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DEGENERATION OF THE INTERVERTEBRAL DISC AS AN INTERDISCIPLINARY CLINICAL PROBLEM IN THE CONTEXT OF NEUROSURGERY: A SUMMARY OF THE STATE OF KNOWLEDGE. PART 1

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A – study design, **B** – data collection, **C** – statistical analysis, **D** – interpretation of data, **E** – manuscript preparation, **F** – literature review, **G** – sourcing of funding

ABSTRACT

Background: Degeneration of the intervertebral disc (IVD), resulting from various factors, including genetic and environmental causes, is a significant musculoskeletal disorder. It is characterized by a gradual deterioration of the structure and a weakening in the function of the IVD, leading to a decline in the quality of life (QOL) or disability.

Aim of the study: This study aimed to perform a review of scientific reports and a summary of the current knowledge regarding IVD degeneration in terms of etiopathogenesis, clinical symptoms, and various diagnostic methods.

Material and methods: The literature review was performed by searching key medical databases such as PubMed, MEDLINE, and Scopus. A search strategy was defined, encompassing keywords, synonyms, and controlled keywords (MeSH terms). The search included the last 20 years and focused on aspects of etiopathogenesis, clinical symptoms, and diagnostic methods. Papers meeting the inclusion criteria underwent a full-text analysis. Data and information gathered from the literature analysis were synthesized to provide a comprehensive overview of the current state of knowledge on the researched subject.

Results: The process of IVD degeneration is extremely complex, involving biochemical, metabolic, and morphological changes that impact its structure and function. It entails a gradual loss of water in the nucleus pulposus (dehydration), resulting in an impaired shock-absorbing function and leading to the formation of breaks in the annulus fibrosus, allowing herniation of the nucleus pulposus. For years, mechanical injuries, especially work-related injuries, have been considered the primary factors causing IVD degeneration. Recent findings emphasize the significant role of genetic factors, with the identification of several genes encoding structural proteins of the IVD. Mutations in these genes may accelerate degeneration. Current clinical research results indicate a correlation between aortic calcifications and episodes of low back pain (LBP) and between lumbar artery stenosis, IVD degeneration, and subsequent LBP.

Conclusions: Understanding the complexity of IVD degeneration and its neurological consequences is crucial in planning effective treatment. It is essential to monitor the latest research to systematically update the

knowledge and practical guidelines in the discussed area. Collaboration between an experienced neurosurgeon and an interdisciplinary team of medical specialists can achieve the best therapeutic outcomes.

KEYWORDS: intervertebral disc degeneration, low back pain, etiopathogenesis, symptomatology, neurosurgery, state of knowledge

Background

Contemporary society, due to a dynamic lifestyle, constant technological progress, and changing health habits, are increasingly confronted with various musculoskeletal disorders [1], including those involving the lumbosacral complex [2]. One of the key issues gaining particular significance, in the context of today's health challenges, is the degeneration of the intervertebral disc (IVD). This condition not only affects a broad population, but it represents a civilization-related problem associated with limited physical activity and presents comprehensive diagnostic, therapeutic, preventive, and rehabilitative challenges for specialists from various medical fields.

Aim of the study

This work aimed to delve into issues related to IVD degeneration, focusing on its etiopathogenesis, clinical symptoms, and various aspects of diagnosis. The paper also summarizes the current state of knowledge based on a review of scientific reports, incorporating the latest research and discoveries in the field of IVD degeneration. This review serves as the foundation for further discussion on the interdisciplinary approach to this clinical problem. Through analysis of the latest scientific data, we aimed to offer the reader a comprehensive view of the evolution of scientific thought regarding IVD degeneration and guide our considerations toward potential areas of further research and improvements in clinical practice.

Material and methods

Information sources

The literature review was meticulously conducted by exploring prominent medical databases, including PubMed, MEDLINE, and Scopus. Recognizing the significance of a comprehensive and systematic approach, a well-defined search strategy was formulated to cast a wide net across the vast landscape of available literature. This involved the incorporation of a diverse array of search elements, including keywords, synonyms, and controlled keywords in the form of Medical Subject Headings (MeSH terms).

Search strategy

The meticulous development of the search strategy played a pivotal role in ensuring a comprehensive and up-to-date exploration of the literature. Encompassing the last 20 years, the search was strategically designed to capture the latest advancements and insights into the etiopathogenesis, clinical symptoms, and diagnostic methods of IVD conditions. The temporal scope was chosen to strike a balance between including recent developments and maintaining a sufficient historical context.

Eligibility criteria

Papers meeting the inclusion criteria underwent a full-text analysis, evaluating the quality and quantity of collected data. This rigorous assessment involved a meticulous examination of study design, participant characteristics, intervention methods, and outcome measures.

Data synthesis

Data and information gathered from the literature analysis were synthesized, providing a comprehensive overview of the current knowledge on the researched subject. Standard ethical principles were applied in the literature review, and all data obtained from the studies were used in accordance with the principles of scientific honesty and integrity.

Reporting bias

An essential aspect of the literature review process involved a vigilant consideration of reporting bias in the selected studies. Recognizing the potential influence of selective reporting on the overall findings and conclusions, the authors systematically assessed the presence of reporting bias in the included papers.

Results

The synthesis of literature exploring crucial aspects of etiopathogenesis, clinical symptoms, and diagnostic methods regarding IVD presented a nuanced panorama of research outcomes. This thorough review spanned studies conducted over the last two decades, providing insights into the etio-

pathogenesis, clinical symptoms, and diagnostic methods associated with IVD. The narrative review encompassed the following components: the etiology of IVD degeneration considering the morphological stages of IVD degeneration, clinical symptoms of IVD degeneration categorized into pain, lumbago, and radicular symptoms associated with discogenic low back pain (LBP), as well as the diagnosis of discopathy, specifying methods and the distribution of pain and muscle strength weakness depending on the level of IVD damage. The amalgamation of data and insights derived from the studies under review established a sturdy groundwork for comprehending the present state of knowledge concerning theoretical grounds and diagnostics methods for IVD conditions. The authors consistently adhered to standard ethical principles during the literature review, underscoring the importance of integrity and honesty in the utilization of data extracted from the chosen studies.

Discussion

Etiology of intervertebral disc degeneration

IVD degeneration is extremely complex and not fully understood. It involves biochemical, metabolic, and morphological changes that impact its functioning. Fragmentation of proteoglycans in the IVD begins in childhood, and with age, there is a decline in their composition and water content, especially in the nucleus pulposus [3]. The turnover of matrix substances also decreases, leading to an increase in stable connections between molecules and collagen fibers, further hindering the processing and repair of the matrix components. This results in the retention of damaged macromolecules, ultimately reducing the quality and properties of the entire tissue [4].

Furthermore, in early childhood, the blood supply to the endplates and, consequently, the IVD decreases. As a result, around the age of 15, the first microstructural cracks and fissures appear, especially in the nucleus and endplates [5]. Cell density decreases, leading to a continuous increase in structural defects, including those in the annulus. With a decrease in cell quantity, the synthesis of matrix substance diminishes, and the individual cells reduce their production [6]. For most individuals, active and passive transport mechanisms within the IVD are unable to continuously and adequately supply nutrients to the cells. As the supply of energy and nutrients diminishes for the IVD, cells produce lower-quality fibers and matrix substances, ultimately leading to their disintegration [7].

The gradual loss of water in the nucleus pulposus (dehydration) leads to an impaired shock-absorbing function and the development of annular tears, into which the nucleus pulposus herniates. Concentric, transverse, and radial tears have been identified. Radial tears most commonly lead to the formation of an IVD herniation [8]. Completely separated fragments of the annular tissue and endplates of the IVD, under the influence of asymmetrical stresses, can move beyond the IVD due to weakened areas. As a result, protrusions and displacements of the IVD develop. As long as the outer layers of the annulus are intact, there is a possibility of the nucleus returning to its original location. In the case of an IVD herniation, its displaced tissues penetrate the annulus fibrosus and are unable to return to their original location. Dorsal or dorsolateral displacement of the IVD brings it into contact with the posterior longitudinal ligament, which has sensory innervation from the meningeal branches of the spinal nerves, resulting in local pain, radicular pain, and chronic discogenic LBP [9]. In 1990, based on morphological studies, Thompson et al. [10] introduced a classification of the degree of IVD degeneration, which perfectly illustrates the natural history of the degeneration process (Table 1).

Table 1. Morphological stages of intervertebral disc degeneration [10]

Stage	Nucleus Pulposus	Annulus fibrosus	Vertebral endplate	Vertebral body
I	"Bulging"	Few fibrous lamellae	Uniform layer of hyaline	Rounded edges
II	Peripheral white connective tissue	Mucous material between lamellae	Irregular layer of hyaline	Sharp edges
III	Consolidation of connective tissue	Significant mucinous infiltration; obliteration of the division between annulus fibrosus and nucleus pulposus	Focal defects in cartilage	Early chondrophytes and osteophytes at the edges
IV	Horizontal breaks, parallel to the vertebral endplate	Focal breaks	Fibrocartilaginous tissue emerging from ossification centers; irregular and focal ossification centers	Osteophytes less than 2 mm
V	Scattered cracks in both the nucleus pulposus and annulus fibrosus		Diffuse sclerotization	Osteophytes greater than 2 mm

Currently, it is accepted that disturbances in trophic factors, as described above, play a primary role in the degenerative process of the IVD. For years, it was believed that mechanical injuries, especially work-related, were the main factor causing IVD degeneration. This hypothesis is supported by the observation that degeneration most often occurs at the L5/S1 level – a segment that bears the greatest load. Furthermore, it has been noted that the motion segment adjacent to the level in which a spinal fusion is performed degenerates much faster (known as adjacent level disease) [11]. Recent findings, however, highlight a significant role of genetic factors. Several genes encoding structural proteins of the IVD have been identified, and mutations in these genes may accelerate degeneration. These include genes for collagen types I, IX, and XI, interleukin genes, vitamin D receptor, IGF-1 receptor, aggrecan genes, and enzymes involved in matrix breakdown [12]. Moreover, postmortem studies have shown a connection between atherosclerotic changes in the aorta and degenerative disc disease, as well as between lumbar artery occlusion and episodes of LBP. Clinical research results have indicated a correlation between aortic calcifications and episodes of LBP, as well as between lumbar artery stenosis and IVD degeneration with subsequent LBP [13].

Clinical symptoms of intervertebral disc degeneration

Pain syndrome

A symptom of IVD degeneration is local and/or radiating pain, which arises from the irritation of various elements of the nervous system. The pain may have a referred character – it is felt in the innervation area of the irritated nerve, in regions distant from the source causing its occurrence. An example is sciatica, which is caused by irritation of the nerve roots forming the sciatic nerve. Organ pain is pain felt at the site of the pathological change, with LBP being an example. Although many spinal disorders are challenging to precisely define, most discomfort associated with disc disease can be attributed to the involvement of specific nerve structures located in the vicinity of the IVD [14].

Lumbago

The pathomechanism of LBP is not fully understood. Sensory nerve endings sensitive to mechanical irritation are primarily located within the posterior longitudinal ligament, facet joint capsules, and the nerves themselves [15]. As mentioned earlier, nociceptive fibers normally penetrate the outer layer of the annulus to a depth of about 1–3 mm, but in significantly diseased discs, they can extend into the nucleus pulposus. In the most external layers of the annulus fibrous, immunohistochemical methods have demonstrated the presence of fibers containing substance P and VIP [16].

It is believed that fissures in the annulus fibrous during degenerative changes stimulate these receptors. Even the deeper layers of the annulus fibrous are pain-innervated, and some studies have demonstrated the presence of receptors in the nucleus pulposus itself [17]. Foci of proliferation have been identified in the anterior and, more recently, in the posterior part of the IVD. It has been observed that damage to the annulus fibrosus initiates the ingrowth of vascularized inflammatory tissue along the fissure, with sensory fibers accompanying blood vessels [18]. Since the inner layers of the annulus fibrosus are not vascularized, it appears that this process occurs concentrically, from the most superficial layers towards the nucleus pulposus. Elevated levels of IL-6 and IL-8 have also been demonstrated in patients with LBP, which may be responsible for sensitizing nociceptors within the painful IVD [19].

Despite clear evidence of the presence of nociceptive nerve endings in the IVD, their role in the development of discogenic pain remains unclear. It often happens that patients with significant degenerative changes in the IVD revealed in magnetic resonance imaging (MRI) do not report any pain at all [20], On the other hand, the most sensitive test aiming to demonstrate the IVD as the source of pain is discography. However, despite its high sensitivity, it is characterized by a low specificity. This examination involves injecting a specific volume of fluid into the nucleus pulposus, causing an increase in intradiscal pressure, which, in turn, irritates nociceptive nerve endings in the annulus fibrous. The prevalence of tears within the inner layers of the IVD using this test is estimated at 39%–42%, while the prevalence of discogenic pain without specifying IVD damage is 26% [21].

As mentioned earlier, changes in the height and volume of the IVD always affect the alignment of the facet joints. Conditions predisposing to excessive loading within the facet joints are almost always associated with instability, changes in volume, or irreversible deformation of the IVD. This leads to an increase in stresses acting on the articular processes, resulting in an inflammatory reaction and damage to the articular cartilage. Pain arises during irritation of sensory and autonomic nerve endings in the joint capsule. Facet joint syndrome is currently one of the most controversial issues in spinal surgery, with some even questioning its clinical significance. Its frequency is estimated at 10–15% [22]. The standard

diagnostic procedures include interventional techniques, such as intra-articular injection of an anesthetic or medial branch block [23], as conventional diagnostic methods like computed tomography (CT) or MRI, which will be discussed below, are not sufficiently sensitive in this case. Research is ongoing regarding the use of SPECT in the diagnosis of facet joint pain [24].

Radicular pain

In the lumbar spine, the anatomical relationships between the IVD, intervertebral foramina, and their corresponding spinal nerve roots are of particular importance. The dorsal root ganglia and ventral roots are in close proximity to the IVD. Within the intervertebral foramen, between the nerve root and the surrounding walls of the bony canal, there is, under normal conditions, sufficient space. However, the nerve root may become compressed if any of the reserve spaces narrow due to disc protrusion, disc herniation, osteophytes, enlarged vessels, or overgrown bone tissue (spinal canal stenosis) [25].

Compression of a spinal nerve root causes distinct, segmental sensory and motor disturbances in the trunk and limbs. The precise location and degree of damage to the root determines whether the most pronounced symptoms will reflect the involvement of the ventral or dorsal branch of the nerve. Pain within the dermatome may vary in character depending on the type and degree of nerve compression [26].

The most common form of radicular pain is sciatica – a set of painful symptoms resulting from irritation of the nerve roots forming the sciatic nerve. The association of sciatica with mechanical compression of the root by a herniated nucleus pulposus has been known for a long time, yet the exact pathomechanism of radicular pain – similar to that in the case of LBP – is not fully understood. However, many factors influencing the development of radicular pain have been described. [27].

Pain associated with an IVD herniation is caused not only by mechanical damage but also by chemical irritation of the nerve root. Under physiological conditions, the IVD is not located within the spinal canal, so if it shifts there, it triggers a foreign body reaction. The mechanical and chemical irritation leads to changes in the nerve root – initially, edema and later atrophy. Compressed nerve fibers within the root may spontaneously generate action potentials. Compression results in demyelination of the root axons and alters their excitability – inflammatory changes in the root increase its sensitivity to additional mechanical irritation [28].

In vitro studies have shown that it is not a direct mechanical injury that causes damage, but rather disturbances in blood supply underlie the impairment of function. It has been demonstrated that venous outflow disturbances occur early, even at pressures ranging from 5 to 10 mmHg. As a consequence of impaired outflow, there is a reduction in capillary flow, potentially leading to local ischemia. Partial impairment of nerve conduction may occur at pressures as low as 10 mmHg [29]. At higher values, reaching 50 mmHg, edema formation occurs. Complete conduction block occurs at pressures of 50–75 mmHg, acting for 1–2 hours. Necrosis occurs at pressure values of 70–130 mmHg [30].

It is accepted that when the pressure on the root is below 200 mmHg, the damage results almost exclusively from impaired circulation. Meanwhile, the pressure exerted on the root in the presence of a herniated nucleus pulposus averages about 50 mmHg [31]. Moreover, it has been observed that local swelling around the root is more pronounced when the pressure rises rapidly than when it is increased gradually. This observation may explain the better adaptation to long-term, slowly progressing pathological processes such as spinal canal stenosis compared to the case of an acute herniated nucleus pulposus, where symptoms are generally much more severe. On the other hand, compression of periradicular venous plexuses easily leads to disturbances in capillary circulation with subsequent ischemia and swelling. A vicious cycle of disease occurs when compression causes swelling and an increase in the volume of the root, consequently increasing the pressure on the root [32].

Currently, it is believed that the widespread hypothesis that pain arises due to mechanical compression is considered an oversimplification. It is not in line with the observation that 20–30% of individuals in the general population, without any history of radicular pain, show imaging features of mechanical compression of nerve roots within the intervertebral foramen [33]. Furthermore, based on pressure measurements on the root during microdiscectomy, it was found that the intensity of symptoms, including radicular symptoms, does not correlate with the degree of root compression [34].

Apart from LBP and radicular pain, which are essential components of the clinical picture of degenerative disc disease, some patients also experience muscle pain. Irritation of the dorsal branches of the spinal nerve can lead to continuous motor impulses to the muscles, resulting in abnormal contractions and pain. Furthermore, the instability of the motion segment associated with disc disease can induce compensatory contraction of the trunk and proximal limb muscles, causing excessive tension and overload [35]. Patients typically perceive a combination of painful symptoms that further evolve with the duration of the disease. From a pathophysiological

perspective, it is presumed that the combination of structural, biochemical, and physiological changes in the peripheral and central nervous systems acts as a common pathway for the "mixed pain" characteristic of chronic LBP and sciatica [36]. A dorsolateral disc herniation compressing the nerve root initially causes purely radicular pain, which later transforms into pain consisting of radicular, pseudoradicular, and myotendinous elements. Spinal pain is characterized by considerable diversity and tends to transform into chronic pain [37].

Diagnosis of discopathic changes

The most common levels for degeneration and herniation of the IVD are L4/L5 and L5/S1. Typically, the disease begins with an acute, burning, stabbing pain in the lumbar region radiating to the back of the lower limb, down to the shin. The pain is usually superficial and localized, often accompanied by numbness. A detailed interview regarding the location of the pain is crucial in determining the spinal level where the nerve root is compressed (Table 2) [38].

In advanced disease, paralysis and weakened deep reflexes can occur. Sometimes, symptoms of cauda equina syndrome may appear in the course of a central disc herniation, which is an urgent indication for surgical intervention. Based on the examination of superficial sensations, motor functions, and deep reflexes, the responsible level for symptoms can be precisely determined according to dermatomes and the knowledge of the innervation of individual muscle groups. For example, the nerve roots of the upper lumbar spine (L1–L3) control the function of the iliopsoas muscle, which is primarily responsible for thigh flexion. Fibers from roots L2–L4 innervate the quadriceps femoris muscle, which is a knee extensor. The L4 root innervates the anterior tibialis muscle, responsible for dorsal flexion of the foot causing a patient with a damaged L4 root to have difficulty walking on their heels. Fibers from the L5 root innervate the extensor hallucis longus muscle and the thigh adductors, and a positive Trendelenburg test may indicate damage [39]. An essential part of the examination is the assessment of stretch signs. In the compression of the L5–S1 roots, the Lasegue sign is often positive. The Lasegue test is highly sensitive in recognizing root syndromes, with a sensitivity ranging from 72% to 97%, albeit with relatively low specificity. In the diagnosis of sciatica, another stretch sign – the Mackiewicz sign – has lower sensitivity, ranging from 43% to 60% [40].

Table 2. Distribution of pain and muscle weakness depending on the level of the disease [41]

Level of the spine	Pain localization	Muscle function
T12–L1	Groin area and medial surface of the thigh	—
L1–L2	Anterior and medial part of the upper thigh surface	Slight weakness of the quadriceps femoris muscle, slight weakness of the knee reflex
L2–L3	Anterolateral surface of the thigh	Weakness of the quadriceps femoris muscle, weakness of the knee reflex
L3–L4	Posterolateral surface of the thigh, anterior surface of the lower leg	Weakness of the quadriceps femoris muscle, weakness of the knee reflex
L4–L5	Dorsal surface of the foot	Weakness of the dorsal flexors of the toe and foot
L5–S1	Lateral surface of the foot	Weakness or the absence of the ankle reflex

The described characteristics of symptoms, unfortunately, apply only to a portion of patients, specifically those with a classic root syndrome caused by compression of nerve roots. As mentioned above, the source of pain in degenerative spinal disease can be the disc itself, as well as structures in its vicinity, such as the longitudinal ligaments or facet joints. The clinical picture can be non-specific and challenging to interpret. Additionally, psychological and socioeconomic factors play a significant role [42]. Elements of a medical examination, such as a positive sign (a maneuver interpreted by the patient as inducing pain), tenderness to touch, or an exaggerated response to medical examinations, may raise suspicions of pain on a psychological basis. It has been shown that such symptoms occur more frequently in patients filing disability claims and in those who have had workplace accidents [43].

LBP can also occur in other, more serious conditions. Therefore, degenerative disc disease should be considered after excluding other medical conditions such as tumors, infections, fractures, or cauda equina syndrome. Alarming symptoms in true lumbar radiculopathy or other conditions resembling radiculopathy are referred to as "red flags." For tumors or infections, "red flags" include age over 50 or under 20, a history of cancer, general symptoms such as fever, chills, unexplained weight loss, immunosuppression, intravenous drug use, and pain worsening in a lying position. On the other hand, a history of trauma, prolonged corticosteroid therapy, osteoporosis, and age >70 years may suggest a vertebral fracture. Alarming symptoms of nerve root compression include urinary

and fecal incontinence, sensory disturbances resembling "pants" in cauda equina syndrome, or significant muscle weakness, as in sudden foot drop [44].

Imaging diagnostic methods are crucial for determining the diagnosis, stage of advancement, and potential indications for surgical treatment in degenerative disc disease. The two most important and commonly used diagnostic methods are conventional radiography and MRI. Other methods, such as CT, CT myelography, bone scintigraphy, ultrasonography, and PET/CT, are currently supplementary methods [45].

Conventional radiography

X-rays are usually the initial diagnostic examination in spinal degenerative disease due to their low cost and widespread availability. They effectively illustrate significant changes such as intervertebral space narrowing, osteophytes, sclerosis within vertebral bodies, degenerative kyphosis, scoliosis, and spondylolisthesis. Degenerative disc changes manifest on X-rays as a reduction in disc height, the vacuum phenomenon (caused by gas accumulation within the degenerating disc), and disc calcifications. Primary projections include anteroposterior and lateral views, supplemented by functional studies [46].

Computed tomography

This examination has lower tissue resolution compared to MRI. Therefore, in practice, an indication for performing CT for the evaluation of spinal pain is in patients with contraindications to MRI. Currently, thanks to significant technological advancements, there is the possibility of reconstructing images in virtually any plane and creating volumetric reconstructions. This allows for a detailed assessment of the structure and pathology of the vertebrae, to a lesser extent the intervertebral discs, and in selected cases, with the use of a contrast agent, the soft tissues, primarily the soft tissue structures around the spine and within the vertebral canal. Multiplanar reconstructions can be used to better assess vertebral canal stenosis and narrowing or pathological widening of intervertebral foramina. CT imaging is less preferred compared to MRI due to the necessity of using ionizing radiation [47].

Magnetic resonance imaging

MRI visualizes soft tissues such as the spinal cord, nerve roots, meninges, IVD, muscles, ligaments, joint effusions, adipose tissue, connective tissue scars, and inflammatory granulation tissue in great detail. This examination also allows for the assessment of the vertebral endplates, the marrow cavity, degenerative changes, and vertebral hemangiomas. Imaging changes to the intervertebral discs are very detailed and allow for the evaluation of disc herniations, sequestrations, disruptions of the annulus fibrous, and compression of vertebral canal structures, which is crucial in making therapeutic decisions [48].

The MRI examination always begins with cross-sectional spin-echo sequences (currently most often in their modifications: fast spin-echo or turbo spin-echo) in T1- and T2-weighted images. The scans should cover the entire spinal canal and both intervertebral foramina. Subsequently, at the levels of identified changes, examinations are performed in axial planes (usually in T2-weighted spin-echo or gradient T2*-weighted images; only in patients with previous lumbar disc herniation surgery, additional T1-weighted images are necessary), with the layers tilted parallel to the intervertebral spaces. Axial scans should cover the entire intervertebral foramen [49].

In addition to conventional spin-echo techniques, numerous additional sequences are used: gradient-echo sequences – helpful in differentiating osteophytes from small disc herniations; fat suppression sequences (FatSat) – useful in cases of unclear images of the vertebral bodies, e.g., to distinguish fatty degeneration from destructive changes. Steady-state sequences (CISS, FIESTA, CBASS, DRIVE) allow for a more accurate assessment of the outlines of the spinal cord and the thecal sac than conventional sequences, enabling, for example, a better determination of the relationship between osteophytes and the thecal sac, spinal cord, and spinal nerve roots. Currently, research is underway on the application of other sequences and imaging with 3T devices [50].

The assessment and classification of degenerative changes in the intervertebral discs on MRI examinations with T2-weighted sequences are performed using the five-grade Pfirrmann scale, first described in 2001. It evaluates the condition of the IVD, its height, and hydration. The latter feature is the best-determining factor in the MR image of the IVD, which correlates well with the degree of degenerative changes. Thus, Grade I indicates a homogeneous disc structure, high signal intensity, and a normal disc height, while Grade V indicates a non-homogeneous disc structure, low signal intensity, a lack of distinction between the annulus fibrous and the nucleus pulposus, and a significantly reduced disc height [51].

In 1987, De Roos et al. first described changes in the MRI images of vertebral bodies adjacent to de-

generated intervertebral discs, and a year later, Modic et al. [52] introduced a formal classification based on differences in imaging using T1 and T2-weighted sequences. According to Modic, the altered signal is not caused by the disease process itself. Instead, it reflects its consequences, such as instability or overload. Based on these observations, a three-stage classification of degenerative changes in vertebral bodies was introduced. In Type I, zones of decreased signal intensity are visible with T1-weighted images and increased in T2-weighted and FatSat images, located adjacent to the endplates. These changes indicate an active inflammatory process and correlate with active spinal pain syndrome. In Type II, changes manifest as zones of increased signal intensity in T1-weighted images, located near the endplates. In T2-weighted images, they are iso-intense or moderately hyperintense, and in the FatSat sequence, the areas with fatty degeneration signaling undergo characteristic suppression – indicating chronic overload changes, with marrow fat replacement corresponding to the chronic stable phase of the disease with a possible conversion to Type III. In Type III, zones of low signal intensity are visible on T1- and T2-weighted images, as well as fat saturation, located around the endplates. This suggests marrow sclerosis, likely corresponding to the clinical phase of healing and recovery, although the exact significance is not known [53].

The assessment of changes in the vertebral bodies over the course of degenerative spine disease, according to Modic, is one of the most valuable diagnostic tools currently available. Its primary advantages include its accessibility and high reliability. Additionally, it facilitates differentiation between degenerative changes and other more serious conditions such as discitis or neoplastic changes. Kijer et al. [54] demonstrated that even advanced degenerative changes in the disc can occur without clinical symptoms, while the presence of Modic Type I changes more often correlates with an acute phase of spinal pain syndromes. However, it is currently emphasized that degenerative changes in the spine have a dynamic nature and that Modic changes may be reversible – raising questions about their clinical value [55].

Another useful feature of MRI is the presence of a high-intensity zone (HIZ) in the annulus fibrous, first described in 1992 by Aprill and Bogduk [56]. These are linear or spherical high-signal zones visible on T2-weighted images in the posterior part of the annulus fibrous. The high signal of the HIZ on T2-weighted images is caused by the presence of fluid within a fissure of the annulus fibrous and neovascularization occurring at the edge of the fissure, reflecting the reparative processes. It has also been shown that within a disc with a HIZ on MR examination, not only are there indeterminate tissues in the annulus fibrous, but also inflammatory tissues with granulocytes, causing a higher signal compared to the signal from the nucleus pulposus [57].

The prevalence of HIZs in a group of patients with LBP, as assessed by MRI, was estimated at 28%, and the positive predictive value of significantly altered findings leading to clinical symptoms of disc degeneration was 86% [56]. However, the presence of a HIZ has also been demonstrated in patients without clinical symptoms, reaching 56%, and its frequency increases with the age of the patients. In a recently published study by Alyas et al. [58], they concluded that a HIZ visualized on MRI represents non-specific changes and may not necessarily be related to the occurrence of back pain symptoms.

The bulging of the IVD is visualized on axial sections as a symmetrical displacement of the outer contour of the disc beyond the outline of the vertebral body, towards the spinal canal or intervertebral foramina. Meanwhile, a focal bulging of the posterior contour of the disc indicates a disc herniation, which can be located centrally, paramedially, or laterally, less commonly far laterally or anteriorly. Based on the relationship of the herniation to the parent disc, it is possible to differentiate the type of herniation into protrusion, extrusion, and sequestration. In the case of protrusion, the dimension of the bulging fragment perpendicular to the disc is smaller than its base, while in extrusion, this dimension is larger than the base of the herniation. Moreover, the position of the separated disc fragment is visualized in detail on the MRI in the case of sequestration [59].

Study limitations

According to the authors, the paper constitutes a valuable summary of the current state of knowledge regarding etiopathogenesis, clinical symptoms, and diagnostic methods of IVD disease. Nevertheless, this review paper has potential methodological limitations that need to be addressed. It is a narrative review developed based on guidelines and the Narrative Review Checklist. However, the authors acknowledge that more appropriate guidelines, such as PRISMA, could be applied in the future for a more precise reporting of individual stages of methodology (eligibility criteria, search strategy, selection process, study risk of bias assessment, effect measures, and synthesis methods) and results (study selection, study characteristics, risk of bias in studies, results of individual studies, results of syntheses, reporting biases, and certainty of evidence). Conducting detailed systematic or scoping reviews on the discussed topic is necessary. Scientific data and clinical reports should be continuously verified due to the high volume of research publications of insufficient quality and evidence strength.

Conclusions

The analysis of an interdisciplinary approach to the degeneration of IVDs, focused on the collaboration of specialists from various medical fields, constitutes a significant concept aimed at improving the effectiveness of treatment and the quality of care. Ultimately, understanding the essence of the problem and its complexity allows for the implementation of more effective therapeutic strategies. This approach requires a holistic view of the condition, taking into account biomedical, psychosocial, and interdisciplinary aspects. Further research and the development of clinical practice are crucial for refining diagnostic, therapeutic, and preventive methods related to this exceptionally complex clinical issue.

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