## HAYDARPAŞA NUMUNE MEDICAL JOURNAL

DOI: 10.14744/hnhj.2023.35219 Haydarpasa Numune Med J 2023;63(3):329–333

ORIGINAL ARTICLE



# Effects of Blood Hemoglobin Levels on Vertebral Discal Degeneration

# Eyüp Çetin<sup>1</sup>, Volkan Şah<sup>2</sup>, Mustafa Arslan<sup>3</sup>, Özkan Arabacı<sup>3</sup>, Mehmet Edip Akyol<sup>3</sup>, İlker Ünlü<sup>4</sup>

<sup>1</sup>Department of Brain and Nerve Surgery, Health Sciences University Haydarpaşa Numune Training and Research Hospital, Istanbul, Türkiye
<sup>2</sup>Department of Sports Medicine, Van Yüzüncü Yıl University Faculty of Medicine, Van, Türkiye
<sup>3</sup>Department of Neurosurgery, Van Yüzüncü Yıl University Faculty of Medicine, Van, Türkiye
<sup>4</sup>Department of Neurosurgery, Esenyurt University, İstanbul, Türkiye

#### Abstract

**Introduction:** Vertebral discopathies are one of the most important causes of low back pain, which is very common in the community. Although there are many biochemical and mechanical causes of discogenic low back pain, the most important is discal degeneration. Many studies have been conducted suggesting that disc degeneration could be associated with various factors. In our study, we aimed to reveal a possible relationship between disc degeneration and levels of hemoglobin (HGB).

**Methods:** We investigated the connection between discal degeneration and certain parameters, especially HGB levels, in a total of 174 patients in two separate groups; those with and without disc degeneration. Age, weight, height, body mass index, chronic diseases, mean HGB, hematocrit (HTC) level, white blood cell (WBC), high-density lipoprotein, low-density lipoprotein (LDL), and triglyceride (TG) values of the patients were recorded.

**Results:** No significant difference was detected (p>0.05) between the groups with and without degeneration in terms of HGB, HTC, WBC, and LDL values. The TG level in the group with disc degeneration was significantly (p<0.05) higher than the group without disc degeneration.

**Discussion and Conclusion:** Lumbar disc degeneration is a public health problem that continues to be discussed in the literature. There was no significant difference in terms of HGB levels between the groups, with and without degenerated disc. **Keywords:** Discal degeneration; discogenic pain; hemoglobin; low back pain.

A fter the discovery of the relationship between discal degeneration and low back pain, studies were conducted to investigate the underlying causes of disc degeneration and to slow down the degeneration process. Significant results have been achieved in this field, thanks to the ever-increasing data on the biology and biomechanical behavior of the intervertebral disc<sup>[1]</sup>.

With the use of spinal magnetic resonance imaging (MRI), which can clearly detect disc degeneration, an opportunity has emerged for dual studies showing the importance of hereditary factors in the formation of disc degeneration. Spinal MRI has also revealed that many people have disc degeneration despite being asymptomatic<sup>[2]</sup>.

The micromolecular composition of the intervertebral disc,

**Correspondence:** Eyüp Çetin, M.D. Department of Brain and Nerve Surgery, Health Sciences University Haydarpaşa Numune Training and Research Hospital, Istanbul, Türkiye



Submitted Date: 13.01.2023 Revised Date: 26.02.2023 Accepted Date: 14.03.2023

Copyright 2023 Haydarpaşa Numune Medical Journal OPEN ACCESS This is an open access article under the CC BY-NC license (http://creativecommons.org/licenses/by-nc/4.0/).



whose main task is to transmit the load-bearing work to a lower vertebra in a balanced way and to provide spinal movement, is formed by the organization of fibrillar collagens, proteoglycan, water, and the extracellular matrix<sup>[3]</sup>. The normal intervertebral disc is a fibrocartilaginous structure of three parts: The outer annulus fibrosus, consisting of fibroblast-like cells, and Type I collagen, the inner soft nucleus pulposus composed of chondrocyte-like cells and water, and the cartilage end plate<sup>[4]</sup>. The production and protection of all these molecules in the disc structure are provided by the few resident cells in that region, and a dynamic change process continues throughout life<sup>[5]</sup>.

Disc degeneration, which is an important cause of discogenic low back pain, is generally considered to be an aging process of disc tissue caused by reduced proteoglycan content, resulting in declined intervertebral disc height, endplate sclerosis, and osteophyte formation<sup>[6]</sup>.

Low back pain, the major cause of which is considered to be intervertebral disc degeneration, is a common public health problem that causes problems in the economic and social quality of life worldwide<sup>[7]</sup>. Although disc degenerations are mostly asymptomatic, they are also associated with sciatic pain, disc protrusion, and herniation<sup>[8]</sup>. Since the height of the intervertebral disc has an important role in the biomechanics of the spine, a disruption here may adversely affect other spinal tissues, including muscles and ligaments<sup>[9]</sup>.

Disc degeneration, which was previously considered to be related only to age-related and mechanical features, has been shown to be associated also with genetic and nutritional factors in the past decade<sup>[10]</sup>. Genes related to collagen, which have important effects on the mechanical features of the disc, and genes related to regulatory pathways that can change the metabolism of disc cells have been identified<sup>[11]</sup>. In this study, we tried to reveal whether there is a connection between some blood measurement parameters and disc degeneration.

# **Materials and Methods**

This retrospective study was approved by the Ethics Committee (with the decision dated April 15, 2021, and numbered 2021/05-10). After obtaining the approval of the Ethics Committee, data on individuals between the ages of 18 and 100 with and without degenerated discs were collected retrospectively through the Enlil HBYS system, which is the patient admission and follow-up system in our hospital, from the records of patients who had lumbar magnetic resonance imaging (MRI) due to low back pain. Data from patients with lumbar disc herniation secondary to lumbar disc degeneration were also included in the study. Patients who applied to our hospital in the past 5 years were randomly selected. Data on a total of 174 patients older than 18 years of age, of both sexes, with and without lumbar degenerated disc were collected. Lumbar MRIs of selected patients were interpreted by a radiologist. Age, weight, height, body mass index, chronic diseases, mean hemoglobin (HGB), hematocrit (HTC) level, white blood cell (WBC), high-density lipoprotein (HDL), lowdensity lipoprotein (LDL), and triglyceride (TG) values of the patients were recorded.

#### **Statistical Analysis**

Mean, standard deviation, median, minimum, maximum, frequency, and ratio rates were used in the descriptive statistics of the data. The dispersion of variables is measured with the Kolmogorov–Smirnov test. Independent sample t-test and the Mann–Whitney u-test were used in the analysis of quantitative independent data. The chi-square test was used in the analysis of qualitatively independent data. The Statistical Package for the Social Sciences 28.0 program was used in the analysis (Table 1).

#### Results

The participants' mean age in the group with disc degeneration was significantly (p<0.05) higher than the group without disc degeneration. The ratio of male patients

Table 1. Evaluation of demographic data						
Min–Max	Median	Mean±SD/n-%				
18.0-85.0	40.0	42.0±15.5				
	106	60.9%				
	68	39.1%				
150.0-185.0	170.0	169.8±8.7				
50.0-100.0	78.0	76.2±15.2				
17.3-35.6	25.9	26.5±5.4				
8.9-18.8	14.4	14.3±1.8				
30.3-58.4	43.2	43.1±4.9				
3.8-21.2	8.4	8.7±2.8				
39.0-400.0	109.0	110.1±49.1				
20.0-92.0	46.0	48.7±15.1				
38.0-555.0	145.5	160.8±103.9				
	99	56.9%				
	75	43.1%				
	76.0	43.7%				
	98.0	56.3%				
	Min-Max 18.0-85.0 150.0-185.0 50.0-100.0 17.3-35.6 8.9-18.8 30.3-58.4 3.8-21.2 39.0-400.0 20.0-92.0 38.0-555.0	Min-Max         Median           18.0-85.0         40.0           18.0-85.0         106           68         68           150.0-185.0         170.0           50.0-100.0         78.0           17.3-35.6         25.9           8.9-18.8         14.4           30.3-58.4         43.2           3.8-21.2         8.4           39.0-400.0         109.0           20.0-92.0         46.0           38.0-555.0         145.5           99         75           76.0         98.0				

1	5 1		
	MR Deger	MR Degeneration (-)	
	Mean±SD	Median	Mean±SD
Age	32.2±10.3	31.0	49.6±14.6
Gender			
Female	56	73.7%	50
Male	20	26.3%	48

170.2±10.3

72.5±17.5

Table 2.	Comparisor	of groups
----------	------------	-----------

Height

Weight

BMI	25.0±5.9	24.7	27.4±5.1	26.1	
HGB	14.2±1.7	14.1	14.4±1.9	14.6	
HTC	42.8±4.8	42.3	43.4±5.0	43.7	
WBC	8.2±2.7	8.0	9.0±2.8	8.5	
LDL	104.7±31.9	110.0	113.9±58.3	98.5	
HDL	56.6±14.3	57.0	42.9±13.2	38.5	
TG	125.8±74.1	111.0	185.5±115.4	165.5	
Chronic disease					
(-)	60	78.9%	39	39.8%	
(+)	16	21.1%	59	60.2%	
<sup>t</sup> t test/ <sup>m</sup> Mann–Whitney U-test / X <sup>2</sup> Chi-square test.					

170.0

74.0

in the group with disc degeneration was significantly (p<0.05) higher than the group without disc degeneration. No significant difference (p>0.05) was found between the groups with and without MR degeneration in terms of height, weight, and BMI values of the patients. The mean HGB, HTC, WBC, and LDL levels did not vary significantly (p>0.05) between groups with and without disc degeneration (Table 2). The mean HDL level in the

group with disc degeneration was significantly (p<0.05) lower than the group without disc degeneration. The mean TG level in the group with disc degeneration was significantly (p<0.05) higher than the group without disc degeneration. The rate of chronic disease in the group with disc degeneration was significantly (p<0.05) higher than the rate in the group without disc degeneration (Table 2).

**MR Degeneration (-)** 

169.5±7.9

78.2±13.9

Median

47.5

51.0%

49.0%

170.0

81.0



Figure 1. The relationship between age and degeneration.



331

р

0.000<sup>m</sup>

0.000<sup>X<sup>2</sup></sup>

0.846<sup>t</sup>

0.350<sup>t</sup>

0.269<sup>t</sup>

0.482<sup>t</sup>

0.481<sup>t</sup>

0.049<sup>t</sup>

0.649<sup>t</sup>

0.001<sup>t</sup>

0.028<sup>t</sup>

0.000<sup>X<sup>2</sup></sup>

Figure 2. The relationship between gender and disc degeneration.

Figure 3. The relationship between TG level and disc degeneration.

### Discussion

Some studies have suggested that intervertebral disc degeneration may be a complex phenomenon caused by decreased nutrient supply in intervertebral disc tissue, changes in extracellular matrix components in intervertebral discs, an increased amount of apoptosis, biomechanical changes, and autoimmunity<sup>[12]</sup>.

Various studies have been conducted on the coexistence of lumbar degeneration and other diseases. Uysal et al.<sup>[13]</sup> revealed that osteoporosis and lumbar spine degeneration are associated with each other. In our study, it was concluded that degeneration increased with age, which is consistent with the literature<sup>[14]</sup> (Fig. 1). Due to progressive apoptosis, the amount of fibrous tissue increases, the composition and amount of proteoglycans change, and the number of cells decreases. Different factors such as mechanical, traumatic, genetic, and nutrition have important roles in the degenerative process<sup>[15]</sup>.

Disc tissue meets the necessary oxygen and nutrient requirements by providing vascular nutrition through diffusion from the cartilage end plates. One of the causes of disc degeneration is the decrease of nutrition provided by diffusion from the cartilage end plates. In addition, disc tissue, such as all body tissues, needs oxygen and glucose<sup>[16]</sup>. We thought that the levels of elements (such as HGB) that carry these substances in the blood may affect disc degeneration indirectly, if not directly.

According to the results of the blood samples taken from the patients included in our study, there was no statistical difference in HGB levels between the groups with and without disc degeneration (Table 2). In the study of Chang



et al.,<sup>[17]</sup> it was shown that there is a relationship between lumbar disc degeneration and anemia. Although there are publications stating that gender is not an influential factor in disc degeneration, it was shown in this trial that disc degeneration is more common in the male gender<sup>[18]</sup> (Fig. 2). We estimate that this result occurred due to the harsh working conditions of the male gender in our region.

The effect of factors such as height, weight, and BMI in patients with disc degeneration is controversial in the literature. In our study, we determined that these factors had no effect on disc degeneration. In a study of patients who underwent surgery for lumbar intervertebral disc herniation, it was demonstrated that excess weight significantly affects disc degeneration<sup>[19]</sup>. However, a cross-sectional Xray study showed that being overweight had no effect on the prevalence of disc degeneration among construction workers and painters<sup>[20]</sup>. This suggests that weight, height, and BMI do not have an effect on disc degeneration alone.

Although no relationship between serum LDL level and disc degeneration was observed in our study, it was concluded that TG levels were related to disc degeneration (Fig. 3). In the literature, it has been reported that serum lipid levels were related to disc degeneration and herniation, and serum lipid levels have been demonstrated to be high in disc degenerations<sup>[21]</sup>.

It is seen that chronic diseases are also a factor in disc degeneration, in addition to degeneration occurring throughout the body (Fig. 4). In all major chronic diseases, it has been proven that cell aging occurs in the cardiovascular system, the nervous system, and especially in the musculoskeletal system<sup>[22]</sup>.

Since the data of our study were obtained retrospec-





tively, we excluded the data of the patients whose HGB values could not be obtained from our study, but the deficiencies in the lipid profile of some patients included in the study constitute a missing aspect of our study. At the same time, we accept that the time-dependent differences in blood values and radiological imaging are another limitation of our study. The studies in which radiological imaging and blood tests are performed simultaneously will undoubtedly reveal more valuable results. There is also a need for studies to be obtained from larger patient series.

#### Conclusion

As a result, we predicted that there may be a relationship between blood parameters and disc degeneration in addition to aging and genetic factors. As in previous studies, we observed in our study that blood lipid levels were parallel to disc degeneration. In our study, we revealed that there was no relationship between blood HGB level and disc degeneration.

**Ethics Committee Approval:** This study was approved by the Ethics Committee (with the decision dated April 15, 2021, and numbered 2021/05-10). It was performed in accordance with the Principles of the Declaration of Helsinki.

#### Peer-review: Externally peer-reviewed.

**Authorship Contributions:** Concept: E.Ç.; Design: E.Ç., M.A.; Supervision: E.Ç., M.A Fundings: M.A; Materials: E.Ç., M.A., Ö.A.; Data Collection and/or Processing: M.A., İ.Ü.; Analysis and/or Interpretation: V.Ş., M.E.A., Ö.A.; Literature Search: İ.Ü.; Writing: E.Ç.; Critical Review: M.E.A., V.Ş.

#### Conflict of Interest: None declared.

**Financial Disclosure:** The authors declared that this study received no financial support.

#### References

- Urban JPG, Fairbank JCT. Current perspectives on the role of biomechanical loading and genetics in development of disc degeneration and low back pain; A narrative review. J Biomech 2020;102:109573.
- Filler A. Magnetic resonance neurography and diffusion tensor imaging: Origins, history, and clinical impact of the first 50,000 cases with an assessment of efficacy and utility in a prospective 5000-patient study group. Neurosurgery 2009;65(Suppl 4):A29–43.
- Sivan SS, Hayes AJ, Wachtel E, Caterson B, Merkher Y, Maroudas A, et al. Biochemical composition and turnover of the extracellular matrix of the normal and degenerate intervertebral disc. Eur Spine J 2014;23(Suppl 3):S344–53
- Rodrigues-Pinto R, Berry A, Piper-Hanley K, Hanley N, Richardson SM, Hoyland JA. Spatiotemporal analysis of putative notochordal cell markers reveals CD24 and keratins 8, 18, and 19

as notochord-specific markers during early human intervertebral disc development. J Orthop Res 2016;34:1327–40.

- 5. Buckwalter JA. Aging and degeneration of the human intervertebral disc. Spine (Phila Pa 1976) 1995;20:1307–14.
- 6. Sharma A. The role of adipokines in intervertebral disc degeneration. Med Sci (Basel) 2018;6:34.
- Liu H, Kang H, Song C, Lei Z, Li L, Guo J, et al. Urolithin a inhibits the catabolic Effect of TNFα on nucleus pulposus cell and alleviates intervertebral disc degeneration in vivo. Front Pharmacol 2018;9:1043.
- 8. Luoma K, Riihimäki H, Luukkonen R, Raininko R, Viikari-Juntura E, Lamminen A. Low back pain in relation to lumbar disc degeneration. Spine (Phila Pa 1976) 2000;25:487–92.
- Pimenta L, Marchi L, Oliveira L, Nogueira-Neto J, Coutinho E, Amaral R. Elastomeric lumbar total disc replacement: Clinical and radiological results with minimum 84 months follow-up. Int J Spine Surg 2018;12:49–57.
- 10. Cheung KM. The relationship between disc degeneration, low back pain, and human pain genetics. Spine J 2010;10:958–60.
- Song YQ, Ho DW, Karppinen J, Kao PY, Fan BJ, Luk KD, et al. Association between promoter -1607 polymorphism of MMP1 and lumbar disc disease in Southern Chinese. BMC Med Genet 2008;9:38.
- 12. Jin L, Balian G, Li XJ. Animal models for disc degeneration-an update. Histol Histopathol 2018;33:543–54.
- 13. Uysal M, Gürün U, Kochai A, Özalay M. The correlation between the degenerative changes and osteoporosis in the lumbar spine of elderly patients. Sakarya Med J 2016;6:207–11.
- Lin D, Alberton P, Delgado Caceres M, Prein C, Clausen-Schaumann H, Dong J, et al. Loss of tenomodulin expression is a risk factor for age-related intervertebral disc degeneration. Aging Cell 2020;19:e13091.
- 15. Gübitz R, Lange T, Gosheger G, Heindel W, Allkemper T, Stehling C, et al. Influence of age, BMI, gender and lumbar level on T1p magnetic resonance imaging of lumbar discs in healthy asymptomatic adults. Rofo 2018;190:144–51.
- 16. Urban JP, Smith S, Fairbank JC. Nutrition of the intervertebral disc. Spine (Phila Pa 1976) 2004;29:2700–9.
- Chang H, Gao X, Li X, Zhao R, Ding W. Anemia was associated with multilevel lumbar disc degeneration in patients with low back pain: A single-center retrospective study. Eur Spine J 2022;31:1897–905.
- Siemionow K, An H, Masuda K, Andersson G, Cs-Szabo G. The effects of age, sex, ethnicity, and spinal level on the rate of intervertebral disc degeneration: A review of 1712 intervertebral discs. Spine (Phila Pa 1976) 2011;36:1333–9.
- 19. Böstman OM. Prevalence of obesity among patients admitted for elective orthopaedic surgery. Int J Obes Relat Metab Disord 1994;18:709–13.
- 20. Riihimäki H, Mattsson T, Zitting A, Wickström G, Hänninen K, Waris P. Radiographically detectable degenerative changes of the lumbar spine among concrete reinforcement workers and house painters. Spine (Phila Pa 1976) 1990;15:114–9.
- Longo UG, Denaro L, Spiezia F, Forriol F, Maffulli N, Denaro V. Symptomatic disc herniation and serum lipid levels. Eur Spine J 2011;20:1658–62.
- 22. Baar MP, Perdiguero E, Muñoz-Cánoves P, de Keizer PL. Musculoskeletal senescence: A moving target ready to be eliminated. Curr Opin Pharmacol 2018;40:147–55.