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REVIEW ARTICLE OPEN ACCESS

Hernia Nucleus Pulposus: Pathophysiology, Risk Factors, and Clinical Management – A Literature Review

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ABSTRACT

Background: Lumbar Herniated Nucleus Pulposus (HNP) is a leading cause of low back pain and radicular symptoms that significantly impair functional capacity and quality of life. The condition arises from a complex interaction of biomechanical stress, inflammatory responses, and degenerative biological changes in the intervertebral disc. This article aims to comprehensively review current evidence on the pathophysiology, risk factors, and clinical management of lumbar HNP using a narrative literature review approach.. **Methode:** A literature search was conducted across PubMed, Google Scholar, and accredited national journals using the keywords "hernia nucleus pulposus," "lumbar disc herniation," "low back pain," "pathophysiology," and "clinical management." Eligible articles were full-text publications in English or Indonesian published between 2015 and 2025, including original research, literature reviews, systematic reviews, and clinical practice guidelines. Studies related to traumatic or non-lumbar disc herniation and those without full-text availability were excluded. Selected articles were analyzed descriptively and synthesized narratively. **Results:** The review indicates that lumbar HNP is a progressive degenerative disorder influenced by altered disc biomechanics, inflammatory cascades, and cellular aging. Major risk factors include advancing age, increased body mass index, sedentary lifestyle, and occupational exposure to heavy physical workload. Clinical management follows a stepwise strategy, prioritizing conservative therapy, while surgical intervention is reserved for selected cases. **Conclusion:** A comprehensive understanding of the pathophysiological mechanisms and associated risk factors of lumbar HNP is essential to support evidence-based and individualized patient management.

Keywords: *Herniated nucleus pulposus, lumbar disc herniation, low back pain, pathophysiology, clinical management*.

INTRODUCTION

Herniated Nucleus Pulposus (HNP) is one of the most common degenerative disorders of the spine and a leading cause of low back pain and radicular pain. This condition occurs due to protrusion or extrusion of the nucleus pulposus through the annulus fibrosus, resulting in compression of adjacent neural structures, particularly in the lumbar segment. Radicular pain typically radiates along the dermatome distribution of the affected nerve. Globally, low back pain are often associated with HNP that represents a significant public health problem, contributing substantially to disability and reduced quality of life[1].

The prevalence of HNP increases with advancing age and is closely associated with intervertebral disc degeneration. Imaging studies have demonstrated that degenerative disc changes, including reduced disc height and disc bulging beyond normal anatomical boundaries (intervertebral

disc protrusion), can be observed even in asymptomatic individuals, indicating that disc degeneration is part of the natural spectrum of spinal aging[2]. However, under certain conditions, these degenerative changes may progress to disc herniation, in which the nucleus pulposus of the intervertebral disc extends beyond the annulus fibrosus and compresses nerve roots or the spinal cord. Such neural compression may become symptomatic and result in clinically significant impairment.

In addition to degenerative factors, various biological mechanisms contribute to the pathogenesis of HNP, including alterations in vertebral endplate structure and local inflammatory processes. Pathological changes such as Modic changes reflect a complex interaction between disc degeneration, inflammation, and the surrounding bone tissue response, which may exacerbate pain symptoms in patients with HNP. Modic changes of the intervertebral disc are signal intensity alterations in the vertebral subchondral bone (endplates and adjacent bone marrow) observed on magnetic resonance imaging (MRI) and are associated with disc degeneration[3]. Genetic factors are also known to influence individual susceptibility to intervertebral disc degeneration and HNP, thereby explaining interindividual clinical variability[4]. Lifestyle-related factors have also been increasingly recognized as contributors to chronic degenerative and metabolic conditions. Nutritional status and sedentary lifestyle patterns, including prolonged gaming behavior among adolescents and young adults, have been associated with increased metabolic health risks[5–7]. These factors may indirectly influence the progression and clinical burden of degenerative spinal disorders such as lumbar Herniated Nucleus Pulposus.

The clinical manifestations of lumbar HNP are not limited to low back pain and radiating pain but may also have a broader impact on daily functioning and overall quality of life. Recent studies have shown that pain intensity in patients with lumbar HNP is significantly associated with sleep disturbances, which may further aggravate both physical and psychological conditions[8]. These findings underscore that HNP is a multidimensional condition requiring a comprehensive clinical approach.

In clinical practice, MRI is the primary imaging modality for establishing the diagnosis of lumbar HNP due to its ability to accurately depict the degree of herniation and the involvement of neural structures[9]. Management of HNP is individualized and follows a stepwise approach, ranging from non invasive therapies to surgical intervention in selected cases. Various clinical guidelines emphasize the importance of conservative management as the first-line treatment, with further interventions considered based on therapeutic response and the severity of neurological deficits [10,11].

MATERIALS AND METHODS

This study was conducted using a narrative literature review approach to examine scientific evidence related to lumbar Herniated Nucleus Pulposus (HNP), including its pathophysiology, risk factors, and clinical management. A literature search was performed across PubMed, Google Scholar, and accredited national journals using the keywords “hernia nucleus pulposus,” “lumbar disc herniation,” “low back pain,” “pathophysiology,” and “clinical management.” The initial search identified 264 articles, of which 98 remained after duplicate removal. These articles were screened through title and abstract review, resulting in 65 studies eligible for full-text assessment. Finally, 41 articles met the inclusion criteria and were included in the analysis. The selection process followed a structured approach similar to PRISMA guidelines, adapted for a narrative review design.

Included studies were full-text articles published between 2015 and 2025, comprising original research, literature reviews, systematic reviews, and clinical practice guidelines in English or Indonesian. Articles not relevant to lumbar HNP, including traumatic or non-lumbar disc herniation and those without full-text availability, were excluded. Screening was conducted in three stages (title,

abstract, and full-text) to ensure study relevance. As a narrative review, this study is subject to potential selection bias and limited reproducibility; however, this was minimized by applying predefined inclusion and exclusion criteria and using multiple databases to ensure comprehensive coverage of relevant literature.

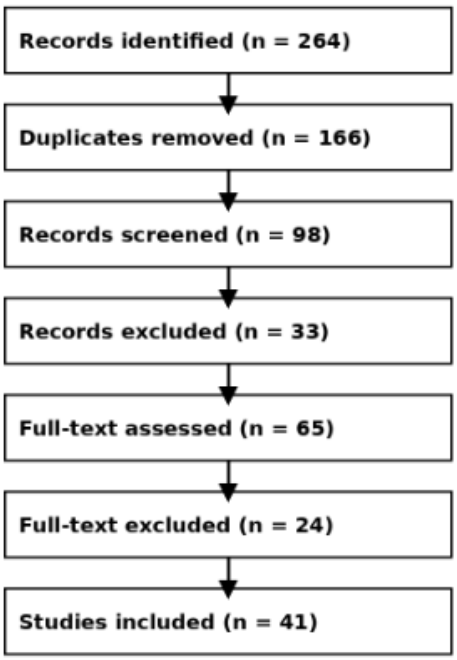

Figure 1. Flow diagram of study selection process

Included studies were full-text articles published between 2015 and 2025, comprising original research, literature reviews, systematic reviews, and clinical practice guidelines in English or Indonesian. Articles not relevant to lumbar HNP, including traumatic or non-lumbar disc herniation and those without full-text availability, were excluded. Screening was conducted in three stages (title, abstract, and full-text) to ensure study relevance. As a narrative review, this study is subject to potential selection bias and limited reproducibility; however, this was minimized by applying predefined inclusion and exclusion criteria and using multiple databases to ensure comprehensive coverage of relevant literature.

RESULTS

The results of this literature review are presented as a narrative synthesis of the main findings from relevant scientific publications on lumbar Herniated Nucleus Pulposus (HNP). The synthesis was conducted by grouping the evidence into three main themes: pathophysiology, risk factors, and clinical management. A summary of the reviewed findings is presented in Table 1, which integrates cross-study evidence along with scientific interpretation and clinical implications, and serves as the basis for further discussion of each theme.

A comparative analysis indicates that most studies consistently support the role of disc degeneration and inflammatory mechanisms in lumbar HNP. However, variability exists in the strength of association of lifestyle-related risk factors, such as BMI and occupational exposure, across

different populations. Evidence from systematic reviews and meta-analyses provides stronger support for biological mechanisms, while lifestyle factors are mainly derived from observational studies. Importantly, these themes are closely interconnected, where underlying pathophysiological processes are influenced by both modifiable and non-modifiable risk factors, which in turn determine the selection and effectiveness of clinical management strategies. These findings highlight the multifactorial nature of lumbar HNP and the heterogeneity of available evidence.

Table 1. Synthesis and Interpretation of the Literature on Lumbar Herniated Nucleus Pulposus

Theme	Synthesis of Literature Findings	Scientific & Clinical Interpretation	Sources
Pathophysiology	Intervertebral disc degeneration involves reduced hydration of the nucleus pulposus, disruption of the annulus fibrosus, impaired extracellular matrix homeostasis, and biomechanical alterations that promote disc herniation. Inflammatory processes contribute through increased levels of proinflammatory cytokines, leading to disc degradation and pain sensitization. Aging and cellular senescence further reduce the regenerative capacity of the intervertebral disc.	HNP represents a progressive degenerative process driven by both mechanical and biological mechanisms. Pain is not solely caused by nerve compression but also by inflammatory and molecular changes, indicating that therapeutic strategies should be multimodal and individualized.	12–17
Risk Factors	Risk factors for HNP are multifactorial and include age, body mass index, body weight changes, sedentary behavior, non-ergonomic working posture, weakness of spinal stabilizing muscles, and long-term exposure to physically demanding work. Occupational factors such as patient handling and repetitive heavy workload significantly increase the risk of HNP requiring surgical intervention.	Prevention of HNP requires a comprehensive approach that includes lifestyle modification, weight control, and ergonomic interventions in the workplace. Risk assessment should be incorporated as part of routine clinical evaluation.	18–28
Clinical Management	Management of lumbar HNP follows a stepwise approach based on pain severity, neurological status, and response to treatment. Conservative therapy is the first-line treatment for patients without neurological red flags and is recommended for 4–12 weeks before considering surgery, except in emergency situations. More than 80% of patients may experience spontaneous improvement, and meta-analyses indicate that disc resorption occurs more frequently in extrusion and sequestration types. Surgical intervention is indicated in cases of progressive or severe neurological deficits, cauda equina syndrome, persistent radicular pain refractory to conservative treatment, or uncontrolled severe pain. The North American Spine Society (NASS) guidelines recommend discectomy for persistent radicular symptoms lasting more than six weeks despite conservative management.	Lumbar HNP management strategies should be individualized and evidence-based, prioritizing conservative treatment in the early phase while reserving surgical intervention for cases with clear and measurable indications. Conservative options include pharmacological therapy, physiotherapy, epidural steroid injections, relaxation techniques, and acupuncture. Common surgical techniques include open discectomy and percutaneous endoscopic lumbar discectomy (PELD).	10,14,16,28–40

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DISCUSSION

3.1 Pathophysiology of Lumbar Herniated Nucleus Pulposus

Lumbar Herniated Nucleus Pulposus (HNP) represents the final manifestation of a gradual and progressive intervertebral disc degeneration process. This process begins with disruption of extracellular matrix homeostasis within the disc, characterized by a reduction in proteoglycan and water content in the nucleus pulposus, which compromises the disc's ability to absorb and evenly distribute mechanical loads. As a consequence, greater axial stress is transmitted to the annulus fibrosus, leading to progressive structural weakening and an increased risk of disc herniation[12,15]. Degenerative changes in the intervertebral disc are closely linked to cellular dysfunction, altered extracellular matrix turnover, and inflammatory signaling, consistent with fundamental principles of biomedical cell biology[41].

In addition to structural degeneration, biomechanical factors play a critical role in the pathogenesis of HNP. Uneven load distribution in the lumbar segments particularly during repetitive flexion and rotational movements accelerates annular damage. Once annular integrity is compromised, the nucleus pulposus may protrude or extrude from the disc space, resulting in direct compression of adjacent nerve roots. This mechanism explains the association between certain physical activities and the onset of clinical symptoms, particularly radicular pain, in patients with lumbar HNP[17].

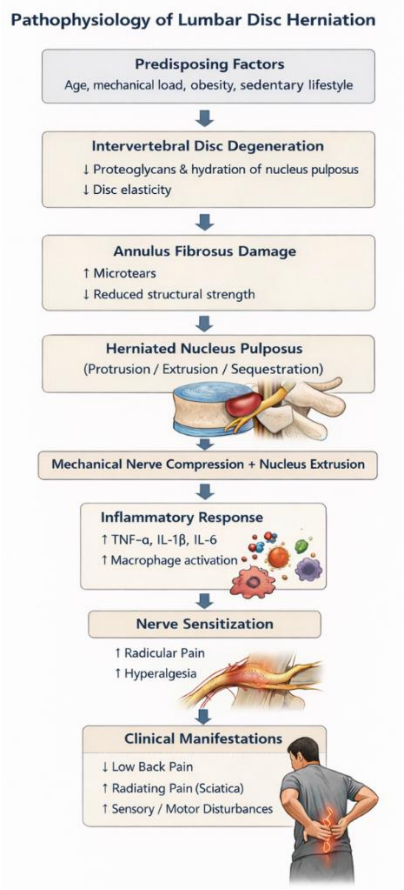

Figure 2. Pathophysiological diagram of lumbar Herniated Nucleus Pulposus (HNP).

Beyond mechanical mechanisms, inflammatory processes contribute substantially to pain generation and disease progression. Degenerated discs exhibit increased expression of proinflammatory cytokines, such as interleukin-1β and tumor necrosis factor-α, which promote extracellular matrix degradation and enhance nociceptive nerve sensitization. These inflammatory pathways help explain why pain severity in HNP does not always correlate with the degree of nerve compression observed on radiological imaging[13,14].

Recent research has further highlighted the role of aging and cellular senescence in the pathophysiology of HNP. Accumulation of senescent cells within disc tissue reduces regenerative capacity and creates a proinflammatory microenvironment. This condition not only accelerates disc degeneration but also limits tissue repair potential, thereby contributing to symptom chronicity in patients with lumbar HNP[16]. Consequently, lumbar HNP should be understood as a complex degenerative disorder involving the interplay of biomechanical stress, inflammatory responses, and biological aging processes.

Importantly, inflammatory mediators such as interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α) play a pivotal role in linking structural disc degeneration to clinical manifestations. These cytokines not only accelerate extracellular matrix degradation but also sensitize nociceptive nerve fibers, contributing to increased pain perception and the development of radiculopathy. This mechanism explains why the severity of clinical symptoms is not always proportional to the degree of nerve compression observed on imaging. Furthermore, these molecular pathways represent potential therapeutic targets, supporting the development of anti-inflammatory and biologic-based treatments in lumbar HNP[13,14,17].

3.2 Risk Factors for Herniated Nucleus Pulposus

Risk factors for lumbar Herniated Nucleus Pulposus (HNP) are multifactorial and reflect the interaction between individual characteristics and environmental exposures. Risk factors for lumbar HNP can be broadly classified into non-modifiable and modifiable factors to enhance clinical applicability and preventive strategies. Non-modifiable factors include age and genetic predisposition, which contribute to progressive intervertebral disc degeneration over time. In contrast, modifiable factors include body mass index (BMI), physical activity level, occupational exposure, and ergonomic posture. Among these, excessive mechanical loading associated with high BMI and prolonged occupational strain appears to exert the greatest clinical impact, as they directly increase intradiscal pressure and accelerate disc degeneration. This classification highlights the importance of targeted preventive strategies focusing on modifiable risk factors to reduce disease burden and progression. [19,20,24]

Systematic reviews indicate that age and body mass index (BMI) are important determinants in the development of intervertebral disc degeneration and lumbar disc herniation. With increasing age, the disc's ability to maintain its structural integrity and biomechanical function declines, thereby increasing susceptibility to HNP[19,26]. Changes in body weight also have significant clinical implications. Population-based cohort studies have shown that increases in BMI over a one-year period are associated with an increased risk of lumbar HNP. Furthermore, elevated BMI has been associated with postoperative recurrence, suggesting that excessive mechanical loading contributes not only to disease onset but also to its clinical course [24,25].

Occupational factors represent another important component of HNP risk. Long-term exposure to heavy physical activity, repetitive lifting, and patient handling among healthcare workers has been shown to increase the risk of lumbar HNP requiring surgical intervention. Meta-analyses and long-term longitudinal studies confirm that cumulative workload over many years contributes significantly to disc degeneration and herniation [20,22]. Although disc herniation is often associated with natural aging-related degeneration, chronic exposure to physically demanding work may

accelerate these degenerative processes at a younger age. High exposure to physical workload during early adulthood (20–40 years) has been linked to an increased risk of disc herniation compared with populations experiencing lighter occupational exposure, a trend consistently observed in long-term cohort studies among construction and manual handling workers[20].

Non-ergonomic working postures, such as prolonged forward bending and sustained non-neutral spinal alignment, increase mechanical stress on spinal structures, including the intervertebral discs. Repetitive compressive loading and non-neutral positioning of the lumbar spine elevate intradiscal pressure, weaken the annulus fibrosus, and promote displacement of the nucleus pulposus. Compared with neutral posture, these biomechanical conditions induce microdamage, accelerate early disc degeneration, and increase the likelihood of disc herniation [21].

Non-ergonomic posture also imposes prolonged static loading on paraspinal muscles, leading to musculoskeletal fatigue and reduced spinal stabilization. Weakness and atrophy of supporting musculature significantly increase compressive forces acting on the intervertebral disc, thereby accelerating disc degeneration. Reduced muscular stabilization shifts excessive mechanical load to the disc, which would otherwise be distributed by active musculature [18,28]. In addition, sedentary lifestyle and low levels of physical activity further increase the risk of lumbar spinal disorders. Longitudinal meta-analyses have demonstrated that sedentary behavior is associated with an increased incidence of low back pain and intervertebral disc degeneration, underscoring the importance of active lifestyle modification as part of preventive strategies for HNP in both the general population and high-risk groups[23,27].

Overall, modifiable risk factors play a critical role in both the onset and progression of lumbar HNP, suggesting that early intervention through lifestyle modification and ergonomic improvement may significantly reduce long-term morbidity. [23,27]

3.3 Clinical Management of Herniated Nucleus Pulposus

Management of lumbar HNP is based on a stepwise approach that considers pain severity, neurological deficits, and response to initial therapy. Evidence-based management of lumbar HNP is supported by a hierarchy of clinical evidence, with randomized controlled trials (RCTs) and meta-analyses providing the highest level of recommendation. Current evidence indicates that more than 80% of patients undergoing conservative treatment experience significant symptom improvement within the first 6–12 weeks. In comparison, surgical intervention such as discectomy may provide faster pain relief, particularly in patients with persistent radiculopathy; however, long-term outcomes between surgical and non-surgical approaches are often comparable. Several systematic reviews have reported similar functional outcomes at 1–2 years follow-up despite earlier symptom resolution in surgically treated patients. These findings support a stepwise and individualized treatment approach. [32,33,37]

Management of lumbar HNP is based on a stepwise approach that consider pain severity, neurological deficit, and response to initial therapy. Current evidence supports conservative management as the first-line treatment for early-stage HNP, aiming to relieve pain and restore function without invasive intervention. This approach includes pharmacological therapy, physiotherapy, and patient education[33,36]. Conservative therapy is recommended for patients who do not present neurological red flags such as cauda equina syndrome or progressive motor deficits. It is particularly appropriate for patients with low back pain and/or mild to moderate radiculopathy, absence of progressive neurological deficits, and symptom duration of less than 6–8 weeks. Several studies report spontaneous symptom improvement within the first few weeks in more than 80% of patients without surgical intervention [32].

MRI findings that support conservative management include mild disc protrusion, disc extrusion, or sequestration without neurological deficits, as well as mild to moderate nerve root

compression. Evidence suggests that spontaneous resorption of herniated disc material occurs more frequently in extrusion and sequestration types[34,35]. Conservative treatment is generally recommended for approximately 4–12 weeks before considering surgery, unless emergency neurological signs are present. Regular clinical evaluation is essential to monitor symptom progression, neurological status, and therapeutic response[10,32]. Several long-term studies indicate that clinical outcomes of surgical and non-surgical treatments may be comparable in certain patients, particularly regarding pain relief and functional improvement over the medium to long term[37,38]. Beyond conventional treatment, adjunctive interventions have demonstrated potential benefits in HNP management. Epidural steroid injections may reduce inflammation and radicular pain and serve as a bridging therapy before surgical decision-making. Prospective studies suggest that this approach can delay or even avoid surgery in selected patients[40].

Multimodal strategies combining non-pharmacological and complementary therapies are increasingly emphasized. Structured physiotherapy programs focus on strengthening lumbar stabilizing muscles to reduce disc loading and support tissue recovery. Common modalities include lumbar mobilization, McKenzie exercises, muscle stretching, and moderate-strength training programs[32]. Structured physiotherapy, relaxation therapy, and acupuncture have been reported to reduce pain and improve quality of life in patients with lumbar HNP. Clinical trials and case reports indicate that integrating conventional and complementary therapies may yield better outcomes than single-modality approaches, particularly in patients with chronic pain[29,31,39].

Severe HNP manifestations such as progressive or significant neurological deficits, cauda equina syndrome, persistent radicular pain unresponsive to conservative treatment, or uncontrolled severe pain that require surgical intervention. Guidelines from the North American Spine Society (NASS) recommend discectomy as an effective treatment for patients with radicular symptoms persisting for more than six weeks despite conservative therapy [10]. The conventional standard surgical procedure remains open discectomy using a posterior approach with a relatively larger incision and partial laminotomy to remove nucleus pulposus fragments compressing the nerve root. A minimally invasive alternative with faster recovery is Percutaneous Endoscopic Lumbar Discectomy (PELD), performed via transforaminal or interlaminar endoscopic approaches. Meta-analyses demonstrate comparable pain outcomes with shorter recovery times compared to open discectomy[10,28,30].

In clinical practice the management of lumbar HNP should be individualized based on clinical presentation and supported by high-level evidence. Conservative treatment remains the cornerstone of early management, while surgical and adjunctive interventions should be reserved for well-defined indications to optimize patient outcomes and resource utilization.

In addition to conventional and surgical approaches, emerging regenerative therapies have gained increasing attention in the management of lumbar HNP. These include mesenchymal stem cell therapy and platelet-rich plasma (PRP), which aim to restore disc structure and modulate inflammatory processes. Early evidence suggests that these approaches may promote extracellular matrix regeneration and reduce pain by targeting underlying biological mechanisms. However, current evidence remains limited, as most studies are preclinical or small-scale clinical trials. Therefore, further high-quality randomized controlled trials are needed to establish their long-term efficacy and safety[14,16,33].

CONCLUSION

Lumbar Herniated Nucleus Pulposus (HNP) is a complex degenerative condition resulting from the interaction of biomechanical alterations, inflammatory processes, and biological mechanisms within the intervertebral disc, with multifactorial risk factors including individual characteristics, lifestyle behaviours, and long-term occupational exposure contributing to its onset and progression. Clinically, these findings highlight the importance of early risk identification,

particularly targeting modifiable factors such as obesity, sedentary behaviour, and occupational strain, to prevent disease progression and reduce long-term disability. Management of lumbar HNP should follow a stepwise, evidence-based and individualized approach, prioritizing conservative therapy as the first-line strategy while reserving surgical and complementary interventions for selected cases to optimize patient outcomes. Furthermore, future research should focus on the development of biomarker-based diagnostic tools and the evaluation of emerging regenerative therapies to support more precise, mechanism-based, and personalized management of lumbar HNP.

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Authors' contributions

All authors contributed equally to the study design, data collection and analysis, and manuscript preparation. All approved the final version and take full responsibility for the accuracy and integrity of the work.

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Conflict of interest

The authors declare that they have no conflict of interest.

REFERENCES

1. Hartvigsen J, Hancock MJ, Kongsted A, et al. What low back pain is and why we need to pay attention. *Lancet*. 2018;391(10137):2356-2367. doi:10.1016/S0140-6736(18)30480-X

2. Brinjikji W, Luetmer PH, Comstock B, et al. Systematic Literature Review of Imaging Features of Spinal Degeneration in Asymptomatic Populations. *Am J Neuroradiol*. 2015;36(4):811-816. doi:10.3174/ajnr.A4173

3. Dudli S, Fields AJ, Samartzis D, Karppinen J, Lotz JC. Pathobiology of Modic changes. *Eur Spine J*. 2016;25(11):3723-3734. doi:10.1007/s00586-016-4459-7

4. Zhang Y, Sun Z, Liu J, Guo X. Advances in Susceptibility Genetics of Intervertebral Degenerative Disc Disease. *Int J Biol Sci*. Published online 2008:283-290. doi:10.7150/ijbs.4.283

5. Hermawati L, Irawati NBU, Zulfa HA, Diana WA. The Role of Balanced Nutrition Knowledge in Influencing Nutritional Status and Health Risks: A Literature Review. *Int J Med Public Heal*. 2025;2(1):1-14. https://jurnal.untirta.ac.id/index.php/inomed/article/view/31567

6. Darifah S, Trisnasari E, Yuniarti TE, Sugiharto A, Wulansari ER, Hermawati L. Skrining gizi dan edukasi nutrisi untuk pencegahan masalah gizi dan metabolik komunitas e-sport. *J Pembelajaran Pemberdaya Masy*. 2025;6(3):983-992. doi:10.33474/jp2m.v6i3.24080

7. Darifah S, Hermawati L, Wulansari ER, Eva A, Agustianti E, Asma F. Remaja dan Gaming : Perkembangan Gaya Hidup Sedentari dan Risiko Kesehatan. 2025;1(3):38-50.

doi:10.58218/jhp.v1i3.1850

8. Sholicha Dyananti AL, Yekti M, Rohmani A. HUBUNGAN INTENSITAS NYERI DENGAN KUALITAS TIDUR: STUDI PADA PENDERITA HERNIA NUKLEUS PULPOSUS (HNP) LUMBAL. *Cerdika J Ilm Indones*. 2023;3(09):850-856. doi:10.59141/cerdika.v3i09.670

9. Ameliya Widya Astuti, I Made Lana Prasetya, Tri Asih Budiarti. Penatalaksanaan Pemeriksaan Magnetic Resonance Imaging (MRI) Lumbal Dengan Kasus Hernia Nukleus Pulposus. *J Anestesi*. 2023;2(1):331-342. doi:10.59680/anestesi.v2i1.806

10. Kreiner DS, Hwang SW, Easa JE, et al. An evidence-based clinical guideline for the diagnosis and treatment of lumbar disc herniation with radiculopathy. *Spine J*. 2014;14(1):180-191. doi:10.1016/j.spinee.2013.08.003

11. 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

12. Oichi T, Taniguchi Y, Oshima Y, Tanaka S, Saito T. Pathomechanism of intervertebral disc degeneration. *JOR SPINE*. 2020;3(1). doi:10.1002/jsp2.1076

13. Risbud M V., Shapiro IM. Role of cytokines in intervertebral disc degeneration: pain and disc content. *Nat Rev Rheumatol*. 2014;10(1):44-56. doi:10.1038/nrrheum.2013.160

14. Molinos M, Almeida CR, Caldeira J, Cunha C, Gonçalves RM, Barbosa MA. Inflammation in intervertebral disc degeneration and regeneration. *J R Soc Interface*. 2015;12(104):20141191. doi:10.1098/rsif.2014.1191

15. Rider SM, Mizuno S, Kang JD. Molecular Mechanisms of Intervertebral Disc Degeneration. *Spine Surg Relat Res*. 2019;3(1):1-11. doi:10.22603/ssrr.2017-0095

16. Silwal P, Nguyen-Thai AM, Mohammad HA, et al. Cellular Senescence in Intervertebral Disc Aging and Degeneration: Molecular Mechanisms and Potential Therapeutic Opportunities. *Biomolecules*. 2023;13(4):686. doi:10.3390/biom13040686

17. Zhou M, Theologis AA, O'Connell GD. Understanding the etiopathogenesis of lumbar intervertebral disc herniation: From clinical evidence to basic scientific research. *JOR SPINE*. 2024;7(1). doi:10.1002/jsp2.1289

18. Alessa F, Ning X. Changes of lumbar posture and tissue loading during static trunk bending. *Hum Mov Sci*. 2018;57:59-68. doi:10.1016/j.humov.2017.11.006

19. Hoffeld K, Lenz M, Egenolf P, et al. Patient-related risk factors and lifestyle factors for lumbar degenerative disc disease: a systematic review. *Neurochirurgie*. 2023;69(5):101482. doi:10.1016/j.neuchi.2023.101482

20. Wahlström J, Liv P, Stjernbrandt A, et al. Occupational risk factors for surgically treated lumbar disc herniation – a 33-year follow-up. *Scand J Work Environ Health*. 2025;51(6):550-558. doi:10.5271/sjweh.4253

21. Jo M, Chae SW. Stress analysis of intervertebral disc during occupational activities. *Comput Methods Programs Biomed*. 2021;208:106298. doi:10.1016/j.cmpb.2021.106298

22. Schröder C, Nienhaus A. Intervertebral Disc Disease of the Lumbar Spine in Health Personnel with

Occupational Exposure to Patient Handling—A Systematic Literature Review and Meta-Analysis. *Int J Environ Res Public Health*. 2020;17(13):4832. doi:10.3390/ijerph17134832

23. Alzahrani H, Alshehri MA, Alzhrani M, Alshehri YS, Al Attar WSA. The association between sedentary behavior and low back pain in adults: a systematic review and meta-analysis of longitudinal studies. *PeerJ*. 2022;10:e13127. doi:10.7717/peerj.13127

24. Fotakopoulos G, Makris D, Kotlia P, Tzerefos C, Fountas K. Recurrence Is Associated With Body Mass Index in Patients Undergoing a Single-Level Lumbar Disc Herniation Surgery. *J Clin Med Res*. 2018;10(6):486-492. doi:10.14740/jocmr3121w

25. Masuda S, Fukasawa T, Matsuda S, Kawakami K. One-year body mass index change and subsequent risk of lumbar disc herniation: a population-based cohort study. *Eur Spine J*. Published online October 30, 2025. doi:10.1007/s00586-025-09508-2

26. Hincapié CA, Kroismayr D, Hofstetter L, et al. Incidence of and risk factors for lumbar disc herniation with radiculopathy in adults: a systematic review. *Eur Spine J*. 2025;34(1):263-294. doi:10.1007/s00586-024-08528-8

27. Pojskic M, Bisson E, Oertel J, Takami T, Zygourakis C, Costa F. Lumbar disc herniation: Epidemiology, clinical and radiologic diagnosis WFNS spine committee recommendations. *World Neurosurg X*. 2024;22:100279. doi:10.1016/j.wnsx.2024.100279

28. Qin B, Baldoni M, Wu B, Zhou L, Qian Z, Zhu Q. Effect of lumbar muscle atrophy on the mechanical loading change on lumbar intervertebral discs. *J Biomech*. 2022;139:111120. doi:10.1016/j.jbiomech.2022.111120

29. Lum UI. Physiotherapy Management of Lumbar Herniated Nucleus Pulposus: A Case Report. *Maj Ilm Fisioter Indones*. 2025;13(3):492-497. doi:10.24843/mifi.000000355

30. MEYER G, DA ROCHA ID, CRISTANTE AF, et al. Percutaneous Endoscopic Lumbar Discectomy Versus Microdiscectomy for the Treatment of Lumbar Disc Herniation: Pain, Disability, and Complication Rate—A Randomized Clinical Trial. *Int J Spine Surg*. 2020;14(1):72-78. doi:10.14444/7010

31. Tu JF, Shi GX, Yan SY, et al. Acupuncture vs Sham Acupuncture for Chronic Sciatica From Herniated Disk. *JAMA Intern Med*. 2024;184(12):1417. doi:10.1001/jamainternmed.2024.5463

32. Yaman O, Gushcha A, Vaishya S, Zileli M, Zygourakis C, Oertel J. The role of conservative treatment in lumbar disc herniations: WFNS spine committee recommendations. *World Neurosurg X*. 2024;22:100277. doi:10.1016/j.wnsx.2024.100277

33. Awadalla AM, Aljulayfi AS, Alrowaili AR, et al. Management of Lumbar Disc Herniation: A Systematic Review. *Cureus*. Published online October 29, 2023. doi:10.7759/cureus.47908

34. Chiu CC, Chuang TY, Chang KH, Wu CH, Lin PW, Hsu WY. The probability of spontaneous regression of lumbar herniated disc: a systematic review. *Clin Rehabil*. 2015;29(2):184-195. doi:10.1177/0269215514540919

35. Zhong M, Liu JT, Jiang H, et al. Incidence of spontaneous resorption of lumbar disc herniation: A meta-analysis. *Pain Physician*. 2017;20(1):E45-E52. doi:10.36076/ppj.2017.1.e45

36. Deyo RA, Mirza SK. Herniated Lumbar Intervertebral Disk. Solomon CG, ed. *N Engl J Med*. 2016;374(18):1763-1772. doi:10.1056/NEJMcp1512658

37. Liu C, Ferreira GE, Abdel Shaheed C, et al. Surgical versus non-surgical treatment for sciatica: systematic review and meta-analysis of randomised controlled trials. *BMJ*. 2023;381:e070730. doi:10.1136/bmj-2022-070730

38. Lurie JD, Tosteson TD, Tosteson ANA, et al. Surgical Versus Nonoperative Treatment for Lumbar Disc Herniation. *Spine (Phila Pa 1976)*. 2014;39(1):3-16. doi:10.1097/BRS.0000000000000088

39. Poquet N, Lin CWC. Management strategies for sciatica (PEDro synthesis). *Br J Sports Med*. 2016;50(4):253-254. doi:10.1136/bjsports-2015-095268

40. Raymaekers V, Roosen G, Put E, et al. Transforaminal epidural steroid injection for radiculopathy and the evolution to surgical treatment: a pragmatic prospective observational multicenter study. *Pain Manag*. 2024;14(4):173-182. doi:10.2217/pmt-2023-0121

41. Kasemen K, Fathimah I, Ahyani A, Alawiyah F. Pelatihan Pengolahan Kulit Melinjo (Gnetum genemon L .) Menjadi Nugget dan Sosis sebagai Upaya Optimalisasi Potensi Lokal di. 2025;5(8):1007-1014.