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LUMBAR DISC DISEASE: A COMPARATIVE REVIEW OF TRANSFORAMINAL AND INTERLAMINAR EPIDURAL STEROID INJECTIONS

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ABSTRACT

Lumbar epidural injections have been widely utilized to treat lumbar radicular pain among the several therapies used to manage persistent spinal pain. Transforaminal epidural injections have achieved rapid and widespread recognition for treating lumbar and lower extremities pain among caudal, inter-laminar, and transforaminal epidural injections. Regarding Roland Morris disability evaluation, Visual Numeric Scale, and Finger Floor Distance assessment, transforaminal epidural steroid medication has a better effect. TFESI is superior to Interlaminar steroid injection since it allows for target-specific delivery. Interlaminar steroid delivery under fluoroscopic supervision is also beneficial. However, because it is usually given blindly, there is a risk of needle misplacement, resulting in a lower success rate.

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1. INTRODUCTION

The epidural corticosteroids in the therapy of sciatica patients at Guy's Hospital in London led to a significant pain reduction in the short term. Fifty patients with chronic cervicobrachialgia and treated with epidural steroids. There were statistically significant results obtained. In chronic resistant cervicobrachialgia, cervical epidural steroid/local anesthetic injection is excellent for instant and long-term pain relief, improved motion, and performance. They hypothesized that the caudal and interlaminar techniques had considerable differences in results due to difficulty in giving medicine to the desired area and the occurrence of epidural ligaments and scarring impacting drug distribution [1,2]. After transforaminal injections, they noticed the injectate flowed towards the anterior and lateral epidural spaces. They thought that transforaminal injection was preferable in reaching the pathological site while allowing them toutilize fewer steroids. The patients received a transforaminal epidural steroid injection or a saline trigger-point injection [3]. The schematic representation of both sites is shown in Figure 1. After an average follow-up of 1.4 years, the group getting transforaminal epidural steroid injections had an 84 percent success rate, compared to 48 percent for the group receiving trigger-point injections [4]. Thirty-one patients with discal radicular pain lasting less than three months were randomly assigned to either radio-guided transforaminal or blindly administered interspinous epidural

corticosteroid injections [5–7]. Clinically, the therapy outcome was assessed solely by a sent questionnaire. With a reduction in the Roland–Morris score, answers to the questionnaire still revealed substantially superior outcomes for transforaminal injection [8].

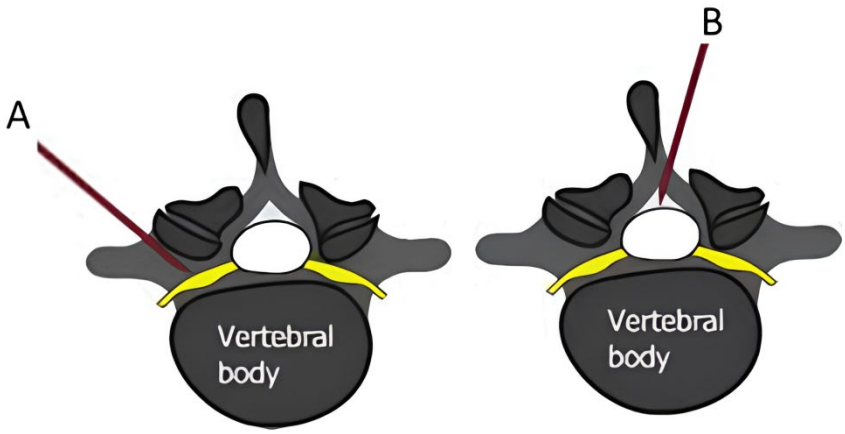

Figure 1. Schematic representation of A. Transforaminal epidural steroid injection and B. Interlaminar epidural steroid injection.

2. NOVEL METHODS IN LUMBAR DISC RESEARCH

Magnetic resonance imaging showed the patients' degenerative lumbar spinal stenosis, graded as mild, moderate, or severe. Before the initial injection, an impartial observer assessed the patients, and they completed questionnaires at six weeks, six months, and 12 months after the injections [9]. According to the researchers, the fluoroscopically guided caudal approach of epidural steroid injections may help lower bilateral radicular pain and enhance patient tolerance to standing and walking [10,11]. A total of 64 people with persistent radiculopathy were enrolled randomly and followed up. Long-term pain alleviation and functional capacity improvements were slightly better with steroids administered via the Transforaminal route. Assumption [12]: For treating low back pain withlumbosacral radicular discomfort, epidural injection delivered using theParasagittal Interlaminar approach is equal to the transforaminal strategy in pain reduction and functional improvement. The PIL approach is a viable alternative to the TF strategy for its equal effectiveness, improved safety profile, and technical ease [13].

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2.1. TREATMENT STRATEGIES

L1-L5 vertebrae form the lumbar region. The laminae, pedicles, and articular processes ofadjacent vertebrae, coupled with the intervertebral discs, create a gap through which spinalnerves leave. The lumbar vertebrae form a lordotic curve once combined [14]. The trabecular bone, containing the red marrow, is surrounded by a thin exterior layer of compact bone in the vertebral body. The vertebral (spinal) canal housing the spinal cord is formed by the arch and the posterior side of the body [6,15–19]. The bony segments form the arch (bilateral pedicles) that connect the transverse and spinous processes, and the bilateral lamina builds most of the arch [20]. A typical vertebra has two superior and two inferior articular processes, which engage neighboring vertebrae's inferior and superior articular processes. Where superior and articular facets meet is a facet or zygapophyseal joint [21]. These are responsible formaintaining spinal alignment, controlling the range of motion, and supporting weight in specific situations [22,23].

The characteristics of typical lumbar vertebrae differ from those of cervical or thoracic vertebrae. The existence of a big vertebral body is the most striking difference. In comparisonto the size of the vertebra, the spinous process is short and thick, and it protrudes perpendicularly from the body [24]. The superior facets of the articular aspects are directed posteromedially and medially, while the inferior facets are directed posteromedially and medially [25]. The facets also have a curved articular surface, a distinctive feature. This is one of the characteristics that distinguishes lumbar vertebrae from thoracic vertebrae. The mammillary process is present in the posterior part of the superior articular process [26]. The height of the lumbar intervertebral disc lies in the middle of the cervical and thoracic intervertebral discs. The nucleus pulposus is a remnant of the notochord. The nucleus contains 70–85 percent water, which declines with aging; collagen fibers are randomly organized in the central nucleus, and elastin fibers are arranged radially; these fibers are embedded in a highly hydrated aggrecan-containing gel. Chondrocyte-like cells, often in a capsule inside the matrix, are interspersed at low density [27]**.** The aggrecan in the nucleus pulposus contains highly sulfated, negatively charged GAG chains. The nucleus possesses the largest concentration of aggrecan, which is hydrated, as well as a lower collagen content than other areas [28]. Because of the restricted space available to accept collagen, only type II collagen is found in the nucleus. A load of axial compression is distributed vertically and radially throughout thenucleus pulposus due to its gelatinous nature [29].

3. FUTURE PERSPECTIVES

The annulus fibrosis has been arranged in 15 to 20 concentric layers. Within each lamella, collagen fibers are arranged parallel. The fibers in adjacent lamellae are orientated at about 60 degrees to the vertical axis, alternating to the left and right. Between the lamellae are elastin fibers, which may help the disc recover to its previous shape after bending, whether in flexion or extension [30]. It contains both type I and type II collagen. The annulus fibers absorb the radial distribution of the vertical force (tangential loading of the disc). Through the hyaline cartilage plate, weight is conveyed to the nucleus. Because it is avascular, hyaline cartilage is perfect for this role. If weight is transmitted through a vascularized structure, such as bone, the local pressure will cut off blood flow, and the bone will die in stages [31]. This condition occurs when the cartilage plate has congenital abnormalities and the nucleus is indirect touch with the spongiosa of bone. The blood supply is cut off, a small area of bone dies, and the nucleus gradually intrudes into the vertebral body. The nucleus becomes more rigid, lesshydrated, or more robust and collagenous due to fewer proteoglycans [32]. The number of viable cells decreases, and the nucleus pulposus turns yellow-brown instead of white. Annulus tears in many areas. The ratio of keratin sulphate to chondroitin sulphate and lactate concentration would increase [33]. The same result is seen in scoliotic discs when the concentration of type X collagen in the matrix of aged people increases. This could be due to aberrant calcification in these stressed discs or deteriorated disc calcification [34].

Failure of the nutrition supply to the disc cells is considered one of the fundamental reasons for disc degeneration. Changes in the nucleus pulposus and vertebral end-plates signify the molecular degenerative process. The regenerative process is thought to be stimulated and maintained by notochordal cells (nucleus pulposus) [35]. Large notochordal cells are active until the second decade of life, when they become scarce. The hyaline cartilage layer that covers the bony vertebral end-plates functions as a diffusion barrier, enabling only small uncharged molecules to migrate into and out of the disc in large

quantities [36]. The vertebral end-plates calcify as they age, and the calcification impacts disc metabolism by reducing nutrient delivery and eliminating waste products [37].

4. CONCLUSIONS

The loss of notochordal cells is the first physical change in the disc, which maytip the balance from regeneration to degeneration. Notochordal cells have been demonstrated to stimulate proteoglycan formation due to the release of soluble mediators; hence their absence may deprive disc health of a crucial stimulus. Cell necrosis and apoptosis (programmed cell death) are two types ofcell death that occur over time [38]. Fas-mediated apoptosis can be triggered in twoways: type I cells have the Fas receptor on their cell membrane, while type II cells contain the Fas receptor intracellularly in the mitochondria. According to a recent study, type II cells predominate in human disc fragments [39]**.** Disc degeneration is aided by disc enzymes such as cathepsin, lysozyme, aggrecanase, and numerous matrix metalloproteinases (MMPs) [40]. Genes related to disc development have been determined. Early disc degeneration is more likely in people with a variation in the aggrecan gene [41].Mutations in structural matrix molecules such as aggrecan, collagen II, and collagen IX have been shown to cause disc degeneration in transgenic mice. Disc degeneration has also been linked to gene mutations other than thosethat code for structural matrix macromolecules [42].

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COMPETING INTEREST

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