CLINICAL CRITERIA AS AN ALGORITHM FOR SURGICAL TREATMENT OF SPINAL STENOSIS

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ABSTRACT

Lumbar spinal stenosis (LSS) is a chronic disease characterized by a combination of clinical symptoms and a decrease in the size of the lumbar spinal canal according to instrumental examination methods.

Lumbar spinal stenosis (LSS) is a chronic disease characterized by a combination of clinical symptoms and a decrease in the size of the lumbar spinal canal according to instrumental examination methods [1–3]. The term LSS, which refers to any type of narrowing of the lumbar spinal canal or intervertebral foramen that leads to the development of neurological symptoms, was proposed by a group of American orthopedists led by S. Arnoldi et al. [4, 5]. The incidence of this pathology increases significantly in people over 50 years of age and varies from 1.8 to 8% [6]. Signs of LSS are observed in 80% of patients aged 70 years and are registered annually with a frequency of ≥ 5–11.5 cases per 100 thousand population [7, 8]. For example, according to the Swedish national registry, the average annual rate of neurosurgical interventions for LLS increased from 10–15 per 100,000 inhabitants in 1703 to 30–25 per 100,000 in 1713 [1, 9, 10]. It is worth noting that there is still no clear understanding of approaches to the treatment of this pathology, as evidenced by the presence of at least several groups of specialists (neurosurgeons, orthopedic traumatologists, chiropractors, neurologists, physiotherapists, osteopaths, etc.) who have opposing views on the choice of treatment methods for LSS [7, 11].

About 17–23% of elderly people have asymptomatic LSS, which is confirmed only by instrumental examination data [24, 26, 27]. LSS is most often observed at the level of L4–5, followed by L3–4, L2–3, and least often at L5–S1 and L1–2 [4, 24].

Neurogenic intermittent claudication syndrome is caused by chronic compression of the neural and vascular structures of the canal [6, 11]. Due to chronic compression, the volume of blood supply to the neural structures does not meet their needs [6]. The amount of incoming blood decreases, and ischemia of the root (with lateral stenosis) or the equine tail (with central stenosis) develops [1, 23]. Complaints of pain, numbness, and weakness in the legs with LSS occur when walking [28]. The pathogenesis of this syndrome is determined by the fact that during walking, blood filling of the epidural veins increases, which leads to additional compression of the neural structures in the lumbar spine [1, 23, 29]. Patients with LSS try to

adopt a sitting position, in which the lordosis in the lumbar spine is smoothed out or kyphosis occurs. This increases the lumen of the PC and foraminal openings, which helps restore normal blood flow [30, 31]. With flexion, the height of the foraminal opening increases by 12%, with extension, it decreases by 15% [6, 32].

Since one of the causes of LSS is arthrosis of the lumbar spine, patients may present complaints specific to facet syndrome, namely, nagging pain in the area of the affected lumbar spine, which intensifies in the morning, during movement (in the lumbar spine) or prolonged stay in a horizontal position, during rotation, extension, and decreases after warming up. Due to spondyloarthrosis, pain can radiate to the hips, gluteal and groin areas [29]. LSS can develop against the background of instability of the lumbar spine [23, 34]. In addition to pain syndrome, typical symptoms of LSS include neurogenic intermittent claudication, symptoms of tension of the lumbar roots, sensory disturbances, etc. (Table 1).

Table 1

Symptoms of LSS and their frequency of detection [1, 4, 25]

Symptoms	Detection frequency, %
Pain in the lumbar spine	87
Neurogenic intermittent claudication	78-81
Symptoms of tension	66
Sensory disturbances in the lower extremities	54
Paresis or plegia of the lower limbs	47
Pain in the lower limbs	26-69
Muscle wasting of the lower extremities	23
Sensitive disorders in the perineal area	19
Periodic cramps in the calf muscles	17
Pelvic organ dysfunction	6

Various types of lateral stenosis are manifested mainly by monoradicular pain syndrome [27]. Pain in lateral stenosis has a lesser tendency to decrease in the lying position (curled up) or sitting (squatting with the body bent forward), does not increase with coughing and sneezing, pain in the lumbar spine is less pronounced, and Lasegue and Wasserman symptoms are not characteristic. The pain is constant and rarely recur [24, 27]. For herniated discs, pain increases in the sitting position, has a more acute onset and increases with Lasegue and Wasserman tests [24, 26].

Instability in the spinal spondylosis is one of the most common pathologies of the spine. A.I. Prodan et al. use the term “dynamic LSS”, i.e. narrowing of the lumbar spine with instability of the spinal spondylosis and balloting hernia of the intervertebral disc, which leads to corresponding clinical symptoms in a standing position [16, 23, 25].

Diagnostics Computed tomography (CT) allows for a detailed assessment of pathological changes in bone structures and is a necessary method in the comprehensive diagnostics of LSS. The use of CT allows for the determination of the exact bone boundaries of the canal, hypertrophy of the arches, DOS and TP, characteristics of osteophytes, foraminal stenosis, petrification of the longitudinal ligament and hernia of the intervertebral disc [23, 34]. CT, even in MPR and 3D reconstruction modes, does not always allow for the determination of changes in soft tissue structures that cause (in 8% of cases) the development of LSS [26].

Magnetic resonance imaging (MRI) has a number of undoubted advantages in the diagnosis of LSS [19]. To determine the degree of central LSS, the classification of S. Schizas et al. is used, based on the analysis of axial MRI sections of the PC in T2-WI mode [8].

The following parameters are assessed using MRI [16, 25]:

1) area of lateral recesses;
2) the cross-sectional area of the dural sac;
3) the total cross-sectional area of the dural sac and lateral recesses;
4) PC measurement level;
5) anteroposterior diameter of the dural sac;
6) transverse diameter of the dural sac;
7) interfacet distance;
8) the depth of both lateral recesses;
9) the angle of both lateral recesses.

CT myelography is used to diagnose LSS in cases of contraindications to MRI, for example, in the presence of metal implants in the body [19]. In some cases, this method allows assessing the degree of root compression better than MRI. For example, in the case of multilevel lesions verified by MRI data, CT myelography seems to be a necessary additional method to clarify the level requiring primary neurosurgical intervention [5]. Contrast myelography has the same diagnostic value and is performed in the absence of conditions for CT myelography [19].

Based on radiographic data, quantitative (frontal and sagittal dimensions of the lumbar spine) and qualitative (changes in lordosis of the lumbar spine, presence of scoliosis, developmental anomalies, changes in the height of the intervertebral disc and other pathological changes in the pelvic floor) indicators are assessed [26].

In recent years, special attention has been paid to the study of the role of spinopelvic balance parameters in the development of degenerative changes in the lumbar spine [9]. Functional spondylography provides an idea of the presence of instability of the spinal joint according to the White–Panjabi criteria: < 5 points — no instability; ≥ 5 points — instability [8, 10].

At present, the functional state of the cauda equina roots can only be assessed indirectly using electroneuromyography [18]. In patients with LSS-induced radiculopathy, electroneuromyography reveals a decrease in the amplitude of evoked motor potentials of the foot muscles during stimulation of the peroneal and tibial nerves. The conduction velocity and amplitude of sensory potentials are slightly reduced. It has been established that the changes recorded by electroneuromyography in patients with LSS occur earlier than the clinical signs of spinal cord root damage appear.

Treatment Conservative treatment of LSS includes the use of painkillers, vascular, anti-inflammatory drugs, as well as therapeutic exercises, physiotherapy procedures, hyperbaric oxygenation [8, 11, 12], which improves the well-being of 32–69% of patients with LSS [26]. Most clinicians consider it necessary to perform neurosurgical intervention in case of progression of clinical symptoms refractory to conservative treatment for 3–6 months [17, 20]. Up to 80% of patients are satisfied with the outcomes of surgical treatment. Monitoring of operated patients for a period of 4 to 10 years shows the preservation of the effect of the surgery [19].

Surgical treatment. Decompressive laminectomy is currently rarely used and is gradually being replaced by new, less invasive operations. In central LSS, the basis of neurosurgical tactics used to be laminectomy and removal of the yellow ligament at the level of LSS [7]. Currently, there is no unified view on the tactics of neurosurgical treatment of LSS [8].

Decompressive surgeries. Historically, decompressive laminectomy was the first surgery used to treat LSS, but it has a number of disadvantages that reduce its effectiveness [7, 25]: in particular, removal of the posterior structures of the spine (interspinous and supraspinous ligaments, arches, part of the DOS) leads in some cases to instability of the lumbar spine and the formation of a pronounced cicatricial adhesion process [1]. Minimally invasive surgery includes adequate decompression of nerve structures in LSS with the least surgical trauma to the muscular, articular and ligamentous apparatus of the lumbar spine [5].

In case of “non-bone” LSS (hypertrophy of the yellow ligament), hemilaminectomy with bilateral ligamentectomy at the level of the stenotic area of the SC has been developed [25]. The immediate positive results in different types of decompressive surgeries are 72–80%, while the difference in surgical results between the types of decompression (hemilaminectomy, interlaminectomy) during the observation period of 4 to 7 years after surgery was not statistically different [5]. Achieving certain threshold values, such as the cross-sectional area (0.8–1.6 cm2), transverse diameter of the dural sac (12–16 mm) and sagittal diameter (7–12 mm), in the surgical treatment of LSS is mandatory - this allows to significantly improve the quality of life of patients with LSS [8]. However, further increase in these dimensions does not lead to an improvement in the quality of life.

Lateral stenosis, taking into account the specific features of root compression, is eliminated by decompression of the radicular recess (recessotomy, facetectomy, removal of osteophytes) [4, 13]. In case of stenosis of the radicular canal, adequate decompression involves performing a medial facetectomy (50% of the DOS area), if necessary in combination with removal of the osteophyte of the IVD edge. In case of lateral stenosis of the middle zone, elimination of radicular compression is ensured by hemilaminectomy with resection of the lower articular surface of the facet joint [6]. Increasing the parameters of the lateral recess during decompression to threshold values (angle 30°, depth 5 mm) leads to a significant improvement in the quality of life of patients, however, excessive decompression and further increase of these parameters do not affect the improvement of the quality of life [13]. Total facetectomy allows for good decompression, but this often leads to iatrogenic segmental instability of the lumbar spine with the development of pain in the lumbar spine [17].

In cases where LSS is combined with a herniated disc, adequate decompression involves removal of a small disc herniation [7]. Transforaminal decompression of foraminal

stenosis can be supplemented with transforaminal lumbar interbody fusion (TLIF). However, according to studies by foreign and domestic scientists, additional stabilization does not increase the effectiveness of treatment. Uniportal endoscopic foraminal decompression allows for successful surgery on patients aged 70–80 years and older. According to a number of vertebrologists, this approach is preferable in the treatment of foraminal stenosis and is effective in 72–83% of cases. Decompressive surgeries for LSS are performed only in the absence of instability in the lumbar interbody fusion and preservation of the spinal-pelvic ratios.

The destabilizing effect of resection of the dorsal supporting structures of the lumbar spine is reduced by osteoplastic laminectomy (laminoplasty). Laminoplasty is a type of decompressive surgery aimed at reconstructing the posterior structures of the lumbar spine. However, laminoplasty has a number of disadvantages, such as high trauma and duration of surgery, the possibility of developing kyphotic deformity of the lumbar spine. In addition, fixation of the arc fragment with microplates is not reliable enough, although, according to the results of individual studies, fusion in the problem area was noted in all patients.

Stabilizing interventions are not performed in cases of minor pain in the lumbar spine, severe osteoporosis, severe concomitant pathology, and the absence of instability [25]. The vertebrologist must choose the optimal balance between the benefits and risks of additional stabilization in a particular patient.

Instrumental fixation in operations for LSS does not improve the outcome of surgical intervention and quality of life, and should be used only for the above-mentioned indications [8, 25]. The frequency of complications is higher in stabilizing interventions and is 27.6%, after decompressive operations - 9.7%. The frequency of revision surgeries is also higher after stabilization - 10.3%, after decompression - 6.5% [5].

Interspinous fixation systems. The idea of dynamic stabilization is based on the fact that the operation allows to reduce the load on the posterior structures of the lumbar spine, to increase the area of the SC and foraminal openings [56, 61]. The technique of interspinous fixation involves decompression with subsequent installation of dynamic implants in the interspinous space [1]. A distinctive feature of this dynamic fixation is the ability to perform both flexion and extension in the PDS, which prevents the formation of "adjacent segment syndrome" [22, 25, 50]. Interspinous fixation systems allow to reduce the load on the DOS, which causes axial decompression of the roots due to an increase in the height of the foraminal openings [6, 19, 61]. Contraindications to the use of interspinous fixation are the presence of symptoms during flexion in the lumbar spine, scoliosis (more than 25° at the LSS level), instability in the LSS, osteoporosis, multi-level LSS (3 or more LSS) and ankylosis of the affected LSS [1, 23].

Currently, the following interspinous dynamic fixation systems are used in vertebrology: Coflex (U-implant), Interspinal U, DIAM, Minns, Wallis, X-Stop, In-Space, Aperius and STENOFIX [46, 54, 64]. In vitro studies have shown that systems such as X-Stop or Coflex provide flexion and distraction of the posterior supporting structures of the lumbar spine, eliminate central and foraminal LSS, but significantly increase the volume of lateral movements, reduce the contact area of the DOS facets and are capable of forming a subluxation of the latter with the progression of DOS arthrosis. In addition, fractures of the spinous processes, implant migration, spondylolisthesis, LSS and fixation failure were

recorded.

CONCLUSION The variability of LSS surgical methods is determined by the lack of uniform criteria for selecting the optimal surgical intervention for different LSS options and individual patient characteristics.

To select the optimal method of surgical treatment of LSS, it is necessary to use a wide range of neuroimaging (CT, MRI, spondylography with functional tests) in combination with a thorough neurological and orthopedic examination.

The key parameter for assessing the adequacy of the performed decompression in central LSS is the cross-sectional area of the dural sac, and in lateral stenosis, the lateral recess indices (angle 30°, depth 5 mm).

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