

Epidural steroid injections for low back pain in rural practice: a 5-year retrospective study

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Introduction: Epidural steroid injections (ESIs) are a safe and accessible therapy for chronic low back pain, one of the most common and challenging chronic conditions seen in primary care. However, the indications for and effectiveness of ESI remain controversial. In rural settings with limited public transportation infrastructure, such a mobility-limiting condition can have even more negative effects on quality of life and function. Furthermore, diagnostic and specialist services are often limited. A paucity of safe, effective and accessible treatments leads to heavy reliance on oral analgesics, especially opioids, which have well-known complications.

Methods: We reviewed the use of ESI for the 2 most common types of chronic low back pain in those with neurologic symptoms: lumbar disc herniation (LDH) and lumbar spinal stenosis (LSS). We did a retrospective chart review of all patients who underwent ESI between Jan. 1, 2005, and Feb. 25, 2010, at our rural hospital in northwestern Ontario.

Results: During the study period, 123 ESIs were administered to 65 patients. After the first injection, 40 patients (62%) reported improvement, 10 (15%) reported worsening or no change, and 15 (23%) had no follow-up documented.

Conclusion: Some patients with neurologic compromise from LDH or LSS have improvement in symptoms after ESI. A prospective study is underway to more rigorously assess the effectiveness of this treatment.

Introduction : Les injections épidurales de corticostéroïdes (IEC) constituent un traitement sécuritaire et accessible de la lombalgie chronique, une des affections chroniques les plus courantes et complexes en médecine de premier recours. Les indications et l'efficacité des IEC ne font cependant pas consensus. En milieu rural, où les infrastructures de transport public sont limitées, une maladie qui affecte autant la mobilité peut avoir des effets encore plus négatifs sur la qualité de vie et le fonctionnement. En outre, les services diagnostiques spécialisés sont souvent restreints dans ces régions. Le manque de traitements sécuritaires, efficaces et accessibles entraîne une importante dépendance aux analgésiques oraux, particulièrement aux opiacés, dont les complications sont bien connues.

Méthodes : Nous avons passé en revue l'utilisation des IEC pour les 2 plus courants types de lombalgie chez des sujets qui en présentent des signes neurologiques : la hernie discale lombaire (HDL) et la sténose spinale lombaire (SSL). Nous avons examiné de manière rétrospective les dossiers de tous les patients qui ont reçu des IEC entre le 1^{er} janvier 2005 et le 25 février 2010 dans notre hôpital rural du Nord-Ouest de l'Ontario.

Résultats : Durant la période de l'étude, 123 IEC ont été administrées à 65 patients. Après la première injection, 40 patients (62 %) ont signalé une amélioration de leur état, l'état de 10 patients (15 %) s'est aggravé ou est resté stationnaire et pour 15 sujets (23 %), nous n'avons pas obtenu de données de suivi.

Conclusion : Certains patients qui éprouvent des difficultés neurologiques en raison d'une HDL ou d'une SSL voient leurs symptômes s'améliorer après des IEC. Une étude prospective est en cours pour évaluer plus rigoureusement l'efficacité de ce traitement.

INTRODUCTION

Low back pain is among the most common presenting symptoms in primary care.^{1,2} Whereas the vast majority of patients presenting with back pain have an excellent prognosis for both pain relief and functional recovery, the smaller number of chronic cases that require prolonged follow-up constitute a disproportionate number of clinic visits. In a rural setting, these patients typically travel long distances for advanced imaging and orthopedic referral.

Detailed pathophysiologic models and classification systems are in stark contrast to the great uncertainty faced by primary care clinicians and specialists in specific cases: an estimated 85% of cases cannot be given a precise diagnosis.^{3,4} The uncertainties arise from both the complexity of the disease entity itself, and from the limitations of our tools and models.

Chronic back pain often interacts with a host of other medical and psychologic impairments, with each condition exacerbating the others and complicating management. In one survey, 20% of patients with lumbar spinal stenosis (LSS) reported symptoms of depression and 25% reported being "generally dissatisfied with life."^{5,6}

Frequently used terms such as "sprain," "strain," and "degeneration" have no widely accepted histologic or anatomic definition and are effectively synonymous with "idiopathic."⁷ The association between clinical and imaging findings on the one hand and patient distress and disability on the other is generally poor. Physical findings, although clear-cut in many acute cases, become increasingly ambiguous in more chronic ones. Inappropriate use of imaging is widespread and well-documented.⁸⁻¹⁰ The functional relevance of "abnormal" findings is often unclear. Potentially significant findings such as bulging or herniated discs turn out to be very common, even among asymptomatic adults,¹¹⁻¹⁴ and are often incidental, even in symptomatic patients. Such findings can lead to overdiagnosis, increased anxiety, and unnecessary and potentially harmful treatments.⁷ These uncertainties manifest as wide variations in diagnostic workup and treatment.⁴

At initial presentation, the first concern is ruling

out rare (1%–3%)⁷ but potentially life- and limb-threatening causes of back and lower extremity pain: cauda equina syndrome, tumour, epidural abscess, spinal osteomyelitis and aortic aneurysm.¹⁵ Fortunately, although these conditions do have their own diagnostic challenges, the key to accurate diagnosis remains an index of suspicion and thorough evaluation, which may involve emergency, long-distance transport for rural patients.

Of the remaining, so-called mechanical, cases, about 70% are short-lived, often labelled as "sprain" or "strain," and another 10% are due to nonspecific degenerative changes in discs and facet joints.⁷ Lumbar spinal stenosis and lumbar disc herniation (LDH) each account for 3%–4% of cases at initial presentation,¹⁵ but can portend a more chronic course and hence constitute a much larger portion of prevalent disease. Whereas 90% of patients presenting with nonspecific back pain within 3 days of onset recover within 2 weeks,¹⁶ those patients with LSS are likely to experience persistent or worsening pain, despite little progression in neurologic dysfunction. Two follow-up LSS studies (which included 32 and 47 patients, respectively) suggested that 50%–75% of patients with this diagnosis experience either persistent or worsening symptoms.^{17,18} In distinction, LDH has a more favourable natural history, with most patients showing significant clinical improvement within the first 6 weeks.¹⁹ A magnetic resonance imaging follow-up study has suggested that the herniation itself resolves at least partially in two-thirds of cases.²⁰

As with other conditions of subacute and chronic pain, there has been little progress in developing safe, accessible and reliably effective therapies. In a rural setting with limited public transportation infrastructure and large distances, the mobility limitations imposed by back pain can have even greater consequences for quality of life and function. In addition, diagnostic and therapeutic facilities are less accessible in such areas. Frequently, opioid analgesics become the only option for symptomatic treatment, with their limited effectiveness and long-term medical and social complications.²¹

Epidural steroid injections (ESIs) represent a fairly economical, accessible and safe alternative in

In general, the effectiveness of surgery for chronic back pain remains controversial, and there is no consensus on specific indications. The general trend in outcomes is a transient improvement in pain scores in the first 1–5 years, with perhaps subtle improvement in disability and functional outcomes in the first 1–2 years. The longest follow-up data on LSS comes from the Maine Lumbar Spine Study,^{39–41} which followed a cohort of 148 patients for 10 years. Patients who received surgical treatment had more severe symptoms and worse functional status at baseline, and better outcomes at 4-year evaluation than the patients who received nonsurgical treatment.

Epidural steroid injections

Epidural steroid injections are a safe and widely available alternative treatment. Three techniques are commonly used: interlaminar (“classic”), transforaminal and caudal. The transforaminal approach requires fluoroscopic guidance, which is not commonly available in many rural centres. Both the caudal and transforaminal techniques require specific skills, whereas most rural anesthesia providers are proficient at interlaminar epidural injections, commonly used in obstetrics.

Overall, the evidence regarding the effectiveness of ESI over placebo is vast, yet inconclusive. The latest Cochrane review on the subject found a lack of evidence, not only for epidural steroids but also for other injection therapies for subacute or chronic low back pain.⁴² Some studies have reported a range of benefits, from reducing symptoms to delaying or reducing the rates of surgery,^{43–45} whereas others have found no benefit.⁴⁶ Difficulties in diagnosing the exact cause of chronic low back pain make classification of patients and identification of clinically relevant subgroups difficult. It may be that certain etiologies of low back pain are more responsive to epidural steroids than others. In addition, comparison of the techniques used in the positive versus negative trials suggests that the choice of steroid may be important, with nearly all studies using methylprednisolone having negative findings and most studies using other steroids finding some benefit.⁴⁵ Thus far, our centre has been using only methylprednisolone for ESIs.

METHODS

Data collection and analysis

Charts for all patients who underwent an ESI between Jan. 1, 2005, and Feb. 25, 2010, were

reviewed by one of the authors (L.M.). Demographic data, presenting symptoms, diagnosis, imaging results, comorbidities, dates and number of ESIs were coded. The patients had been referred by their family physicians to 2 local general practitioner–anesthetists. We were primarily interested in patients’ diagnosis at the time of injection and their response to treatment.

Outcome at follow-up was documented as “improved,” “no difference/worse” or “no follow-up,” as recorded in the chart. This represented physicians’ overall impression based on the patient’s reports of symptom severity, as well as physical findings. There was substantial variation in the type and detail of follow-up data available, as this was a retrospective chart review.

Data were collected in Excel and imported into IBM SPSS software (version 19.0 for Windows). Data were initially analyzed descriptively, with frequencies and percentages for categorical data and means and standard deviations for continuous data. The characteristics of those who saw improvement, saw no improvement or were lost to follow-up after the initial injection were compared using Pearson χ^2 tests for categorical data, and one-way analysis of variance for continuous data.

Research ethics approval was obtained from the Sioux Lookout Meno Ya Win Research Review Committee.

Method of injection

Before the injection, the physician discussed the risks and procedure with each patient. All patients sat in lumbar flexion on an operating table. The iliac crest was used as a reference point for the L3–4 interspace. From there, the level of injection was identified based on imaging results and patient anatomy. Using sterile technique, after subcutaneous lidocaine injection, the epidural space was identified using loss-of-resistance to air with a 17-gauge Tuohy needle, and 80 mg of methylprednisolone in 5 mL of 0.9% saline was injected. All patients received advice on postinjection management and instructions to follow up with their family physicians.

Previous back surgery, such as spinal fusion, laminectomy or discectomy, often alters access to the epidural space. As a result, in some cases the injection was given 1 level above or below the affected site.

RESULTS

During the 5-year study period, 123 ESIs were administered to 65 patients. Characteristics of the

the rural setting. Their effectiveness, however, has been controversial since their introduction in 1953.²² Most ESI research to date focuses on LDH, often excluding patients with LSS.

In this paper, we briefly review the literature on ESI for low back pain related to LSS and LDH, and present 5 years of clinical experience in a rural hospital. We have included both LDH and LSS patients in our study. To our knowledge, this is the first study to be undertaken in a rural setting using general practitioner–anesthetists undertaking classic epidural technique without advanced imaging. This is currently the only widely available delivery model for ESI in the rural setting.

LOW BACK PAIN, LDH AND LSS

Lumbar disc herniation results from degenerative tears in the annulus fibrosus of the intervertebral discs, leading to herniation of nucleus pulposus into the spinal canal, or neural foramina.²³

Lumbar spinal stenosis is the gradual narrowing of the spinal canal, or the lateral recesses and neural foramina, leading to radicular or chordal neurologic dysfunction. It can arise congenitally²⁴ (primary disease) or, far more commonly, secondary to hypertrophic degenerative changes, degenerative disc disease or less common conditions.²⁵ The most frequent sites of clinically significant stenosis are the lumbar followed by the cervical spine.

In both cases, the resulting mechanical stress and tissue injury can trigger a complex of still poorly understood neurologic, inflammatory, microcirculatory, immune and endocrine changes. Compression can lead to local inflammation; sensitization of surrounding tissue and changes in the excitatory state of nerves;²⁶ impairment of arterial supply and venous return, leading to ischemia and further inflammation;^{27,28} autonomic dysregulation and further impairment of circulation;²⁷ and exacerbation of pain.

The classic manifestation of LSS is neurogenic claudication: a constellation of uni- or bilateral weakness, dull pain and fatigue involving the legs and lower back, worsening with activity and backward extension (e.g., walking downhill, looking up) and improving with rest and forward flexion (e.g., walking uphill, pushing a cart).²⁹ It is typically accompanied by sensory abnormalities, such as numbness and paresthesia.⁷ Consistent with a slow degenerative etiology, patients usually present with a history of months to years of gradually increasing symptoms.

Unlike most cases of LSS, the source of mechanical stress in LDH tends to arise more suddenly and

regress in most cases.^{19,20} However, despite differences in initial etiology, chronic cases of LDH likely involve the same pathophysiologic processes as LSS, with stenosis being caused by the herniated disc and the associated inflammatory and degenerative changes.

MANAGEMENT

Nonsurgical treatment

In addition to treatment for medical and psychiatric comorbidities, conservative management for low back pain consists of activity modification (to reduce spinal extension), conditioning exercise, stretching, physiotherapy, meditation and relaxation techniques, and transcutaneous electrical nerve stimulation.³⁰ Trials of conservative management regimens typically use multimodal strategies and report some improvement in up to 70% of cases.^{31–33}

Oral analgesics, usually nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids, are used frequently but with limited effectiveness. There is no clear evidence for the benefit of one class over another, and both have potentially substantial side effects with prolonged use. NSAIDs are associated with renal impairment and gastrointestinal bleeding and, in the case of cyclooxygenase-2 inhibitors, potentially elevated cardiovascular risk.³⁴ The high incidence of diabetes and diabetic renal compromise in many rural populations, such as in our own catchment area in northern Ontario, limits the use of NSAIDs. Opioids, on the other hand, are associated with tolerance, potential abuse and dose-related risk of death.²¹ One recent study suggests that the use and dose of opioids for nonmalignant pain in socioeconomically disadvantaged patients has substantially increased in the province of Ontario,²¹ leading to increased mortality.

Surgery

For LDH without symptoms of cauda equina syndrome or foot drop, conservative management is recommended for at least 1 month, as only 10% of patients have sufficient pain after 6 weeks to consider surgery.⁷ Lumbar discectomy is the most common surgical treatment for refractory pain from LDH and has been documented to offer improved pain relief for up to 4 years.⁷

Lumbar spinal stenosis has a much higher rate of surgical intervention and has become the most frequent indication for spinal surgery in adults older than 65 years.^{35–37} Lumbar laminectomy for LSS is better supported by outcome evidence than the more complex instrumented fusion procedures.^{35,38}

patients are provided in Table 1. Given the small sample and our aim of identifying factors for future investigation, we highlight all findings with $p < 0.15$. Slightly more than half of all patients were female, one-third self-identified as Aboriginal and the aver-

age age at first injection was 55 years.

The most common comorbidities were hypertension (52%), osteoarthritis (49%), psychosocial conditions (37%) and type 2 diabetes (32%). The most commonly documented initial symptoms were back

Table 1. Characteristics of patients, by outcome after first epidural steroid injection

Characteristic	No. (%) of patients*†				p value‡
	Total, n = 65	No improvement, n = 10	Improvement, n = 40	Lost to follow-up, n = 15	
Age, yr, mean (SD)	54.8 (18.5)	48.6 (20.9)	56.2 (17.4)	55.1 (19.9)	0.51
Sex					0.70
Male	27 (42)	3 (11)	17 (63)	7 (26)	
Female	38 (58)	7 (18)	23 (61)	8 (21)	
Aboriginal (self-identified)	24 (37)	4 (17)	16 (67)	4 (17)	0.64
Location					0.26
Sioux Lookout, Ont.	42 (65)	5 (12)	24 (57)	13 (31)	
Northern community	15 (23)	4 (27)	10 (67)	1 (7)	
Other/unknown	8 (12)	1 (13)	6 (75)	1 (13)	
Radiographic diagnosis					
Lumbar spinal stenosis	33 (51)	4 (12)	22 (67)	7 (21)	0.65
Lumbar disc herniation	52 (80)	9 (17)	30 (58)	13 (25)	0.44
Spondylolisthesis	16 (25)	3 (19)	10 (63)	3 (19)	0.85
Back pain NYD	5 (8)	1 (20)	2 (40)	2 (40)	0.58
Symptoms					
Leg pain	54 (83)	8 (15)	35 (65)	11 (20)	0.44
Leg weakness	31 (48)	8 (26)	17 (55)	6 (19)	0.08
Leg numbness	33 (51)	8 (24)	19 (58)	6 (18)	0.11
Positive "shopping cart" test	8 (12)	2 (25)	6 (75)	0 (0)	0.23
Bowel or bladder incontinence§	9 (14)	3 (33)	4 (44)	2 (22)	0.26
Limited exercise tolerance	36 (55)	6 (17)	22 (61)	8 (22)	0.95
Abnormal deep tendon reflexes	25 (39)	5 (20)	15 (60)	5 (20)	0.60
Back surgery	15 (23)	4 (27)	9 (60)	2 (13)	0.29
Analgesic use					
Narcotics	37 (57)	7 (19)	23 (62)	7 (19)	0.51
NSAIDs	42 (65)	5 (12)	26 (62)	11 (26)	0.49
Other analgesics	28 (43)	4 (14)	17 (61)	7 (25)	0.94
Physiotherapy					0.37
No physiotherapy	31 (48)	3 (10)	21 (68)	7 (23)	
Education/exercise	16 (25)	2 (13)	10 (63)	4 (25)	
Physiotherapy	16 (25)	5 (31)	7 (44)	4 (25)	
Comorbidities					
Type 2 diabetes	21 (32)	2 (10)	15 (71)	4 (19)	0.49
Osteoarthritis	32 (49)	4 (13)	20 (63)	8 (25)	0.78
Rheumatoid arthritis	3 (5)	1 (33)	1 (33)	1 (33)	0.51
Peripheral vascular disease	6 (9)	0 (0)	5 (83)	1 (17)	0.46
Psychosocial (anxiety, depression, stress)	24 (37)	5 (21)	13 (54)	6 (25)	0.61
Hypertension	34 (52)	3 (9)	25 (74)	6 (18)	0.08
Coronary artery disease	12 (19)	1 (8)	7 (58)	4 (33)	0.57
Cerebrovascular disease	6 (9)	0 (0)	3 (50)	3 (50)	0.21
Scoliosis	11 (17)	2 (18)	5 (46)	4 (37)	0.52

Note: Findings with p values < 0.15 are highlighted in bold.

NSAID = nonsteroidal anti-inflammatory drug; NYD = not yet diagnosed; SD = standard deviation.

*Unless stated otherwise.

†For the full sample, percentages are out of 65; for the 3 outcome groups, percentages are by row. Percentages do not always total 100 because of rounding.

‡Tests of significance are based on the t test (age) and the χ^2 test (all others, Pearson or Fisher Exact test as appropriate).

§All cases of bowel and bladder incontinence were chronic. None of the patients in this series presented with cauda equina syndrome.

pain (all), leg pain (83%), limited exercise tolerance (55%), leg numbness (51%) and leg weakness (48%). Sixty-one of the patients had computed tomography or magnetic resonance imaging; of these, 80% had disc herniation or bulging in at least 1 level, 51% had LSS and 25% spondylolisthesis. Only 1 patient had no abnormalities on imaging. There were no cases of acute cauda equina syndrome in the group.

Outcomes of ESI

Of the 65 patients who received a first injection, 33

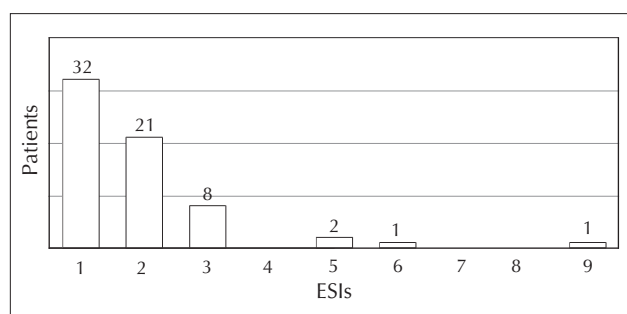


Fig. 1. Number of epidural steroid injections (ESIs) per patient (total 123 ESIs in 65 patients).

(50%) had subsequent injections, with 12 (18%) having 3 or more (Fig. 1). The interval between injections ranged from 9 days to 4 years, with a median of 109 days (Fig. 2).

After the first injection, 40 (62%) reported improvement, 10 (15%) reported worsening or no change and 15 (23%) had no follow-up documented. Of the patients who had multiple injections, 58%–67% reported improvement. The outcome data for the 123 ESIs are presented in Figure 3. Patients whose symptoms improved with their first ESI were somewhat more likely to receive future injections ($p = 0.17$).

Comparison of groups

Factors associated with improvement ($n = 40$), no improvement ($n = 10$), or loss to follow-up ($n = 15$) are presented in Table 1. Those with no improvement tended to be somewhat younger than those who did improve or were lost to follow-up (49 years of age compared with 56 and 55 years, respectively), but this association was not significant ($p = 0.51$).

Two symptoms showed a potentially significant

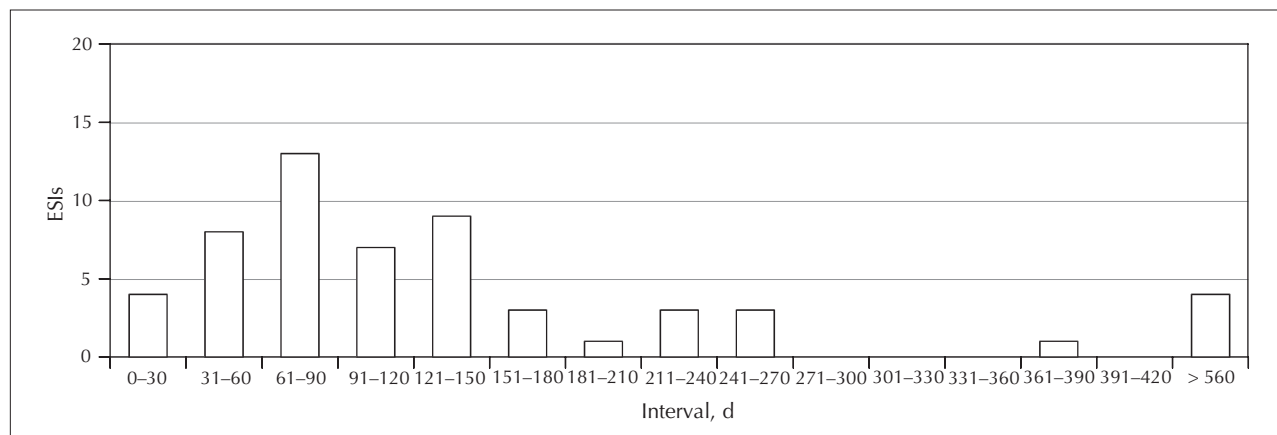


Fig. 2. Interval between procedures in patients who had more than 1 epidural steroid injection (ESI; 59 ESIs in 33 patients).

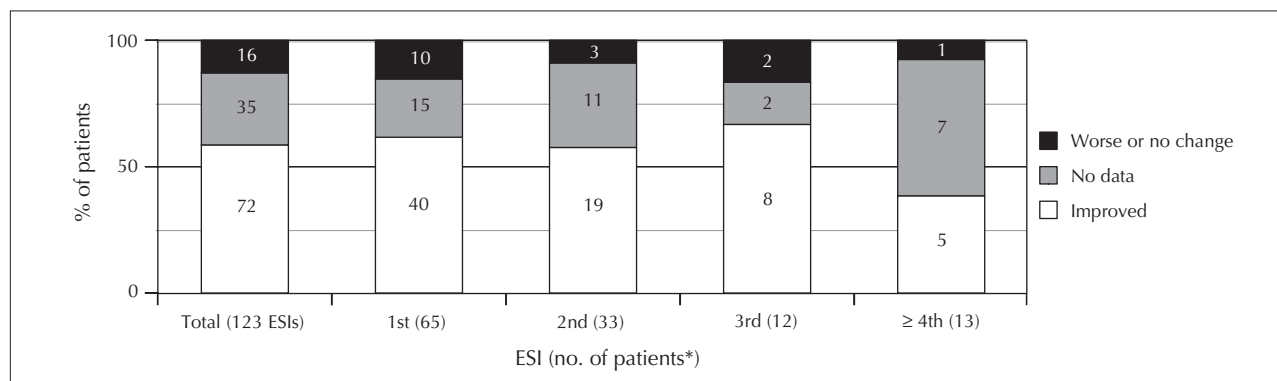


Fig. 3. Outcome in epidural steroid injections (ESIs), by total ESIs performed and by number performed in patients. *Unless stated otherwise.

association with a negative outcome: leg weakness ($p = 0.08$) and leg numbness ($p = 0.11$). Both are suggestive of neurologic dysfunction. Hypertension was the only comorbidity potentially associated with injection outcome ($p = 0.08$), with the improved group having a higher proportion of patients with hypertension. More than half of those whose symptoms improved did so despite having no access to physiotherapy. There were no apparent associations between diagnosis and ESI outcome, or between analgesic use and ESI outcome. More patients with LSS had improved symptoms than those with LDH, although many patients had both LSS and LDH (Table 1).

Surgery

Fifteen of 65 patients had documented back surgery. Six patients had undergone back surgery before the study period and 10 had surgery during the study period (1 patient had both). Three patients underwent 2 surgeries. The type of surgery was not documented for 2 of the 15 patients. Of the remaining 13 patients, 2 underwent spinal fusion, and 11 had 1 or 2 laminectomy/discectomy procedures.

One patient had undergone 2 previous surgeries and received 9 ESIs during the period under review. Excluding this patient, the number of injections for patients who underwent surgery (either before or after) ranged from 1 to 6 with an average of 1.8, similar to the overall average.

DISCUSSION

Inferences from these data regarding the effectiveness of ESI are limited by the retrospective nature of the study, the incompleteness and potential inconsistency of follow-up data, and the real-world complexities of copathology and diagnostic uncertainty.

Although the sample is small, and therefore underpowered, the data do suggest that symptoms of leg weakness and numbness may be associated with poorer outcomes. Among those with follow-up data, 80% had documented improvement. We cannot say whether this is an improvement over the natural history of the disease.

Epidural steroid injections can be a useful therapy for a common and symptomatic condition, in settings where other options are limited. Whereas fluoroscopically guided techniques are a common standard in the literature, our study highlights that commonly used obstetric anesthesia techniques available in many rural areas can provide positive results. As such, the pre-

cise indications and effectiveness of epidural steroids in rural areas deserves more rigorous study.

CONCLUSION

We have documented that some patients with neurologic compromise, from either LSS or LDH, seem to have improvement of symptoms after an ESI, but the retrospective nature of our study does not allow us to draw any clear causal links. This requires larger, prospective pools of clinical data, as well as improved understanding of the pathophysiologic mechanisms of chronic back pain and the potential points of therapeutic intervention. We have already begun a multicentred prospective rural study of the effectiveness of ESIs.

Competing interests: None declared.

REFERENCES

1. Andersson GB. Epidemiological features of chronic low-back pain. *Lancet* 1999;354:581-5.
2. Hart LG, Deyo RA, Cherkin DC. Physician office visits for low back pain. Frequency, clinical evaluation, and treatment patterns from a U.S. national survey. *Spine* 1995;20:11-9.
3. Cherkin DC, Deyo RA, Loeser JD, et al. An international comparison of back surgery rates. *Spine* 1994;19:1201-6.
4. Cherkin DC, Deyo RA, Wheeler K, et al. Physician variation in diagnostic testing for low back pain. Who you see is what you get. *Arthritis Rheum* 1994;37:15-22.
5. Sinikallio S, Aalto T, Airaksinen O, et al. Depression is associated with a poorer outcome of lumbar spinal stenosis surgery: a two-year prospective follow-up study. *Spine (Phila Pa 1976)* 2011;36:677-682.
6. Sinikallio S, Aalto T, Airaksinen O, et al. Depression is associated with poorer outcome of lumbar spinal stenosis surgery. *Eur Spine J* 2007;16:905-12.
7. Deyo RA, Weinstein JN. Low back pain. *N Engl J Med* 2001;344:363-70.
8. Carey TS, Garrett J. Patterns of ordering diagnostic tests for patients with acute low back pain. The North Carolina Back Pain Project. *Ann Intern Med* 1996;125:807-14.
9. Swedlow A, Johnson G, Smithline N, et al. Increased costs and rates of use in the California workers' compensation system as a result of self-referral by physicians. *N Engl J Med* 1992;327:1502-6.
10. Keller RB, Atlas SJ, Soule DN, et al. Relationship between rates and outcomes of operative treatment for lumbar disc herniation and spinal stenosis. *J Bone Joint Surg Am* 1999;81:752-8.
11. Boden SD, Davis DO, Dina TS, et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am* 1990;72:403-8.

12. Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med* 1994;331:69-73.
13. Weishaupt D, Zanetti M, Hodler J, et al. MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. *Radiology* 1998;209:661-6.
14. Stadnik TW, Lee RR, Coen HL, et al. Annular tears and disk herniation: prevalence and contrast enhancement on MR images in the absence of low back pain or sciatica. *Radiology* 1998;206:49-55.
15. Siebert E, Pruss H, Klingebiel R, et al. Lumbar spinal stenosis: syndrome, diagnostics and treatment. *Nat Rev Neurol* 2009;5:392-403.
16. Coste J, Delecoeuillerie G, Cohen de Lara A, et al. Clinical course and prognostic factors in acute low back pain: an inception cohort study in primary care practice. *BMJ* 1994;308:577-80.
17. Johnsson KE, Rosén I, Udén A. The natural course of lumbar spinal stenosis. *Clin Orthop Relat Res* 1992;(279):82-6.
18. Swezey RL. Outcomes for lumbar stenosis. *J Clin Rheumatol* 1996;2:129-34.
19. Delauche-Cavallier MC, Budet C, Laredo JD, et al. Lumbar disc herniation. Computed tomography scan changes after conservative treatment of nerve root compression. *Spine* 1992;17:927-33.
20. Bozzao A, Gallucci M, Masciocchi C, et al. Lumbar disk herniation: MR imaging assessment of natural history in patients treated without surgery. *Radiology* 1992;185:135-41.
21. Gomes T, Juurlink DN, Dhalla IA, et al. Trends in opioid use and dosing among socio-economically disadvantaged patients. *Open Medicine* 2011;5(1). Available: www.openmedicine.ca/article/view/Article/421/373 (accessed 2011 Apr. 13).
22. Goebert HW Jr, Jallo SJ, Gardner WJ, et al. Painful radiculopathy treated with epidural injections of procaine and hydrocortisone acetate: results in 113 patients. *Anesth Analg* 1961;40:130-4.
23. Kallewaard JW, Terheggen MAMB, Groen GJ, et al. 15. Discogenic low back pain. *Pain Pract* 2010;10:560-79.
24. Singh K, Samartzis D, Vaccaro A, et al. Congenital lumbar spinal stenosis: a prospective, control-matched, cohort radiographic analysis. *Spine J* 2005;5:615-22.
25. Fogel GR, Cunningham PY 3rd, Esses SI. Spinal epidural lipomatosis: case reports, literature review and meta-analysis. *Spine J* 2005;5:202-11.
26. Kobayashi S, Kokubo Y, Uchida K, et al. Effect of lumbar nerve root compression on primary sensory neurons and their central branches: changes in the nociceptive neuropeptides substance P and somatostatin. *Spine* 2005;30:276-82.
27. Chosa E, Sekimoto T, Kubo S, et al. Evaluation of circulatory compromise in the leg in lumbar spinal canal stenosis. *Clin Orthop Relat Res* 2005;(431):129-33.
28. Kobayashi S, Baba H, Uchida K, et al. [Blood circulation of cauda equina and nerve root]. [Article in Japanese] *Clin Calcium* 2005;15:63-72.
29. van Gelderen C. An orthotic (lordotic) cauda syndrome. *Acta Psychiatr Scand* 1948;23:57-68.
30. Hilibrand AS, Rand N. Degenerative lumbar stenosis: diagnosis and management. *J Am Acad Orthop Surg* 1999;7:239-49.
31. Murphy DR, Hurwitz EL, Gregory AA, et al. A non-surgical approach to the management of lumbar spinal stenosis: a prospective observational cohort study. *BMC Musculoskelet Disord* 2006;7:16.
32. Vo AN, Kamen LB, Shih VC, et al. Rehabilitation of orthopedic and rheumatologic disorders. 5. Lumbar spinal stenosis. *Arch Phys Med Rehabil* 2005;86(SUPPL. 1):S69-76.
33. Simotas AC. Nonoperative treatment for lumbar spinal stenosis. *Clin Orthop Relat Res* 2001;(384):153-61.
34. Roelofs PDDM, Deyo RA, Koes BW, et al. Nonsteroidal anti-inflammatory drugs for low back pain: an updated cochrane review. *Spine* 2008;33:1766-74.
35. Deyo RA, Gray DT, Kreuter W, et al. United States trends in lumbar fusion surgery for degenerative conditions. *Spine* 2005;30:1441-5, discussion 1446-7.
36. Weinstein JN, Tosteson TD, Lurie JD, et al. Surgical versus non-surgical therapy for lumbar spinal stenosis. *N Engl J Med* 2008;358:794-810.
37. Deyo RA, Mirza S, Martin BI. Trends, major medical complications, and charges associated with surgery for lumbar spinal stenosis in older adults. *JAMA* 2010;303:1259-65.
38. Carragee EJ. The increasing morbidity of elective spinal stenosis surgery: Is it necessary? *JAMA* 2010;303:1309-10.
39. Atlas SJ, Keller RB, Wu YA, et al. Long-term outcomes of surgical and nonsurgical management of lumbar spinal stenosis: 8 to 10 year results from the Maine Lumbar Spine Study. *Spine* 2005;30:936-43.
40. Atlas SJ, Deyo RA, Keller RB, et al. The Maine Lumbar Spine Study, part III: 1-year outcomes of surgical and nonsurgical management of lumbar spinal stenosis. *Spine* 1996;21:1787-94.
41. Atlas SJ, Keller RB, Robson D, et al. Surgical and nonsurgical management of lumbar spinal stenosis: four-year outcomes from the Maine Lumbar Spine Study. *Spine* 2000;25:556-62.
42. Staal JB, de Bie RA, de Vet HCW, et al. Injection therapy for subacute and chronic low back pain: an updated Cochrane review. *Spine* 2009;34:49-59.
43. Roberts ST, Willick SE, Rho ME, et al. Efficacy of lumbosacral transforaminal epidural steroid injections: a systematic review. *PM R* 2009;1:657-68.
44. Riew KD, Yin Y, Gilula L, et al. The effect of nerve-root injections on the need for operative treatment of lumbar radicular pain. A prospective, randomized, controlled, double-blind study. *J Bone Joint Surg Am* 2000;82-A:1589-93.
45. Carette S, Leclaire R, Marcoux S, et al. Epidural corticosteroid injections for sciatica due to herniated nucleus pulposus. *N Engl J Med* 1997;336:1634-40.
46. Iversen T, Solberg TK, Romner B, et al. Effect of caudal epidural steroid or saline injection in chronic lumbar radiculopathy: multicentre, blinded, randomised controlled trial. *BMJ* 2011;343:d5278.