Epidural Steroid Injections for Radicular Pain Due to Spinal Stenosis Caused by Lipomatosis

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Myth: Epidural steroid injection is contraindicated in patients with spinal stenosis due to epidural lipomatosis.

Fact: There is low-level evidence of an association between epidural steroid injections (ESIs) and the development and/or worsening of spinal epidural lipomatosis (SEL). However, there is insufficient evidence to establish whether ESIs independently result in an increase in spinal stenosis with neurological compromise in individuals with pre-existing SEL.

Spinal epidural lipomatosis (SEL) is a condition of excess, non-encapsulated adipose tissue deposition circumferentially within the epidural space (1). The etiology of SEL remains unclear; however, studies have highlighted four broad categories, which include: exogenous steroid administration, endogenous steroid hormonal disease, obesity, and idiopathic (1). SEL can cause symptoms such as axial low back pain, radiculopathy, neurogenic claudication, myelopathy, or cauda equina syndrome, depending on its location and extent, while also being asymptomatic in many cases (1). Spine magnetic resonance imaging (MRI) can identify the presence of SEL. Although well-defined and reproducible grading systems exist (2,3), they are utilized inconsistently in studies on this subject matter. There is no broadly accepted definition or cutoff measure to define SEL. Treatment includes reduction of exogenous steroid dose, treatment of underlying endocrinopathy, dietary changes, and surgical decompression/laminectomy in severe circumstances (4). A literature review identified 49 cases of idiopathic SEL and 62 cases of secondary SEL treated with surgical decompression resulting in full recovery of symptoms in 60% of lumbar cases and 15-50% in cases involving the thoracic spine (5). The authors did not report on the rate of surgical complications.

Systemic exogenous corticosteroid administration is the most common etiology of SEL (6). Systemic corticosteroids stimulate glucocorticoid receptors within adipose tissue, which may result in the expansion of adipose tissue in the Cushingoid fat distribution and pre-existing epidural adipose tissue (6,7). Conversely, local steroid administration, such as epidural steroid injections (ESIs), is not as clearly associated with the development of or increase in SEL. Thus, while ESI is a common treatment offered to patients with radicular pain secondary to disc herniation and degenerative spinal stenosis (8,9), it is unclear if this treatment is appropriate for patients with radicular pain primarily due to spinal stenosis caused by SEL.

Does ESI increase adipose deposition in the epidural space?

A total of four case reports (10-13), two case series (14,15), and three observational studies (16-18) comprising 861 patients discussed SEL after ESI administration. All studies reported the progression of SEL after ESI or an association between SEL and ESI. The largest study reviewed the MRIs of 28,902 patients and identified that the rate of SEL was 2.5% (731 patients) (18). The authors reported that incidental SEL was present in 168 cases (0.6%), SEL with spine-related symptoms was present in 526 cases (1.8%), and symptomatology specific to SEL was present in 37 cases (0.1%). Multivariate logistic regression revealed that the most important risk factor associated with
overall SEL (both incidental SEL detected on imaging and symptomatic SEL) was prior ESI (Odds Ratio [OR] 3.48, p<0.001). This study also identified other risk factors associated with SEL, including older age, higher modified Charlson comorbidity index (19), male sex, African American race, and systemic corticosteroid use. A subgroup analysis stratified patients with incidental SEL, SEL with spine-related symptoms (e.g., radiculopathy, neurogenic claudication, spinal cord compression), and symptomatic SEL with SEL-specific symptoms (e.g., SEL responsible for symptoms). Multivariate logistic regression revealed that SEL with spine-related symptoms was associated with prior ESI (OR 3.96, p<0.001), though no sub-analysis was performed to assess the association between prior ESI and patients with SEL-specific symptoms.

A case-control study compared 70 patients with SEL diagnosed by MRI and 34 randomly selected control patients (16). This study defined SEL based on compression or distortion of the thecal sac and/or nerve sheath by lipoid on MRI T1 films. This study identified a strong correlation between the number of ESIs and radiographic evidence of worsening SEL. The absence of ESI delivery or a single ESI was not associated with the radiographic evidence of SEL. After three ESIs and four ESIs, the probability for radiographic evidence of SEL was 98% and 100%, respectively. Similarly, a cross-sectional study reported that among patients with SEL, 33% (17/52) had previously received an ESI (17). There was no matched control group.

The findings from these observational studies are further substantiated by case reports (10-13) and case series (14,15) highlighting the progression of SEL with serial MRI imaging after ESI. In one case report, new numbness and dysesthesias developed in the lower extremity with new focal SEL causing thecal compression at the same spinal level where the ESI was previously performed (10). In another case, serial MRIs revealed a circumferential increase in SEL after 13 ESI procedures were performed over a 5-year span, with subsequent resolution of SEL 7 months after cessation of steroid injections (11).

In summary, all included studies demonstrate either the development or progression of SEL after ESI. One such publication reported an association between a greater degree of SEL and the number of ESIs (16). The level of evidence for these findings is low given the type of study design (retrospective observational studies and case reports/series), the presence of confounding variables that were not adjusted within observational studies, and sources of heterogeneity between studies. Causality cannot be established in the absence of prospective studies.

**Does ESI worsen pain and neurological symptoms in patients with pre-existing SEL?**

A total of five case series (14,15,20-22) and one case report (10) comprising 12 patients discussed pain severity and neurological symptoms after ESI administration in patients with pre-existing SEL. Favorable outcomes were reported in two case series (20,21). In two patients with pre-existing SEL causing pain symptoms, there was an 80-85% improvement in pain intensity at 2 weeks after ESI with triamcinolone, and the neurological examination remained stable at follow-up appointments spanning 8-18 months (20). Three patients with lumbosacral radiculopathy experienced a 50-75% decrease in pain scores and an improvement in pain disability index by 13-44 points after ESI with dexamethasone (21). Both of these case series that reported favorable outcomes utilized the transforaminal approach when performing ESI (20,21), though the ability to extrapolate these implications is limited by a small sample size.

Two case series reported worse outcomes after ESI in patients with SEL (14,22). Two patients with pre-existing SEL had a progression of neurological deficits less than 5 months after ESI (14). Similarly, another case series highlighted a patient who had received 103 ESI procedures over a 12-year period and abruptly developed T10 paraplegia due to spinal cord compression and required T10-L2 laminectomy and decompression with removal of epidural fat (22). Notably, the number of ESIs administered in this case series was more than double the maximum limit for the number of ESIs recommended by most society guidelines during that period of time (23).

Equivocal outcomes were reported in one case series (15) and a case report (10). One patient with radicular pain symptoms obtained short-term benefit after three ESIs; serial MRI revealed progression to borderline grade II SEL without worsening neurological symptoms associated with spinal stenosis (15).
Conclusion And Recommendations

1) There are limited data from observational studies and case reports/case series investigating ESIs in patients with SEL (24). Prospective and appropriately-powered studies are needed to establish if there is a causal relation between ESI and both radiographic and symptomatic progression of SEL.
2) Low-level evidence indicates an association between SEL documented on MRI and a history of prior ESI. Physicians may consider advising patients about the potential for an increase in SEL radiographically and an increase in spinal stenosis-related symptoms due to the progression of SEL after receiving an ESI.
3) The number of ESIs performed and the dosage of corticosteroid utilized before the onset or progression of SEL is highly variable. SEL has been reported even after one ESI (14). Low-level evidence demonstrates a correlation between the number of ESIs performed and the subsequent development of SEL.
4) Some case series that included patients with pre-existing SEL report modest, short-term improvement in pain severity and disability following ESI. In contrast, others report worsening neurological deficits.
5) Several studies do not clearly describe the ESI approach (interlaminar versus transforaminal) utilized in their patient cohort (24). Two case series (n=5) utilized a transforaminal approach for managing symptomatic radiculopathy due to SEL, demonstrating improved pain severity and disability (20,21). Additional studies evaluating the ESI approach and outcomes are needed.
6) In the referenced publications, high doses of corticosteroid were commonly administered, exceeding currently recommended doses in contemporary clinical practice (25). There are no studies specifically assessing corticosteroid type or dose utilized in ESI as it relates to the initiation or progression of SEL.

References