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RESEARCH

Effects of Type 1 diabetes mellitus disease on iron metabolism and hepcidin level abstract

Tip 1 diyabet mellitus hastalığının demir metabolizması ve hepsidin düzeyi üzerine etkileri

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Abstract

Purpose: The aim of this study was to reveal the relationship between iron metabolism, hepcidin, and hemoglobin A1c (HbA1c) in type 1 diabetes mellitus (T1DM) patients with better and poor metabolic control.
Materials and Methods: The study included 30 T1DM patients with poor metabolic control (HbA1c value >7.5%), 30 T1DM patients with better metabolic control (HbA1c value ≤7.5%), and 30 healthy controls who were of similar age and gender. Routine, hemogram, biochemical tests, iron, transferrin, and ferritin levels were evaluated.
Results: The hepcidin level of the poor metabolic patient group (519.52±114.58) was significantly higher than the control group (431.94±124.05). The ferritin level of the poor metabolic patient group (52.10±36.53) was significantly higher than that of the better metabolic patient group (35.13±36.18) and the control group (28.63±18.08). Positive relationships were found between HbA1c and hepcidin and ferritin. In receiver operating characteristic (ROC) analysis, hepcidin was at the threshold value of 445.20 and had 61.7% sensitivity and 56.0% specificity in diagnosing T1DM.
Conclusion: Hepcidin and ferritin levels have been shown to increase in T1DM patients and both show a positive correlation with HbA1c. Hepcidin may be a promising marker contributing to the follow-up and diagnosis of T1DM.
Keywords:. Type 1 diabetes mellitus, hepcidin, iron metabolism

Öz

Amaç: Bu çalışmanın amacı daha iyi ve kötü metabolik kontrole sahip tip 1 diabetes mellitus (T1DM) hastalarında demir metabolizması, hepsidin ve hemoglobin A1c (HbA1c) arasındaki ilişkiyi ortaya koymaktır.
Gereç ve Yöntem: Çalışmaya kötü metabolik kontrole sahip 30 T1DM hastası (HbA1c değeri >%7,5), daha iyi metabolik kontrole sahip 30 T1DM hastası (HbA1c değeri ≤%7,5) ve benzer yaş ve cinsiyete sahip 30 sağlıklı kontrol dahil edildi. Rutin, hemogram, biyokimyasal testler, demir, transferin ve ferritin düzeyleri değerlendirildi.
Bulgular: Kötü metabolik hasta grubunun hepsidin seviyesi (519,52±114,58) kontrol grubundan (431,94±124,05) anlamlı olarak yüksekti. Kötü metabolik hasta grubunun ferritin seviyesi (52,10±36,53) daha iyi metabolik hasta grubundan (35,13±36,18) ve kontrol grubundan anlamlı olarak yüksekti (28,63±18,08). HbA1c ile hepsidin ve ferritin arasında pozitif ilişkiler bulundu. Alıcı çalışma karakteristiği (ROC) analizinde, hepsidin 445,20 eşik değerindeydi ve T1DM'yi teşhis etmede %61,7 duyarlılık ve %56,0 özgüllüğe sahipti.
Sonuç: Hepsidin ve ferritin düzeylerinin T1DM hastalarında arttığı ve her ikisinin de HbA1c ile pozitif korelasyon gösterdiği gösterilmiştir. Hepsidin T1DM hastalığının takibine ve teşhisine katkıda bulunan umut verici bir belirteç olabilir.

Anahtar kelimeler: Tip 1 diabetes mellitus, hepsidin, demir metabolizması

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INTRODUCTION

Type 1 diabetes mellitus (T1DM) is a type of diabetes with an absolute deficiency of insulin, also known as "insulin-dependent diabetes"[1]. T1DM patients may face the risk of many complications throughout their lives. One of these complications is related to iron metabolism. It is thought that the iron metabolism in T1DM patients can be better clarified, the more benefit it will provide in the treatment and follow-up of the disease[2].

All mammals need a certain amount of iron. Both deficiency and excess of iron at the cellular level are undesirable and cause pathological consequences. Therefore, the concentration of iron in biological fluids is strictly controlled to ensure that it is used when necessary and to prevent its toxicity. This control is mainly achieved by regulating intestinal absorption[3,4].

Hepcidin is a peptide hormone that is synthesized in the liver, circulates, and excreted in the urine, and is an important regulator of systemic iron balance. It has this effect by reducing iron absorption from the small intestines, ensuring that the iron formed as a result of macrophage breakdown of expired erythrocytes is retained in macrophages, and preventing the release of iron from the liver into the circulation. Increasing the rate of erythropoiesis, decreasing oxygen concentrations, and decreasing iron stores reduce hepcidin synthesis. Iron accumulation in the body and inflammation are conditions that cause increased hepcidin synthesis[4,5].

Since T1DM is known to be a chronic inflammatory disease characterized by low-grade inflammation, we aimed to reveal the relationship between iron metabolism, hepcidin, and hemoglobin A1c (HbA1c) in T1DM patients with better and poor metabolic control in the current study.

MATERIALS AND METHODS

Sample

Sixty T1DM patients who were followed up in the Pediatric Endocrinology department of KSU Faculty of Medicine between February 1, 2022, and February 1, 2024, were included in the study. As a healthy control group, 30 children of similar age and gender to the case group who applied to the Child Health and Diseases and Child Endocrinology Polyclinics for routine check-ups between the same dates and who did not have any acute or chronic disease as a result of their examinations and tests and who were not using any medication were included.

Those who had an infection in the last 4 weeks, those with hemoglobinopathy, those with congenital malformations, those with oncological diseases, those who received iron therapy in the previous 6 weeks, those who received erythrocyte suspension replacement in the last 6 months, those who did not give written consent, those who have missing data were excluded from the study.

According to International Society for Pediatric and Adolescent Diabetes (ISPAD), the patient group includes 30 T1DM patients in the better metabolic group with HbA1c value ≤7.5%, which is the most valuable indicator for glycemic control in patients with T1DM, and 30 T1DM patients in the poor metabolic group with HbA1c value >7.5%[6,7]. The control group was planned to be 30 healthy patients.

Demographic data (age, body mass index (BMI), height, gender, weight), mean corpuscular volume (MCV), red blood cell count (RBC), hemoglobin, hematocrit, red cell distribution width (RDW), transferrin, C-reactive protein (CRP), total iron binding capacity (TIBC), ferritin, reticulocyte, iron, HbA1c, erythrocyte sedimentation rate (ESR), fasting blood glucose, and hepcidin levels were examined. Transferrin saturation was calculated and all data was recorded.

Procedure

The Kahramanmaraş Sütçü İmam University (KSU) Clinical Research Ethics Committee approved this work (2021/17-03). All families were informed about the purpose and content of the study and their informed written consent was obtained.

Measurements

Study participants' height and weight were measured by the same physician during the clinical examination using a precision scale and tape measure. Body mass index (BMI) was then calculated and recorded by dividing weight by height in meters squared.

Sample collection

For hematological and biochemical analyses of the

patients and controls, after a minimum of 8 hours of fasting, 2 cc of blood was collected into three biochemistry tubes and 2 cc of blood was collected into two EDTA tubes. The blood samples collected in the biochemistry tubes were centrifuged at 3500 rpm for 10 minutes, and the separated serum was placed in Eppendorf tubes and stored at -80ºC in the Biochemistry Laboratory of Kahramanmaraş Sütçü İmam University, Faculty of Medicine, Health Practice and Research Hospital.

Reagent kits, methods, and devices used

Iron level, iron-binding capacity, and fasting blood glucose levels were measured using a Roche Hitachi Cobas C702 fully automated biochemistry autoanalyzer. Ferritin, vitamin B12, and folic acid levels were measured using an electrochemiluminescence device using a Cobas 602 device. Transferrin levels were measured using a nephelometric method using a BN-II device. CRP was measured using a nephelometric method using a BN-II device. Hemogram and reticulocyte count were measured using a Sysmex XN-3000 fully automated blood counting device. Erythrocyte sedimentation rate (ESR) was measured using an HPLC device. Photometrically, the Vision-c device was used. Hepcidin levels were measured using the ELISA method using a Biotek 800 microplate reader with a detection range of 62.5 to 4000 pg/mL. For the evaluation of hepcidin, the USCN Hepcidin hormone ELISA (E91979Hu) (Enzyme-Linked Immunosorbent Assay Kit for Hepcidin) kit was used.

Statistical analysis

The SPSS 22 package application was used. Categorical data were displayed as n (%), and continuous data as mean ± standard deviation. To ascertain whether continuous variables were appropriate for a normal distribution, the Kolmogorov-Smirnov test was employed. To compare categorical variables, the Pearson Chi-square method was utilized. If the data were normally distributed, the "One Way Anova Test" was used for comparing independent groups, and the Tukey Test was used for pairwise comparisons. If the data were not normally distributed, the "Kruskal Wallis Test" was used, for pairwise comparisons the Mann-Whitney U Test was used, and statistical significance was evaluated by applying Bonferroni correction. In pairwise comparisons, test results were considered significant if $P<0.017$. The relationship between variables was evaluated with the Spearman Correlation Coefficient. For the differential diagnosis of the patient group from the control group, the cut-off value was estimated by making a Receiver Operator Characteristic (ROC) curve of HbA1c and Hepcidin measurement values, and sensitivity and specificity values were found. The significance was set at $p<0.05$.

RESULTS

Between the poor metabolic, better metabolic, and control groups, there were no significant differences in demographic data ($p>0.05$) (Table 1).

Table 1. Demographic and anthropometric characteristics of groups

Parameter	Poor metabolic T1DM (n=30)	Better metabolic T1DM (n=30)	Healthy Control (n=30)	p-value
Sex (n %)				0.065*
Female	12 (40.00)	16 (53.30)	21 (70.00)	
Male	18 (60.00)	14 (46.70)	9 (30.00)	
Age (years)	11.63±3.89	11.10±3.93	12.10±3.07	0.571
Height (cm)	145.30±22.40	142.20±21.82	148.53±16.52	0.489
Weight (kg)	42.78±19.65	40.34±16.45	43.95±14.79	0.707
BMI (kg/m2)	18.89±4.26	18.83±3.55	19.37±3.17	0.827

*: Chi-Square Test. **: One-way Anova Analysis; T1DM, Type 1 diabetes mellitus; BMI, Body mass index.

In the laboratory analyses of the poor metabolic, better metabolic, and control groups, a significant difference was determined between TIBC, transferrin, ferritin, HbA1c, glucose, and hepcidin levels ($p=0.002$, $p<0.001$, $p=0.010$, $p<0.001$, $p<0.001$, $p=0.028$, respectively) (Table 2).

Glucose, HbA1c, and ferritin levels of the poor metabolic patient group were found to be significantly higher than the better metabolic patient group and the control group ($p<0.001$ and $p<0.001$; $p<0.001$ and $p<0.001$; $p=0.009$ and $p=0.009$, respectively). Glucose and HbA1c levels of the better

metabolic patient group were higher than the control group (p=0.031, p<0.001, respectively).

The TIBC value of the poor and better metabolic patient groups was significantly lower than the control groups (p=0.002, p=0.018, respectively). Transferrin was higher in the poor metabolic group than in the control group (p<0.001).

The hepcidin level of the poor metabolic patient group was significantly higher than the control group (p = 0.015) (Table 2).

Table 2. Laboratory findings of groups

Parameter	Poor metabolic T1DM (n=30)	Better metabolic T1DM (n=30)	Healthy Control (n=30)	p-value
HGB (gr/dl)	14.03±1.71	14.02±1.45	13.89±1.08	0.923**
HCT (%)	41.82±4.90	41.73±3.68	41.01±2.73	0.854*
RBC (x10^6/uL)	5.07±0.55	5.25±0.36	5.06±0.33	0.159**
MCV (fL)	82.49±5.30	80.16±6.10	81.17±4.07	0.410*
RDW-CV (%)	13.23±1.10	13.44±1.26	13.46±0.88	0.497*
Retikulocyte (%)	1.16±0.46	1.19±0.35	1.04±0.32	0.245**
CRP (mg/L)	3.50±1.53	3.59±1.60	3.00±0.00	0.163**
ESR (mm/h)	7.47±6.92	6.50±6.74	7.50±5.22	0.795**
Iron (mg/dl)	87.14±41.71	79.50±34.17	69.52±28.84	0.214*
TIBC (mg/dl)	250.90±67.95†	263.73±63.39†	311.93±70.37	0.002**
Transferrin (gr/L)	2.95±0.51†	2.69±0.46	2.48±0.31	P<0.001**
T. Sat. (%)	40.99±31.29	39.21±31.81	25.04±15.73	0.067*
Ferritin (ng/ml)	52.10±36.53†,‡	35.13±36.18	28.63±18.08	0.010*
Glucose (mg/dl)	242.53±130.08†,‡	144.47±53.03†	90.47±8.80	P<0.001**
HbA1c (%)	10.21±2.44†,‡	6.88±0.45†	5.26±0.38	P<0.001*
Hepcidin (pg/ml)	519.52±114.58†	461.33±90.55	431.94±124.05	0.028*

*: Kruskal Wallis Analysis, **: One-way Anova Analysis. †: There is a significant difference between the control group and the control group. ‡: There is a significant difference between the good metabolic group and the good metabolic group. T1DM: Type 1 diabetes mellitus, HGB: hemoglobin, HCT: hematocrit, RBC: red blood cell count, RDW-CV: red blood cell distribution percentage, T. sat: transferrin saturation, MCV: mean corpuscular volume, TIBC: total iron binding capacity ESR: erythrocyte sedimentation rate.

Table 3. Correlation analysis of the parameters

		Hepcidin	Iron	TIBC	Trf.	T. Sat	Ferritin	HbA1c	Glucose
Hepcidin	r		0.264	-0.161	0.009	0.248	0.125	0.261	0.162
	p		0.012	0.129	0.931	0.019	0.241	0.013	0.126
Iron	r	0.264		-0.661	-0.041	0.899	0.380	0.095	0.078
	p	0.012		p<0.001	0.703	p<0.001	p<0.001	0.372	0.464
TIBC	r	-0.161	-0.661		0.217	-0.808	-0.525	-0.233	-0.167
	p	0.129	p<0.001		0.040	p<0.001	p<0.001	0.027	0.117
Transferrin	r	0.009	-0.041	0.217		-0.131	-0.039	0.348	0.293
	p	0.931	0.703	0.040		0.218	0.712	0.001	0.005
Transferrin Saturation	r	0.248	0.899	-0.808	-0.131		0.485	0.161	0.111
	p	0.019	p<0.001	p<0.001	0.218		p<0.001	0.130	0.298
Ferritin	r	0.125	0.380	-0.525	-0.039	0.485		0.281	0.081
	p	0.241	p<0.001	p<0.001	0.712	p<0.001		0.007	0.449
HbA1c	r	0.261	0.095	-0.233	0.348	0.161	0.281		0.668
	p	0.013	0.372	0.027	0.001	0.130	0.007		p<0.001
Glucose	r	0.162	0.078	-0.167	0.293	0.111	0.081	0.668	
	p	0.126	0.464	0.117	0.005	0.298	0.449	p<0.001	

*Spearman correlation coefficient; Trf: transferrin, T. saturation: transferrin saturation, TIBC: total iron binding capacity; r: correlation coefficient; p: p-value

The correlation analysis of the parameters is given in Table 3. There was a positive relationship between HbA1c and glucose, hepcidin, ferritin, and transferrin (p<0.001, p=0.013, p=0.007, p=0.001, respectively)

(Table 3). There was a negative relationship between HbA1c and TIBC (p=0.027). There was a positive relationship between hepcidin and iron and transferrin saturation (p=0.012, p=0.019 respectively) (Table 3).

In receiver operating characteristic (ROC) analysis, hepcidin was at the threshold value of 445.20 and had 61.7% sensitivity and 56.0% specificity in diagnosing T1DM (p=0.043) (Table 4).

Table 4. Results of the receiver operating characteristic curve analysis for the HbA1c, and Hepcidin

	AUC	95% CI	Cut-off	Sensitivity	Specificity	p-value
HbA1c	0.990	0.971-1.010	6.55	91.7	100	**<0.001**
Hepcidin	0.631	0.493-0.769	445.20	61.7	56.7	**0.043**

DISCUSSION

The results of this study demonstrated a positive correlation between hepcidin and HbA1c in patients with T1DM, and that hepcidin could be used as a promising biomarker in the diagnosis and monitoring of T1DM.

Hepcidin plays an important role in iron metabolism and is a recently discovered low-molecular-weight liver peptide. Iron must be in the correct balance to maintain normal cellular function[4,8]. There are very few studies in the literature examining iron metabolism and hepcidin levels in children with T1DM[2,9,10,11].

The results of studies investigating hepcidin levels in T1DM are contradictory. Sam et al.[9] reported that there was no difference between hepcidin levels of patients with T1DM and controls. Rusak et al.[2] showed that hepcidin levels were higher in T1DM patients than in controls. It has been reported that the reasons for such differences are not clear and are likely to be due to various factors that may affect hepcidin levels[12]. Venkatesan et al.[12], when these factors are controlled for, showed that serum hepcidin levels were significantly higher in diabetic patients compared to control subjects and stated that they believe their results provide evidence that hepcidin levels are high in diabetics. In the current study, the hepcidin levels of the poorly metabolic T1DM group, like others[2,12], were significantly higher than the control group. Furthermore, there is a positive correlation between hepcidin and HbA1c.

It has been reported that the TIBC level of the T1DM patient was significantly lower than control[2]. Similarly, in the current study, TIBC levels were lower in the patient group. Additionally, the current study found that there was a negative relationship between TIBC and HbA1c.

Venkatesan et al.[12] reported that serum ferritin levels were significantly higher in the diabetic group than in the control group. Akkermans et al.[13] did not find a relationship between HbA1c and ferritin. They attributed this to the fact that they could not reach the required power to detect HbA1c levels between patients with and without iron deficiency due to the low prevalence of iron deficiency and anemia in their study. Like Metwalley et al.[14], in this study, ferritin levels of T1DM patients were higher than controls, and a positive relationship was detected between HbA1c and ferritin. Since we investigated iron status in patients with T1DM, a chronic inflammatory disease, in our study, we did not have any patients with iron deficiency.

Similar to our study, serum ferritin and hepcidin levels were reported to be higher, and TIBC was lower in patients with gestational diabetes mellitus (GDM)[15]. Furthermore, Meka et al.[16] concluded in their meta-analysis that high hepcidin is associated with the pathogenesis of GDM and that high hepcidin levels may help in early GDM diagnosis. Increased hepcidin expression and increased ferritin levels have been shown to increase the risk of developing type 2 DM[17]. Furthermore, it has been reported that high serum ferritin and hepcidin levels in newly diagnosed diabetic patients indicate that iron homeostasis is impaired[12].

T1DM is a chronic inflammatory disease[18]. Diabetes is quite common in people with iron overload, and iron is recognized to have a function in glucose regulation. Serum ferritin serves as an indirect indicator of the body's total iron storage capacity. Ferritin is the primary protein in cells that stores iron. Serum ferritin, on the other hand, is an acute-phase

protein that rises during an inflammation. The liver is the primary source of hepcidin, a crucial regulator of iron homeostasis. Inflammation or high iron levels can raise hepcidin levels[19]. In the current study, it is thought that hepcidin and ferritin levels increase in T1DM patients due to the chronic inflammatory process. In this regard, studies using inflammatory cytokines such as IL-1, IL-6, and TNF-α and higher sensitivity inflammatory markers such as high sensitivity C-reactive protein (hs-CRP) are needed.

In conclusion, it has been observed that hepcidin and ferritin levels increase in T1DM patients due to the chronic inflammatory process and both show a positive correlation with HbA1c. Furthermore, TIBC levels were significantly lower in T1DM patients and there was a negative relationship between TIBC and HbA1c. Hepcidin can be considered a promising marker that contributes to the follow-up and diagnosis of T1DM disease. However, our study has several limitations. The relatively small sample size we used can be considered a limitation of our study. Another limitation of our study is the absence of any histological or cellular analysis and the lack of supporting data at the immunohistochemical or cell-based level (e.g. liver tissue expression, inflammatory cell analysis). The final limitation of our study is that most of the studies were conducted in the last few years, and since the hepcidin biomarker is relatively new, not much evidence was found. Why and by which mechanisms hepcidin and ferritin increase in T1DM have not yet been fully elucidated and need further investigation.

Considering the multifactorial nature of T1DM, it is thought that there may be different factors affecting hepcidin levels in this process and that studies are needed with the participation of more patients and inflammatory cytokines such as IL-1, IL-6 and TNF-α and high-sensitivity inflammation markers such as hs-CRP (high-sensitivity C reactive protein).

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