



Original Article

Association between nutritional status and Modic classification in degenerative disc disease

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Abstract. [Purpose] This study was conducted to examine the association between Modic classification and the eating habits in patients with degenerative disc disease (DDD) and to determine the influence of nutrition on disease severity. [Subjects and Methods] Sixty patients with DDD visiting a low back pain outpatient clinic were enrolled. Through face-to-face interviews, they completed questionnaires regarding their demographics, disease activity, smoking and alcohol use, concomitant diseases, disease duration, and nutritional status. Exclusion criteria were age <20 years or >65 years, other comorbidities, missing MRI data, and inability to speak Turkish. [Results] Forty patients were finally included in the study. The frequency with which they consumed water, salt, fast food, eggs, milk, yogurt, cheese, whole wheat bread, white bread, butter, and margarine was recorded. A weak negative correlation was observed between the Modic types and fish and egg consumption. [Conclusion] Modic changes, which indicate the severity of DDD, seem to be correlated to patients' dietary habits. However, studies with comparison groups and larger samples are needed to confirm our promising results before any cause-and-effect relationship can be proposed.

Key words: Degenerative disc disease, Modic changes, Low back pain

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INTRODUCTION

Chronic low back pain (LBP) has long been one of the most common causes of disability in adults and is a very frequent condition among early retirees in industrialized societies. Degenerative disc disease (DDD) is the most frequent problem in patients with LBP. In 1988, Modic et al. summarized the changes occurring in DDD and classified them into three types. Thereafter, the medical term Modic changes (MC) has appeared in various studies on spinal degenerative diseases. The prevalence of MC among patients with DDD of the lumbar spine varies between 19% and 59%. MC of types 1 and 2 are more common than those of type 3 and mixed changes. MC are considered a magnetic resonance imaging (MRI) parameter determining morphological changes in spinal degenerative diseases. Nonetheless, the etiology of MC remains poorly understood¹⁾. MC are thought to occur because of environmental, genetic, hormonal, mechanical, and degenerative factors, as

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well as because of the interaction of several unknown factors. Among the environmental factors, dietary habits and trends of DDD patients are considered one of the main ones. However, their significance in the monitoring and treatment of the disease remains unclear.

Lumbar DDD is the most common cause of LBP throughout the world²⁻⁴. LBP is especially common in industrialized regions. It is the single most common cause of disability in individuals aged over 45 years and the second most common reason for primary care physician visits⁵⁻⁷.

The risk factors associated with DDD include advanced age, socioeconomic status, torsional stress, smoking, obesity, heavy lifting, vibration, trauma, immobilization, psychosocial factors, gender, height, hereditary and genetic factors, and occupations like machine drivers, carpenters, and office workers⁸⁻¹².

MC have been described as being strongly associated with LBP¹³⁻¹⁵. According to Modic et al., these changes visible on MRI scans can be classified into three different types^{14, 15}. Type 1 changes are seen on T2-weighted MRI as areas of high signal intensity and on T1-weighted MRI as areas of low signal intensity extending from the vertebral endplates. Histological examination of material harvested during surgery has shown that MC type 1 manifest as disruption and fissuring of the endplate with regions of degeneration, regeneration, and vascular granulation tissue¹⁴. Type 2 are seen as areas of high signal intensity on both T1- and T2-weighted images, manifesting as disruption of the endplates with increased reactive bone and granulation tissue. Hematopoietic elements in the vertebrae are replaced by abundant fat (yellow marrow)¹⁴. Type 3 MC presumably represent bone sclerosis and are visualized on MRI as low-signal intensity areas on both T1- and T2-weighted images¹³⁻¹⁶.

The aim of the present study is to investigate the effects of eating habits on the Modic classification in patients with DDD.

SUBJECTS AND METHODS

This randomized, prospective, controlled, single-blinded study was conducted at an outpatient clinic for physical medicine and rehabilitation of LBP patients. This department is part of the rheumatology outpatient clinic. Via face-to-face interviews, 60 consecutive patients diagnosed with DDD completed forms regarding their nutritional status. This form recorded the patients' demographics, MC, smoking and alcohol use, concomitant diseases, disease duration, and nutritional status. The questions related to tobacco and alcohol use, frequency of breakfast consumption, daily water consumption, salt consumption, and frequency of consumption of fast food, eggs, milk, yogurt, cheese, whole wheat bread, white bread, butter, and margarine. For each food choice, the frequency of consumption was rated as every meal, once a day, 3 days a week, 2 days a week, 1 day a week, once every 15 days, once every 1 month, and not sure. In addition to demographic characteristics (age, gender, weight, height, and body mass index [BMI]), patients were also questioned about their occupation, main symptoms, and time of diagnosis.

Only patients between 20 and 65 years old were included. Other exclusion criteria included other comorbidities, unavailable MRI data, and inability to speak Turkish.

All the recruited subjects signed informed consent forms before participating in the study, and the approval of the local ethics committee was obtained. Further, all subjects consented to their random assignment to groups.

The erythrocyte sedimentation rate (ESR; mm/h) was measured using the Westergren method, and the serum C-reactive protein (CRP; mg/dl) level was determined using nephelometry.

All statistical analyses were performed using Statistical Package for Social Sciences for Windows software version 16.0 (SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was used to confirm that the data were within the ranges of normal distribution in both groups. A non-parametric test was used for variables outside normal distribution. Data were compared between groups using the independent-samples *t*-test. Statistical significance was set at $p < 0.05$ with a 95% confidence interval.

RESULTS

Forty DDD patients were finally included in the study (Fig. 1). Most were female (85%), and the mean age of the entire cohort was 43.65 ± 11.36 years. The mean disease duration was 4.16 ± 1.59 years, and mean BMI was 27.52 ± 5.32 . In terms of smoking and alcohol use, 15 patients (37.5%) were smokers and 2 (5%) consumed alcohol. The mean daily tea consumption was 1.65 ± 1.27 cups, while the mean water consumption was 1.12 ± 0.92 l. Thirty percent of the patients did not know exactly how much milk they consumed; 20% said they consumed it on 2 days a week, while 20% stated that they never consumed it. The frequency of yogurt consumption was 3 days a week for 47.5% patients, frequency of cheese consumption was 3 days a week for 92.5% patients, and frequency of white bread consumption was 3 days a week for 62.5% patients. Half the patients did not know how frequently they consumed whole wheat bread, while 30% reported eating it 3 days a week. The frequency of margarine consumption was unknown by 72.5% patients; 30% did not know how often they ate butter, while 27.5% reported that they ate it once every 15 days. The frequency of fish consumption was once a month among 37.5% patients. The frequency of egg consumption was 1 day per week for 30% patients and once every 15 days for 27.5%. Then, 60% did not know how often they consumed fast food, and 20% reported that they did not consume fast food. The frequency of red meat consumption was 2 days a week for 35% patients and once every 15 days for 35% patients. The frequency of

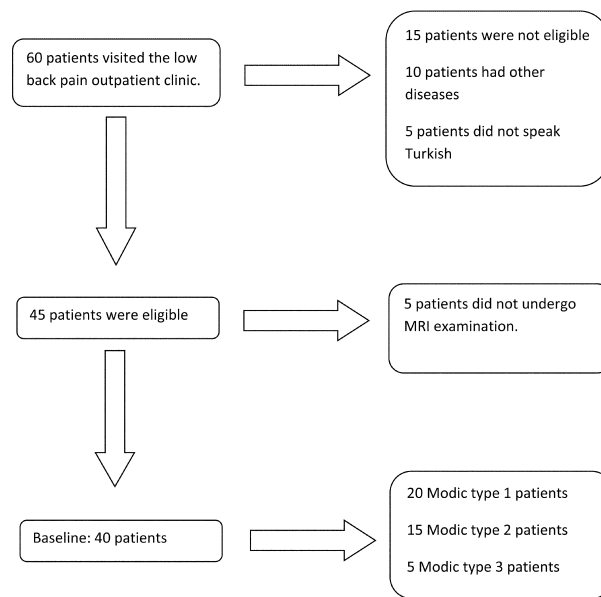


Fig. 1. Study flowchart

Table 1. Frequency of consumption of various foods in terms of number of days

	Do not know	Never	One day a month	Once every 15 days	Once a week	Twice a week	3 days a week	Every day
Milk	12 (30%)	8 (20%)	4 (10%)	3 (7.5%)	3 (7.5%)	8 (20%)	2 (5%)	0 (0%)
Yogurt	2 (5%)	1 (2.5%)	0 (0%)	2 (5%)	7 (17.5%)	9 (22.5%)	19 (47.5%)	0 (0%)
White bread	5 (12.5%)	3 (7.5%)	1 (2.5%)	0 (0%)	1 (2.5%)	0 (0%)	25 (62.5%)	5 (12.5%)
Whole wheat bread	20 (50%)	1 (2.5%)	1 (2.5%)	0 (0%)	1 (2.5%)	0 (0%)	12 (30%)	5 (12.5%)
Margarine	29 (72.5%)	4 (10%)	2 (5%)	2 (5%)	3 (7.5%)	0 (0%)	0 (0%)	0 (0%)
Butter	12 (30%)	1 (2.5%)	0 (0%)	11 (27.5%)	9 (22.5%)	7 (17.5%)	0 (0%)	0 (0%)
Fish	1 (2.5%)	9 (22.5%)	15 (37.5%)	10 (25%)	4 (10%)	1 (2.5%)	0 (0%)	0 (0%)
Eggs	2 (5%)	0 (0%)	0 (0%)	5 (12.5%)	12 (30%)	10 (25%)	11 (22.5%)	0 (0%)
Fast food	24 (60%)	8 (20%)	3 (7.5%)	2 (5%)	2 (5%)	1 (2.5%)	0 (0%)	0 (0%)
Cheese	0 (0%)	1 (2.5%)	0 (0%)	0 (0%)	1 (2.5%)	1 (2.5%)	37 (92.5%)	0 (0%)
Red meat	0 (0%)	1 (2.5%)	2 (5%)	14 (35%)	9 (22.5%)	14 (35%)	0 (0%)	0 (0%)
White meat	0 (0%)	2 (5%)	5 (12.5%)	14 (35%)	12 (30%)	7 (17.5%)	0 (0%)	0 (0%)

white meat consumption was once every 15 days for 35% and 1 day a week for 30% patients (Table 1).

The mean body fat percentage of the patients was $31.22\% \pm 9.58\%$ and fat amount was 23.37 ± 10.22 kg. The average ESR was 24 ± 8 mm/h, and mean CRP level was 1.42 ± 1.2 mg/dl. The cervical disc was affected in 11 patients, the lumbar disc in 18, and both lumbar cervical discs in 11 patients. The Modic classifications according to the type of disc degeneration are given in Table 2. A weak negative correlation was noted between fish and egg consumption and the Modic types ($r = -0.361$, $p = 0.42$; $r = -0.428$, $p = 0.14$, respectively).

DISCUSSION

The main aim of this study was to investigate the relationship between the diet of DDD patients and the degree of disease as assessed from the Modic classification. The most important feature of our study is that the extent or frequency of water, salt, fast food, eggs, milk, yogurt, cheese, whole wheat bread, white bread, butter, and margarine consumption was recorded. The consumption of cheese, yogurt, and white bread is significantly more than that of other food. Although cheese and yogurt are rich in calcium, they are also abundant in fats. Further, consumption of white bread is associated with obesity. This direction is consistent with the literature. In the present study, a weak negative correlation was found between fish and

Table 2. Modic classification according to degenerated disc type

	Region			Total
	Cervical	Lumbar	Both cervical and lumbar	
Modic Type 1	8 (2.5%)	8 (20%)	4 (10%)	20 (50%)
Modic Type 2	2 (5%)	8 (20%)	5 (12.5%)	15 (37.5%)
Modic Type 3	1 (2.5%)	2 (5%)	2 (5%)	5 (12.5%)
Total (n, %)	11 (27.5%)	18 (45%)	11 (27.5%)	40 (100%)

egg consumption and the Modic classification. These food products contain essential amino acids along with omega-3 and omega-6 fatty acids.

In most cases in the present study, disc degeneration was observed in 4th and 5th decades of life, a finding similar to those of other studies^{17, 18}.

The prevalence of Modic Type 1 changes in this study was 50%, which is comparable what is reported in some previous studies^{19, 20}, and some studies have reported a prevalence ranging from 43 to 59%. Thus, our findings are consistent with the literature. MC did not seem to influence the clinical course of pain and function and were not prognostic factors for recovery. In fact, educational level was a strong predictor of recovery. Clinicians need to be reminded to treat patients with chronic LBP by using a biopsychosocial model of recovery²¹.

The pathogenetic mechanisms underlying MC are not completely understood. A suggested cause is disc degeneration, which increases the shear force on the lumbar vertebral endplates, leading to microfractures. MC could be either edema-initialized via endplate microfractures or an inflammatory response caused by proinflammatory chemicals seeping from the nucleus pulposus through such microfractures²².

The current study is based on the assumption that MC could be the singular cause of LBP in some patients. However, these changes are often detected along with other MRI findings (i.e., degeneration, bulges, and herniation), which could also cause LBP. In the present study, patients were excluded if they had a competing somatic disease such as disc herniation with symptomatic root compression; nevertheless, it is still very likely that the pain in some cases could have another pathological cause or a combination of causes. A study conducted in the general population showed that the clinical profile of individuals with both disc degeneration and MC was more pronounced than that of individuals with only disc degeneration, suggesting that MC are the crucial element connecting LBP and clinical findings.

To our knowledge, few studies have examined the relationship between diet and Modic types. Johansen et al. studied the relationship between vitamin D and MC and surprisingly found that MC were more common in individuals with normal levels of vitamin D than in those with low levels. However, the mechanisms underlying the development of MC remain unclear at present. Findings suggest that the link between vitamin D and MC is perhaps related to inflammation, though further confirmatory studies are needed²³. According to our theoretical framework, individuals with MC are expected to have low levels of vitamin D because of an increased susceptibility to inflammation and/or because microfractures occur in the vertebrae because of increased levels of parathyroid hormone^{24, 25}.

In conclusion, although the results of our study are promising, further studies with comparison groups and larger samples are needed before a cause-and-effect relationship can be proposed.

REFERENCES

- 1) Albert HB, Manniche C: Modic changes following lumbar disc herniation. *Eur Spine J*, 2007, 16: 977–982. [[Medline](#)] [[CrossRef](#)]
- 2) Choi J, Lee S, Hwangbo G: Influences of spinal decompression therapy and general traction therapy on the pain, disability, and straight leg raising of patients with intervertebral disc herniation. *J Phys Ther Sci*, 2015, 27: 481–483. [[Medline](#)] [[CrossRef](#)]
- 3) Lee JH, Park YH, Jang SH: The effects of stabilization exercise with an oral assistive device on pain and functionality of low back pain patients. *J Phys Ther Sci*, 2015, 27: 3031–3034. [[Medline](#)] [[CrossRef](#)]
- 4) Bo GH, Park SH: Kinematic analysis of lumbar spine depending on three McKenzie's extension exercises in prone. *J Phys Ther Sci*, 2012, 24: 271–274. [[CrossRef](#)]
- 5) Choi J, Hwangbo G, Park J, et al.: The effects of manual therapy using joint mobilization and flexion-distraction techniques on chronic low back pain and disc heights. *J Phys Ther Sci*, 2014, 26: 1259–1262. [[Medline](#)] [[CrossRef](#)]
- 6) Kong BJ, Lim JS, Kim K: A study on dispersion and rate of fat infiltration in the lumbar spine of patients with herniated

nucleus pulposus. *J Phys Ther Sci*, 2014, 26: 37–40. [[Medline](#)] [[CrossRef](#)]

- 7) Lehtola V, Luomajoki H, Leinonen V, et al.: Efficacy of movement control exercises versus general exercises on recurrent sub-acute nonspecific low back pain in a sub-group of patients with movement control dysfunction. Protocol of a randomized controlled trial. *BMC Musculoskelet Disord*, 2012, 13: 55. [[Medline](#)] [[CrossRef](#)]
- 8) Baldwin NG: Lumbar disc disease: the natural history. *Neurosurg Focus*, 2002, 13: E2. [[Medline](#)] [[CrossRef](#)]
- 9) Katz JN: Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. *J Bone Joint Surg Am*, 2006, 88: 21–24. [[Medline](#)] [[CrossRef](#)]
- 10) Kanayama M, Togawa D, Takahashi C, et al.: Cross-sectional magnetic resonance imaging study of lumbar disc degeneration in 200 healthy individuals. *J Neurosurg Spine*, 2009, 11: 501–507. [[Medline](#)] [[CrossRef](#)]
- 11) Liuke M, Solovieva S, Lamminen A, et al.: Disc degeneration of the lumbar spine in relation to overweight. *Int J Obes*, 2005, 29: 903–908. [[Medline](#)] [[CrossRef](#)]
- 12) Battié MC, Videman T, Gibbons LE, et al.: 1995 Volvo Award in clinical sciences. Determinants of lumbar disc degeneration. A study relating lifetime exposures and magnetic resonance imaging findings in identical twins. *Spine*, 1995, 20: 2601–2612. [[Medline](#)] [[CrossRef](#)]
- 13) Mitra D, Cassar-Pullicino VN, McCall IW: Longitudinal study of vertebral type-1 end-plate changes on MR of the lumbar spine. *Eur Radiol*, 2004, 14: 1574–1581. [[Medline](#)] [[CrossRef](#)]
- 14) Modic MT, Steinberg PM, Ross JS, et al.: Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. *Radiology*, 1988, 166: 193–199. [[Medline](#)] [[CrossRef](#)]
- 15) Modic MT, Masaryk TJ, Ross JS, et al.: Imaging of degenerative disk disease. *Radiology*, 1988, 168: 177–186. [[Medline](#)] [[CrossRef](#)]
- 16) Weishaupt D, Zanetti M, Hodler J, et al.: Painful lumbar disk derangement: relevance of endplate abnormalities at MR imaging. *Radiology*, 2001, 218: 420–427. [[Medline](#)] [[CrossRef](#)]
- 17) Miller JA, Schmatz C, Schultz AB: Lumbar disc degeneration: correlation with age, sex, and spine level in 600 autopsy specimens. *Spine*, 1988, 13: 173–178. [[Medline](#)] [[CrossRef](#)]
- 18) de Schepper EI, Damen J, van Meurs JB, et al.: The association between lumbar disc degeneration and low back pain: the influence of age, gender, and individual radiographic features. *Spine*, 2010, 35: 531–536. [[Medline](#)] [[CrossRef](#)]
- 19) Modic MT, Ross JS: Lumbar degenerative disk disease. *Radiology*, 2007, 245: 43–61. [[Medline](#)] [[CrossRef](#)]
- 20) Jensen TS, Karppinen J, Sorensen JS, et al.: Vertebral endplate signal changes (Modic change): a systematic literature review of prevalence and association with non-specific low back pain. *Eur Spine J*, 2008, 17: 1407–1422. [[Medline](#)] [[CrossRef](#)]
- 21) Keller A, Boyle E, Skog TA, et al.: Are Modic changes prognostic for recovery in a cohort of patients with non-specific low back pain? *Eur Spine J*, 2012, 21: 418–424. [[Medline](#)] [[CrossRef](#)]
- 22) Albert HB, Kjaer P, Jensen TS, et al.: Modic changes, possible causes and relation to low back pain. *Med Hypotheses*, 2008, 70: 361–368. [[Medline](#)] [[CrossRef](#)]
- 23) Johansen JV, Manniche C, Kjaer P: Vitamin D levels appear to be normal in Danish patients attending secondary care for low back pain and a weak positive correlation between serum level Vitamin D and Modic changes was demonstrated: a cross-sectional cohort study of consecutive patients with non-specific low back pain. *BMC Musculoskelet Disord*, 2013, 14: 78. [[Medline](#)] [[CrossRef](#)]
- 24) D'Ambrosio D, Cippitelli M, Cocciolo MG, et al.: Inhibition of IL-12 production by 1,25-dihydroxyvitamin D3. Involvement of NF-kappaB downregulation in transcriptional repression of the p40 gene. *J Clin Invest*, 1998, 101: 252–262. [[Medline](#)] [[CrossRef](#)]
- 25) Mosekilde L: Primary hyperparathyroidism and the skeleton. *Clin Endocrinol (Oxf)*, 2008, 69: 1–19. [[Medline](#)] [[Cross-Ref](#)]