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Review Article

Epidural steroid therapy for back and leg pain: mechanisms of action and efficacy

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Abstract

BACKGROUND CONTEXT: Epidural steroid injection (ESI) is one of the most common nonsurgical interventions prescribed for back and leg pain symptoms. Although the use of ESI is widespread, proof of efficacy among the broad population of low back pain patients is lacking and use is predicated to a great extent on the cost and morbidity of the perceived "next step" in many patient's care—surgery.

PURPOSE: To review the relative indications and clinical features that predict success with ESI therapy, and to provide a physiological rationale to guide clinical decision-making.

STUDY DESIGN/SETTING: Review of literature and clinical experience.

RESULTS: Clinical studies have alternately supported and refuted the efficacy of ESI in the treatment of patients with back and leg pain. Steroid medications do benefit some patients with radicular pain, but the benefit is often limited in duration, making efficacy difficult to prove over time. Steroids appear to speed the rate of recovery and return to function, however, allowing patients to reduce medication levels and increase activity while awaiting the natural improvement expected in most spinal disorders. Fluoroscopic verification of needle placement, with contrast injection, greatly improves steroid delivery while reducing risks. Although it is assumed that the benefit of steroids is related to their effect on inflammation, that remains unproven, and it is possible that benefit is gained through an unrecognized action.

CONCLUSIONS: Randomized, controlled trials are needed to conclusively identify those patients most likely to benefit from ESI, and when and for how long. Until then, epidural steroids provide a reasonable alternative to surgical intervention in selected patients with back and/or leg pain, whose symptoms are functionally limiting. When appropriate goals are established and proper patients are selected, sufficient short-term benefit has been documented to warrant continued use of this tool. © 2005 Elsevier Inc. All rights reserved.

Keywords: Back pain; Radiculopathy; Epidural steroid injection; Lumbar spine; Nonoperative management

Introduction

Whereas mechanical compression of the spinal nerve root is usually the precipitating cause of sciatic pain in patients with lumbar disc herniation, it is unlikely that pressure on the nerve root is the only cause of radicular pain in patients with disc disease [1–3]. The association between pressure and pain is not so direct. Severe nerve root compression may not produce pain in every patient, and surgical decompression may fail to relieve symptoms in others. Although decompression may provide more rapid improvement than nonsurgical care, nonoperative modalities can provide excellent pain relief in many patients [4,5]. The key factor determining the extent and severity of leg and back pain in these patients is often inflammation, in combination with nerve root pressure or mechanical irritation. Modalities that alleviate this inflammatory process can reduce pain symptoms and improve function in patients with a variety of spinal disorders.

FDA device/drug status: not applicable.

Nothing of value received from a commercial entity related to this research.

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History of epidural steroid use

Medications were first injected into the epidural space to treat back and leg pain in the early part of the last century. Viner began injecting large volumes of saline and procaine into the lumbar epidural space to treat back pain and lumbar radiculopathy in the 1920s [6]. Evans reported results with a similar procedure in 1930, but achieved only a 14% clinical success rate in 40 patients [7]. The effects of epidural steroid injection were first reported in 1960. Brown reported complete transient relief in four patients with long-standing (6–24 months) sciatica treated with methylprednisolone [8]. Goebert et al. gave injections of procaine and hydrocortisone to 239 patients with sciatica, and reported greater than 60% relief of symptoms in 58% of patients [9]. Since that time, the technique and indications of epidural steroid injection have been changing constantly.

A variety of anesthetics have been used (procaine, lidocaine, bupivacaine) as well as a number of corticosteroid agents (hydrocortisone, methylprednisolone, triamcinolone). Physicians have tried epidural injections with saline alone, anesthetic alone, steroid alone, and with combinations of each [6–10]. Dosages of each medication, and the number and timing of injections have varied widely. Both caudal and lumbar interlaminar approaches have been used to reach the epidural space [11,12]. Intrathecal injections were popular for a time, but the incidence of steroid-induced arachnoiditis caused by either the steroid or its carrier substance [13], and the less common occurrence of meningitis (aseptic, septic, cryptococcal, and tuberculous) [14,15], have made this approach uncommon in modern pain management.

Indications for use have also changed over time. Whereas most authorities continue to emphasize rest, anti-inflammatory medication, and physical therapy as the initial treatment for radiculopathy and back pain [16], physicians have advocated epidural steroid therapy for both acute and chronic pain, back or leg pain, and for diagnoses ranging from acute disc herniation to end-stage disc degeneration. Because epidural injections are usually prescribed along with a number of other modalities, ranging from prolonged bed rest and lumbar traction to passive and active physical therapy programs, therapeutic modalities (heat, cold, ultrasound), and any of a variety of anti-inflammatory medications, it is always difficult to attribute treatment benefit to the injection alone. This constant variation in clinical indications, applications, and outcome measures makes it difficult to compare results from one study to another in the literature. This inconsistent approach to epidural steroid therapy has made it difficult to prove efficacy.

Causes of sciatica

Nerve root compression, through disc herniation or stenosis, is the most common cause of sciatic pain symptoms [17–19]. Other factors contribute significantly to the overall experience of radicular pain, however. Although Lindblom and Rexed proposed nerve root compression as a cause of radicular pain after demonstrating pathological changes in nerve roots compressed by herniated disc material [20], other investigators have revealed disc protrusion or herniation on post-mortem examinations in roughly 40% of patients with no history of sciatic pain [21]. Thirty-five percent of asymptomatic individuals demonstrate myelographic abnormalities [22] and up to 60% have magnetic resonance imaging findings [23], evidence that compression alone is not sufficient to generate symptoms in every patient. Likewise, relief of pressure is not always enough to eliminate symptoms of radicular pain. Patients presenting with acute sciatic symptoms often improve greatly over the next 2 to 6 weeks, long before resorption of extruded disc material can relieve the pressure naturally. Other patients continue to experience severe symptoms despite complete surgical decompression. Still others, with no evidence of direct nerve root compression on magnetic resonance imaging or myelographic examination, suffer persistent sciatic pain despite an apparent absence of the mechanical compression.

Acute compression of the nerve root typically produces weakness and numbness first. Pain is usually not the first symptom. Commonly, radicular pain becomes intense a few hours or days after acute compression.

Inflammation plays a major role in the evolution of symptoms from paresis to true radiculopathy. The nucleus pulposus contains materials that are inflammatory and neuroexcitatory. Application of autologous nucleus pulposus material to the dorsal root ganglion produces sustained nerve discharges consistent with nociceptive function [24,25]. Howe demonstrated that the nerve root does not become sensitized or begin to transmit pain signals until an inflammatory process has been generated [18,26]. Once inflammation is established, however, the nerve becomes exquisitely sensitive to pressure, producing prolonged, pain-generating discharges with either gentle manipulation or pressure [27]. The inflammatory and neurochemical components of the inflammatory cascade serve as principal modulators, if not precipitators, of radicular pain. The dorsal root ganglion may be the initial source of pain in response to mechanical pressure, if that pressure is delivered to the ganglion itself. When compared with the nerve root, the ganglion responds early and with prolonged after-discharges to mechanical stimuli, whereas the root responds similarly only when already irritated or inflamed. This may be explained by the higher concentration of sodium channels in the cell body [28]. It is this inflammatory arm of the pain pathway that physicians try to influence when they inject corticosteroids into the spinal epidural space (Fig. 1).

Scientific rationale for corticosteroid use

Lindahl and Rexed first noted inflammation, edema, and proliferative or degenerative changes in biopsy samples from posterior nerve roots of patients undergoing laminectomy

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