Research Article
Volume 13:01, 2024

Journal of Metabolic Syndrome

ISSN: 2167-0943 Open Access

Association between Serum Ferritin Levels and Risk Factors of Obesity among Mongolian Men

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Abstract

Objective: Serum ferritin is a sensitive and key indicator of iron accumulation, and serum ferritin levels can be elevated due to risk factors for many diseases. Obesity is a manifestation of metabolic dysfunction, and our study purpose was to assess the relationship between body adiposity indexes and serum ferritin level and estimate risk.

Materials and methods: A total of 227 Mongolian men between the ages of 30 and 60 participated (45.5 ± 8.4 years) in the study and were classified as normal ferritin, hyperferritinemia, and iron overload based on serum ferritin levels.

Results: Anthropometric measurements that indicate obesity and indexes that indicate visceral adiposity, lipid accumulation, and insulin resistance increased significantly in the groups with an increased ferritin level ($p<0.05$). A positive correlation was observed between serum ferritin and the Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP), Triglyceride-Glucose Index (TyG) indexes, and logistic regression analysis showed that increased serum ferritin was significantly associated with increased VAI, LAP and TyG. Even after adjusting for possible cofounders, TyG remained a significant risk factor for elevated serum ferritin.

Conclusion: Our study results concluded that body fat is one of the risk factor to increase the serum ferritin level and alters iron metabolism for Mongolian men. Body adiposity indexes, which are formulas for anthropometric measurements and functional parameters, are indicators of the prevention of increased iron accumulation in serum, so it is necessary to study them in conjunction with other factors in the future.

Keywords: Ferritin • Hepcidin • Iron accumulation • Metabolism • Risk factor of obesity

Introduction

Serum iron is a trace element that plays an important role in oxygen transport, cellular respiration, the synthesis of deoxyribonucleic acid, which stores genetic information, and cell growth, and if it accumulates in excess in the liver, pancreas, and heart, it produces harmful reactive oxygen species. Iron-induced oxidative stress is the cause of metabolic disturbances and damage to target organ cells, such as dyslipidemia and glucose metabolism disorders [1-4]. Pathologically, the hepcidin released from liver cells increases due to fatty liver and inflammation caused by metabolic disorders and obesity. An increased level of hepcidin reduces the level of iron in serum, causing iron accumulation in tissues and the worsening of metabolic disorders [5-7]. On the other hand, several studies conducted among the population have determined that insulin tolerance, hyperglycemia, and lipid metabolism disorders increase the risk of increased ferritin, an indicator of iron metabolism [7-11]. Recent studies have determined changes in lipid metabolism using VAI and LAP indexes, which are formulated as simple parameters representing human organs and functions. Elevated serum iron increases the risk of developing primary liver tumors by 55% in individuals with hemochromatosis, and clinical and experimental studies have shown that control of iron levels reduces the incidence of liver tumors [12].

Ferritin accumulation is related to liver cirrhosis and cancer, which are the leading causes of morbidity and mortality among Mongolian men. The average life expectancy of Mongolian men is 9.67 years shorter than that of women, and the death rate is 45-65 years higher (42.1%) [13]. According to the 2019 survey, the prevalence of overweight and obesity, which is a risk factor for noncommunicable diseases among men in urban areas, has increased by 49.4% or 9.6% compared to 10 years ago [14]. In terms of lifestyle, the consumption of red meat, which provides the iron intake from the food, is 17 times higher than recommended in the World Health Diet for Mongolians [15]. Therefore, in this study, our objective was to determine the effect of fat metabolism and risk factors on serum iron accumulation in men at high risk for non-communicable diseases.

Materials and Methods

This study was conducted using a population-based snapshot survey design. In the study, 45.5 ± 8.4 years (average age) 227 Mongolian men living in Ulaanbaatar were included in the study with an informed consent form approved by the Research Ethics Committee of the Mongolian National University of Medical Sciences (MNUMS) (Research Ethics Committee Meeting Decision No. 21/0613). The research was carried out between October 2021 and July 2022 at the Bio-Medical School of the Mongolian National University

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Received: 30 January, 2024, Manuscript No. JMS-24-126277; **Editor Assigned:** 01 February, 2024, PreQC No. P-126277; **Reviewed:** 18 March, 2024, QC No. Q-126277; **Revised:** 23 March, 2024, Manuscript No. R-126277; **Published:** 30 March, 2024, DOI: 10.37421/2167-0943.2024.13.349

of Medical Sciences. In the study, 1. rejected the informed consent form, 2. was diagnosed with anemia, 3. was infected with hepatitis B and C virus, diagnosed with cancer, 4. had chronic or acute diseases of the digestive tract, 5. underwent surgical treatment in the last 3 months, 6. regular blood donors, 7. undergoing drug treatment for hemodialysis and cardiovascular disease, 8. regular use of iron supplements and iron-enriched products in the last 6 months, or 9. Weight loss programs in the last 6 months are not included.

Methodology of anthropometric measurements

Body Mass Index (BMI) in the study, height and weight were measured according to the standard with a BSM370 automatic measuring device and Waist Circumference (WC) and hip circumference was measured twice each with a tape measure "SECA“. Abdominal circumference was measured around the middle of the lower edge of the rib cage and the upper edge of the hip bone. Hip circumference was measured around the widest part of the hip, the Trochanter Major.

BMI=kg/m^2,

Waist hip ratio (WHR) WHR=WC/HC [16].

The following formula was used to calculate the indexes of body adiposity [17].

Body fat percentage (BFP) $BFP_{men=}$ (1.20 × BMI)+(0.23 × Age)-16.2;

$VAI_{male=}$ (WC/(39.68+(1.88 × BMI))) × (Triglyceride/1.03) × (1.31/HDL);

LAP= (WC-65) × Tg;

TyG=Ln(Triglyceride[mg/dl] × glucose [mg/dl]/2)

Methods of laboratory measurements

Participants on an empty stomach (fasted for 8 hours) and fasting glucose and lipid parameters (triglyceride, High-Density Lipoprotein (HDL)) were analyzed by biochemical analysis. Iron metabolism parameters such as serum-free iron, serum ferritin, Transferrin Saturation (TSAT), Total Iron Binding Capacity (TIBC), and Unsaturated Iron Binding Capacity (UIBC) were determined using a Roche-brand Cobas-6000 fully automatic analyzer (Cobas-c-601, Cobas-c-501). Hepcidin by the ELISA method (Shanghai Coon Koon Biotech Co., Ltd. Human Hepcidin-25 (HEP25) ELISA kit, Cat No: CK-bio-11865 and the Biobase EL-10B ELISA microplate reader) were used for determination.

In British Columbia-Investigation and Management (2021) divided serum ferritin into three groups: <300 ng/ml as normal ferritin, 300-600 ng/ml as hyperferritinemia, and >600 ng/ml as iron overload [18].

Statistical analysis

Statistics analysis was performed using IBM SPSS Statistics 26.0 software and the 95% confidence interval (95% CI) was calculated by expressing the quality variables as a percentage, and the chi-square test was used to calculate the p-value. The difference between the scale variables, depending on the distribution, was that an independent sample T test was used for the normal distribution and the Mann-Whitney U test was used for the non-normal distribution, and a value of p A less than 5% was considered significant. When evaluating the relationship between the data, Pearson's correlation was calculated if the numerical values were normally distributed, and Spearmin's correlation coefficient was calculated if the qualitative indicators and quantitative values were not normally distributed. When assessing risk factors, a covariate adjusted logistic regression analysis was performed.

Results

The general parameters of body measurements, some parameters of biochemical analysis, and body adiposity indexes of the study participants were compared according to the serum ferritin group. From Table 1, the WC, BMI, WHR, BFP, triglyceride, fasting glucose, VAI, LAP and TyG statistics in the group with elevated serum ferritin and iron overload were statistically significantly higher, but HDL was significantly lower from the normal ferritin group (p<0.05) (Table 1).

In comparing iron metabolism parameters between groups, serum free iron and TSAT decreased in hyperferritinemic group compared to normal serum ferritin group but increased in iron overload group (p<0.05). UIBC was higher in the hyperferritinemic group than in the normal serum ferritin group but lower in the iron overload group (p<0.05). Serum Hepcidin concentration was significantly increased with increasing ferritin concentration (Table 2).

In assessing the correlation between parameters, serum ferritin level had a significant positive correlation with Hepcidin (r=0.483) and TSAT (r=0.142), but no correlation was observed with other parameters of iron metabolism. Furthermore, ferritin level was positively correlated with triglyceride (r=0.244), glucose (r=0.187), BMI (r=0.223), BFP (r=0.256), WHR (r=0.201), VAI (r=0.235), LAP (r=0.279), TyG (r=0.278), which was statistically significant (p<0.05) negatively correlated with HDL (r=-0.165). HDL has a strong negative correlation (p<0.01) not only with ferritin level but also with body adiposity indexes (VAI r=-0.647, LAP r=-0.456, TyG r=-0.419). Triglycerides were strongly associated with body adiposity indexes (VAI r=0.916, LAP r=0.896, TyG r=0.932); Glucose has a weak correlation with VAI (r=0.177), LAP (r=0.137), and moderately strong positive correlation with TyG (r=0.447),

Table 1. Comparison of some biochemical and anthropometric parameters.

Parameters		Ferritin Group				p-value
		Total	Normal	Hyperferritine mia	Iron Overload	
		M ± SD	M ± SD	M ± SD	M ± SD	
		(n=227)	(n=114)	(n=77)	(n=36)	
General parameters	Weight (kg)	82.2 ± 14.7	80.9 ± 15.7	83.3 ± 13.9	84.5 ± 13.1	0.124
	Height (cm)	170.4 ± 6.5	170.7 ± 5.9	170.1 ± 7.1	170.4 ± 7.4	0.78
	WC (cm)	98.8 ± 13.1	97.3 ± 14.6	99.3 ± 11.3	102.9 ± 11.3	0.022
	BMI[a] (kg/cm^2)	28.1 ± 4.1	27.5 ± 4.2	28.6 ± 3.9	29.1 ± 4.2	0.019
	BFP[b] (%)	28.1 ± 5.4	27.1 ± 5.4	28.6 ± 5.1	29.7 ± 5.4	0.016
	WHR[c] ratio	1.02 ± 0.06	1.01 ± 0.07	1.02 ± 0.05	1.05 ± 0.06	0.035
Biochemical parameters	Triglyceride (mg/dL)	1 43.2 ± 96.3	124. 2 ± 81.3	158.5 ± 102.7	170.5 ± 114.7	0.002
	HDL[d] (mg/dL)	44.7 ± 10.4	45.9 ± 10.5	44.5 ± 10.6	41.6 ± 9.1	0.041
	Glucose (mg/dL)	80.1 ± 20.1	74.4 ± 14.9	80.4 ± 15.6	78.6 ± 9.2	0.087
Body fat indexes	VAI[e]	2.05 ± 1.6	1.81 ± 1.58	2.30 ± 1.72	2.28 ± 1.27	0.003
	LAP[f]	69.6 ± 56.1	59.1 ± 51.1	77.9 ± 59.7	85.3 ± 57.9	0.001
	TyG[g]	4.57 ± 0.33	4.49 ± 0.33	4.64 ± 0.33	4.71 ± 0.31	0.0001

Note: p-values were calculated using one-way ANOVA for variables with normality or the Kruskal-Wallis test for nonparametric variables. **a)** BMI: Body Mass Index, **b)** BFP: Body Fat Percentage, **c)** WHR: Waist Hip Ratio, **d)** HDL: High-Density Lipoprotein, **e)** VAI: Visceral Adiposity Index, **f)** LAP: Lipoprotein Accumulation Product and **g)** TyG: Triglyceride-Glucose index

which is significant (p<0.05). During the evaluation of the correlation between indexes, TyG has a strong and positive correlation (p<0.01) with VAI (r=0.802), LAP (r=0.815), and VAI with LAP (r=0.846). No significant correlation was observed between Hepcidin and body adiposity (VAI r=0.012, LAP r=0.093, TyG r=0.090) (p>0.05). Figure 1 summarizes the relation between serum ferritin, Hepcidin, and body adiposity (Figure 1).

The regression analysis with adjust based on the correlation analysis showed that all of them are positively correlated (p < 0.05) or risk factors in the unadjusted model when assessing the risk of serum ferritin level with body adiposity indexes. The risk of increased serum ferritin level is increased the visceral adiposity index by 23%, the lipid accumulation product by 2%, and the insulin resistance index by 4.91 times. Linear regression analysis showed that model 2, adjusted for WHR (OR=4.31, 95% CI: 1.797-10.348), model 3, adjusted for WHR, age, WC, BMI, BFP (OR=3.74, 95%CI: 1.521-9.218) respectively, TyG index still has a strong effect (p<0.05) (Table 3).

Table 2. Comparison of some iron metabolism parameters.

Parameters of Iron Metabolism	Ferritin Group				p-value
	Total	Normal	Hyperferritine mia	Iron overload	
	M ± SD	M ± SD	M ± SD	M ± SD	
Iron (mg/dL)	114.5 ± 42.01	114.9 ± 47.8	102.5 ± 23.5	136.6 ± 46.4	0.0001
UIBC[a] (mg/dL)	209.2 ± 42.2	209.7 ± 43.8	221.3 ± 39.9	185.03 ± 31.9	0.0001
TIBC[b] (mg/dL)	323.8 ± 49 .1	324.6 ± 51.3	323.8 ± 43.8	321.6 ± 54.1	0.958
TSAT[c] (%)	35.1 ± 10.1	34.9 ± 11.6	31.8 ± 6.4	41.9 ± 9.07	0.0001
Hepcidin (ng/dL)	4.17 ± 0.87	3.84 ± 0.77	4.15 ± 0.63	5.03 ± 0.93	0.0001

Note: p-values were calculated using one-way ANOVA for variables with normality or the Kruskal-Wallis test for nonparametric variables. **a)** UIBC is the Unsaturated Iron Binding Capacity, **b)** TIBC is the Total Iron-Binding Capacity and **c)** TSAT is the Transferrin Saturation

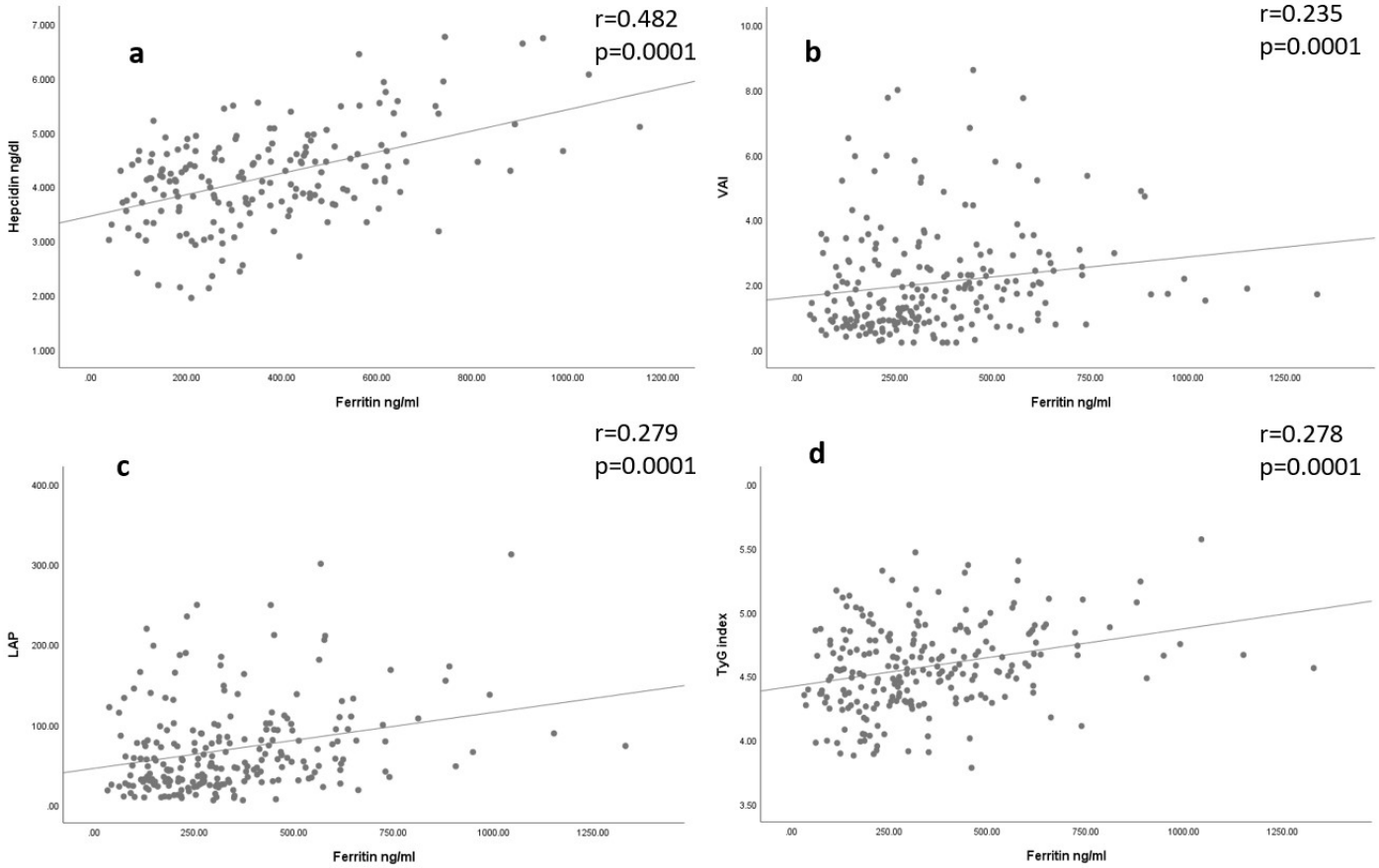

Figure 1. Correlation between the indexes of ferritin, Hepcidin and body adiposity. **a)** Ferritin *vs.* Hepcidin, **b)** Ferritin *vs.* Visceral Adiposity Index (VAI), **c)** Ferritin *vs.* Lipid Accumulation Product (LAP) and **d)** Ferritin *vs.* Triglyceride-Glucose Index (TyG). Correlations among variables with normal distributions are shown as Pearson's correlation coefficients, and for variables with skewed distribution Spearman's correlation coefficients are used.

Table 3. Logistic regression analysis of the association of VAI, LAP and TyG with serum ferritin (ng/ml).

Ferritin	Model 1		Model 2		Model 3	
	ß (95%CI)	p	ß (95%CI)	p	ß (95%CI)	p
VAI[a]	1.23 (1.032-1.466)	0.021	1.17 (0.973-1.410)	0.095	1.15 (0.951-1.392)	0.148
LAP[b]	1.02 (1.002-1.013)	0.005	1.006 (1.000-1.012)	0.037	1.006 (1.000-1.012)	0.065
TyG[c]	4.91 (2.095-11.502)	0.0001	4.31 (1.797-10.348)	0.001	3.74 (1.521-9.218)	0.004

Note: Model 1 –unadjusted. Model 2–adjusted for WHR. Model 3–WHR, age, WC, BMI and BFP. **a)** VAI: Visceral Adiposity Index, **b)** LAP: Lipoprotein Accumulation Product and **c)** TyG: Triglyceride-Glucose index

Discussion

A rise in serum iron accumulation from the normal level leads to the Fenton reaction in the cells, causing cell damage and causing further non-communicable diseases. Red meat consumption is 17 times higher than the world health diet recommended by the Mongolian people, and in the last 10 years, the obesity rate has increased 9.6 times [15]. Therefore, Mongolians need to clarify serum iron level and its related factors among men in the high-risk group.

In our study, 49.8% of the subjects showed elevated serum ferritin levels, of which 15.9% had iron overload, which included Mongolian men aged 30 to 60 years with a high mortality rate due to non-communicable diseases. Then with an increase in serum ferritin, the anthropometric and functional parameters that determine body fatness increase and the HDL level decreases. In a Chinese study that included 7,488 subjects, the average BMI of 3,485 male participants had an mean results of BMI, WC, triglycerides, HDL and glucose levels were corresponding to a lower than the results of our study [19]. Similarly, in a study of 1,120 people aged 18 to 84 in Ireland, men in a group at risk of iron accumulation had mean BMI, WC, WHR, and the indexes of visceral adiposity and lipid accumulation product were higher than the other groups. However, a positive correlation between body fat parameters and serum ferritin in men was similar to our results [7]. Similar to the results of other researchers, the visceral adiposity index and the lipid accumulation index were directly related to loss of lipid and carbohydrate metabolism, according to the study of Kavaric N, et al. [20]. Metabolism dysfunction accounts for a significant proportion of the cause of iron overload, and insulin resistance is a risk factor for hyperferritinemic and iron overload, both in adults and adolescents [21-23].

Serum ferritin level is associated with obesity, visceral adiposity, and central obesity in men, and the prevalence of obesity increased in the group with increased ferritin level [7]. Adipose tissue accumulation is responsible for metabolic loss and imbalance of iron metabolism. Body adiposity indexes are a risk factor for elevated serum ferritin in our study, but no association with Hepcidin was found. However, Rodríguez-Mortera R, et al. conducted a study among adolescents and the loss of lipid metabolism was positively correlated with Hepcidin, which differs from our results [21]. This suggests that adolescent obesity affects iron metabolism by increasing Hepcidin secretion. However, in our study, Hepcidin was positively correlated with ferritin levels but not with adiposity indexes, possibly due to dysregulation of iron metabolism in overweight or obese subjects.

Hepcidin, a peptide hormone secreted by liver cells, regulates the iron metabolism. In case of increased iron level in the serum, the balance of iron metabolism is maintained by reducing the mechanism of absorption through the intestinal wall. However, in the obese group, inflammatory mediators released from adipose tissue increase the release of hepcidin, which may lead to an increase in serum ferritin, causing the imbalance of iron metabolism [12]. In the future, the causes of iron accumulation in the serum of Mongolian people need to be investigated in relation to lifestyle characteristics, consumption of red meat, physical activity, and other inflammatory diseases.

Conclusion

For Mongolian men, one of the factors that increases the serum ferritin level and alters the balance of iron metabolism is body adiposity. The adiposity indexes, also known as anthropometric parameters and clinical parameters, are a preventive indicator of increased iron accumulation in the serum and, therefore, in the future it is necessary to study it in conjunction with other factors.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Bolortsetseg, Zorigtbaatar, M. Lutzul, G. Munkhtuul and N. Sugar, et al. "Association between Serum Ferritin Levels and Risk Factors of Obesity among Mongolian Men." *J Metabolic Synd* 13 (2024): 349.