Original article

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Surgical treatment of degenerative stenotic lesions of the lumbar spine. Own experience of using minimally invasive techniques

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Lumbar spinal stenosis is a multifactorial progressive condition mainly affecting older individuals, characterized by narrowing of the natural anatomical pathways passage of nerve structures, resulting in typical clinical symptoms. The disease affects about 103 million people in worldwide with an incidence of absolute lumbar spinal stenosis 19.4% among individuals aged 60-69 years. In the United States, about 600,000 surgical interventions for lumbar spinal stenosis are performed annually.

This disease is one of the most common causes of lumbar pain and lower limbs in the elderly individuals, accompanied by claudication and ultimately leading to disability. The main cause of clinical symptoms of the lumbar spinal stenosis is the discrepancy between the sizes of nerve structures and their osteofibrous sheaths, resulting from gradually developing degenerative-dystrophic changes.

Objective: To compare the outcomes of surgical treatment of patients with degenerative stenotic lesions of the lumbar spine using open and minimally invasive methods.

Materials and methods. The results of surgical treatment of 97 patients with lumbar spinal stenosis aged from 28 to 81 years on the basis of the Department of Minimally Invasive and Laser Spinal Neurosurgery of the State Institution " A.P. Romodanov Neurosurgery Institute, Ukraine", " Neurosurgical Department №20 of Vinnytsia Regional Clinical Psychoneurological Hospital named after Acad. O.I. Yushchenko" of Vinnytsia Regional Council and "Spinex" Medical Center following all clinical research protocols. Patients were divided into four groups depending on the surgical intervention method.

Results. The development of concepts regarding the mechanisms of occurrence and treatment methods of lumbar spinal stenosis are highlighted, as well as personal experience of using minimally invasive treatment techniques for this pathology in combination with the use of the ERAS (Enhanced Recovery After Surgery) protocol of perioperative patient management. It was found that the duration of hospital stay for patients with lumbar spinal stenosis statistically significantly desreased when using minimally invasive decompression ($p \leq 0.05$) and the ERAS rehabilitation protocol ($p \leq 0.05$). When comparing the average length of stay in the hospital of patients of the four groups, a statistically significant ($p \leq 0.05$) shorter length of stay in the hospital was observed for patients who underwent minimally invasive decompression using the ERAS rehabilitation protocol. The greatest reduction in pain intensity (according to the Numeric Pain Scale (NPS)) at 6 months post- intervention and rehabilitation was also noted in patients of this group.

Conclusions. The use of minimally invasive techniques, the correct choice of surgical procedure volume (interbody fusion is desirable in surgery for degenerative spinal diseases) combined with Enhanced Recovery after Surgery protocol (ERAS) significantly improves postoperative well-being of patients, accelerates patient mobilization, and reduces the length of stay in the hospital.

Key words: *lumbar spinal stenosis; spondylolisthesis; stabilization; minimally invasive stabilization*

Relevance

Lumbar spinal stenosis (LSS) is a multifactorial progressive condition that predominantly affects elderly individuals and is characterized by narrowing of the natural anatomical pathways of nerve structures, leading to typical clinical symptoms. The condition affects about 103 million people worldwide. In the United States, about 600,000 surgical interventions for lumbar spinal stenosis are performed annually [1].

This disease is one of the most common causes of lumbar pain and lower limbs in the elderly individuals, accompanied by claudication and ultimately leading to disability [2]. Facet joint deformity, hypertrophy of the ligamentum flavum, intervertebral disc degeneration, and osteophytes lead to narrowing of the spinal canal, resulting in compression of the spinal cord and nerve roots [3].

A cross-sectional observational study [4] showed that the prevalence of acquired relative LSS is 22.5 cases per 100 adults, while absolute stenosis is 7.3 cases per 100 adults. Among individuals aged 60–69 years, relative and absolute LSS occurs with a frequency of 47.2 and 19.4%, respectively. A population-based study conducted in Japan [5] showed that the incidence rate of LSS increases with age: 1.7–2.2% – among the population aged 40–49 years and 10.3–11.2% – among the population aged 70-79 years old. Another study found that the frequency of symptomatic LSS is about 10.0% [6].

The main reason for the manifestation of clinical symptoms of LSS is the discrepancy between the sizes of nerve structures and their osteofibrous sheaths, resulting from gradually progressive degenerative-dystrophic changes (hypertrophy of the posterior longitudinal ligament, ossification of intervertebral disc protrusions, osteophytes, hypertrophy of the facet joints, hypertrophy of ligamentum flavum).

Diagnosis is typically based on the evaluation of the patient's clinical history of back and lower limb pain, which worsens with lumbar exertion, improves during rest, and is confirmed by imaging methods such as spiral computed tomography (CT) or magnetic resonance imaging (MRI).

Historical Background

The symptoms of LSS were first described in 1803 by the French pathologist Antoine Portal. He was also the first to report on the spinal canal stenosis caused by curvature of the spinal column [7]. The main cause of scoliotic deformities were rickets and sexually transmitted diseases. In one of the observations of this researcher, the lumen of spinal canal was narrowed by half. He noted the development of weakness, muscle atrophy, and even paralysis of the lower limbs in some patients.

Possibly, LSS was described as early as the 1880s, but the modern description dates back to 1949 when H. Verbiest (Netherlands) reported on a "particular form of lumbar spinal canal narrowing, not associated with any other spinal anomaly. These patients exhibited signs of cauda equina syndrome during walking and standing: bilateral radicular pain, sensory and motor disturbances in the legs. When the patient was in the supine position, the symptoms immediately disappeared, and there were no neurological abnormalities during rest. Myelography revealed block with the appearance of extradural compression" [8]. Lumbar spinal canal stenosis is now defined as "a clinical syndrome of buttock or lower extremity pain, with or without back pain, associated with reduced space available for nerve and vascular elements in the lumbar spine," and specific characteristic provocative signs.

In 1893, W. A. Lane in the UK first performed decompressive laminectomy for cauda equina syndrome due to LSS [9].

As early as 1982, it was found that conservative treatment approach with traction and bed rest was rarely successful in patients with symptomatic LSS [10]. Furthermore, if the spine is supported in extension with a lumbar support or hyperextended in a relaxed state under anesthesia, there is a risk of further cauda equina damage. For many patients, the period between the onset of symptoms and surgery was 5 years or more. The condition of others worsened due to poorly performed myelography or limited median laminectomy [11].

In the 1980s, the best surgical treatment option was partial facetectomy with decompression of the spinal canal by removing hypertrophied bony foramina, ligamentum flavum, and disc sequestration [12]. The surgery had an additional advantage of allowing visualization of venous plexus compression and observation of the restored blood flow of these vessels after surgery. It was noted that the success rate of surgical treatment was maintained even in the presence of adhesive arachnoiditis, if decompression was performed radically enough, and repeated operations using this approach may also be beneficial.

In 2005, in patients with preoperative degenerative spondylolisthesis, scoliosis, or kyphosis, as well as the development of stenosis in a previously decompressed segment, it was recommended that stabilization surgery should be considered [15].

According to a large meta-analysis conducted in the United States, from 2002 to 2007, the frequency of lumbar spinal fusion surgeries sharply increased. Although the number of operated patients with spinal stenosis remains constant, the frequency of stabilization has increased 15 times - from 1.3 to 19.9 per 100,000 population [13]. By 2011, the average rate of spinal fusion for lumbar spinal canal stenosis in the USA had increased to 41.1 per 100,000 population [14]. Such a difference arises from the lack of consensus among surgeons regarding the indications for surgery and evidence that decision-making in practice often depends on surgeons' preferences and enthusiasm rather than patient characteristics [16].

Lumbar spinal canal stenosis is distinguished into stable stenosis (due to hypertrophy of the ligamentum flavum and facet joints, degeneration, and protrusion of the intervertebral disc, ***Fig. 1***) and unstable stenosis, combining these pathologies with instability due to degenerative spondylolisthesis, scoliosis, etc. ***(Fig. 2)***.

This article contains some figures that are displayed in color online but in black and white in the print edition.

According to localization, the following are distinguished:

1) stenosis of the central canal:

a) hypertrophy of the ligamentous apparatus;

b) hypertrophy of facet joints;

c) congenital narrow canal ("short pedicle syndrome");

d) intervertebral discs protrusion;

e) osteophytes originating from the posterior surface of the vertebral body;

f) spondylolisthesis;

2) lateral recess stenosis;

3) foraminal stenosis:

a) protrusion/hernia of the intervertebral foramen;

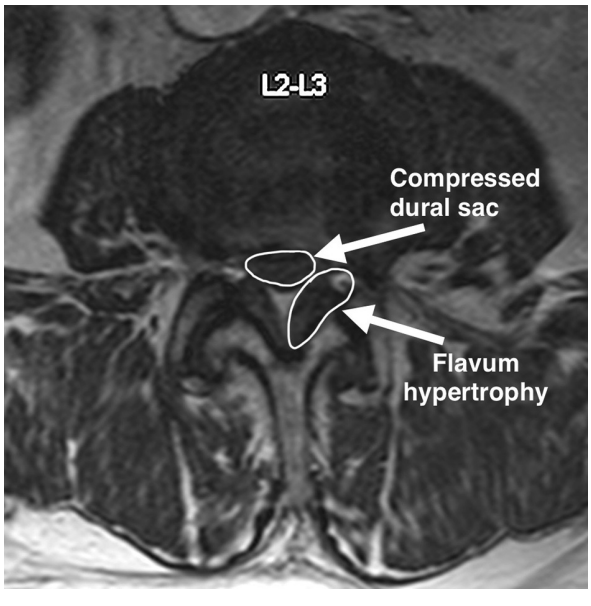

Fig. 1. MRI. Stenotic segment on axial section

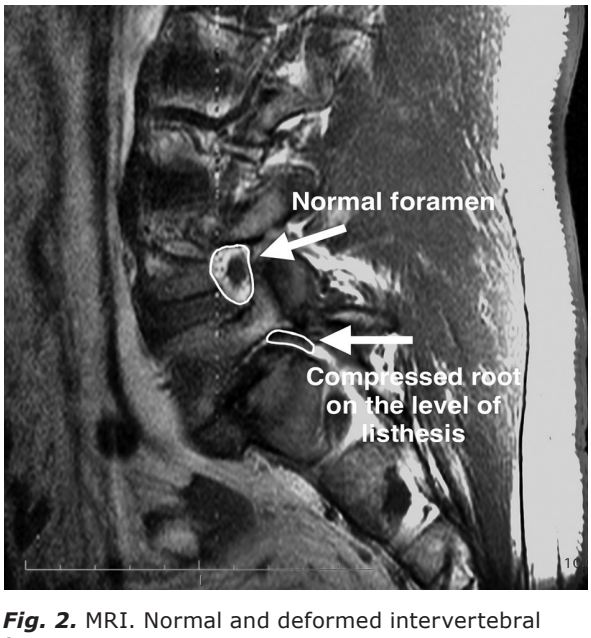

Fig. 2. MRI. Normal and deformed intervertebral foramen

b) spondylolisthesis;

c) hypertrophy of the facet joint;

d) reduction in the height of the disc space;

e) synovial cyst of the facet joint.

Depending on the localization of the compression factor, the clinical presentation of the disease will vary.

According to etiology, LSS is divided into:

1) congenital:

a) idiopathic;

b) achondroplastic;

2) acquired:

a) degenerative;

b) post-traumatic;

c) caused by additional formations of the spinal canal.

Congenital stenosis, primary and relatively rare, affects younger patients (30–50 years old). The narrowing of the spinal canal occurs due to dysplasia of bone structures. Imaging reveals a reduction in the length of the pedicle and the cross-sectional area of the spinal canal. The exact etiology is unknown, but it is often associated with achondroplasia (achondroplastic variant) [17]. This condition is easier to diagnose because patients are younger and usually do not have other medical problems, such as diabetes mellitus or vascular insufficiency. Stenosis can develop at multiple levels of the spinal column and often leads to serious neurological deficits.

Patients with congenital LSS are often asymptomatic in childhood. The shorter pedicle length in cases of congenital LSS is associated with a smaller diameter of the anteroposterior canal, which may lead to earlier clinical manifestations. In middle-aged individuals, in addition to the combination with degenerative changes in bone and soft tissues, signs such as a decrease in anteroposterior and lateral diameter of the spinal canal and foraminal stenosis are detected. Due to altered biomechanical effects in these cases, degenerative changes appear earlier than signs of acquired LSS and clinically manifest at the age of 4-5 decades.

Achondroplasia is an autosomal dominant genetic disorder characterized by abnormal functioning of the fibroblast growth factor receptor-3 *(FGFR3*) gene and in some populations manifests as a *de novo* mutation [18]. The impact on intracartilaginous ossification during spinal embryonic development leads to the formation of anatomical anomalies, such as thoracolumbar kyphosis, shortening of the pedicles, and decreased interpedicular distance in the craniocaudal direction, which progresses and leads to the formation of a narrowed spinal canal [19]. Other anomalies, such as underdeveloped and narrow sacra, are also possible. The iliac wings are relatively higher, and the L5 vertebra is deeply embedded (below the iliac wings) [4, 5]. These changes may cause spinal canal stenosis in patients with achondroplasia, so symptoms appear earlier than in the general population, at the age of 3-4 decades [6]. Some of the most common symptoms include intermittent claudication, nerve roots compression and paraparesis depending on the level of stenosis. Screening of the entire spine is recommended for patients with achondroplasia, as a combination of

lumbar, cervical, thoracic, and thoracolumbar spinal stenosis is most common.

Another genetically determined cause of stenosis is ligamentum flavum ossification, a rare disease more commonly found in Asian and Caucasian populations [20]. The conducted studies confirmed that the development of ossified ligamentum flavum syndrome is primarily due to endochondral ossification, which is also controlled by the *FGFR3* gene [21]. The presence of this anomaly complicates the technique of surgical decompression and more often leads to intraoperative damage to the dura mater during surgery [22]. The gold standard treatment for LSS is early decompression at the pathology level, which can prevent complications such as accidental durotomy and spinal cord injury during decompression.

Acquired stenosis results from a cascade of changes initiated by degeneration of the nucleus pulposus of the intervertebral disc with age, thus more commonly manifesting at the age of 50 and older. Degeneration and atrophy of the stabilizing axial musculature, repeated trauma to the axial spine due to daily wear and tear, and potential occupational exposures lead to dehydration of the nucleus pulposus and collapse of the disc space. This process can be enhanced by weakness or degeneration of the axial muscles, especially in combination with fatty infiltration of the muscles, and excessive body weight [23]. Disc space collapse reduces the segment height, often combined with a local kyphotic deformity, altering the sagittal balance, shifting the axial stress towards the posterior elements (facet joints, interspinous, ligamentum flavum and subarticular ligament). Chronic excessive stress leads to joint hypertrophy, accompanied by the appearance of synovial cysts and the osteophyte formation, as well as curvature and thickening of the posterior longitudinal ligament. The combination of these factors entails spinal canal narrowing. Thus, the central stenosis results from intervertebral disc collapse, lateral stenosis results from facet joint and subarticular ligament proliferation, and is complemented posteriorly by ligamentum flavum thickening. Degenerative spondylolisthesis - vertebral body displacement with/without a bone tissue defect may also be an important factor in the development of LSS [24].

Obviously, apart from acquired degeneration that can cause LSS, genetic factors play an important role in the development of LSS and may significantly influence the clinical course. Genetic predisposition may explain the difference in prevalence estimates of moderate (24 and 78%) and severe (8 and 30%) stenosis in individuals over 40 years of age in different populations [25]. In such cases, patients have degenerative changes that, against the background of a congenital narrow canal, lead to complex LSS due to the combination with other spinal deformities, such as spondylolisthesis, scoliosis, or lumbar kyphosis.

Central canal stenosis is associated primarily with axial back pain and neurogenic claudication, motor or sensory radicular symptoms are possible. Pain associated with central canal stenosis is usually bilateral, with lumbar levels L4–L5 most commonly affected, followed by L3–L4 and L5–S1. There are two theories explaining the mechanism by which central stenosis leads to neurogenic claudication. According to the ischemic theory, compression leads to decreased blood flow to the nerve roots, resulting in ischemic pain and weakness. The venous stasis theory suggests that venous blood stasis leads to inadequate oxygenation of the capillary bed, accumulation of metabolites in the cauda equina, and subsequent onset of pain and claudication [26]. Lateral recess stenosis and foraminal stenosis can be unilateral and cause compression of the nerve root in the subarticular recess and intervertebral foramen, respectively. With stenosis of the lateral recess, compression of the segment of the nerve root passing through it occurs due to the facet joint and hypertrophy of the subarticular ligament. Foraminal stenosis can be caused by scoliosis, lateral or foraminal disc herniation, or synovial cyst of the facet joint, which may compress a nerve or sensory ganglion [27]. Such compression leads to unilateral radiculopathy with pain and possibly weakness in the corresponding myotome.

The diagnosis of LSS is usually made on the basis of a combination of clinical symptoms and the presence of stenosis with dural sac compression visualized during an X-ray examination. A systematic review of E.I. de Schepper et al. found that the most specific and sensitive clinical manifestation is pain in the lower limbs, which radiates and worsens during standing [28]. Bilateral buttock or leg pain that resolves when sitting or bending forward and a wide-based gait were also found to be sufficiently sensitive and specific symptoms, whereas more well-defined clinical signs (positive straight leg raising test (Lasègue's sign)) had less diagnostic value.

An international Delphi study proposed a set of seven core items in the patient's history to help professionals identify LSS with increased accuracy in both clinical and research settings [29].

Differential diagnosis between neurogenic and vascular claudication is crucial. If in the first case relief occurs when bending forward and sitting, then in the second one - when the affected limb is at rest. Examination of the peripheral vessels often reveals the absence of a pulse on the dorsal surface of the foot with a positive Burger's test result. Arterial imaging (duplex scan) is performed if necessary. However, sometimes a combination of vascular and neurogenic pathology may be observed.

In the era of computed tomography, the cross-sectional area of the thecal sac has become the reference measurement. N. Schönström et al. during studies conducted on cadavers, pressure changes during cauda equina compression were evaluated and the cross-sectional area of the thecal sac was determined to be <75 mm^2 and <100 mm^2 for absolute and relative LSS, respectively [30]. Several additional indices and classification systems have been proposed, but they are too time-consuming to calculate and correlate poorly with clinical manifestations of the disease [31]. In general, there is a low correlation between the reduction in the area of the spinal canal that can be visualized by instrumental examination and the patient's clinical symptoms. That is why imaging alone is insufficient for

diagnosis, its data must be compared with the patient's symptoms and history [32].

Usually, MRI is the preferred diagnostic imaging tool due to its high resolution for soft tissues. If there are contraindications to MRI, then SCT is performed, but this technique is associated with ionizing radiation, and provides poorer visualization of soft tissue structures. Sometimes a combination of both methods is necessary to make a well-informed decision, especially in patients with a long history of the disease, as SCT allows the assessment of the degree of ossification of pathological tissues and the selection of a surgical approach with the optimal angle of attack.

Electrodiagnostic testing (electroneuromyography) is not recommended as a routine screening of patients with suspected LSS, but in individuals with an atypical course, inconclusive imaging, or in case of doubt regarding mixed etiology (lumbar plexopathies, peripheral nerve injury syndrome, intermittent claudication, or metabolic neuropathies), these tests may be useful in combination with clinical examination [33].

Another important diagnostic method is functional radiography, which allows the choice of surgical strategy (decompression or decompression-stabilization surgery) in complex cases, to detect dynamic spondylolisthesis or segment instability due to kyphotic deformity, which is amplified by maximum forward bending. Complementing the diagnostic data with the results of functional radiography makes it possible to significantly reduce the frequency of repeated surgical interventions in LSS of various etiologies.

Treatment options for LSS vary considerably: medication, exercise, physical therapy, transcutaneous electrical nerve stimulation (TENS), epidural steroid injections, and surgical decompression. The choice of tactics usually depends on evidence-based medicine recommendations, individual patient characteristics and patient preferences.

So far, there is no evidence level C or higher regarding the efficacy of conservative treatments. A systematic review published in 2013 noted that the available evidence is insufficient to provide formal recommendations for clinical practice [34]. However, some patients report both short-term and long-term symptomatic relief after conservative treatment.

Surgical decompression is generally indicated in patients with moderate to severe disease, with persistent progressive deterioration, or those in whom conservative treatments have not been effective. Open surgical treatment of lumbar spinal stenosis has been standard practice for decades, and recently several minimally invasive treatment options have expanded the available surgical treatment options [35]. Most importantly, these minimally invasive options are supported by prospective randomized trials [36].

A systematic review of the literature showed that delaying surgical intervention while undergoing conservative treatment is not associated with worse future outcomes. It is also noted that surgical intervention is more effective than continuing conservative treatment if conservative options do not yield results within 3–6 months [37].

There are various surgical approaches to treating LSS (open, minimally invasive and endoscopic procedures). Currently, there are no recommendations with a sufficient level of evidence for choosing an approach in specific cases or for a particular category of patients. The best option for surgical intervention is chosen based on the anatomical location of the stenosis, the number of involved levels, involvement of the thoracolumbar transition, presence of abnormal anatomy, instability or deformity. The goal of the approaches is to decompress compromised neural elements, provide symptomatic relief, and prevent further degeneration so as not to destabilize the spine [38].

A series of randomized trials have been conducted to evaluate the effectiveness of surgical decompression and conservative treatment. Spine Patient Outcomes Research Trial (SPORT) is the largest study comparing standard posterior decompressive laminectomy with conservative treatment in patients with LSS without spondylolisthesis. It was found that surgical intervention was significantly more effective in reducing pain syndrome. A difference in decreased pain and improved function after 2 years of surgical treatment was also found [39]. Four years later, the authors published additional data confirming sustained functional improvement and reduction in pain syndrome after the application of surgical treatment methods [40].

The role of spinal fusion combined with decompression is controversial. In the 1990s, two studies showed that patients with LSS and degenerative spondylolisthesis had better outcomes when decompression was combined with spondylodesis [41, 42]. Subsequently, decompression and spondylodesis became standard practice for LSS with degenerative spondylolisthesis, and the incidence of lumbar spondylodesis surgery increased significantly [47]. However, a large cohort study (5390 patients) published in 2013 found no difference in satisfaction with treatment outcomes using spinal fusion compared to decompression alone [43].

Two randomized clinical trials with controversial results were published in 2016. A large Swedish cohort randomized clinical trial of spinal stenosis comparing treatment outcomes using decompression combined with spondylodesis and decompression alone. No significant difference in clinical outcomes or reoperation rates between the two groups after 2 and 5 years is observed regardless of the presence of degenerative spondylolisthesis [44]. Similar results were obtained in the registry study of three Scandinavian countries [45]. However, in the randomized clinical trial of Spinal Laminectomy versus Instrumented Pedicle Screw (SLIP) involving patients with degenerative spondylolisthesis and LSS, improved physical health, quality of life, and lower rates of reoperation after decompression combined with spondylodesis compared to decompression alone [46].

Objective: To compare the outcomes of surgical treatment of patients with degenerative stenotic lesions of the lumbar spine using open and minimally invasive methods.

Materials and methods. The study was carried out on the basis of the Department of Minimally Invasive and Laser Spinal Neurosurgery of the State Institution "A.P. Romodanov Neurosurgery Institute, Ukraine", Neurosurgical Department №20 of "Vinnytsia Regional Clinical Psychoneurological Hospital named after Acad. O.I. Yushchenko" of Vinnytsia Regional Council and "Spinex" Medical Center.

Study participants

In 2020–2024, a comprehensive in-depth examination was conducted involving 97 individuals aged 28 to 81 years old.

For detailed patient assessment, the e-form "Medical record of in-patient" (form No. 003/o) and the "Individual patient examination card" developed by us, in which passport details, general, hereditary and allergic history, complaints, disease course characteristics, as well as the results of clinical laboratory and clinical investigations.

When conducting the study, ethical principles of scientific research involving human subjects (Declaration of Helsinki) and recommendations for good clinical practice were followed. The study design was approved by the Ethics Committee of the State Institution "Institute of Neurosurgery named after Acad. A. P. Romodanov of the National Academy of Sciences of Ukraine" (Minutes No. 3 dated December 16, 2020).

Inclusion and exclusion criteria

According to the chosen study design, each patient must meet all inclusion criteria and have no exclusion criteria.

Inclusion criteria:

- lumbar spinal canal stenosis;
- informed consent of the patient to cooperate with the researcher within the study framework.

Exclusion criteria:

- stage 3 heart failure;
- acute thrombosis of the veins of lower extremities;
- thromboembolism of pulmonary artery branches;
- the presence of chronic foci of infection;
- septic condition;
- acute cerebrovascular accident with severe neurological disorders;
- presence of psychopathology rendering surgical intervention impossible.

When included in the study, each patient received an informed consent form for participation. Bioethical examination was conducted by a local independent ethics committee.

Scientific sources cited in Scopus, WoS, Google scholar, etc., using databases such as UpToDate, PubMed, etc., were analyzed.

Study design

The presented work is a prospective cohort study. Diagnosis and treatment were performed according to the guidelines of the Ministry of Health of Ukraine "00436. Stenosis of the spinal canal in the lumbar region" dated June 30, 2017 (https://guidelines.moz.gov.ua/documents/3293). Preliminary order No. 317 dated June 13, 2008 of the Ministry of Health of Ukraine "On the approval of clinical protocols for the provision of medical care in the specialty "Neurosurgery" became invalid on September 1, 2023.

Patients were divided into four groups: group I – patients with LSS who underwent open decompression without using of ERAS rehabilitation protocol, group II – patients with LSS who underwent open decompression with the use of ERAS (Enhanced Recovery After Surgery) rehabilitation protocol, group III – patients with LSS who underwent minimally invasive decompression without using of ERAS rehabilitation protocol, group IV - patients with LSS who underwent minimally invasive decompression with the use of ERAS rehabilitation protocol. A comparison of treatment outcomes in the groups was carried out, determining the average length of hospital stay and pain syndrome assessment before and after treatment using a Numeric Pain Scale (NPS).

Statistical analysis

Statistical processing of the obtained results was performed using the IBM SPSS Statistics program, version 12 (20) (license number 9593869, belonging to the Department of Infectious Diseases of Vinnytsia National Pirogov Memorial Medical University, Ministry of Health of Ukraine) using parametric and non-parametric methods of evaluating the obtained results. Arithmetic mean (M) and standard error (m) were calculated. In the case of qualitative signs, the frequency of manifestation (%) and its standard error (m%) were calculated. Checking the distribution for compliance with the Gauss's law was performed using the Shapiro–Wilk test. Reliability of the difference between independent quantitative values in case of normal distribution was determined using the Student's test for independent values, for data presented in percentages - using the Fisher's exact test, in other cases - using the Mann-Whitney U-test. Results were considered statistically significant at a statistical significance level $p<0.05$.

Results and discussion

The average age of the examined patients was (53.32 ± 3.39) years. 40 (41.2%) men and 57 (58.8%) women participated in the study.

The length of hospital stay of patients with LSS was studied and comparisons were made among the study groups. It was found that the length of hospital stay was statistically significantly decreased when using minimally invasive decompression ***(Table 1).***

Surgical decompression is usually indicated for patients with a moderate to severe course of the disease, persistent deterioration of condition, or ineffectiveness of conservative treatment methods. Open surgical treatment of LSS has been a standard practice for decades. Recently, several minimally invasive treatment options have added to the surgical treatment arsenal [35]. The effectiveness of these options has been confirmed in prospective randomized studies. Proper patient selection for new treatment options is of paramount importance [36].

A statistically significant decrease in the length of hospital stay in patients with LSS when using ERAS rehabilitation protocol has also been recorded ***(Table 2).***

Over the past 70 years, the treatment approach for this condition has evolved from traction and immobilization

to minimally invasive surgical interventions, allowing patient mobilization on the day of surgery and reducing hospital stay to 2-3 days. The combination of minimally invasive techniques with a modern Enhanced Recovery After Surgery (ERAS) protocol holds great promise, considering the significant reduction in treatment costs, faster rehabilitation, and long-term treatment outcomes comparable to those following traditional "major" decompressive surgeries.

When comparing the average length of hospital stay among patient groups, a statistically significant reduction in this indicator was noted among patients in Group IV ***(Table 3).***

Before surgical intervention, the average score on the NPS scale in the groups was (9.23±1.19) points. Six months after surgical treatment, the score of group IV was statistically significantly different compared to the others, especially compared to group I ***(Table 4)***. Thus, performing surgical treatment using minimally invasive decompression in combination with the ERAS rehabilitation protocol contributes to better reduction of the pain syndrome compared to the open method.

Table 1. The average length of hospital stay of patients with LSS depending on the type of intervention (M ± m)

Intervention	Length, bed-day
Open decompression (n=63)	6,51±1,65*
Minimally invasive decompression (n=34)	4,5±1,74

Note. * - The difference is statistically significant (p ≤ 0.05) compared to the index of patients who underwent minimally invasive decompression.

Table 2. The average length of hospital stay in patients with LSS depending on the use of ERAS protocol (M ± m)

Group of patients	Length, bed-day
ERAS rehabilitation protocol was not used (n=26)	9,20±4,13*
ERAS rehabilitation protocol was used (n=71)	4,6±1,13

Note. * - The difference is statistically significant (p ≤ 0.05) compared to the rate of patients who were treated with the ERAS rehabilitation protocol.

Table 3. Comparison of the average length of hospital stay in patients with LSS (M±m)

Group of patients	Length, bed-day
I (n=18)	9,11±1,28
II (n=45)	6,83±1,41
III (n=8)	5,52±1,20
IV (n=26)	4,40±1,12*

Note. * – The difference is statistically significant (p ≤ 0.05) compared to the rate of patients of the I group.

Table 4. Severity of pain syndrome according to the NPS scale in patients with LSS before and after surgical treatment (M±m)

Group of patients	NPS score, point	
	Before surgical treatment	After surgical treatment
I (n=18)	9,12±1,23	5,2±0,89
II (n=45)	9,38±1,02	4,1±1,12
III (n=8)	9,25±1,10	3,4±1,15
IV (n=26)	9,43±1,18	2,0±0,91*.**

Note. The difference is statistically significant (p ≤ 0.05) compared to the index of: * - patients of group I; ** - patients of group IV before surgery.

We present a case report.

A patient born in 1957 presented with complaints of lower back pain radiating along the posterior surface of both lower limbs, weakness in both lower limbs, predominantly on the right side. The complaints had been bothering him for years, but a significant exacerbation occurred 2 months prior to presentation. Conservative treatment proved to be ineffective.

MRI and CT scans of the lumbar spine were performed, revealing degenerative spondylolisthesis at the L5-S1 level with complete collapse of the intervertebral disc and significant foraminal stenosis, predominantly on the right side ***(Fig. 3)***. The presence of this pathology warranted decompressive-stabilizing neurosurgical intervention.

The surgery included: minimally invasive bilateral facetectomy L5–S1, discectomy, open reduction of spondylolisthesis, bilateral TLIF, and transpedicular spinal fusion L5–S1. The duration of surgery was 3 hours and 15 minutes. Two paravertebral incisions approximately 3 cm in length were made ***(Fig. 4)***, with a blood loss volume of 200 ml.

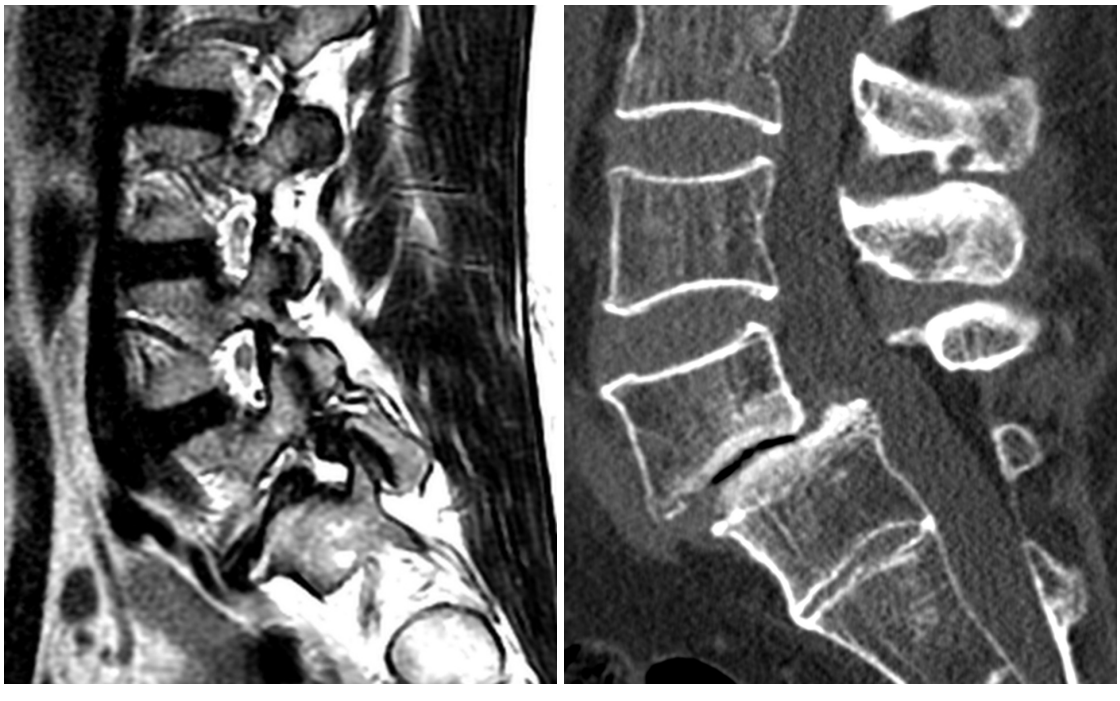

Fig. 3. Preoperative MRI and CT scans of the lumbar spine

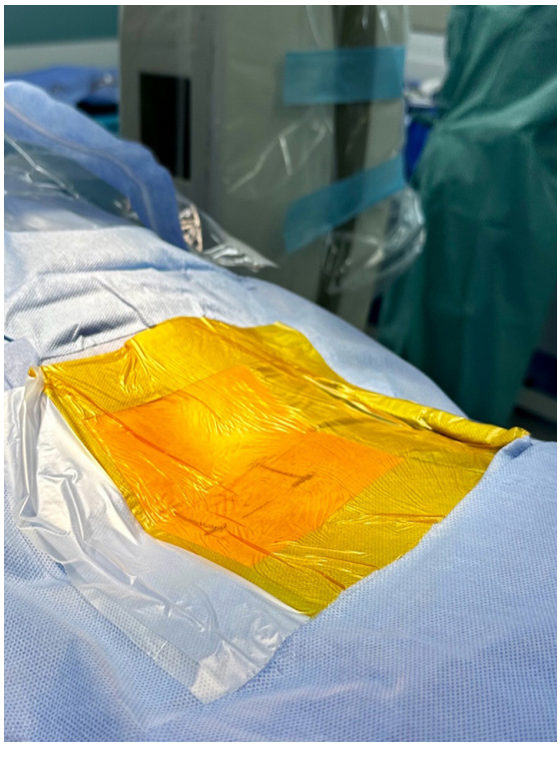

Fig. 4. Marking of the surgical field

During the perioperative period, a standard ERAS protocol was employed. To prevent postoperative complications, the patient was "warmed" with a ventilated blanket ***(Fig. 5).*** He was mobilized on the day of surgery.

The patient was discharged on the 3rd day after surgery. By the time of discharge, there was complete regression of pain in the lower limbs. Upon admission, the pain score on the NPS scale was 8 points, and the quality of life impairment according to the Oswestry questionnaire was 55.0%. At the time of discharge, the patient is fully mobilized, capable of self-care, can move independently, climb stairs without assistance and aids.

The control examination was carried out 5 months after the surgical intervention. Assessment of pain syndrome according to the NPS scale - 2 points, impaired quality of life according to the Oswestry questionnaire - 17.7%. Assessment of fusion and postoperative wound healing was performed ***(Figs. 6 and 7).***

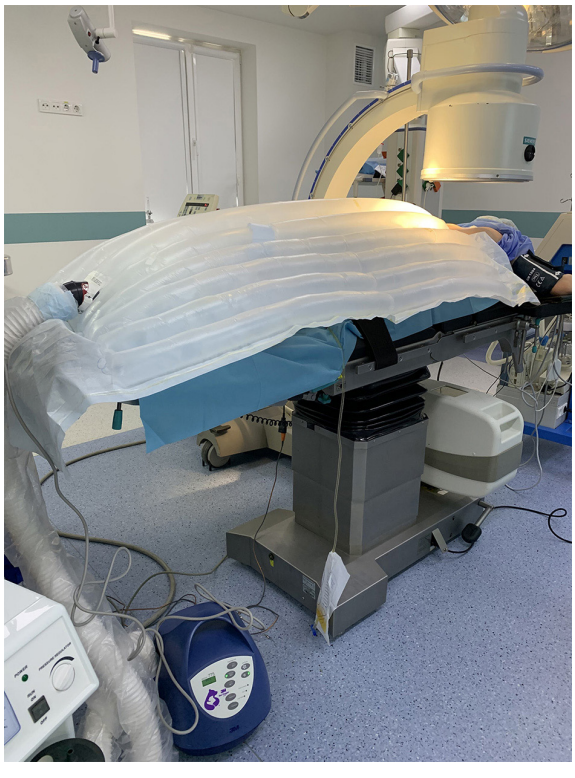

Fig. 5. "Warming" the patient with a ventilated blanket

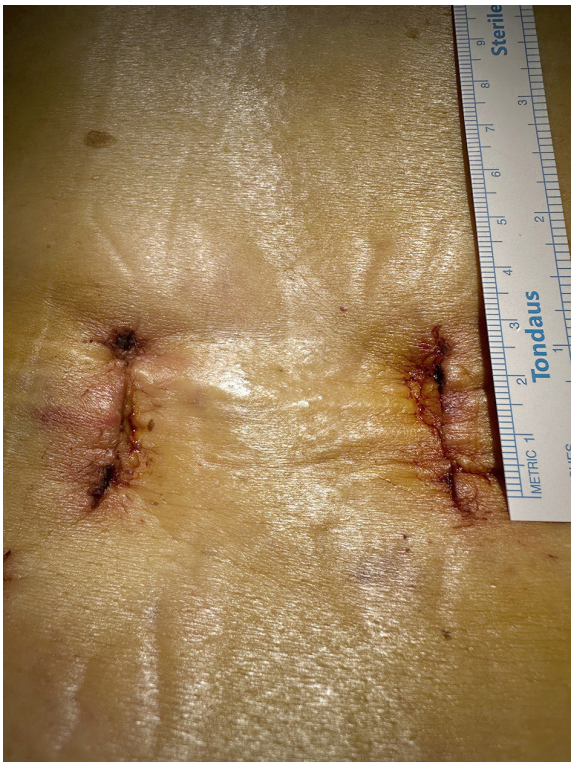

Fig. 6. Postoperative wound condition

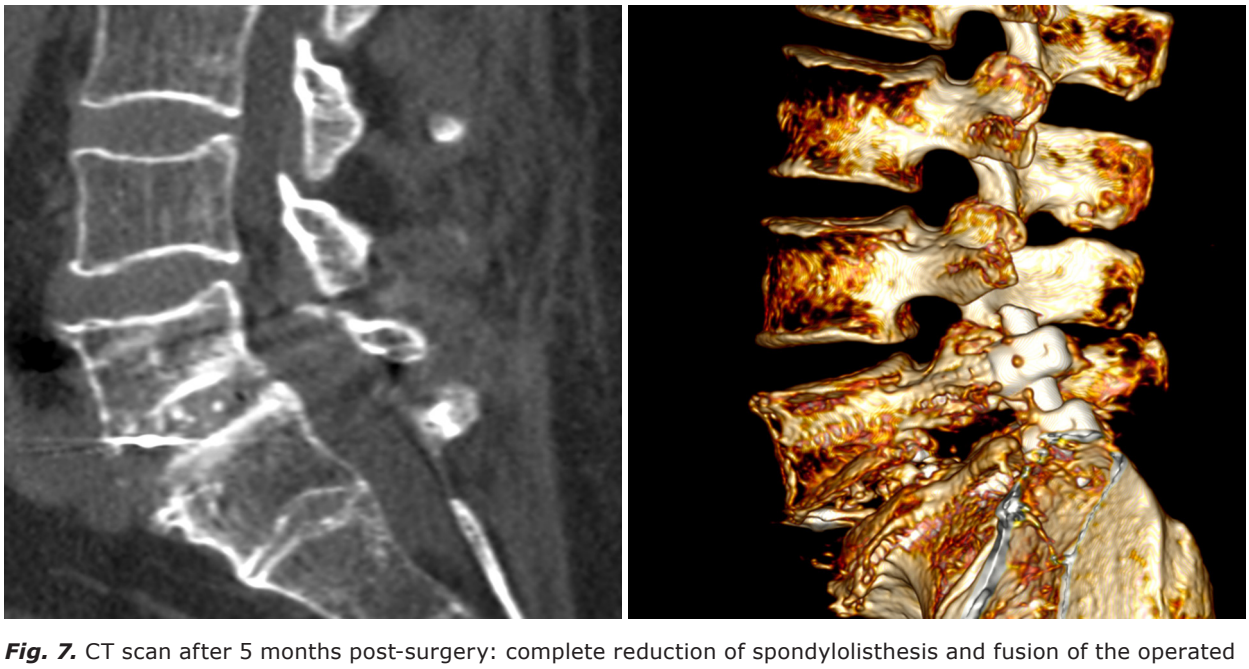

Fig. 7. CT scan after 5 months post-surgery: complete reduction of spondylolisthesis and fusion of the operated segment

Conclusions

The use of minimally invasive techniques, appropriate surgical procedure selection (interbody fusion is preferable in degenerative spinal surgery), combined with an Enhanced Recovery After Surgery (ERAS) protocol significantly improves postoperative well-being of patients, accelerates their mobilization, and reduces hospital stay.

Given the insufficient data on the application of minimally invasive surgical methods, it is relevant to thoroughly study and compare them with conventional techniques, as they allow for reducing hospital stay, lowering treatment costs, and mitigating potential consequences and disability factors.

Disclosure

Conflict of interest

The authors declare no conflict of interest and no personal financial interest in the preparation of the article.

Ethical approval

All procedures performed on patients comply with the ethical standards of institutional and national ethics committees, the 1964 Declaration of Helsinki and its amendments or similar ethical standards.

Informed consent

Informed consent for the study and publication of data and any accompanying images was obtained from each of the patients.

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