

Leptin Hormone Level Effect on the Nodal Osteoarthritis Patients

Al-Jashamy K^{1*}, Mustafa SS², Ghadir KF¹, Hmood KT¹, Doustjalali SR³ and Sabet NS³

¹Bilad Alrafidain University, Diyala, 32001, Iraq

²Middle Technical University, Medical Institute - Baghdad, Iraq

³Faculty of Medicine, SEGi University, Malaysia

*Corresponding author:

Karim Al-Jashamy,

Bilad Alrafidain University, Diyala, 32001, Iraq

Received: 16 Sep 2024

Accepted: 09 Oct 2024

Published: 15 Oct 2024

J Short Name: JCM

Copyright:

©2024 Al-Jashamy K, This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Citation:

Al-Jashamy K, Leptin Hormone Level Effect on the Nodal Osteoarthritis Patients. J Clin Med Img. 2024; V8(4): 1-4

Keywords:

Nodal Osteoarthritis; Osteoarthritis; Leptin; Pathophysiology; Hormone; Pathology

1. Abstract

1.1. Background and Objective: Nodal Osteoarthritis (NOA) is known as cartilage loss disease in hand-finger joints that relates to the production many of inflammatory responses. The objective of this study was to determine the role serum leptin level in the development of nodal osteoarthritis disease.

1.2. Materials and Methods: The body weight and BMI were recorded, and 10 mL of blood was collected from all individuals. Serum Leptin level was selected using measurement parameters in both groups of this study via a chemical measurement technique called Sandwich ELISA. 10 ml of blood was collected from 30 patients in group NOA and 30 patients in the control group.

1.3. Results: A significant increase in serum leptin level in the NOA patient group compared with the control group at a p-value of 0.0219, a non-significant value among genders when comparing the NOA patients group with the control group. The BMI in the NOA was 32.8 ± 7.6 which showed a significant increase compared with the control group.

1.4. Conclusion: The current study demonstrates that serum leptin level can act as a promoter for inflammation development in NOA disease.

2. Introduction

Osteoarthritis (OA) is considered one of the most common joint disorders. There are accumulating etiology that suggest that osteoarthritis is an inflammatory disease of the entire cartilaginous synovial joint. Nodal osteoarthritis (NOA) is a chronic disease classified as one of the subtypes of the cartilaginous synovial

joints, this disease is considered a commonly geriatric disease due to the physiologic changes processes with age [1]. NOA is characterized by damage of chondrocytes of the hand fingers joints, the joint cartilage covering the end bones at the joint, and this joint might interact with inflammatory factors. The NOA pathology was recognized by two causes of this chronic disease that classified it into primary NOA, which correlates with inherited conditions, and secondary NOA which has a correlation with acquired conditions [2]. The commonly acquired cause of secondary NOA is obesity. The most important NOA signs and symptoms in hand joints are stiffness, swelling, decreased range of motion, pain, and others. Nodal osteoarthritis is diagnosed according to international guidelines such as the American College of Rheumatology (ACR) that depend on laboratory and radiology examinations [3].

Leptin is an inflammatory hormone mainly produced via adipose tissue and it is encoded by ob/ob gene and classified as a subtype of cytokine type-1. Leptin has many receptors but an important receptor is oRb which is active through JAK/STAT pathway [4]. Leptin has various functions such as the role of homeostasis, decreased intake of food, and mediated immune responses. Many previous studies demonstrated the correlation of leptin levels with various diseases such as obesity and arthritis [5]. Many different cases might be similar clinically such as gout, arthritis, or osteoarthritis, however, each clinical case needs for specific diagnostic test. Leptin is a product of the obese gene that secreted from adipose tissue, which binds to the leptin receptor (LEP-R) that might play important roles with diabetic patients, Hence, the objective of this study was to determine the correlation between serum leptin levels in the development of nodal osteoarthritis disease from.

3. Materials and Methods

The present study was achieved on 60 individuals classified into two groups, 30 patients group NOA (12 males and 18 females) and 30 control group (14 males and 16 females). All individuals were diagnosed as NOA or control via depended on the X-ray and clinical examination. The age of all subjects was more than 50 years old for both genders. This study was conducted at Al-Yarmouk Teaching Hospital in June-July 2023, after the agreement of the researchers steering the committee on this study. The body weight and BMI were recorded, and 10 mL of blood was collected from all individuals, then the serum was immediately used for the quantity method of an immunoassay for measuring the serum leptin. The sample of serum was obtained after the blood sample centrifugation process without preservative addition. Serum Leptin level was selected using measurement parameters in both groups of this study via a chemical measurement technique called Sandwich ELISA (Leptin kit-Cat. No. RD191001100 - BioVendor company-Czech Republic) using the standard curve of leptin parameter. The excluded cases were those patients with positive rheumatoid arthritis, gout, and acute arthritis.

3.1. Statistical Analysis: The statistical significance of the serum leptin level was evaluated using a t-test of mean + standard deviation (SD), and a p-value of less than 0.05 is considered a significant value.

tion (SD), and a p-value of less than 0.05 is considered a significant value.

3.2. Ethical Consideration: The human ethical approval of this study was approved by the ethical committee under NO. Bila-Rf2022-B118.

4. Results

Current study results demonstrated the qualitative analysis, a non-significant value when comparing NOA patients group with the control group (Table 1). A significant increase in serum leptin levels in the NOA patient group compared with the control group. This comparison used the t-test method according to mean + standard deviation (SD), which for the NOA group, was 11.1 ± 6.7 while for the control group was 8.4 ± 7.7 . The BMI in the NOA was 32.8 ± 7.6 which showed a significant increase compared with the control group (Table 2, Figure 1).

The features of X-ray images in the NOA showed swelling joint and narrowing joint synovial space, bone spurs formation, articular surface cortical irregularity, and joint sclerosis. These specific features of NOA might show aggregation of the tissues formed around the interphalangeal joint, as well as the formation of some bone spurs and fibrosis (Figure 2).

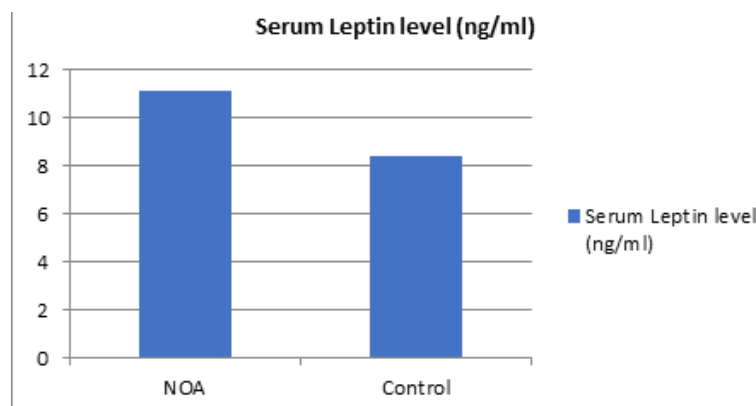


Figure 1: Increase of serum leptin level in NOA group compare with control group.



Figure 2: The features of X-ray images in the NOA showed swelling joint and joint sclerosis formed around the interphalangeal joint (arrow).

Table 1: Comparison of gender between (NOA group) and control group)

P-value	(control group)	(NOA group)	Gender
0.0896*	14	12	Males No.
0.0722*	16	18	Females No.

*None significant value >0.05

Table 2: Comparison according to the serum level of leptin and BMI between NOA and control groups

P-value	Control group mean+SD	NOA group mean+SD	Parameter (ng/ml)
0.0219*	8.4+7.7	11.1+6.7	Serum leptin level
0.032*	24.2±6.5	32.8±7.6	BMI

*Significant value <0.05

5. Discussion

The results of this study showed a significant increase in serum leptin levels in NOA and BMI patients. NOA is a high prevalence in geriatric diseases characterized by cartilage degeneration at the bones of the hand joint. The pathophysiology of NOA is coupled with various factors such as mechanical and non-mechanical interactions that cause inflammation conditions with cytokines present [2]. Cytokines are an inflammatory factor or some called inflammatory hormones such as leptin that are secreted from fatty tissues and promote the generation process of inflammation in joints [6]. The various studies demonstrated a correlation between NOA and serum leptin level with all signs and symptoms of NOA, this study's results confirm this pathogenesis of the NOA and agreed with previous reports demonstrated that elevated leptin accelerated the inflammatory process of the hand finger joints [7]. The current study result agreed with Bilski et al.8 and also confirmed the elevation and correlation of leptin levels with NOA patients. These specific features of NOA might show aggregation of the tissues formed around the interphalangeal joint, as well as the formation of some spurs caused by fibrosis due to sensitivity to the serum leptin, which helped to form this tumor towards the joint [9]. These spurs and tissues formed led to a restriction in the movement of the finger, which was caused by the tumor occurring around the interphalangeal joint, as mentioned previously [4, 9, 10]. Leptin is a product of the obese (ob) gene and following synthesis and secretion from fat cells in white adipose tissue, binds to and activates its cognate receptor, the leptin receptor (LEP-R) that might play important roles with diabetic patients [11]. For the future, we recommend that apply the study of on the patients those suffer from NOA and diabetic comparing with BMI.

6. Conclusion

The current study demonstrates that leptin level in serum is high in NOA BMI patients, this shows that leptin hormone includes pathological processes that lead to cartilage loss in hand finger joints, as well as that serum leptin level can act as a biomarker for inflammation development in NOA diagnosis. Feature study on the NOA with more cases that might recommend on the patients those suffer from NOA and diabetic comparing with BMI.

United Prime Publications. LLC., clinandmedimages.com

7. Acknowledgments

The authors would like to thank the University of Diyala and the Iraqi Center for Cancer and Medical Genetic Researches (ICC-MGR) cell bank unit, Al-Mustansiriyah University.

8. Conflict of Interest

The authors have no conflict of interest in this research.

9. The Human Ethical Approval

This study was approved by the ethical committee under NO. Bila-Rf2022-B118.

10. The Author's Contribution

This study was KAJ, and KDF lab work analysis and writing the article. AMK, NAE, JOK followed up on the statistical analysis and shared them in the discussion.

11. Source of Fund

«This research did not receive any specific grant from funding agencies in the public, commercial, or not for-profit sectors»

References

1. El-Najjar AR, Abu-Elsoaud AM, Mohammed HT, Shawky KM. Diagnostic potential of magnetic resonance imaging (MRI) of the first carpometacarpal joint in hand osteoarthritis. *The Egyptian Rheumatologist*. 2021; 43(1): 59-64.
2. Favero M, Belluzzi E, Ortolan A, Lorenzin M, Oliviero F, Doria A, et al. Erosive hand osteoarthritis: latest findings and outlook. *Nat Rev Rheumatol*. 2022; 18(3): 171-183.
3. Pottabattula B, Sattari M. Giant cell tumour of tendon sheath mimicking nodal osteoarthritis. *BMJ Case Rep*. 2022; 13(2): e231902.
4. Ahn MB, Kim SK, Kim SH, Cho WK, Suh JS, et al. Clinical significance of the Fetuin-A-to-Adiponectin ratio in obese children and adolescents with diabetes mellitus. *Children (Basel)*. 2021; 8(12): 1155.
5. Jiliang C, Zhiping X, Zou B. The association between serum leptin levels and cardiovascular events in patients with rheumatoid arthritis. *Laboratory Medicine*. 2021; 52: 86-92.
6. Fajgenbaum DC, June CH. Cytokine storm. *New England Journal of Medicine*. 2020; 383(23): 2255-2273.

7. Plotz B, Bomfim FM, Sohail A, Samuels J. Current epidemiology and risk factors for the development of hand osteoarthritis. *Current Rheumatology Reports*. 2021; 23(8): 1-14.
8. Bilski J, Pinkas M, Wojcik-Grzybek D, Magierowski M, Korbut E, Mazur-Bialy, et al. Role of obesity, physical exercise, adipose tissue-skeletal muscle crosstalk and molecular advances in Barrett's esophagus and esophageal adenocarcinoma. *Int J Mol Sci*. 2022; 23(7): 3942.
9. Herren D. The proximal interphalangeal joint: arthritis and deformity. *EFORT Open Rev*. 2019; 4(6): 254-262.
10. Kamnerdnakta S, Huetteman, Chung KC. Complications of proximal interphalangeal joint injuries: prevention and treatment. *Hand Clin*. 2018; 34(2): 267-288.
11. Obradovic M, Sudar-Milovanovic E, Soskic S, Essack S, Arya AJ, Stewart T, Gojobori E. Leptin and obesity: role and clinical implication. *Front Endocrinol (Lausanne)*. 2021; 12: 585-887.