THE NEW ARMENIAN MEDICAL JOURNAL
Volume18 (2024), Issue 3, p. 35-41

DOI: https://doi.org/10.56936/18290825-3.v18.2024-35

COMPARISON OF LEPTIN AND FERRITIN LEVELS IN BETA-THALASSEMIA MAJOR AND HEALTHY INDIVIDUALS

Nourbakhsh S.M.K.[1], Hashemi E.[2], Kheyri M.[3], Bahadoram M.[4], Keikhaei B.[4], Hassanzadeh S.[4]*

[1] Department of Pediatric Hematology and Oncology, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran
[2] Department of Pediatrics, Child Growth and Development Research Center, Research Institute for Primordial Prevention of Non-Communicable Disease, Isfahan University of Medical Sciences, Isfahan, Iran
[3] Deputy of Curative Affairs, Ministry of Health and Medical Education, Tehran, Iran
[4] Thalassemia and Hemoglobinopathy Research Center, Research Institute of Health, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran

Received 02.019.2022; Accepted for printing 04.08.2024

Abstract

Introduction: *Beta-thalassemia major is an inherited hematologic disorder that is characterized by decreased or lack in the synthesis of beta-hemoglobin sequence. An increase in ferritin leads to a decrease in leptin in healthy individuals.*

This study aimed to compare leptin and ferritin levels in the patients with beta-thalassemia major and healthy individuals.

Material and Methods: *This cross-sectional study was conducted in 2018-2019. The study participants were divided into two groups including 57 patients with beta-thalassemia major in the case group and 57 healthy volunteers in the control group. Leptin and ferritin levels were measured using the ELISA kit. The data were entered into SPSS 21 software and were compared and analyzed using the T-test.*

Results: *The mean leptin level in the healthy individuals and patients with beta-thalassemia major was 10.17 ± 8.29 ng/ml and 3.23 ± 3.22 ng/ml, respectively. The ferritin level in healthy individuals was 99.14 ± 90.5 ng/ml compared to 1233.456 ± 0.701 ng/ml in the patients with beta-thalassemia major.*

Conclusion: *In beta-thalassemia major patients, the leptin level was lower compared to healthy individuals. In addition, an increase in ferritin levels leads to a decrease in leptin levels in healthy individuals. However, this finding was not observed in patients with beta-thalassemia major.*

Keywords: *beta thalassemia major, leptin, ferritin, hematologic disorder, iron overload.*

Introduction

Beta thalassemia major (β-TM) is a hematologic disease caused by decreased synthesis of the beta-hemoglobin sequence (the main deficiency in the patients) [*Thein S, 2013*]. The decrease in the synthesis of the beta-hemoglobin sequence causes a relative increase in the alpha sequence inside erythroid cells. As a result, abnormal molecule alpha 4 emerges which is unstable and precipitates

Cite This Article as:

Nourbakhsh S.M.K., Hashemi E., Kheyri M., Bahadoram M., Keikhaei B., Hassanzadeh S. (2024). Comparison of leptin and ferritin levels in beta-thalassemia major and healthy individuals: A systematic review, The New Armenian Medical Journal, vol.18(2), 35-41; https://doi.org/ 10.56936/18290825-3.v18.2024-35

Address for Correspondence:

Shakiba Hassanzadeh
Thalassemia and Hemoglobinopathy Research Center
Research Institute of Health, Ahvaz Jundishapur University of Medical Sciences Ahvaz 15794-61357, Iran
Tel.: +98-61-33750410
E-mail: shak.hassanzadeh@gmail.com

in the membrane of the red globules. These globules are lysed and destroyed in the reticuloendothelial system. Therefore, β-TM is classified as hemolytic anemia [*Loukopoulos D, 1991; Viprakasit V, Ekwattanakit S, 2018*]. β-TM is more prevalent in the Mediterranean populations as well as in Iran. β-TM is the most severe form of thalassemia during which the complete lack of beta-protein in hemoglobin leads to life-threatening anemia. These individuals are dependent on regular blood transfusion and continuous medical care [*Kliegman R et al.,2017; Hadipour Dehshal M et al., 2019*]. The normal range of hemoglobin defers depending on the age and gender of the individuals; children aged between 7 to 12 years (11-16 *gr/dl*), adult women (12-16 *gr/dl*), and adult men (12-18 *gr/dl*) [*Glader B, 2004*]. On the other hand, the repeated blood transfusion for survival in patients with β-TM causes iron accumulation in different tissues and organs leading to fibrosis, cell death, and organ failure (particularly the heart and secreted glands) [*Lukens J, 1999; Crisponi G et al., 2019*]. The main complications of patients with β-TM include delayed development, delayed sexual puberty, short height, thinness, and hypogonadism [*Karachaliou F et al., 2006; Badiger S, Barauh A, 2019*] These are related to the low levels of hemoglobin, high levels of ferritin, and insufficient treatment with iron chelators [*Saxena A, 2003*].

Leptin is a polypeptide hormone with 16 kDa and 146 amino acids that was discovered in 1994 [*Gainsford T, Alexander W, 1999; Izadi V et al., 2014*]. This hormone is secreted through lipid cells and has an important contribution to the survival of the body in the long term. Leptin decreases appetite and increases energy intake by affecting the hypothalamus. In addition, leptin prevents neuropeptide Y (stimulates appetite) and promotes gamma-melanocyte-stimulating hormone decreasing appetite through the hypothalamus [*Ebbeling C et al., 2006; Kliegman R, 2007; Zhang Y, Chua S, 2017*]. Leptin concentration is higher in women than men and is age-dependent. In both genders, the increase in leptin concentration is accompanied by a decrease in leptin receptor concentration and leptin concentration in women is lower than in men [*Gainsford T, Alexander W, 1999*].

The toxic effects of iron overload on cellular membrane and proteins have raised a hypothesis that iron overload decreases the leptin level by destroying lipid cells [*Youson J, Sargent P, 1984; Gao Y et al., 2019*]. Ferritin is the most important iron reserve in the body and is used to assess iron metabolism-related disorders. Ferritin is an intracellular protein and a globular protein complex consisting of 24 protein subunits that save iron as a nontoxic solution. It is responsible for reserving iron in the body [*Braunwald M et al., 2001; Wang W et al., 2010*]. The normal level of ferritin is 300-400 *ng/ml* in men and 15-200 *ng/ml* in women. The level of this protein in the serum of individuals is directly associated with iron reserves [*Tsiotra P et al., 2000; Saito H, 2014*].

This study aimed to determine the potential association between leptin and β-TM as well as the potential association between the iron reserve and leptin levels.

MATERIAL AND METHODS

Study design: This analytical, cross-sectional study was conducted in 2014. The sample size included two groups. The case group consisted of 57 β-TM patients (50.9% men and 49.1% women) with a mean age of 18.8 years. The control group included 57 healthy individuals (45.6% men and 54.4% women) with a mean age of 18 years.

3cc of blood sample with no anti-coagulation agent was obtained from the participants before blood transfusion. The serum was obtained after coagulation and kept at -20ºC until the performance of the analysis. Before blood taking, written informed consent was obtained from the participants or their parents. The patients' personal information such as age, gender, height, weight, and duration of treatment were documented. The healthy volunteers were matched with the patients by age, gender, and weight. The ELISA kit was used for measuring the serum leptin and ferritin levels. This kit is ELISA-based and its sensitivity is 0.5 *ng/ml*.

Ethical issues: The research follows the principles of the Declaration of Helsinki. The Ethics Committee of Shahrekord University of Medical Sciences, Shahrekord, Iran has approved all of this study's protocols (IR.SKUMS.REC.1392.5.14). Written informed consent was obtained from all participants before any intervention.

Data analysis: The data were analyzed with the SPSS 21 software using the statistical tests of T-test and Pearson correlation. The $p< 0.05$ was considered statistically significant.

Results

In this study, 57 patients with β-TM including 29 males (50.9%) and 28 females (49.1%) as well as 57 healthy individuals consisting of 26 males (45.6%) and 31 females (54.4%) were evaluated. The mean leptin level in the female and male patients with β-TM was 4.85 ± 3.5 *ng/ml* and 1.47 ± 1.6 *ng/ml*, respectively. The leptin levels in the patients with β-TM were significantly lower compared to the control group (p=0.0001). The mean leptin level in healthy women and men was 14.5 ± 8.32 *ng/ml* and 5.01 ± 1.49 *ng/ml*, respectively. The mean leptin level had a significant difference between the two study groups ($p< 0.05$) (Fig. 1). In total, the leptin level in healthy individuals was 10.17 ± 8.29 *ng/ml*, which was notably different from that in the patients with β-TM (3.22 ± 3.23 *ng/ml*) (Table 1). The level of leptin was significantly lower compared to healthy individuals. In addition, the leptin level in the patients with β-TM was positively associated with their age. As the age advanced, their leptin level increased (p= 0.002) and vice versa (Fig. 2).

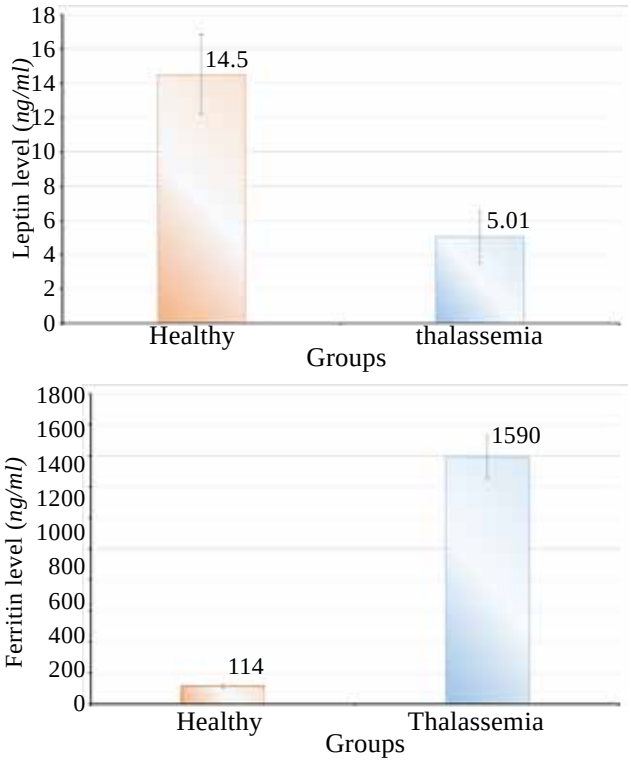

Figure 1. *The serum levels of leptin (a) and ferritin (b) in the study groups (healthy individuals and thalassemia patients.*

Table 1.

The study of leptin and ferritin levels in the two study groups.

Healthy		
Leptin	Mean	10.18±8.29
	p	< 0.0001
Ferritin	Mean	99.14±90.5
	p	< 0.0001
Major thalassemia		
Leptin	Mean	3.23±3.22
	p	0.0
Ferritin	Mean	1233.46±701
	p	0.335

There was a significant difference in the ferritin level between the two study groups (p= 0.0). The ferritin level in healthy individuals was 99.14 ± 90.5 *ng/ml*. This was notably different from that in the patients with β-TM (1233.456 ± 0.701 *ng/ml*) (Table 1). The ferritin level in the patients with

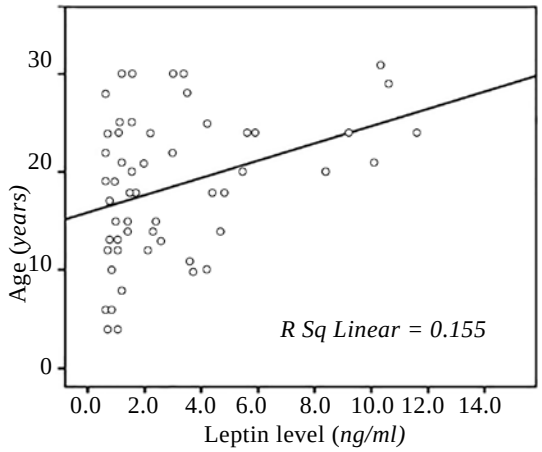

Figure 2. *The association between leptin and age in the patients.*

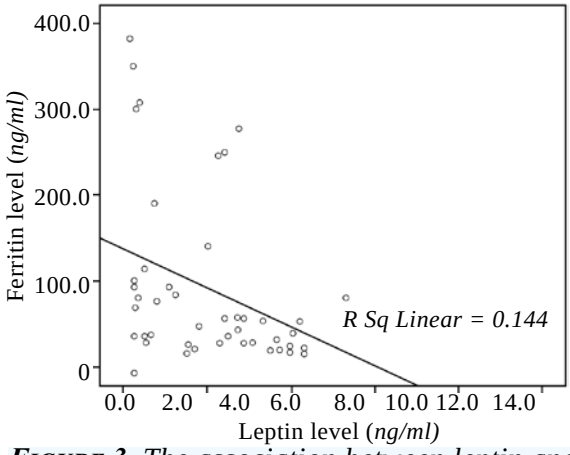

Figure 3. *The association between leptin and ferritin in healthy individuals.*

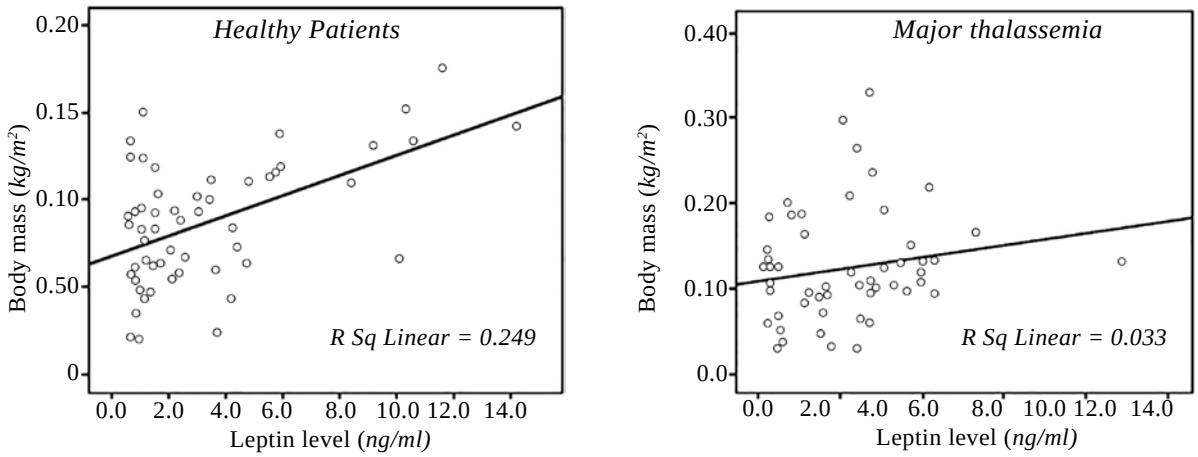

FIGURE 4. *The association of leptin level with body mass in the two study groups.*

β-TM (1590±142.5 *ng/ml*) was higher compared to the healthy individuals (114±14.6 *ng/ml*) (Fig. 3).

The ferritin level in the patients with β-TM had no significant association with their age. However, as the age of the healthy individuals advanced, the ferritin level increased. The leptin and ferritin levels were negatively associated with healthy individuals. As the ferritin level increased and decreased, the leptin level decreased and increased (p=0.004) (Fig. 3). However, there was no significant association between leptin and ferritin levels in the patients with β-TM.

The leptin level in healthy individuals and the patients with β-TM was positively associated with the body mass. Along with the increase and/or decrease in leptin, the body mass increased and/or decreased (p=0.004). In addition, individuals with lower leptin in the body had lower body mass index (BMI) (Fig. 4). There was no significant association between the ferritin level and the body mass in the two study groups.

DISCUSSION

Major thalassemia occurs in all parts of the world and blood transfusion is the best option of controlling the disorder. The effects of this disease change the body hormone levels and the iron load rate. The ferritin level and the rate of a hormone derived from the body fat (such as leptin) constantly affect the development (height and weight) and sexual puberty in these patients.

The results of the present study indicated that the mean leptin level in these patients was lower than normal and the mean ferritin level was higher than normal compared to healthy individuals (Fig. 1). As the age of the patients advanced, the leptin level increased (Fig. 2). In this study, the leptin level was higher in women than men. In addition, there was a positive significant association between leptin and BMI and a significant negative association between leptin and ferritin in healthy individuals, but not in the patients with β-TM (Fig. 3, 4, 5).

In a study on 162 patients with major thalassemia and 138 healthy individuals, the serum level of leptin was evaluated. There was a significant difference in the serum leptin level between the male patients (2.69 ± 1.23 µgr/l) and the control group (6.86 ± 2.71 µgr/l) (p<0.0005). There was also a significant difference in the serum leptin level between the female patients (6.37±2.9 µgr/l) and normal individuals (9.37 ± 5.2 µgr/l) (p<0.05) [*Miraglia del Giudice E et al., 1999*]. Dedousis et al. (2002) reported the leptin levels of the patients with thalassemia and healthy individuals as 1.9 *ng/dl* and 14.6 *ng/dl*, respectively. This indicates that the patients with thalassemia had lower leptin levels [*Dedoussis G. et al., 2002*]. This is in agreement with the present study. The mean leptin level in these patients was lower than the normal level and equal to 3.22 *ng/dl*, with a significant difference from that of healthy individuals (10.17 *ng/dl*). Another study showed that leptin was significantly lower in the patients compared to controls. The low serum leptin in beta-thalassemia was possibly due to the toxic effect of iron overload on the adipose tissue [*Chaliasos N et al., 2010*]. In the present study, the leptin level in male patients was lower with a mean of 1.6 *ng/dl* and different from that in the healthy individuals with a mean of 4.85 *ng/dl*. In addition, the leptin level in female pa-

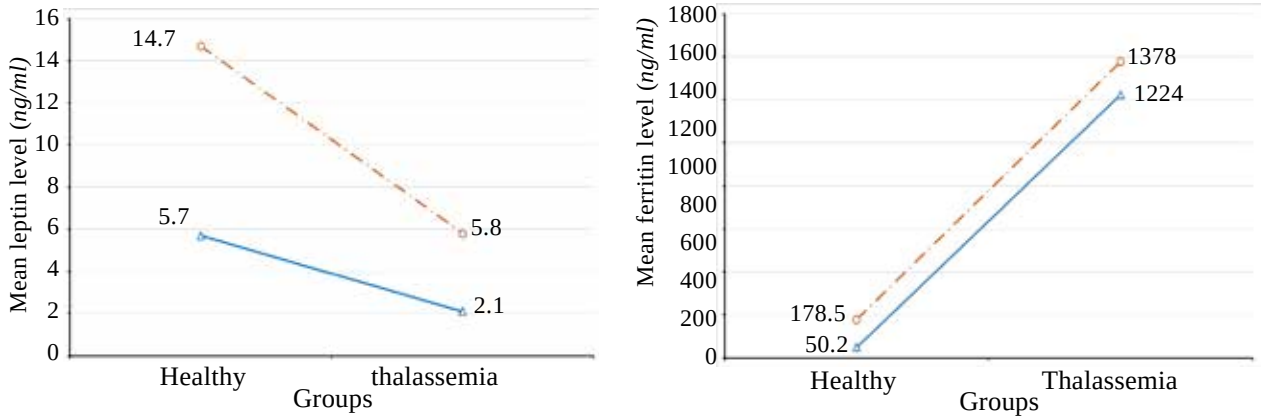

Figure 5. *The mean serum levels of leptin and ferritin during the study in the two study groups based on the gender of the patients (Women: dotted line; Men: solid line).*

tients was significantly different and lower (mean of 5.01 *ng/dl*) compared to healthy individuals (14.5 *ng/dl*). The most probable reason for this difference is the toxic effects of iron overload on the cellular membrane and proteins. Free iron (in the cases of iron overload) stimulates hydroxyl free radicals and per oxidative products that damage the lipid membrane and proteins. Therefore, in overload cases (such as in thalassemia), leptin declines because of the destruction of lipid cells. Moreover, the lipid cells that contain yellow marrow and are replaced by red marrow, which occurs in hemolytic anemia conditions, could be the reason for the decline in leptin in patients with thalassemia. Investigation of the association between leptin and BMI in healthy individuals revealed that they were significantly and positively associated which is consistent with previous studies [*Moller N et al., 1998; Suzuki K et al., 2003*].

In addition, the results of the present study indicated that leptin in the group of patients with β-TM had a positive association with their BMI. Their BMI increased as the leptin increased. Based on the previous studies and the present study, there is a strong association between serum leptin and BMI. As both serum leptin and BMI are low in thalassemia patients, iron precipitation in lipid cells seems to decrease lipid and the production of leptin. We also found that the leptin level was positively associated with age in the patients with β-TM. As the age of the patients advanced, their leptin levels increased. This is probably caused by the increase in the lipid tissue as the patients become older. A study reported that the decreased serum leptin level in patients with β-TM had a significant positive association with age and the levels were higher in females compared to males. Moreover, there was a significant negative association between serum ferritin and serum leptin [*Ebtesam R et al., 2018*]. Another study reported that major thalassemia reduces the serum levels of leptin regardless of age and body mass. In addition, they found an inverse statistical correlation between the serum levels of leptin and ferritin among the studied individuals [*Shahramian I et al., 2013*]. In the present study, there was a strong association between leptin and gender in healthy individuals and patients with β-TM, which is in line with the previous studies [*Saad M et al., 1997*]. Leptin was higher in female participants which might be attributed to the physiologically higher lipid tissue in women compared to men. However, another possible reason may be the sexual hormones that influence leptin. Al-Naama et al. (2016) revealed that the serum leptin levels were significantly lower in patients with β-TM than in healthy individuals. In addition, the low serum leptin was sensitive to predict short stature and was significant in β-TM females only [*Al-Naama L et al., 2016*]. Choobineh et al. (2009) reported that the adipocytes of patients with β-TM were unable to produce sufficient leptin. This suggests that the dysfunction of the adipose tissue can be considered as an endocrinopathy that affects the patients with β-TM [*Choobineh H et al., 2009*]. Since leptin increases with the initiation of cardiac involvement, it can also be a predictive marker of cardiac involvement in patients with β-TM [*Shahramian I et al., 2015*].

CONCLUSION

In this study, leptin and ferritin were measured in the same blood sample and compared between healthy individuals and patients with β-TM. The leptin and ferritin levels were negatively associated with healthy individuals. As ferritin increased or decreased, the leptin level decreased or increased (p=0.004). However, there was no significant association between the levels of ferritin and leptin in the patients with β-TM. This is probably due to the medications such as desferal that affect the patients' ferritin level and, in turn, change the findings. In addition, another reason for this could be the fact that leptin varies in a specific interval in the patients. As ferritin increases, the decline in leptin does not exceed a certain limit. However, the leptin and the ferritin levels are constantly lower and higher in patients with β-TM, respectively. This indicates the necessity and significance of controlling the serum iron as well the leptin hormone in these patients.

ACKNOWLEDGEMENT: *We would like to thank the Research and Technology Deputy of Shahrekord University of Medical Sciences, Shahrekord, Iran for funding the project (Grant No 1458) and also the Research Unit of Hajar Hospital and all staff for cooperating with us in implementing the project.*

REFERENCES

1. *Al-Naama LM, Hassan MK, Abdul Karim MM (2016).* Evaluation of Serum Leptin Levels and Growth in Patients with β-Thalassaemia Major. Anemia. 2016: 8454286 DOI: 10.1155/2016/8454286
2. *Badiger S, Barauh A (2019).* A study of growth pattern in regularly transfused thalassemic children of age group of 2 years to 12 years. Int J Contemp Pediatr. 6: 1575-1581 DOI: 10.18203/2349-3291.ijcp20192758
3. *Braunwald MD, Eugene, Fauci MD, Anthony S, Kasper MD (2001).* Harrison's Principles of Internal Medicine: 15th Edition, 2-Volume Set (Harrison's Principles of Internal Medicine (2v.) McGraw Hill
4. *Chaliasos N, Challa A, Eleftheria H, Koutsouka F, Bourantas DK. Vlahos AP., et al (2010).* Serum adipocytokine and vascular inflammation marker levels in Beta-thalassaemia major patients. Acta Haematol. 124(4): 191-196 DOI: 10.1159/000320274
5. *Choobineh H, Dehghani SJ, Alizadeh Sh, Ghobadi Dana V, Saiepour N, Meshkani R, Einollahi N (2009).* Evaluation of Leptin Levels in Major beta-Thalassemic Patients. International Journal of Hematology-Oncology and Stem Cell Research. 3(4): 1-4
6. *Crisponi G, Nurchi VM, Lachowicz JI (2019).* Iron Chelation for Iron Overload in Thalassemia. Met Ions Life Sci. 19 DOI: 10.1515/9783110527872-009
7. *Dedoussis G, Kyrtsonis MC, Andrikopoulos N, Voskaridou E, Loutradis A (2002).* Inverse correlation of plasma leptin and soluble transferrin receptor levels in β-thalassemia patients. Annals of hematology. 81(9): 543-547 DOI: 10.1007/s00277-002-0499-7
8. *Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. (2006).* Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. Pediatrics. 117(3): 673-680 DOI: 10.1542/peds.2005-0983
9. *Ebtesam R, Hagag AA, Badraia IM, Maaly MM (2018).* Study of Serum Leptin in Children with Beta Thalassemia: Correlation with Iron Overload. The Medical Journal of Cairo University. 3037-3045 DOI: 10.21608/mjcu.2018.59871
10. *Gainsford T, Alexander WS (1999).* A role for leptin in hemopoieses? Molecular biotechnology. 11(2): 149-158 DOI: 10.1007/BF02915808
11. *Gao Y, Liu J, Bai Z, Sink S, Zhao C, Lorenzo FR, McClain DA (2019).* Iron down-regulates leptin by suppressing protein O-GlcNAc modification in adipocytes, resulting in decreased levels of O-glycosylated CREB. Journal of Biological Chemistry. 294(14): 5487-5495 DOI: 10.1074/jbc.RA118.005183

12. *Glader B (2004).* Iron deficiency anemia. Dalam: Behrman RE, Kliegman RM, Jenson HB, Penyunting. Nelson Textbook of pediatrics. Edisi-17. Philadelphia: WB Saunders
13. *Hadipour Dehshal M, Tabrizi Namini M, Hantoushzadeh R, Yousefi Darestani S (2019).* β-Thalassemia in Iran: Things Everyone Needs to Know About This Disease. Hemoglobin. 43(3): 166-173 DOI: 10.1080/03630269.2019.1628774
14. *Izadi V, Saraf-Bank S, Azadbakht L (2014).* Dietary intakes and leptin concentrations. ARYA Atheroscler. 10(5): 266-272
15. *Karachaliou F, Vlachopapadopoulou E, Theochari M, Konstandellou E, Michalados S (2006).* Leptin levels in patients with thalassemia major. Minerva pediatrica. 58(4): 373-378
16. *Kliegman RM, Behrman RE, Jenson HB, Stanton BMD., et al (2007).* Nelson textbook of pediatrics e-book. Elsevier Health Sciences
17. *Kliegman RM, Lye PS, Bordini BJ, Toth H, Basel D (2017).* Nelson Pediatric Symptom-Based Diagnosis E-Book. Elsevier Health Sciences
18. *Loukopoulos D (1991).* Thalassemia: genotypes and phenotypes. Annals of hematology. 62(4): 85-94 DOI: 10.1007/BF01702920
19. *Lukens J (1999).* The thalassemias and related disorder: an overview. Wintrobe's Clinical Hematology. 405-433
20. *Miraglia del Giudice E, Perrotta S, Carbone MT, Calabro C, Esposito L, De Rosa C., et al (1999).* Evaluation of leptin protein levels in patients with Cooley's anaemia. British journal of haematology. 105(3): 839-840 DOI: 10.1046/j.1365-2141.1999.01484.x
21. *Moller N, O'Brien P, Nair KS (1998).* Disruption of the relationship between fat content and leptin levels with aging in humans. The Journal of Clinical Endocrinology & Metabolism. 83(3): 931-934 DOI: 10.1210/jcem.83.3.4620
22. *Saad MF, Damani S, Gingerich RL, Riad-Gabriel MG, Khan A., et al (1997).* Sexual dimorphism in plasma leptin concentration. The Journal of Clinical Endocrinology & Metabolism. 82(2): 579-584 DOI: 10.1210/jcem.82.2.3739
23. *Saito H (2014).* Metabolism of iron stores. Nagoya J Med Sci. 76(3-4): 235-254
24. *Saxena A (2003).* Growth retardation in thalassemia major patients. International Journal of Human Genetics. 3(4): 237-246 DOI: 10.1080/09723757.2003.11885858
25. *Shahramian I Akhlaghi E, Ramezani A, Rezaee A, Noori N, Sharafi E (2013).* A study of leptin serum concentrations in patients with major Beta-thalassemia. Iran J Ped Hematol Oncol. 3(2): 59-63
26. *Shahramian I, Noori NM, Teimouri A, Akhlaghi E, Sharafi E (2015).* The Correlation between Serum Level of Leptin and Troponin in Children with Major Beta-Thalassemia. Iran J Ped Hematol Oncol. 5(1): 11-17
27. *Suzuki K, Ito Y, Ochiai J, Kusuhara Y, Hashimoto S., et al (2003).* Relationship between obesity and serum markers of oxidative stress and inflammation in Japanese. Asian Pacific Journal of Cancer Prevention. 4(3): 259-266
28. *Thein SL (2013).* The molecular basis of β-thalassemia. Cold Spring Harb Perspect Med. 3(5): a011700-a011700 DOI: 10.1101/cshperspect.a011700
29. *Tsiotra PC, Pappa V, Raptis SA, Tsigos C (2000).* Expression of the long and short leptin receptor isoforms in peripheral blood mononuclear cells: implications for leptin's actions. Metabolism-Clinical and Experimental. 49(12): 1537-1541 DOI: 10.1053/meta.2000.18519
30. *Viprakasit V, Ekwattanakit S (2018).* Clinical Classification, Screening and Diagnosis for Thalassemia. Hematol Oncol Clin North Am. 32(2): 193-211 DOI: 10.1016/j.hoc.2017.11.006
31. *Wang W, Knovich MA, Coffman LG, Torti FM, Torti SV (2010).* Serum ferritin: Past, present and future. Biochim Biophys Acta. 1800(8): 760-769 DOI: 10.1016/j.bbagen.2010.03.011
32. *Youson J, Sargent P (1984).* Iron deposition in the integument of lampreys. The Anatomical Record. 209(4): 461-468 DOI: 10.1002/ar.1092090406
33. *Zhang Y, Chua S, Jr (2017).* Leptin Function and Regulation. Compr Physiol. 8(1): 351-369 DOI: 10.1002/cphy.c160041

THE NEW ARMENIAN MEDICAL JOURNAL

Volume18 (2024). Issue 3

The Journal is founded by
Yerevan State Medical
University after M. Heratsi.

Rector of YSMU

Armen A. Muradyan

Address for correspondence:

Yerevan State Medical University
2 Koryun Street, Yerevan 0025,
Republic of Armenia

Phones:

(+37410) 582532 YSMU
(+37493 588697 Editor-in-Chief
Fax: (+37410) 582532
E-mail:namj.ysmu@gmail.com, ysmiu@mail.ru
URL:http//www.ysmu.am

Our journal is registered in the databases of Scopus, EBSCO and Thomson Reuters (in the registration process)

Scopus *EBSCO*
Reuters

Copy editor: Tatevik R. Movsisyan

Printed in "LAS Print" LLC
Director: Suren A. Simonyan
Armenia, 0023,Yerevan,
Acharyan St. 44 Bulding,
Phone: (+374 10) 62 76 12,
E-mail: las.print@yahoo.com

THE NEW ARMENIAN MEDICAL JOURNAL
Volume18 (2024). Issue 3

CONTENTS