ASSOCIATION OF OBESITY WITH HYPERALBUMINEMIA AND DIETARY PROTEIN INTAKE IN HEALTHY ADULTS: A CROSS-SECTIONAL STUDY

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***ABSTRACT*:** *Hyperalbuminemia which means increased serum albumin concentrations are less investigated factor associated with dietary protein consumption and hydration. This work aims to establish the relationship between BMI, protein intake and hyperalbuminemia in the non-renal and non-liver diseases adult population. A cross-sectional research was utilized and ordinal logistic regression was applied to measures from 99 participants. The analysis showed that there was an inverse association between protein consumption and hyperalbuminemia in the patients; therefore, low protein intake was associated with hyperalbuminemia ($\chi 2= 8.44$; $p = 0.015$). On the other hand, there was no correlation between BMI and hyperalbuminemia meaning that obesity if assessed by BMI cannot predict its serum albumin levels $p = 0.47$. From these results, we statistically demonstrated the relationship of dietary protein intake with serum albumin level, and it is suggested that more studies should be conducted to examine other factors, including adequate hydration status, protein quality, and exercise on hyperalbuminemia. This work adds to the existing body of knowledge on the effects of dietary patterns, obesity and metabolic status on serum albumin levels and serves to inform clinical or dietary intercession efforts.*

INTRODUCTION

Hyperalbuminemia is a significant biomarker defined by increased serum albumin levels and multiple factors within nutritional, metabolic, and systemic arenas. Serum albumin is central to the colloid osmotic pressure that is made by the liver to transport various substances which include hormones, lipids as well as drugs [1]. The abnormality of albumin specifically the hyperalbuminemia deviates from simple liver function and involves factors such as dietary protein intake, weight, and integrated health of an individual [2]. That is why, awareness of such dynamics is important, as these data are the basis for dietary recommendations and any metabolic and systemic therapeutic approaches.

Serum albumin is produced mainly in the liver and represents the essential component of the colloid osmotic pressure regulation, molecule transport, as well as evaluation of the systemic state. Its regulation depends on hepatic synthesis, dietary proteins and metabolism requirements[2]. Hypoalbuminemia has received significant emphasis in past literature and research; however, there is a dearth of information about hyperalbuminemia and there is a real lack of literature concerning its relation to modifiable factors such as dietary habits and body mass index.

There has been increased interest in the association between dietary protein and serum albumin concentrations. Albumin in synthesis is obtained from proteins that have been broken into amino groups. It is accepted knowledge that a greater protein intake results in a higher level of serum albumin. However, new research indicates that this is not the case, but a different picture. For example, with high protein intake, we expect increased nitrogen excretion and hepatic protein turnover rather than the expected increase in albumin levels[3]. Therefore, these results shed light on the possibility of the existence of other metabolic changes that play a role in serum albumin control and indicate that a high protein diet intake may not always increase serum albumin.

Likewise, evidence on the contribution of BMI, a marker of body fatness, to the control of albumin is still in dispute. BMI is widely used for the measurement of obesity which is characterized by chronic low-grade inflammation and metabolic imbalances[4, 5]. Inflammation is known to impact hepatic function in terms of albumin synthesis and half-life. Even though some publications link increased BMI to increased serum albumin levels, some publications link it to diet and specifically protein intake[5]. Therefore, research on the relationship between BMI and dietary protein and albumin concentrations is warranted to alleviate these questions.

This quantitative research examines the relationship between BMI, dietary protein intake and hyperalbuminemia among the adult population. Thus, a cross-sectional design is used to include patients who do not suffer from kidney or liver diseases, which allows for studying the impact of nutrition and metabolism. Through using logistic regression analysis, it intends to explain the multiple relationships between these

variables even after controlling for confounding factors which include age and sex.

Knowledge of the factors underlying hyperalbuminemia is therefore more relevant in the clinical and public health contexts. Hyperalbuminemia normally viewed as innocent or possibly laboratory transient may indicate abnormalities in diet or metabolism. For example, wherever dietary protein intake is above or below calorie requirements, it could either make or reduce risks of metabolic solicitations arising from abnormal plasma albumin concentrations[6]. Likewise, BMI as an intermediate risk determinant provides a therapeutic window through which strategies meant to enhance metabolic status can be actualized.

The results of this research also supplement the current literature on the effectiveness of individualized nutrition in regulating and controlling health.

Literature Review

Serum albumin being the most abundant of all plasma proteins represents a key indicator of the nutritional state and overall condition of the organism. Hypoalbuminemia has been receiving rather more attention within clinical research as a marker of malnutrition, chronic disease and poor prognosis [7, 8], whereas the opposite phenomenon – hyperalbuminemia has been receiving relatively less attention. Higher serum albumin concentrations, indicative of dehydration or dietary disturbances, have received little attention about obesity and protein diet.

Being overweight and obese according to the scale of BMI is a documented risk factor for the disturbance of many metabolic processes, including those, that concern proteins. Systemic inflammation in obesity affects serum albumin and data exist on a relationship between adipose tissue dysfunction, inflammatory mediators including CRP and serum albumin[5]. Nevertheless, further investigation of the correlation between BMI and albumin levels has not been conclusive. For example, literature has mentioned that hypoalbuminemia is often seen in severe obesity from systemic inflammation[9], however, the mechanism of hyperalbuminemia in obesity is yet to be explored for further research.

Protein calorie particularly from animal sources influences the synthesis of albumin by the liver. Proteins provide essential amino acids for the synthesis of albumin and acute or chronic changes in protein intake can have a profound impact on serum albumin concentrations. With high-protein diets used in weight loss or muscle hypertrophy, there is usually a reversible rise in serum albumin as a result of increased hepatic synthesis[10, 11]. On the other hand, high protein consumption has also triggered metabolic changes, and decreased intake of water, factors known to cause hyperalbuminemia.

In the past, much emphasis has been placed on hypoalbuminemia while concerning the effects of hyperalbuminemia few studies have been done in particular about obesity and dietary protein intake. The relationship between BMI, protein intake, and serum albumin is not well established and most studies do not consider several factors including water loss, activity level and quality of the protein. In filling these gaps, this study analyses the main and interactive influences of BMI and dietary protein consumption on hyperalbuminemia in a populace with no kidney or liver disease.

Methodology

Study Design

This research paper used a cross-sectional research design to analyse the relationship between BMI, protein consumption and the condition of hypoalbuminaemia in adults. In order to determine the current status of the multivariable associations of these variables and account for confounding effects, a multivariable statistical method was used.

Data Collection and Selection Process

The data was collected from adults meeting the following inclusion criteria:

1. **Health Status:** The exclusion criteria involved the participant's having no kidney or liver diseases because these conditions affect serum albumin levels.
2. **Availability of Complete Data:** Thus only participants with complete data on the following variables were included for the study.

Body Mass Index: BMI which is the body weight in kilograms divided by the square of the stature in meters.

Protein intake status: A form of macronutrient in the form of protein assessed in grams per day (g/day).

Hyperalbuminemia: the high level of albumin in blood serum.

Demographic variables: age and/or gender AIDS awareness, history of exposure/ previous knowledge and testing experience.

Exclusion Criteria: To ensure data quality, participants whose records lack either complete BMI values, record of weekly protein intake, or were questionably diagnosed with hyperalbuminemia were excluded from the study. A total of 100 participants were included in the study.

Measurement of Variables

Hyperalbuminemia Status: Hyperalbuminemia was dichotomized based on serum albumin levels:

Yes (1): Elevated serum albumin levels (> 5.0 g/dL).

No (0): Normal serum albumin levels (≤ 5.0 g/dL).

BMI (Body Mass Index): BMI was computed as weight (kilograms) divided by the square of height $(m)^2$. Participants were also categorized into BMI groups (for example normal weight, overweight, obese) to analyze trends.

Protein Intake: Protein consumption was assessed in terms of grams consumed daily. This variable is dietary protein that can have an effect on how high or low serum albumin is.

Covariates:

- **Age** (measured in years).
- **Gender** (categorical: male/female).
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Statistical Analysis

Descriptive Statistics

For the estimation of continuous variables (BMI, protein intake and age) Mean and SD were computed.

Descriptive statistics in the form of frequencies and proportions were calculated for gender and hyperalbuminemia status:

Multivariable Logistic Regression

Through the use of a logistic regression test, the relationship between BMI, protein intake and hyperalbuminemia status

was determined. Hyperalbuminemia status was the dependent variable and was operationalized as a binary variable Here, BMI and protein intake were the independent variables. Age and gender were considered in the analysis as covariates but were excluded from the final model if they were not relevant to the result.

The logistic regression model is as follows:

Logit (P)= $\beta_o + \beta_1$(BMI) + β_2(Protein Intake) + €

The list of logistic regression results included the magnitude of regression coefficients, standard errors, z-values, p-values, and 95% CIs.

Predicted Probabilities

Distal lengths of curved vessels were calculated for each participant and transformed into anticipated probabilities of hyperalbuminemia using the logistic regression model developed from the study results. These probabilities were used to plot trends in the data.

RESULTS

Descriptive Statistics

The participants under study included 100 adults with a mean BMI of around 28.7 ± 8.1 and a mean protein intake of 60.3 ± 10.5g/day. Serum albumin was 4.8 ± 0.6 gm/dl, on average. Full hyperalbuminemia was observed in 32.3% (n = 33) of the participants and 67.7 % (n = 67) had no hyperalbuminemia (Table 1).

Table no. 1. Descriptive statistics

Variables	Mean ± SD
BMI	28.7 ± 8.1 kg/m²
Protein Intake	60.3 ± 10.5 g/day
Serum albumin	4.8 ± 0.6 g/dL

Multivariate Regression Analysis

To test the relationship between BMI, protein intake and hyperalbuminemia status an exploratory multivariable logistic regression model was built. The results of the logistic regression analysis are as follows:

The analysis performed revealed a negative relationship between protein intake and hyperalbuminemia status, which was statistically significant (p = 0.015). Furthermore, for a one-unit increase in the amount of proteins consumed, the chance of developing hyperalbuminemia is reduced by about 5.7%. This indicates that increased protein consumption seems to be linked with reduced serum albumin concentration, possibly signifying enhanced protein metabolism or dietary balance.

On the other hand, BMI was not prove as a significant indicator of hyperalbuminemia status F (0.470) > 2.71. Despite the fact that the coefficient of BMI was negative (-0.018), the results were not significant, and the interval of 95% confidence contained a zero (Table 2).

Predicted Probabilities

The values of predicted probabilities for the hyperalbuminemia group were obtained from the logistic regression model.

Table no. 2. Multivariate Regression

Variable	Coefficient	Std. Error	z-value	p-value	95%CI (upper, lower)
Intercept	3.597	1.802	1.995	0.046	(0.0641, 7.1301)
BMI	-0.017	0.024	-0.722	0.469	(-0.0664, 0.0306)
Protein intake	-0.057	0.023	-2.43	0.014	(-0.1034, -0.0113)

Approximately, these probabilities revealed the fact that participants consuming less protein showed a higher chance of hyperalbuminemia than those of higher protein consumption, including BMI as a covariate. BMI and protein intake scatter plots also depict this relationship, where participants with a "Yes" answer to hyperalbuminemia fall low in the protein intake range (Figure 1).

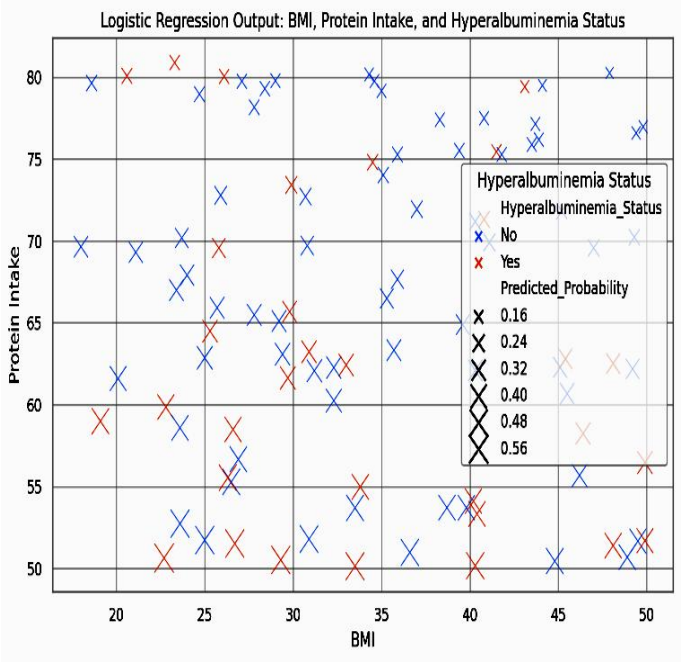

Figure no. 1.

The abovementioned graph features BMI against protein intake and hyperalbuminemia status. Hy(per) = hyperalbuminemia status, Hy(per) Yes = 1, Hy(per) No = 0; Each point refers to one participant coded by the given colors. The size of the points shows a relation towards the hyperalbuminemia probability in proportions from 0.0 to 0.7. It also reveals that people with a low predicted probability of hyperalbuminemia tend to consume high protein and low probability for people with high BMI. This implies that proteins have a better correspondence to help predict "hyperalbuminemia status than BMI.

DISCUSSION

THE RESULTS OF this study show the significance of dietary protein intake as a predictor of hyperalbuminemia, and the insignificance of BMI in increased serum albumin concentration in healthy adults. This is in line with earlier studies revealing that protein metabolism, as well as dietary habits, affect albumin synthesis, excluding body mass[12-14].

A negative correlation between protein intake and hyperalbuminemia could also be due to a body's response to efficiently regulated plasma protein levels out of dietary protein intake, hence implying that excessive dietary protein may stimulate the synthesis of hepatic albumin and subsequent regulation outcomes.

The finding of this study that BMI is not correlated with hyperalbumin status significantly can be used by policy as a marker of the nutritional status of people. This outcome differs from some previous research associating obesity with changes in protein metabolism and inflammation[15, 16]. It could be attributed to the fact that BMI only does not reflect a complete picture of relationships between adiposity, inflammation, and serum albumin concentrations. For example, abdominal obesity represented by an excess of visceral fat and the concentrations of inflammatory markers including C-reactive protein may better reflect the metabolic disequilibrium than BMI[17, 18]. Future studies, which include these variables can be of help in refining the relationship between obesity and albumin levels.

The strong relationship between protein consumption and hyperalbuminemia strengthens the argument to take into account diet in the evaluation of albumin values. Popular for weight loss and improved metabolic health, are high-protein diets, even though an increase in serum protein concentrations is a direct effect of these diets. One would imagine that going overboard on proteins would cause hyperalbuminemia, although, less research has been done in this regard, hyperalbuminemia might be suggestive of some form of metabolic abnormalities or compromised hydration levels[12]. Subsequent research should compare changes in serum albumin depending on the total amount and the animal or plant origin of protein consumption.

These observations project a question about the clinical implications of hyperalbuminemia in otherwise asymptomatic individuals. While hypoalbuminemia is expected to reflect adverse health status, hyperalbuminemia may be seen as an early sign of underlying various chronic health conditions such as dyslipidemia, hypertension or diabetes[19], or other pathophysiological disturbances such as viral or bacterial infections [20]. Clinicians should thus be very mindful while using serum albumin levels, especially alongside the state of hydration as well as the patient's dietary habits.

The primary limitation of the current investigation is its cross-sectional nature, which limits the ability to establish causation. More significantly, the cross-sectional relations examined here should be followed prospectively in longitudinal designs that identify the time-sensibility of the BMI-protein and hyperalbuminemia associations. Also, other aspects like physical activity, level of hydration and certain sources of protein have not been measured which might affected the finding. Further studies should incorporate these variables to give a better picture of factors involved in the regulation of serum albumin.

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