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Risk factors of recurrence lumbar intervertebral disk herniation after primary endoscopic transforaminal discectomy. Part 1 (literature review)

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Percutaneous endoscopic transforaminal discectomy (PETD) is a minimally invasive technique that improves clinical outcomes. However, limited visibility of the surgical field and the widespread use of endoscopic technology have led to complications after discectomy, among which recurrence of lumbar disc herniation is the main reason for repeated surgical interventions. The aim is to study epidemiological risk factors that potentiate the recurrence of lumbar intervertebral disc herniation after primary percutaneous endoscopic transforaminal discectomy. Methods. The study material is professional articles containing the definition of epidemiological risk factors for recurrence of lumbar disc herniation after primary PETD, for the period 2015–2024. A systematic review of relevant literature sources was performed using the following keywords: "recurrent lumbar disc herniation", «risk factor for recurrent lumbar disc herniation», "minimally invasive lumbar spine surgery", "percutaneous endoscopic lumbar discectomy», "percutaneous endoscopic transforaminal discectomy», "re-discectomy". Results. Research data on the reliability of the relationship between the recurrence of lumbar disc herniation after primary PETD and epidemiological risk factors are contradictory. The most significant among them was older age, ruptures of the annulus fibrosus. Conclusions. The most reliable epidemiological risk factors for rGMD of PVC after primary PETD are age > 50 years, body mass index > 25 kg/m². The reasonable time for performing primary PETD of lumbar disc herniation from a medical and financial point of view is ≤ 8 weeks from the moment of clinical manifestation of the disease.

Перкутанна ендоскопічна трансфорамінальна дискектомія (ПТЕД) є малоінвазивною методикою, яка дозволяє покращити клінічні результати. Проте обмежений огляд операційного поля й широке застосування ендоскопічної технології призвели до ускладнень після дискектомії, серед яких рецидив грижі міжхребцевого диска (рГМД) поперекового відділу хребта (ПВХ) є основною причиною повторних втручань. Мета. Проаналізувати епідеміологічні фактори ризику, які потенціюють рецидив грижі міжхребцевого диска поперекового відділу хребта після первинної перкутанної ендоскопічної трансфорамінальної дискектомії. Методи. Розглянуто фахові статті, які містять визначення епідеміологічних факторів ризику рГМД ПВХ після первинної ПТЕД, за період 2015–2024 р. Виконано систематичний огляд релевантних джерел літератури за ключовими словами як українською, так і англійською мовами: «рецидивуюча грижа міжхребцевого диска поперекового відділу хребта», «малоінвазивні хірургічні втручання на поперековому відділі хребта», «перкутанна ендоскопічна поперекова дискектомія», «перкутанна ендоскопічна трансфорамінальна дискектомія», «повторна дискектомія». Результати. Дані досліджень щодо достовірності взаємозв'язку рецидиву грижі міжхребцевого диска поперекового відділу хребта після первинної ПТЕД із епідеміологічними факторами ризику суперечливі. Найбільш значущими серед них виявилися старший вік, порушення цілості фіброзного кільця. Висновки. Найдостовірнішими епідеміологічними факторами ризику рГМД ПВХ після первинної ПТЕД вважаються вік > 50 років, індекс маси тіла > 25 кг/м². Доцільним терміном виконання первинної ПТЕД грижі міжхребцевого диска поперекового відділу хребта з медичної та фінансової точки зору є термін ≤ 8 тижнів із моменту клінічної маніфестації захворювання. Ключові слова. Первинна трансфорамінальна ендоскопічна дискектомія, рецидив грижі міжхребцевого диска поперекового відділу хребта, епідеміологічні фактори ризику.

Keywords. Primary transforaminal endoscopic discectomy, recurrence of lumbar intervertebral disc herniation, epidemiological risk factors

Introduction

Percutaneous (complete) endoscopic surgery for lumbar disc herniation using transforaminal or interlaminar approaches is an emerging technique that has taken its place in the current “gold standard” of microsurgical discectomy [1–4]. The use of endoscopic techniques for the removal of lumbar disc herniations (LDH) has been shown to improve clinical outcomes by reducing intraoperative blood loss [5, 6] and postoperative pain intensity with a corresponding reduction in the dose of opioid analgesics after surgery, a shorter recovery period [7] with earlier return to daily activities [8–11] and high patient satisfaction, and relatively low hospital costs [5]. Percutaneous endoscopic transforaminal discectomy (PETD) is considered a minimally invasive procedure because the posterior support structures of the spinal segment remain intact [11, 12].

However, the widespread use of endoscopic technology has led to complications after discectomy, among which recurrence of lumbar disc herniation (rLDH) is the main reason for reoperation [13]. The incidence of rLDH, reaching 5–18 % [14–16], is comparable to the failure rate in open microdiscectomy [11]. The main reason for most recurrences is limited intraoperative access, which may result in the retention of a subligamentous or sequestered component of the LDH, and the difficulty of controlling decompression of neural structures [17–19]. Failure of PETD is also the conversion of endoscopic discectomy into open surgery due to the technical impossibility of transforaminal access. In such cases, the conversion of the operation is most often caused by a structural change in the shape and size of the working area in the area of the intervertebral foramen, which is associated with the peculiarities of the orientation and deformation of the articular processes of the corresponding lumbar segment [20, 21].

A sufficiently high frequency of unsatisfactory PETD results requires the identification of clinical and radiological risk factors for rLDH with more careful selection of patients for transforaminal discectomy, because repeated surgical interventions lead to a deterioration in the clinical outcome with a decrease in the quality of life compared with the consequences of the primary surgical intervention [14], and subsequent significant financial costs (estimated costs: direct — 34,242 US dollars, indirect — 3,778) [22].

Purpose: To study the epidemiological risk factors that potentiate the recurrence of lumbar intervertebral disc herniation after primary percutaneous endoscopic transforaminal discectomy.

Material and Methods

The research material is professional articles that contain definitions of risk factors for recurrent lumbar disc herniation after primary PETD.

The literature search was conducted in the PubMed, Google Scholar and Medline databases for the period 2015–2024 using medical subject headings and keywords “recurrent lumbar disc herniation”, “risk factors for recurrent lumbar disc herniation”, “minimally invasive lumbar spine surgery”, “percutaneous endoscopic lumbar discectomy”, “percutaneous endoscopic transforaminal discectomy”, “re-operation”, “re-discectomy”.

An additional search for articles from the bibliographic lists of selected literature sources was also carried out. If necessary, in some cases, sources published outside the search period were used.

Inclusion criteria: articles on risk factors for recurrence of monosegmental LDH after primary PETD. Exclusion criteria: publications on the results of open surgical treatment of herniated intervertebral discs of the lumbar spine, open and endoscopic discectomy of polysegmental and recurrent lumbar disc herniations.

Research method: systematic review of relevant literature sources.

Results and Discussion

Age

There is no single point of view in the literature on the relationship between the age of patients and recurrence of LDH after primary PETD. Some authors define young age of patients as a risk factor for the development of rLDH [23–25]. Other studies have not found a significant difference in age between groups of patients with recurrent disc herniation and uncomplicated postoperative course [14, 26]. Most experts believe that the frequency of recurrence of LDH is positively correlated with age > 50 years [27, 29–31]. Increased risk of rLDH [32] in elderly patients is associated with a decrease in mechanical stiffness of disc tissues due to a combination of involutive and degenerative changes with concomitant loss of part of the gelatinous nucleus, disruption of the integrity of the fibrous ring as a result of disc extrusion. In such biomechanical conditions, in the presence of intraoperative incision of the annulus fibrosus, the intervertebral disc becomes more susceptible to stress loads with the formation of irreversible deformation, which subsequently leads to ineffective healing of the outer layer of the annulus fibrosus after PETD and recurrence of LDH [32].

Gender

The results of studies on the gender effect on the incidence of rLDH also vary widely. Most publications have not found such an effect [14, 29, 31, 33–35]. At the same time, a predisposition to disc recurrence in males has been reported [16, 23, 36]. Some experts identify female gender as a risk factor for rLDH [37]. Interesting results of multivariate logistic regression analysis on the interaction of sex and age are given in the publication by F. Martens et al.: in women under 51 years of age, the risk of recurrent lumbar disc herniation was approximately 10 times higher than in other age groups [38]. The significantly higher frequency of rLDH in young women may be associated with the degradation of type II collagen (the predominant form of collagen in the intact intervertebral disc) with a decrease in the biomechanical properties of the disc under the influence of rapid changes in estrogen concentration, as well as changes in estrogen receptor expression in the premenopausal period [39].

Comorbidity

Increased body mass index (BMI) is a generally accepted risk factor for LDH [19, 25, 30–33, 36, 40–42], although some experts have reported different results [14, 37]. The negative impact of excess body weight on the lower lumbar spinal segments may be due to increased biomechanical loads with cumulative damage to the intervertebral discs [43, 44], especially in the case of abdominal obesity [40]. Overweight (BMI ≥ 25.0 – 29.9 kg/m²) and obesity (BMI ≥ 30.0 kg/m²) increased the absolute risk of LDH by 1.8 and 2.3 times, respectively [40].

Biochemical changes in the intervertebral disc tissues are also possible, associated with a decrease in the level of glycosaminoglycans in the nucleus pulposus [45] and with an increased concentration of leptin. It is a prototypical adipokine produced by adipose tissue [46] and is considered a major biochemical mediator of the inflammatory, degradative, and nociceptive effects of obesity and can be produced by other tissues, including elements of the intervertebral disc [47, 48]. *In vitro* studies have shown that an increase in the level of leptin and its receptors localized in the intervertebral disc can initiate a degenerative and inflammatory cascade through the activation of proteases involved in the degradation of aggrecan and proteases capable of destroying collagen and other matrix macromolecules at the gene and protein levels. In the nucleus pulposus, leptin activated the expression of proinflammatory molecules, especially the cytokines IL-6 and TNF- α [49]. This action of leptin may be of importance *in vivo*, as it and its receptors

have been identified in degeneratively altered areas of the nucleus pulposus [47] and in cells of the annulus fibrosus [48, 50], which themselves produce leptin in the event of degeneration [48].

The results of studies on the relationship between rLDH and diabetes mellitus are contradictory. One group of authors did not find such relationships [13, 23, 27, 29, 37], while other experts consider diabetes mellitus as a risk factor for recurrent disc herniation [28, 51–53]. The possible influence of diabetes mellitus on the formation of rLDH may be due to the limitation of sulfation of glycosaminoglycan molecules [54] and a decrease in the content of proteoglycans [55] in the extracellular matrix of the intervertebral disc, which is accompanied by a weakening of its collagen matrix [54]. Hypertension may increase the frequency of rLDH [33, 56], although other reports deny such a relationship [14, 29, 37]. The specific mechanism linking hypertension to rLDH has not been studied; the assumption of the influence of arterial hypertension *per se* [56] is made.

Smoking

Active smoking is considered a modified risk factor for rLDH and can lead to an increase in absolute risk by 1.6–2.8 times [40, 57]. The clinical course of LDH in smokers is characterized by a low pain threshold, an increased incidence of postoperative complications, delayed healing of the postoperative wound and a long rehabilitation period, and reduced satisfaction with the results of surgical treatment [57–59].

The mechanisms by which the risk of recurrent disc herniation in active smokers increases are related to the fact that nicotine significantly inhibits diffusion and worsens the trophism of the intervertebral disc due to vasoconstriction of the endplate capillaries and subchondral bone [60]. The vasoconstrictive effect of nicotine can also lead to the suppression of cell proliferation and inhibition of the synthesis of extracellular matrix proteoglycans [61, 62], significantly impairing the rate of cellular absorption and production of metabolites in the disc [62, 63]. Such biochemical changes reduce the absorption capacity of the disc and potentiate the acceleration of degenerative processes [40]. In addition, nicotine inhibits the synthesis of collagen in the annulus fibrosus, which reduces the strength of the annulus and increases the risk of traumatic injuries and degenerative changes [64]. Chronic cough of a smoker can increase intradiscal pressure, which increases the risk of recurrent disc herniation under conditions of reduced mechanical stiffness [57]. At the same time, a number of authors deny the existence of a connection between smoking

and rLDH [37, 65]. It is noteworthy that under conditions of cessation of passive smoking, an increase in the content of mucin proteoglycan in the nucleus pulposus and annulus fibrosus was observed in mice, which reflects the possibility of correcting degenerative changes in the intervertebral disc caused by smoking [66].

Duration of “herniated” follow-up

Performing surgery within ≤ 8 weeks of clinical manifestation of LDH is considered preferable to PETD, which is used in a more distant period [67]. Despite the higher initial financial costs associated with conservative treatment in the acute and subacute periods of the disease, the final costs of surgical treatment in this category of patients are on average less than \$11,200 compared to cases of surgical care in later periods. In such patients, additional financial costs are associated with prolonged conservative treatment during the chronic stage of LDH, decreased labor productivity with periodic loss of work capacity [68]. Based on this, it is logical that there is no relationship between rLDH and the duration of “herniated” follow-up in studies in which the average waiting time for PETD was on average 5.5; 7; 30 months. [20, 29, 32].

Conclusions

The most reliable epidemiological risk factors for recurrence of lumbar intervertebral disc herniation after primary PETD include age > 50 years, body mass index $> 25 \text{ kg/m}^2$.

The appropriate time for performing primary PETD of lumbar intervertebral disc herniation from a medical and financial point of view is ≤ 8 weeks from the moment of clinical manifestation of the disease.

Conflict of interest. The authors declare the absence of a conflict of interest.

Prospects for further research. An analysis of the literature on the influence of radiological, including radiometric indicators on the frequency of recurrent lumbar intervertebral disc herniations is relevant.

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RISK FACTORS OF RECURRENCE LUMBAR INTERVERTEBRAL DISC HERNIATION AFTER PRIMARY ENDOSCOPIC TRANSFORAMINAL DISCECTOMY. PART 1 (LITERATURE REVIEW)

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