FEATURES OF THE COURSE OF LUMBAR SPINAL STENOSIS

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https://doi.org/10.5281/zenodo.15111690

ARTICLE INFO

Qabul qilindi: 25-Mart 2025 yil
Ma'qullandi: 28-Mart 2025 yil
Nashr qilindi: 31-Mart 2025 yil

KEYWORDS

lumbar spinal stenosis, lumbosacral instability, stenosis surgical treatment with the imposition of the stabilizing systems.

ABSTRACT

Lumbar spinal stenosis (LSS) is the narrowing of the central spinal canal (central stenosis) or of the concave and intervertebral lateral foramens. LSS is one of the most common causes of back pain in the elderly. Clinical signs of the disease are localized lumbar muscle pain and naughty - radical symptoms that occur when walking. A diagnosis of LSS is most likely to reveal paralysis during walking and stopping, regression during advance or sitting in elderly patients. Treatment strategies are determined by clinical conditions, not by changes identified in imaging studies. The most important diagnostic criterion is walking distance. Treatment strategies for patients with LSS are not fully established, as there is insufficient evidence that different treatment options work.

Lumbar stenosis (PS) - the narrowing of the central vertebral canal or the lateral sections of the vertebral canal and the interphonic hole - is one of the most common causes of lumbar pain in the elderly [1, 2]. The frequency of PS development in the elderly is about 10% [3], while the exact frequency of clinically significant PS is unknown.

The following forms of PS are distinguished: 1) idiopathic - achondroplastic, or innate; (2) acquired, including degenerative, as a result of spondylolisthesia, post-traumatic, combined, iatrogenic or other causes [4, 5].

Clinical manifestations of PS occur in people over 50 years of age in the presence of degenerative changes in the spine. The role of congenital narrowing of the vertebral canal cannot be accurately evaluated, so most authors consider the different variants of combined PS as a consequence mainly deg.

There is central PS, in which the central canal of the spine is narrowed, and lateral PS - narrowing of the lateral pocket or intervertebral opening (the so-called foraminal stenosis). Normally, the anteroposterior (sagittal) size of the spinal canal at the lumbar level is 15-25 mm, and in diameter - 26-30 mm. With a narrowing of the anteroposterior size to 10-15 mm, clinical symptoms may appear, but more often they occur with a narrowing of less than 10 mm; a more reliable criterion for PS is a decrease in the cross-sectional area of the spinal canal to less than 100 mm.^{2}[9].

The development of neurological manifestations of PS is caused by compression of the radicular structures, with ischemia occurring as a result of compression of the radicular vessels, especially the veins, being of great importance. The appearance of clinical symptoms during movement and extension and a decrease during flexion of the lumbar spine are caused by the following mechanism: an increase in epidural pressure over 30–40 mm Hg causes compression of the veins and capillaries of the spinal roots. Against the background of PS, during extension and walking, an additional narrowing of the spinal canal occurs, the epidural pressure increases to over 80 mm Hg; flexion leads to a decrease in pressure to 15–18 mm Hg and the elimination of venous congestion. At the same time, morphological changes develop in the roots, in particular edema, thickening of the arachnoid membrane, degeneration of axons and demyelination of nerve fibers, narrowing and a decrease in the number of intradural vessels [10].

Clinical manifestations and diagnostics of PS PS is characterized by local musculoskeletal pain and claudication - radicular symptoms that occur when walking. Spondylolisthesis often causes spinal deformity in the form of hyperlordosis. Movements in the lumbar region are not limited.

The most striking clinical manifestation of central PS is neurogenic (claudogenic) intermittent claudication. This term is used to describe the phenomenon of claudication: pain, numbness, and weakness in the legs occur when walking a distance of less than 500 m. The pain is usually bilateral, often of the paresthesia type, appears in the lower back, spreading distally, and may first appear in the feet, spreading upward. To reduce the symptoms of claudogenic claudication, the patient must not only stop, but also take a specific pose with flexed legs and forward lean of the body. Claudication can be provoked by prolonged standing and back extension. The disease tends to progress very slowly (in 80% of cases), pelvic disorders develop rarely [1, 2].

To diagnose intermittent claudogenic claudication, a "march test" is used, with an assessment of the appearance of paresthesia, sensory disturbances, decreased reflexes, weakness in the legs, dysfunction of the pelvic organs that occur when walking short distances (up to 500 m), and regression of these symptoms when bending the body forward or in a sitting position. It is possible to use an extension test - the occurrence of neurological manifestations with forced extension of the back [11].

Lateral stenosis has different clinical symptoms and is manifested mainly by monoradicular syndrome. Typical are radicular pains, sometimes in combination with sensitivity disorders in the area of the dermatome corresponding to the root, with paresis in the area of the corresponding myotome and loss of reflexes. Lateral stenosis is characterized by radicular pains that occur at rest and during movement, which distinguishes them from pains caused by herniated intervertebral discs. With PS, pains persist in the supine position, are constant, less often with periodic exacerbations, do not increase with coughing and sneezing, vertebral syndrome is not so pronounced. Lasegue's symptom is not typical for lateral stenosis [6].

Additional diagnostic methods for PS include X-ray spondylography, MRI and/or CT, CT myelography. X-ray imaging can detect spondylolisthesis. Functional tests with maximum flexion and extension are performed to detect spinal instability. CT and MRI can evaluate the parameters of the canal, both bone and soft tissue, and determine their role in the formation

of stenosis. They evaluate the anteroposterior size of the canal (normally more than 11.5 mm), transverse (normally more than 16 mm), as well as the area of the transverse size (at least 145 mm), the thickness of the yellow ligament (normally less than 4–5 mm), the height (anteroposterior size) of the lateral pocket (more than 3 mm). Using the T2 mode, MRI can produce a visual image of stenosis similar to myelography.

In 2013, the North American Spine Society (NASS) clinical practice guideline for the diagnosis and evidence-based treatment of lumbar degenerative stenosis was presented. According to these recommendations, the presence of lumbar degenerative stenosis should be suspected in elderly patients in whom claudications occur during walking and standing, and regress during bending and sitting. There is no generally accepted clinical examination scheme for the diagnosis of lumbar degenerative stenosis. MRI is the best non-invasive method for assessing the size of the spinal canal and the degree of radicular compression, and CT and CT myelography are recommended if there are contraindications to MRI; assessment under axial loading is possible. According to these recommendations, stenosis is considered to be a decrease in area of less than 110 mm^2at one or more levels. Currently, there are no data on significant correlations between structural changes and clinical manifestations. Electroneuromyography of paraspinal muscles can be used as an additional diagnostic method [12, 13].

Claudogenic intermittent claudication in PS should be distinguished from a more life-threatening condition for the patient — intermittent claudication caused by stenotic lesions of the arteries of the lower extremities. A bicycle test can be used for diagnosis: the lumen of the spinal canal becomes larger in the flexion position, which leads to a decrease in intracanal pressure, so when pedaling a bicycle in this position, claudication does not occur with claudogenic claudication, unlike arterial stenosis. More informative non-invasive methods for diagnosing stenosis of the main vessels of the legs are ultrasound and MRI in angio mode. Other pathological situations that imitate the symptoms of claudogenic claudication may be a deep arteriovenous fistula, lesions of the joints of the lower extremities, venous claudication or claudication in myxedema.

Approaches to the treatment of PS: To date, therapeutic strategies for patients with PS have not been fully defined, since there is insufficient convincing evidence of the effectiveness of various treatment options. Treatment tactics are based on existing clinical symptoms, rather than on CT or MRI data. The patient's walking distance is the most significant criterion for the effectiveness of therapy [14, 15].

Treatment of PS involves a comprehensive and individualized approach, including recommendations for lifestyle and physical activity changes, prescription of medications from the NSAID group, anticonvulsants, vasoactive drugs, in particular venotonics, prostaglandin E1, calcitonin, epidural administration of glucocorticosteroids and local anesthetics, and B vitamins.

Modern guidelines for the treatment of chronic lumbar pain recommend the active use of non-drug methods. In the management of patients with subacute and chronic back pain, rational psychotherapy and various types of exercise therapy are of great importance [16]. Currently, the effectiveness of physiotherapeutic methods for the treatment of back pain is being studied, including the effects of infrared radiation and a magnetic field [17–19]. The therapeutic analgesic patch NANOPLAST forte contains two components: rare earth metals

and a powder producing infrared radiation. Thus, NANOPLAST forte combines both of these effects: it affects the affected area with a constant magnetic field and gentle heat. In a prospective comparative randomized placebo-controlled study involving 60 patients with acute/exacerbation of chronic back pain syndrome, N.A. Shostak et al. (2017) found that the use of the NANOPLAST forte patch had a more pronounced analgesic effect compared to the use of placebo [20]. In 2013, a Cochrane systematic review was published, according to which the use of calcitonin in the treatment of patients with intermittent claudogenic claudication was comparable to placebo. Some effectiveness in improving walking parameters was also found with the administration of prostaglandin E1 (low-quality studies), as well as gabapentin and vitamin B_{12}(very low-quality evidence). Epidural glucocorticoids are effective in reducing pain, improving functional status, and improving quality of life in the short term compared with home exercise or physiotherapy. Exercise therapy is effective in the short term in reducing leg pain and disability compared with no treatment [21]. There is some evidence that anticonvulsants are ineffective in intermittent claudogenic claudication, but the review does not provide data on the effectiveness of this group of drugs in the presence of neuropathic pain [22].

According to the 2013 NASS Clinical Guideline, there is insufficient evidence to determine the efficacy of drugs and non-pharmacological interventions for the treatment of clinical manifestations of PS. At the same time, according to the recommendations of the working group experts, a short-term course of active exercise therapy is indicated for patients with PS. For the treatment of claudogenic intermittent claudication, epidural blockades with corticosteroids are effective in the short term (up to 6 months), and the interlaminar method of administration has a higher level of evidence (B) than the sacral and transforaminal approaches (evidence level C). Data on the long-term efficacy of epidural blockades are contradictory. The use of a corset to reduce pain and improve motor activity, in particular walking, in patients with PS is effective (evidence level B). Conservative therapy leads to an improvement in the condition over 2–10 years in many patients [13].

The use of various types of epidural blockades in the treatment of PS is controversial. A systematic review by Manchikanti et al. (2016) demonstrated the effectiveness of epidural blockades with lidocaine or in combination with glucocorticosteroids in the treatment of this category of patients, while epidural administration of other agents, such as bupivaquine or saline, did not show a significant effect [23]. A meta-analysis of 13 randomized controlled trials involving 1465 patients with PS demonstrated the high effectiveness of epidural blockades with local anesthetics and in combination with glucocorticosteroids to reduce pain (by more than 50%), as well as the degree of vital activity determined by the Oswestry scale, and the need to use narcotic analgesics [24]. A recent review showed high efficacy of epidural blocks (level of evidence A) in both short-term relief of PS symptoms and long-term prognosis, while emphasizing that the addition of corticosteroids does not significantly affect treatment outcomes [25].

At the same time, the British clinical guideline (NICE UK 2016) does not recommend the use of epidural blockades for the treatment of claudogenic claudication in PS [26]. More than half of the patients have a favorable prognosis [27] when conservative therapy is prescribed, however, if treatment is ineffective, the neurological deficit progresses, further neurosurgical intervention is suggested, usually laminectomy, sometimes in combination with the use of

fixation systems [2, 8, 28]. At the same time, the data obtained with various types of neurosurgical interventions are quite contradictory. Kovacs et al. (2011) indicate that surgical treatment showed a greater effect compared to conservative therapy in terms of reducing the severity of pain, the degree of impairment of daily activities, improving the quality of life, but did not have a significant effect on walking parameters. The Spine Patient Outcomes Research Trial (SPORT) also demonstrated greater efficacy of surgical procedures for PS and spodylolisthesis than conservative treatment during a 4-year follow-up, with complications or repeated interventions occurring in 13% of cases [28]. A longer follow-up period for patients with moderate to severe PS who underwent decompression surgery showed a better outcome, including in patients over 75 years of age [13]. Several prospective studies have convincingly demonstrated the advantage of neurosurgical intervention in patients with PS over conservative therapy. The frequency of favorable outcomes after surgical intervention ranged from 64 to 85% [6].

At the same time, a Cochrane systematic review [14] showed that to date there is no convincing data on the effectiveness of surgical treatment in relation to pain syndrome and the degree of impairment of daily activities in patients with clinically significant PS. There is also no data on the greater effectiveness of the use of fixation systems than standard decompression, and when using an interspinous fixator, a higher percentage of repeated interventions is observed [14]. In another Cochrane systematic review [17], an analysis of the outcomes of neurosurgical interventions for PS compared with a multimodal conservative approach did not find convincing data on the greater effectiveness of one or another treatment method. At the same time, it was shown that complications such as hematomas, fractures, respiratory disorders, as well as strokes and myocardial infarctions were observed during surgical operations in 10–24% of cases and were absent during conservative treatment. Thus, it is important for clinicians to inform patients about possible treatment methods and to take a balanced approach to the choice of further management tactics [7]. According to a meta-analysis, exercise therapy has a similar effect in spinal PS when compared with decompression surgery [18].

There is no convincing evidence for early surgical intervention, since the duration of conservative therapy conducted before surgery does not affect the subsequent outcome of neurosurgical treatment [19]. The installation of stabilizing systems in patients with stenosis without instability is not recommended [20]. In patients with PS and intermittent claudication that arose against the background of spondylolisthesis, the use of stabilizing systems is most justified. However, there is no convincing evidence to recommend a standard treatment method to achieve durable arthrodesis. When choosing the tactics of neurosurgical management, an individual and balanced approach is recommended, taking into account both the patient's characteristics and the experience of the doctor [13].

Conclusion: PS is a significant cause of low back pain, radiculopathy and claudications, especially in the elderly and senile. Due to the aging of the population, the issues of diagnosis and treatment of PS are becoming increasingly relevant. To date, therapeutic strategies for patients with PS have not been fully defined and systematized, since there is insufficient convincing evidence of the effectiveness of various treatment options. The tactics of managing patients with PS should be based on clinical symptoms, and not on imaging data. The patient's

walking distance is the most significant criterion for the effectiveness of therapy. Further studies are needed to assess the effectiveness and safety of various methods of treating PS.

Literature:

1. Parfenov VA, Isaykin AI Pain in the lower back: myths and reality. M.: IMA-PRESS; 2016 (in Russ.)].
2. Issack PS, Cunningham ME, Pumberger M. Degenerative lumbar spinal stenosis: evaluation and management. J Am Acad Orthop Surg. 2012 Aug;20(8):527–535. DOI: 10.5435/JAAOS-20-08-527.
3. Ishimoto Y., Yoshimura N., Muraki S. Osteoarthritis Cartilage. Prevalence of symptomatic lumbar spinal stenosis and its association with physical performance in a population-based cohort in Japan: the Wakayama Spine Study. 2012 Oct;20(10):1103–1108.
4. Stephen J. Textbook of spinal disorders. Philadelphia; 1995.
5. Battié MC, Ortega-Alonso A., Niemelainen R. et al. Lumbar spinal stenosis is a highly genetic condition partly mediated by disc degeneration. Arthritis Rheumatol. 2014;66(12):3505–3510. DOI: 10.1002/art.38823.
6. Smirnov A. Yu. Clinic, diagnosis and surgical treatment of lumbar stenosis. Neurosurgery. 1999;2:59–64.
7. Smirnov A. Yu., Evzikov G. Yu. Surgical treatment of lumbar stenosis. Neurosurgery. 1998;1:34–38.
8. Covaro A., Vilà-Canet G., de Frutos AG Management of degenerative lumbar spinal stenosis: an evidence-based review. EFORT Open Rev. 2017;1(7):267–274. DOI: 10.1302/2058-5241.1.000030.
9. Steurer J., Roner S., Gnannt R., Hodler J. LumbSten Research Collaboration. Quantitative radiologic criteria for the diagnosis of lumbar spinal stenosis: a systematic literature review. BMC Musculoskeletal Disord. 2011;12:175. DOI: 10.1186/1471-2474-12-175.
10. Kobayashi S. Pathophysiology, diagnosis and treatment of intermittent claudication in patients with lumbar canal stenosis. World J Orthop. 2014;5(2):134–145.
11. Takahashi N., Kikuchi S., Yabuki S. et al.. Diagnostic value of the lumbar extension-loading test in patients with lumbar spinal stenosis: a cross-sectional study. BMC Musculoskeletal Disord. 2014;15:259. DOI: 10.1186/1471-2474-15-259.
12. Tomkins-Lane C., Melloh M., Lurie J. et al. ISSLS prize winner: consensus on the clinical diagnosis of lumbar spinal stenosis: results of an international Delphi study. Spine (Phila Pa 1976). 2016;41(15):1239–1246.
13. Kreiner DS, Shaffer WO, Baisden JL An evidence-based clinical guideline for the diagnosis and treatment of degenerative lumbar spinal stenosis (update). Spine J 2013;13(7):734–743. DOI: 10.1016/j.spinee.2012.11.059.
14. Budithi S., Dhawan R., Cattell A. Only walking matters-assessment following lumbar stenosis decompression. Eur Spine J. 2017;26(2):481–487.
15. Messiah S, Tharian AR, Candido KD, Knezevic NN Neurogenic Claudication: a Review of Current Understanding and Treatment Options. Curr Pain Headache Rep. 2019;23(5):32. DOI: 10.1007/s11916-019-0769-x.
16. Qaseem A, Wilt TJ, McLean RM, Forciea MA Noninvasive Treatments for Acute, Subacute, and Chronic Low Back Pain: A Clinical Practice Guideline From the American College of Physicians Ann Intern Med. 2017;166(7):514–530. DOI: 10.7326/M16-2367.

17. Lee PB, Kim YC, Lim YJ et al. Efficacy of pulsed electromagnetic therapy for chronic lower back pain: a randomized, double-blind, placebo-controlled study. J Int Med Res. 2006;34(2):160–167.
18. Arneja AS, Kotowich A., Staley D. et al. Electromagnetic fields in the treatment of chronic lower back pain in patients with degenerative disc disease. Future Sci O.A. 2016;2(1): FSO105. DOI: 10.4155/fsoa-2015-0019.
19. Gale GD, Rothbart PJ, Li Y. Infrared therapy for chronic low back pain: a randomized, controlled trial. 2006;11(3):193–196.
20. Shostak NA, Klimenko AA, Andriyashkina D. Yu. Possibilities of local therapy for back pain. Clinician. 2017;2(11):74–79 (in Russ.)].
21. Ammendolia C., Stuber KJ, Rok E. Nonoperative treatment for lumbar spinal stenosis with neurogenic claudication. Cochrane Database Syst Rev. 2013;8: CD010712. DOI: 10.1002/14651858.CD010712.
22. Enke O., New HA, New CH et al. Anticonvulsants in the treatment of low back pain and lumbar radicular pain—a systematic review and meta-analysis. CMAJ. 2018;190(26): E786–E793. DOI: 10.1503/cmaj.171333.
23. Manchikanti L., Knezevic NN, Boswell MV et al. Epidural Injections for Lumbar Radiculopathy and Spinal Stenosis: A Comparative Systematic Review and Meta-Analysis. Pain Physician. 2016;19(3): E365–410.
24. Meng H., Fei Q., Wang B. et al. Epidural injections with or without steroids in managing chronic low back pain secondary to lumbar spinal stenosis: a meta-analysis of 13 randomized controlled trials. Drug Des Devel Ther. 2015;9:4657–4667. DOI: 10.2147/DDDT.S85524.
25. John NB, Hodgden J. Epidural Injections for Long Term Pain Relief in Lumbar Spinal Stenosis. J Okla State Med Assoc. 2019;112(6):158–159.
26. Low Back Pain and Sciatica in Over 16s: Assessment and Management. London: National Institute for Health and Care Excellence (UK); 2016. National Institute for Health and Care Excellence: Clinical Guidelines.
27. Trigg SD, Devilbiss Z. Spine Conditions: Lumbar Spinal Stenosis. FP Essent. 2017;461:21–25.
28. Inoue G., Miyagi M., Takaso M. Surgical and nonsurgical treatments for lumbar spinal stenosis. Eur J Orthop Surg Traumatol. 2016;26(7):695–704. DOI: 10.1007/s00590-016-1818-3. Epub 2016 Jul 25.