PATHOGENETIC ASPECTS AND RISK FACTORS FOR RECURRENT LUMBAR DISC HERNIATION: LITERATURE REVIEW

O.N. Dreval[1], A.V. Kuznetsov[1], V.A. Chekhonatsky[1], A.V. Baskov[1], A.A. Chekhonatsky[2], A.V. Gorozhanin[3]
[1]*Russian Medical Academy of Postgraduate Education, Moscow, Russia*
[2]*Saratov State Medical University n. a. V.I. Razumovsky, Saratov, Russia*
[3]*City Clinical Hospital n.a. S.P. Botkin, Moscow, Russia*

One of the main causes of the development of debilitating pain syndrome after surgical treatment of a herniated disc is herniation recurrence. This pathology dictates the need to perform reoperation on an already operated segment of the spinal column, which complicates the technique of surgical intervention and negatively affects the relief of pain syndrome. In the presented review of scientific publications selected from the medical literature databases PubMed, E-library and Cochrane, the current problems of the pathogenesis of recurrent herniated discs in the lumbar spine are considered. The concept of risk factors for the development of recurrent disc herniation is highlighted, their characteristics are given, and the significance of each of them in the development of recurrent disc herniation is analyzed.
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The steadily increasing rate of degenerative diseases of the lumbar spine and the development of neurological manifestations associated with this group of pathologies are among the most pressing issues in modern neurosurgery. Although various modern diagnostic methods have been introduced into neurosurgical routine, the problem related to the outcomes of surgical treatment of lumbar disc herniation has not lost any of its relevance because wrong diagnosis makes surgeons choose an inadequate treatment tactics. Furthermore, the rate of poor surgical treatment outcomes in patients with disc herniation presenting as pain recurrence is also steadily growing.

Benzakour et al. [1] analyzed the postoperative outcomes of 552 patients who had undergone microdiscectomy to treat lumbar disc herniation in 1993–2013 by assessing the clinical outcomes 3, 6, 12 months, 5 years, and then every five years after the surgery. The outcomes were found to be good or very good (regression of radiculopathy by 70–90 % and patients' ability to return to their original work) in 87.3 % of cases within one-year follow-up. These results were worsened after 14-year follow-up but remained satisfactory (pain regression by 30–70 % and patients' ability to perform less difficult work) in 63.7 % of patients. Twenty-nine (5.2 %) patients developed a new symptomatic disc herniation at the adjacent level, with discectomy required in 8 (1.44 %) cases. Furthermore, Benzakour et al. [1] reported 51 (9.2 %) cases of postoperative recurrence of herniation at the same level; the first and second revisions showed that 6.52 % and 1.08 % of patients, respectively, required vertebral fixation. This fact proves that surgical treatment of this neurosurgical pathology is effective and raises the following question: what treatment strategy should be chosen for the developing herniation recurrence?

Recurrent disc herniation is defined as the recurrence of disc herniation at the site of a previous surgery after the initial symptomatic improvement. Despite the tight definition of the term "recurrent disc herniation" where both ipsilateral and contralateral herniation is possible, several researchers argue that this term should refer only to cases of ipsilateral herniation [2].

Development of recurrent disc herniation is a common problem; according to the current literature [3], its rate ranges from 2 to 25 %. Recurrent disc herniation is the main cause of failed back surgery syndrome that causes disability among working-age population and necessitates re-operation at the spinal segment that has already been operated. Ambrossi et al. [4] showed that in patients requiring revision surgery for recurrent disc herniation, the cost of diagnosis and treatment was 17 times higher than that of conservative treatment in patients having the same pathology (USD 39,386 vs. USD 2,315).

Today, there is much controversy over the pathogenesis of recurrent disc herniation. Fibrosis is generally believed to be the most favorable outcome of surgical treatment of disc herniation [5–7] as the development of fibrous tissue in an intervertebral disc to some extent prevents the prolapse of nucleus pulposus; intradiscal displacement is no longer observed during the fibrosis stage. According to A.D. Oleinik and V.N. Malyshko [8], the underlying cause of recurrent disc herniation at the spinal segment operated earlier is incomplete removal of the nucleus pulposus during primary surgery (i.e., failure to perform curettage), since the bulging herniated disc mostly involves the degenerated portion of the nucleus pulposus (which should have been completely resected during primary surgery)

and only partially the fibrous ring. During the postoperative period, the remaining portion of the nucleus pulposus continues to undergo degeneration, and herniation recurrence can eventually occur.

A number of researchers [9–11] suggest that recurrent disc herniation also depends on the development of segmental instability that prevents fibrous tissue formation in the intervertebral disc after the primary surgical intervention. The risk of disc herniation recurrence in patients who had undergone surgical intervention and fixation surgery is 1.8 %, while being 12.5 % in those who had not undergone fixation of the operated segment. This fact pathogenetically substantiates the role played by segmental instability in the development of recurrent disc herniation.

According to Motsumoto et al. [12], some of the main risk factors for the development of recurrent disc herniation involve rupture of the posterior longitudinal ligament, caudal migration of the sequestered disc, and a large defect of the fibrous ring.

Moreover, there is an opinion that the intervertebral disc has been evolutionarily excluded from immune tolerance processes due to its location. However, during its degeneration and extrusion accompanied by integrity disruption of the fibrous ring, the nucleus pulposus interacts with the immune system. The immune system perceives the nucleus pulposus as an antigenic structure and triggers the cascade of biochemical and immune responses that are elicited by activation of B cells and cytotoxic T cells [13].

Therefore, it can be assumed that this process causes chronic inflammation in the operated disc, thus preventing prompt formation of disc fibrosis at the operated level and leading to recurrent prolapse of the remnants of the nucleus pulposus and annulus fibrosus, resulting in herniation recurrence [13, 14].

In their recent studies, Russian researchers have revealed that disorders in biochemical and biomechanical processes directly contribute to the pathogenesis of recurrent disc herniation. E.S. Baikov et al. [14] analyzed the data for 78 patients with disc herniation and inferred that the amount and structure of proteoglycans/glucosaminoglycans contained in the nucleus pulposus and fibrous ring display the features of metabolic processes occurring in the intervertebral disc tissue and undergo specific changes in patients with recurrent disc herniation, which significantly affect the biomechanical properties of intervertebral disc tissue [14].

Hanaei et al. [15] suggested that patients with verified recurrent disc herniation have mutations in both inflammatory and anti-inflammatory genes or congenital connective tissue disorders, which weaken the ligamentous apparatus of the functional spinal unit. In turn, this leads to prolapse of nucleus pulposus remnants and subsequent compression of the nerve structures, even if primary discectomy was successful. It is currently known that mutations in genes encoding the synthesis or degradation of the components of the extracellular matrix of connective tissue are responsible for the development of hereditary disorders of connective tissue [16]. A large group of monogenic syndromes of connective tissue dysplasia associated with mutations in genes encoding extracellular matrix proteins (different types of collagens, fibrillin, tenascin, etc.) and growth factor receptor genes is currently known. However, the studies analyzing the causes of degenerative disc diseases and their progression are most often devoted to TGF-β (transforming growth factor beta) [15–18]. In their experiments on mice, Zheng et al. [17] proved that increasing TGF-β activity effectively reduces the severity of disc degeneration in aged mice and maintains the homeostasis of the intervertebral discs. Other researchers [15, 18] have also proved that TGF-β activity is associated with impaired homeostasis in the intervertebral discs and its degenerative changes. This fact may initiate the development of recurrent disc herniation.

The effect of chronic inflammation in the intervertebral disc on the development of recurrent disc herniation is also being actively discussed. Immunohistochemical analysis of the samples intraoperatively harvested from the disc revealed high levels of phospholipase A2, inflammatory interleukins, chemokines, and various groups of matrix metalloproteinases [19].

Taking into account the prevalence and social significance of this disease, it is important to early identify patients at high risk of recurrent herniation in order to determine the further treatment strategy [20].

Currently, researchers have identified several possible risk factors for the development of recurrent disc herniation [21]. However, contradictory results are often obtained.

Age. Yurac et al. [22] studied 1,028 patients and found that age younger than 30 years is a significant risk factor for developing recurrent disc herniation. However, Yao et al. [23] analyzed 111 patients and revealed that age over 50 years was a direct predictor of herniation recurrence, while other authors revealed no relationship to age at all [3].

Male sex. A study of the data from 627 patients showed that the risk of recurrent lumbar disc herniation was predominantly associated with male sex [24, 25].

Body mass index. Meredith et al. [26] studied 75 patients and inferred that high BMI (33.6 ± 5.1 kg/m^2) is a direct predictor of disc herniation recurrence, but Moliterno et al. [27] (who performed a retrospective study of 217 patients) suggested that low BMI should also be considered a risk factor for recurrent disc herniation.

Height of the intervertebral disc. According to Yaman et al. [28] ($n = 600$), patients with recurrent disc herniation had a relatively greater disc height than those without the recurrence (19.1 ± 4.6 and 15.0 ± 3.3 mm, respectively). Therefore, this parameter can also be regarded as a risk factor for disc herniation recurrence.

Smoking. Miwa et al. [29] analyzed the clinical data from 32 re-operated patients and inferred that smokers were at a 18.5 % higher risk of disc herniation recurrence compared to non-smokers. Other researchers have obtained similar results [3, 30–32].

Occupation. Miwa et al. [29] and D.N. Kinshin et al. [33] found that physically demanding work is also among the key predictors of recurrent lumbar disc herniation. Shimia et al. [24] arrived at a conclusion that surgeons should recommend limiting strenuous physical work during the postoperative period for their patients to prevent herniation recurrence of the operated intervertebral disc. However, Meredith et al. [26] believe that the risk of disc herniation recurrence among manual workers is not significantly higher than that among patients whose occupation is unrelated to physical labor. Daly et al. [34] analyzed the outcomes of lumbar microdiscectomy in patients who had early returned to work and excluded those operated on for recurrent disc herniation from their studies.

Modic changes. The number of studies focused on changes in vertebral bodies adjacent to the degenerated disc is currently growing. The first thorough description of changes in MR signal intensity in the subchondral areas of the vertebral bodies during degenerative processes of the spine was made by Modic in 1998. His name has been used for classification of these changes ever since. Modic changes correspond to the inflammatory phase and are seen as reduced signal intensity in T1-weighted MR images and increased signal intensity in T2-weighted MR images. The researchers analyzing this issue are currently noting the relationship between having Modic changes and back pain; however, the role played by such factors as aseptic inflammation and anaerobic bacteria from Propionibacterium genus (*Propionibacteria acnes*) in the development of the aforementioned changes also remains debatable [35–39]. According to Yaman et al. [28], Modic changes were observed statistically more often in patients with recurrent disc herniation compared to the control group involving patients without herniation recurrence.

Diabetes mellitus. Researchers currently believe that diabetes mellitus of both types plays a crucial role in the pathogenesis of disc herniation recurrence and note that the rate of recurrent disc herniation is significantly increased among patients having this disease [28, 40–43]. Patients with diabetes mellitus are likely to have worse outcomes after primary lumbar intervertebral disc surgery compared to those without diabetes mellitus. Mobbs et al. [44] reported that in the group of patients with diabetes mellitus who had been operated to treat lumbar disc herniation, the recurrence rate was 28 %, while being as low as 3.5 % in the control group of patients without diabetes. NaPier et al. [40] conducted experiments using laboratory rats subjected to posterolateral spinal fusion using a coccyx autograft. Twenty-two of 42 rats received an implantable insulin tablet. Compared to that in healthy rats that were subjected to spinal fusion, the rate of bone block formation was significantly decreased in animals with insulin-dependent diabetes (16.7 % versus 43.0 %). The mean bone mineral density, bone volume and volume fraction of bone tissue were also significantly reduced and negatively correlated with blood glucose levels. This fact proves that insulin-dependent diabetes plays an important role in the pathogenesis of disc herniation recurrence even after fixation surgery.

Conclusion

Today, improving the efficiency of diagnostic measures and treatment of recurrent lumbar disc herniation remains one of the most pressing problems in neurosurgery. The rate of unfavorable outcomes of surgical treatment of disc herniation is as high as 15–20 %, demonstrating that it is necessary to understand new pathogenetic features and risk factors of recurrent lumbar disc herniation that are independent of previous surgical intervention. Furthermore, new treatment modalities need to be developed and the existing ones need to be improved.

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Address correspondence to:
Chekhonatsky Andrey Anatolyevich
Saratov State Medical University n. a. V.I. Razumovsky,
112 Bolshaya Kazachya str., Saratov, 410012, Russia,
fax-1@yandex.ru

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Oleg Nikolayevich Dreval, DMSc, Prof., Head of Department of Neurosurgery, Russian Medical Academy of Continuous Professional Education, 2/1 Barricadnaya str., Moscow, 125993, Russia, ORCID: 0000-0002-8944-9837, odreval@nsi.ru;
Aleksey Vitalyevich Kuznetsov, PhD, Associate Professor of Department of Neurosurgery, Russian Medical Academy of Continuous Professional Education, 2/1 Barricadnaya str., Moscow, 125993, Russia, ORCID: 0000-0002-9487-6008, akuznetsov@nsi,ru;
Vladimir Andreyevich Chekhonatsky, postgraduate student of Department of Neurosurgery, Russian Medical Academy of Continuous Professional Education, 2/1 Barricadnaya str., Moscow, 125993, Russia, ORCID: 0000-0001-6155-1154, fax-0@yandex.ru;
Andrey Vladimirovich Baskov, DMSc, Professor of Department of Neurosurgery, Russian Medical Academy of Continuous Professional Education, 2/1 Barricadnaya str., Moscow, 125993, Russia, ORCID: 0000-0002-7542-6300, abaskov@mail.ru;
Andrey Anatolyevich Chekhonatsky, DMSc, Associate Professor, Head of Department of Neurosurgery, Saratov State Medical University n. a. V.I. Razumovsky, 112 Bolshaya Kazachya str., Saratov, 410012, Russia, ORCID: 0000-0003-3327-1483, fax-1@yandex.ru;
Aleksandr Vadimovich Gorozhanin, MD, PhD, Head of Neurosurgery Department No. 19, City Clinical Hospital n.a. S.P. Botkin, 5 2nd Botkinsky passage, Moscow, 125284, Russia, ORCID: 0000-0002-3593-7034, agorozhanin@list.ru.