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Original Article

Correlation of Leptin Levels With Obesity and Insulin Resistance

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ABSTRACT

Objective: To find the correlation of leptin levels with obesity and insulin resistance.

Methodology: The study was an Analytical Cross-sectional study with 208 participants. It was conducted in Civil Hospital of Quetta Pakistan, from December 2022 to July 2023, after ethical approval and informed consent obtained. Participants were selected based on age and BMI. Demographic characteristics were collected through a questionnaire and laboratory investigations. Data analysis was performed using SPSS version 22, with T-test, Chi-square test, and logistic regression used to determine the association between leptin levels and insulin resistance.

Results: The study involved 208 participants, with a mean age of 48.23±2.16 years. Among them 79.80% were obese, and 20.20% were non-obese, 62.02% were insulin resistant, with 96 being obese and 96 having a mean leptin level of 50.90±3.23. There were 25.59% were non-obese patients among the insulin-resistant individuals.

Conclusion: In conclusion, the findings highlighted the development of key elements that are associated with obesity like leptin levels, and these levels are shown to correlate with high BMI and ultimately lead to the development of various metabolic disturbances like Diabetes mellitus. So, weight management would be the key factor to reduce the burden of metabolic disorders globally.

Keywords: Body Mass Index, Insulin, Insulin resistance, Leptin, Obesity.

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INTRODUCTION

When leptin became known around 20 years ago, there was a lot of excitement surrounding its potential to cure obesity. Leptin is the name of the protein cytokine released by the prototypical adipocyte. Its name comes from the Greek word "leptos," which means thin. It also introduced the idea that adipose tissue is a functional endocrine organ in addition to an inert organ for storing energy.[1] The 167 amino acids that make up the human leptin gene provide leptin, which was initially identified at Jackson Laboratories using positioning sequencing of ob/ob mice, as a mouse model.[2] It is mostly secreted from white adipose tissue, which means there is a positive correlation between fat in the body and its levels. Due to its intermittent secretion as well as its circadian variation, such as greater concentrations in the late hours and early in the morning, leptin is produced in a manner typical of a hormone. Acute variations in energy intake, primarily from calories and fats, are reflected in the human body's circulation of leptin.[3,4] Furthermore, leptin's basic effect on glucose homeostasis is extremely significant. Following leptin administration, streptozotocin-related diabetic mice displayed normalized glucose levels and a significant improvement in insulin sensitivity.[5,6] It is believed that leptin resistance within the neural systems results in hyperleptinemia. Given the significant connections between leptin and insulin, leptin connections are pertinent to changes in glucose metabolism.[7] There is a connection between resistance to insulin, leptin resistance, and obesity. According to studies, those with hyperinsulinemia may be at risk for developing leptin resistance and obesity, which can ultimately result in metabolic syndrome.[8] Elevated leptin levels, which are usually found in obese persons, are connected with both body fat percentage and body mass index. Nevertheless, there is a connection between BMI and leptin. Men's BMI was found to have the highest association with leptin,[9] however, there were no appreciable differences between BMI and leptin and diabetes in a small number of studies.10 Type 2 diabetes mellitus (T2DM) is a leading cause of death and morbidity worldwide, with its incidence increasing rapidy. T2D is caused by varying degrees of insulin resistance (IR) and relative insulin deficiency. Leptin, an adipokine whose main function is to

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regulate energy balance, is involved in insulin secretion and sensitivity of peripheral tissues. Therefore, we sought to determine the role of leptin in the development of insulin resistance.

METHODOLOGY

The design used for measuring the objectives of the study was analytical cross-sectional study. Total participants for the study were 208, calculated by Rao-soft calculator online with parameters of confidence level 0.95 and 0.05 error margin. All the participants were selected conveniently and the study was conducted in the Physiology department Civil Hospital of Quetta from December 2022 to July 2023. Before data collection, ethical approval was granted from the Ethical Committee of the department. In addition, the informed consent was obtained from each participant of the study.

Inclusion Criteria

The age criteria were kept for inclusion as 40 years and above to achieve the purpose and voluntary subjects were included in the study.

The participants were ensured before data collection that their information and all data would be kept confidential. A questionnairewas used for demographic characteristics and further data was taken from laboratory investigations of participants. SPSS version 22 was used for data analysis. The frequencies and percentages were calculated for demographic variables, while the mean and standard deviation were for continuous variables. Differences between groups were deter-mined by using t-test and Chi-square test and logistic regression analysis was used to find out association between leptin levels and insulin resistance.

RESULTS

In the current study, a total of 208 participants were selected through a convenient sampling technique including both genders. The mean age of the participants was 48.23±2.16 years. There were 63.46% were males and 36.53% were female participants. Among the study participants, 166(79.80%) were obese and 42(20.20 %) non-obese individuals.

In Table-II highlights the Comparison of insulin resistance in obese versus non-obese. About 129 (62.02%) of the participants were insulin resistant, of which 96 (74.41%) of them were obese having a mean leptin level of 50.90±3.23 with a *p*-value of 0.002. Consequently, only 33(25.59%) of them were non-obese patients among the insulin-resistant individuals with a mean leptin level was 17.50±1.9, in addition to the *p* value of 0.06.

Table-I: Demographic Characteristics

Age (Mean) 48.23±2.16 years	n (%)
Gender	
Male	132 (63.46)
Female	76 (36.53)
Weight of the participants (BMI)	
Obese (BMI >30 kg/m^2)	166 (79.80)
Non-Obese (BMI <30 kg/m^2)	42 (20.20)

Table-II: Comparison of Insulin Resistance in Obese Versus Non-Obese.

	Insulin-resistant	Mean leptin level	*p*-value
Obese	96(74.41%)	50.90±3.23	0.002
Non-obese	33(25.59%)	17.50±1.9	0.06

DISCUSSION

Numerous pieces of evidence support the hypothesis that leptin plays a major role in modulating the activity of sympathetic nerves in certain locations, which may have pathophysiological implications for obesity. Leptin regulates energy balance by decreasing appetite and increasing consumption of energy via sympathetic activation. Leptin levels in the blood decrease during fasting and increase with overfeeding.[11] Research has indicated that individuals as well as rodents deficient in leptin or its associated receptors exhibit severe hyperphagia and increase in weight, while leptin treatment is shown to reduce appetite.[12] In the current study, 166(79.80%) were obese and 42(20.20%) non-obese individuals, moreover, a study conducted by Zulfania *et al*, 2020 revealed that serum leptin levels were significantly greater in non-obese (7.21±3.78 ng/ml) and obese (9.42±1.87 ng/ml) patients than in controls (5.38±2.20 ng/ml; $p<0.05$) patients. Serum leptin levels were significantly correlated ($p<0.001$) with BMI, FBG, glycated hemoglobin, and fasting blood glucose,[13] however, the present study shows that 129(62.02%) of the participants were insulin resistant, of which 96(74.41%) of them were obese having a mean leptin level of 50.90±3.23 with a *p*-value of 0.002. Consequently, only 33(25.59%) of them were non-obese patients among the insulin-resistant individuals with a mean leptin level was 17.50±1.9, in addition to the *p*-value of 0.06. Similarly, conducted by Ambad *et al* 2020, the BMI of 56% of the subjects is higher than 30."The cases group had higher leptin levels than the controls, and the variations were of statistical significance ($p<0.0001$), additionally, in comparison to a healthy control group without diabetes, people with diabetes had considerably higher

serum leptin levels.[14] In the present study 96 (74.41%) of them were obese having a mean leptin level of 50.90±3.23 with a *p*-value of 0.002. Consequently, only 33(25.59%) of them were non-obese patients among the insulin-resistant individuals with a mean leptin level was 17.50±1.9, on the other hand Kumar *et al* 2020, patients who were obese had higher serum levels of leptin (51.24±18.12 compared. 9.10±2.99: the significance level, <0.0001), cholesterol (198.2±32.1 vs. 151.2±21.2, *p*-value <0.0001), along with the insulin resistance (7.9±2.1 vs. 6.3±1.9, the *p*-value <0.0001).[15-18]

CONCLUSION

In conclusion, the findings highlighted thedevelopment of key elements that are associated with obesity like leptin levels, and these levels are shown to correlate with high BMI and ultimately lead to thedevelopment of various metabolic disturbances like Diabetes mellitus. So, weight management would be the key factor to reduce the burden of metabolic disorders globally. Further research is needed to elucidate the bivariant relationship leading to metabolic issues that help in developing therapeutic approaches to address these problems.

Conflict of Interest: None.

Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

SN & AB: Study design, drafting the manuscript, data interpretation, critical review, approval of the final version to be published.

MM & IAY: Data acquisition, data analysis, approval of the final version to be published.

RA & SI: Critical review, concept, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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