BUN of 23 mg/dL, serum creatinine of 0.8 mg/dL, and blood glucose of 132 mg/dL. She was given 10mg of intravenous dexamethasone prior to obtaining a lumbar spine magnetic resonance imaging (MRI) study with 20 mL of intravenous contrast.

The lumbar MRI showed a large 8 cm by 1.4 cm thick (anterior to posterior dimension) fluid collection, consistent with a posterior epidural hematoma from T9 to L1, with signs of spinal stenosis with anterior cord displacement and flattening and dextroscoliosis (Fig. 2). During her evaluation, her symptoms began to resolve spontaneously. Within one hour of arrival in the emergency department, her numbness regressed from her waist down to her legs, and she had regained some movement of the left leg. After 2 hours in the emergency room, she could move both her legs but continued to have some lower extremity numbness. By 3 hours, she regained most of her strength and at



Fig. 3. Three-month follow-up imaging.

4 hours, she regained full lower extremity strength, lower extremity sensation, and was at her baseline.

When neurosurgery evaluated her, she had no deficits and wanted to return home. Given her remarkable recovery, the neurosurgical team discussed with the patient the decision to monitor her clinically instead of immediate surgery for decompression. She was monitored over 48 hours without worsening symptoms and was discharged home. Due to the concern for undiagnosed coagulopathy, she was referred to hematology after discharge. Hematology found the patient to have both a normal coagulation profile and platelet function test. At her 3-month follow-up she reported no persistent weakness, numbness, or new difficulty with ambulation. A repeat MRI study was obtained that revealed no abnormalities aside from her known degenerative disc disease and scoliosis (Fig. 3).

DISCUSSION

Spinal epidural hematomas are exceedingly rare complications with potentially devastating long-term consequences. Previous case reports detailing spinal epidural hematomas in patients receiving epidural

steroid injections often involve injecting patients who are either on anticoagulants or have spinal stenosis (6-9). Generally, anticoagulants (such as warfarin) are discontinued in patients prior to the epidural steroid injection and normal international normalized ratio verified as advocated by the American Society of Regional Anesthesia and Pain Medicine and the American Society of Interventional Pain Physicians (4,5). The duration of medication cessation depends on the patient's comorbidities and the type of medication they are on. Studies have been performed to evaluate the frequency of occurrence of spinal epidural hematomas with epidural catheters, but the frequency of spinal epidural hematomas after epidural steroid injections is unknown. From these studies, 3 prominent risk factors were identified including patients who are anticoagulated (either through medications or disease states such as liver cirrhosis), traumatic access, or difficult access (3). All 3 of these risk factors highlight the risk of potential epidural vascular damage during the procedure, which could result in hematoma formation.

Compared to other recent case reports (Table 1), our case is similar in that the patient had mild spinal

stenosis. Unique to this patient is that she was not on anticoagulation or antiplatelet therapy at the time of the procedure, and her symptoms spontaneously resolved. Though a fluid collection was seen radiographically, this may represent blood or persistent injectate. Normally blood is diagnosed via MRI by comparing T1 weighted and T2 weighted scans. Whereas saline would appear less hyperintense on T1 imaging compared to T2 imaging, blood would remain hyperintense in both, but to varying degrees depending on the age of the blood. The imaging for our patient was consistent with blood. However, given the speed of her clinical improvement, it is likely that the fluid collection was a combination of blood with persistent injectate. A formal diagnosis was not obtained because surgery was not performed, as her symptoms spontaneously resolved.

Given the delayed onset of the symptoms and the speed of her recovery, we hypothesize that this fluid collection may indeed have been a combination of injectate and blood compressing her spinal cord, which resulted in her transient lower extremity paralysis. A literature search for partition coefficients of saline and blood from epidural space did not yield

Table 1. Characteristics of spinal epidural hematoma case reports.

Paper	Age	Gender	AC	Time stop	ASA/Plavix	Time stop	Level	Spinal stenosis	Volume	Time to intervention	Persistent symptoms
Page et al (6)	67	F	Yes	7 days	No	N/A	Lumbar NOS	Yes	14 mL	35 hours	Bilateral foot drop, impaired ambulation
Loomba et al (7)	63	F	Yes	7 days	Yes	N/A	T12-L1	No*	1 mL contrast + 80 mg methylprednisolone + 9 mL NSS	136 hours	T4 sensory level, lower extremity paraplegia, urinary/bowel incontinence
Shanthanna et al (8)	65	M	Yes	4 days	N/A	N/A	L3-L4	Yes	4 mL	N/A	None
Benyamin et al (9)	81	F	No	N/A	Yes	12 days	C7-T1	No	2 mL contrast + 2 mL steroid + 1 mL NSS	2 hours	None
Lam et al	76	F	No	N/A	Yes	N/A	T12-L1	Yes	8 mL (steroid and saline)	N/A	None

any articles directly discussing this topic. Maugeri et al (10) recently published a case report with literature review regarding post-traumatic cranial epidural hematomas with rates of blood reabsorption. Though the volume involved and the anatomic location of the epidural hematoma is different from the region of interest of this patient, the quoted time of reabsorption ranged from 3 hours (the patient died from thoracic trauma) to several months before the blood was fully reabsorbed. Buffington et al (11) published a study in pigs illustrating direct venous drainage of the epidural space in the high thoracic and cervical region. It is conceivable, given the severity of her lumbar dextroscoliosis, that immediate absorption of the steroid injection was not possible, resulting in the neurologic sequelae she experienced post-procedurally.

A subsequent MRI study was not obtained during

her 48-hour hospitalization; however, a follow-up imaging study, obtained 3 months after the incident, revealed no signs of neurologic damage to her spinal cord. Fortunately, her symptoms resolved and never recurred when she was assessed at subsequent appointments. Without surgical intervention and direct visualization, the diagnosis cannot be definitively confirmed. However, due to both the time of onset and resolution, it is likely that her symptoms were caused by cord compression due to a combination of injectate and blood present from the known venous trauma that occurs during epidural steroid injection. We report the first case of presumed epidural hematoma, likely from persistent injectate and venous blood compression, resulting in bilateral lower extremity paralysis in a patient with dextroscoliosis who had spontaneous resolution without surgical intervention.

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