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Research

Correlation of Urine Haptoglobin and Albumin Levels in Predicting Early Nephropathy in Individuals with Obesity

Obezitesi Olan Bireylerde Erken Nefropatiyi Öngörmede İdrar Haptoglobin ve Albümin Düzeylerinin Korelasyonu

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ABSTRACT

Objective: Urine haptoglobin, an alpha-2 sialoglycoprotein, is associated with renal failure even in the early stages. We aimed to determine the correlation between the urinary haptoglobin-to-creatinine ratio (HCR) and the albumin-to-creatinine ratio (ACR) in diabetic, prediabetic, and non-diabetic obese patients without chronic renal failure.

Methods: A total of 102 obese individuals aged ≥18 years, including diabetic patients, normoglycemic patients, and patients with impaired fasting glucose disorders, were included in this cross-sectional study. Biochemical and urine parameters, including fasting blood glucose, glycated hemoglobin, renal and liver function tests, and spot urine ACR and HCR, were analyzed.

Results: Among study participants, 17.6% (n=18) had impaired fasting glucose, 46.1% (n=47) had type 2 diabetes mellitus, and 36.3% (n=37) were normoglycemic. Urinary ACR was significantly higher and urinary HCR was significantly lower in the diabetic group compared with the other two groups. A moderate, significant positive correlation was observed between ACR and HCR in the diabetic group (r=0.454; p=0.001). Although HCR was significantly higher in non-diabetic individuals with obesity (p=0.011) compared with other groups, no significant relationship was found between ACR and HCR.

Conclusion: We determined a relationship between HCR and ACR levels in diabetic patients with obesity. As a result, evaluation of urinary haptoglobin may be useful for predicting nephropathy related to diabetes mellitus and obesity. Further large-sample prospective studies are warranted to reach more precise conclusions regarding variations in HCR in the obese population.

Keywords: Haptoglobinuria, albuminuria, renal function, obesity, type 2 diabetes

ÖZ

Amaç: Bir alfa-2 sialoglikoprotein olan haptoglobinin idrardaki düzeyi, erken evrelerde bile böbrek yetmezliği ile ilişkilidir. Biz bu çalışmada, tümü obeziteli olan diyabetik, prediyabetik ve normoglisemik hastalarda idrar haptoglobin/kreatinin oranı (HKO) düzeylerini ölçmeyi ve albümin/kreatinin oranı (AKO) ilişkisini değerlendirmeyi amaçladık.

Gereç ve Yöntem: Diyabetik, normoglisemik ve bozulmuş açlık glukozu olan 18 yaş üstü toplam 102 obeziteli hasta kesitsel çalışmaya dahil edildi. Açlık kan şekeri, glikozillenmiş hemoglobin, böbrek ve karaciğer fonksiyon testleri, spot idrar AKO ve spot idrar HKO içeren biyokimyasal ve üriner parametreler analiz edildi.

Bulgular: Katılımcılar arasında %46,1'inde (n=47) tip 2 diyabet, %36,3'ünde (n=37) normoglisemik ve %17,6'sında (n=18) bozulmuş açlık glukozu olan obeziteli hasta mevcuttu. Diyabetik grupta AKO diğer 2 gruba kıyasla anlamlı derecede yüksek, HKO anlamlı derecede düşüktü. AKO ile HKO arasında ise sadece diyabetik grupta istatistiksel olarak orta derecede anlamlı bir korelasyon gözlendi (r=0,454; p=0,001; p<0,01). Diyabetik olmayan obeziteli bireylerde HKO diğer gruplara göre anlamlı olarak yüksek olmasına rağmen (p=0,011; p<0,05), AKO ile HKO arasında anlamlı bir ilişki saptanmadı.

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ÖZ

Sonuç: Çalışmamızda obeziteli diyabetik hastalarda HKO ve AKO düzeyleri arasında istatiksel olarak anlamlı bir ilişki saptandı. Sonuç olarak idrar haptoglobin düzeyinin değerlendirilmesinin diyabet ve obeziteye bağlı nefropatiyi öngörmede faydalı olabileceğini düşünüyoruz. Obeziteli popülasyonda haptoglobin varyasyonları açısından daha kesin sonuçlara varmak için daha büyük örneklemli prospektif çalışmalara ihtiyaç vardır.

Anahtar Kelimeler: Haptoglobinüri, albüminüri, renal fonksiyon, obezite, tip 2 diyabet

INTRODUCTION

Diabetes mellitus (DM) is a significant public health problem, with its prevalence steadily rising (1). The most common cause of end-stage renal disease in developing countries is diabetic nephropathy. Early diagnosis of diabetes and management of blood pressure and glucose levels can prevent or delay its progression (2). Obesity, defined as a body mass index (BMI) of 30 kg/m^2 or higher, is among the leading metabolic risk factors for global disease burden, and its prevalence has continued to increase in recent decades (3-5). It is also a recognized risk factor for the development of chronic kidney disease (CKD) (6,7). It is associated with an increased total glomerular filtration rate (GFR) (8,9), suggesting a potential direct pathogenic relationship with kidney disease such that long-term kidney damage may ensue without intervention.

Urinary albumin-to-creatinine ratio (ACR) is a well-established marker of diabetic nephropathy. However, microalbuminuria (30-300 mg albumin/gram creatinine) is neither a specific nor a sensitive predictor of renal progression, as it does not always precede macroalbuminuria (10,11). Moreover, studies have shown that diabetic nephropathy can develop in individuals with normal albuminuria (12-14).

Haptoglobin is an acute-phase reactant which, beyond its role in hemolysis, contributes to inflammation and immune responses (15). A two- to fivefold increase in haptoglobin occurs in response to inflammatory stimuli (16). Recent studies have linked urinary haptoglobin levels to clinical outcomes in both individuals with and without type 2 DM. Elevated urinary haptoglobin has been identified as a potential predictor of kidney disease progression in patients with type 2 DM (17-19). However, the relationship between urinary haptoglobin and kidney disease in individuals with obesity without DM remains unexplored.

This study aims to evaluate the potential of haptoglobinuria as an alternative to albuminuria for predicting nephropathy by comparing urinary haptoglobin and albumin levels among individuals with diabetes and obesity, obese individuals with normal glucose levels, and individuals with impaired fasting glucose (IFG).

METHODS

A cross-sectional study was performed in the internal medicine outpatient service of our hospital between February and May 2017. Participants aged 18 years or older with a BMI >30 who volunteered were included in the study. Exclusion criteria included estrogen treatment, hepatocellular disease, nephrotic syndrome, active infection, malignancy, and CKD (GFR <60).

Recruited participants were stratified into diabetic, prediabetic, and normoglycemic groups based on their glycemic status. The participants' weight and height were measured with the assistance of the physician, and their BMI was calculated. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2) (3). Fasting blood glucose (FBG), glycated hemoglobin (HbA1c), aspartate aminotransferase (AST), alanine aminotransferase (ALT), urea, creatinine, C-reactive protein, sodium, potassium, uric acid, calcium, phosphorus, and complete blood count were collected. Spot urine samples were subjected to a complete urine examination and analyzed for creatinine, albumin, and haptoglobin levels. Blood samples were collected after an eight-hour fast and centrifuged at 4,000×g for eight minutes. The ACR and the haptoglobin-to-creatinine ratio (HCR) were calculated. "IGF was defined as FBG levels from 100 to 125 mg/dL (from 5.6 to 6.9 mmol/L)" (20). "The abbreviated MDRD equation is: GFR=186×Scr (serum kreatinine) (-1.154)×age(-0.203)×(0.742 if female)×(1.210 if African-American), where Scr is measured in mg/dL and age in years" (21). We compared the biochemical parameters, ACR, and HCR between groups and evaluated the relationship between the ACR and the HCR within groups.

The University of Health Sciences Türkiye, Bağcılar Training and Research Hospital Non-Interventional Clinical Research Ethics Committee (approval number: 2016-554, date: 27.12.2016) approved the study, and all participants gave written informed consent.

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, version 20.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics, including mean, standard

deviation, median, minimum, maximum, first quartile, and third quartile, were calculated for quantitative variables. Frequency and percentage distributions were used for categorical variables. The Shapiro-Wilk test and box plots were employed to assess data normality. For comparisons among three or more groups, one-way analysis of variance was used for data that were normally distributed, followed by the Games-Howell test for pairwise comparisons.

Kruskal-Wallis test and Dunn's test were used for anomalously distributed data. Associations between categorical variables were assessed using the chi-square test. Spearman's correlation analysis was conducted to examine relationships between variables. The results were evaluated at the 95% confidence level, and statistical significance was defined as $p<0.05$.

RESULTS

A total of 102 participants were enrolled in the study, with 84.3% (n=86) being female and 15.7% (n=16) being male. The age range was 18-74 years, with a mean age of 51.66±10.36 years. BMI values ranged from 27.4 to 66.9 kg/m^2, with a mean of 36.45±5.52 kg/m^2 (Table 1). Regarding glycemic status, 17.6% (n=18) had IFG, 46.1% (n=47) had DM, and 36.3% (n=37) were normoglycemic (Figure 1).

No significant gender differences were observed (p>0.05), but age differed significantly among groups (p=0.001). Specifically, the normoglycemic group was significantly younger than both the IFG and DM groups (p=0.035 and p=0.001, respectively) (Table 2). The groups were homogeneous in terms of sex and anthropometric measurements.

Table 1. Distribution of descriptive features

		n (%)
Sex	**Female**	86 (84.3)
	Male	16 (15.7)
Age	Mean±SD	51.66±10.36
	Median (min-max)	52 (18-74)
Height	Mean±SD	158.23±8.31
	Median (min-max)	157 (140-183)
Weight	Mean±SD	90.98±14.89
	Median (min-max)	88.8 (64.4-160)
BMI	Mean±SD	36.45±5.52
	Median (min-max)	36.1 (27.4-66.9)
Groups	**IFG**	18 (17.6)
	DM	47 (46.1)
	NG	37 (36.3)

SD: Standard deviation, IFG: Impaired fasting glucose, DM: Diabetes mellitus, NG: Normoglycemic, BMI: Body mass index

HbA1c levels differed significantly among groups (p=0.001), with the DM group exhibiting higher values compared to both the IFG and normoglycemic groups (p=0.001 for both comparisons). No significant differences in urea, creatinine, GFR, AST, or ALT levels were observed among groups (p>0.05) (Table 3).

ACR levels differed significantly among groups (p=0.016); the DM group showed higher values than the normoglycemic group (p=0.013) (Table 4). HCR levels also differed significantly among groups (p=0.014), with the normoglycemic group exhibiting higher values than those in the IFG group (p=0.011) (Table 4).

No significant correlation was found between ACR and HCR in the IFG or normoglycemic groups (p>0.05). However, a positive, moderate, and significant correlation was observed between ACR and HCR in the DM group (r=0.454; p=0.001) (Table 5).

DISCUSSION

In the present study, ACR was significantly elevated in the DM group compared to the other two groups, whereas HCR was significantly lower in the DM group. A moderate correlation between ACR and HCR levels was observed in the DM group. Although the presence of DM did not increase haptoglobinuria in individuals with obesity, it was more pronounced in albuminuric diabetic patients.

Previous research has investigated the role of urinary haptoglobin in diabetic nephropathy. Bhensdadia et al. (17) identified elevated haptoglobinuria as a strong predictor of early renal functional decline among normoalbuminuric patients with type 2 DM. Yang et al. (18) proposed urinary haptoglobin as a novel biomarker, complementary to

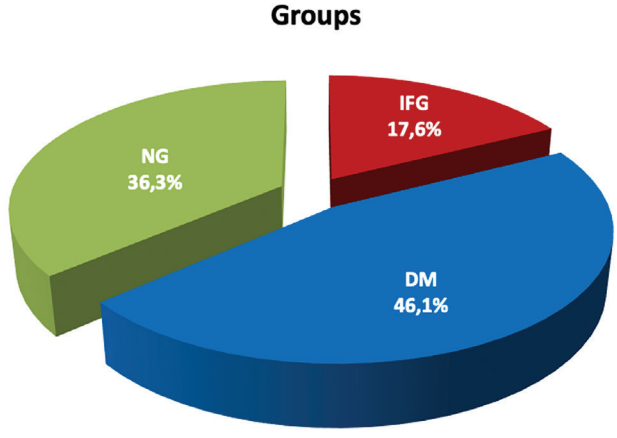

Figure 1. Distribution of the groups
NG: Normoglycemic, IFG: Impaired fasting glucose, DM: Diabetes mellitus

Table 2. Comparison of descriptive characteristics by groups

		Groups			p-value
		IFG (n=18)	DM (n=47)	NG (n=37)	
Sex	Female	16 (88.9)	38 (80.9)	32 (86.5)	[a]**0.656**
	Male	2 (11.1)	9 (19.1)	5 (13.5)	
Age	Mean±SD	53.28±8.72	55.40±8.12	46.11±11.38	[b]**0.001****
	Median (min-max)	55 (29-67)	53 (39-74)	49 (18-67)	
Height	Mean±SD	153.28±6.34	158.32±8.86	160.51±7.53	[b]**0.009****
	Median (min-max)	153 (140-162)	158 (145-183)	158 (149-177)	
Weight	Mean±SD	86.77±13.30	91.67±16.53	92.14±13.34	[b]**0.417**
	Median (min-max)	84.8 (67.3-120)	88.9 (64.4-160)	89.4 (70.8-137)	
BMI	Mean±SD	36.94±5.22	36.58±6.32	36.04±4.62	[b]**0.834**
	Median (min-max)	36.5 (30.5-48.9)	35.7 (27.4-66.9)	36 (30-52.3)	

[a]: Pearson chi-square, [b]: One-way ANOVA test and Games-Howell test, **: p<0.01, SD: Standard deviation, IFG: Impaired fasting glucose, DM: Diabetes mellitus, NG: Normoglycemics, BMI: Body mass index, ANOVA: Analysis of variance

Table 3. Comparison of biochemical parameters in blood by groups

		Total	Groups			p-value*
			IFG (n=18)	DM (n=47)	NG (n=37)	
FBG	Mean±SD	145.78±74.02	103.39±7.84	203.15±75.66	93.54±5.06	[c]**0.001****
	Median (Q1-Q3)	106 (96-168)	104.5 (101-106)	176 (137-257)	95 (92-97)	
HbA1c	Mean±SD	7.21±2.24	5.95±0.27	8.94±2.28	5.62±0.27	[c]**0.001****
	Median (Q1-Q3)	6.1 (5.8-8)	6 (5.8-6.1)	8.4 (7.2-9.7)	5.7 (5.4-5.8)	
Urea	Mean±SD	26.88±6.76	26.72±7.47	27.77±7.43	25.84±5.41	[b]**0.433**
	Median (Q1-Q3)	27 (23-31)	24.5 (22-33)	28 (22-32)	25 (23-28)	
Creatinine	Mean±SD	0.69±0.12	0.64±0.11	0.7±0.12	0.7±0.11	[b]**0.170**
	Median (Q1-Q3)	0.7 (0.6-0.8)	0.6 (0.6-0.7)	0.7 (0.6-0.8)	0.7 (0.6-0.8)	
GFR	Mean±SD	100.08±12.52	101.98±10.16	96.91±11.51	103.19±14.04	[b]**0.056**
	Median (Q1-Q3)	100.5 (91.9-108.3)	103.5 (96-108.3)	98.9 (89.6-104.2)	101.7 (94.8-113.9)	
AST	Mean±SD	21.75±12.1	22.17±18.87	23.34±12.54	19.51±5.77	[c]**0.452**
	Median (Q1-Q3)	19 (15-23)	17.5 (16-20)	19 (15-27)	19 (15-23)	
ALT	Mean±SD	23.58±16.98	23.5±26.06	26.53±16.54	19.86±10.65	[c]**0.092**
	Median (Q1-Q3)	18 (13-26)	16 (13-25)	22 (15-34)	18 (13-23)	
NA	Mean±SD	140.51±3.31	140.61±2.59	139.34±3.36	141.95±3.05	[b]**0.001****
	Median (Q1-Q3)	141 (138-143)	140.5 (139-142)	139 (137-142)	142 (140-144)	
K	Mean±SD	4.57±0.37	4.49±0.36	4.59±0.40	4.60±0.34	[b]**0.554**
	Median (Q1-Q3)	4.6 (4.3-4.8)	4.5 (4.3-4.7)	4.6 (4.3-4.9)	4.5 (4.4-4.8)	
Uric asid	Mean±SD	4.20±1.14	4.43±1.07	4.18±1.12	4.10±1.21	[b]**0.611**
	Median (Q1-Q3)	4.2 (3.3-5)	4.3 (3.5-5.1)	4 (3.3-4.8)	4.2 (3.1-5)	

[b]: One-way ANOVA test and Games-Howell test, [c]: Kruskal-Wallis test and Dunn-Bonferroni test, Q1: First quartile, Q3: Third quartile, SD: Standard deviation, IFG: Impaired fasting glucose, DM: Diabetes mellitus, NG: Normoglycemics, FBG: Fasting blood glucose, HbA1c: Glycated hemoglobin, GFR: Glomerular filtration rate, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, Na: Sodium, K: Potassium, *:p<0.05, **: p<0.01

albumin, for predicting kidney damage in patients with type 2 DM. Liu et al. (14) reported urinary haptoglobin as a predictor of renal progression and of rapid renal function decline in patients with type 2 DM. Consistent with these findings, our study demonstrated a correlation between increased haptoglobinuria and increased albuminuria in patients with diabetes. Unexpectedly, normoglycemic obese individuals exhibited higher HCR levels than those

Table 4. Comparison of biochemical parameters in urine by groups

		Total	Groups			p-value*
			IFG (n=18)	DM (n=47)	NG (n=37)	
Urine albumin (mg/L)	Mean±SD	5.12±12.19	2.32±2.55	7.70±16.45	3.21±7.20	[c]**0.032***
	Median (Q1-Q3)	1.7 (0.6-3.6)	1.5 (0.7-2.5)	2.2 (0.9-5.3)	1.1 (0.5-2.4)	
Albumin/creatinine ratio (mg/g)	Mean±SD	44.43±112.77	21.46±26.65	72.42±159.95	20.04±27.49	[c]**0.016***
	Median (Q1-Q3)	13.5 (6.3-31)	13.2 (7.8-20.2)	20 (6.9-60.4)	8.7 (4.5-24.7)	
Urine creatinine (mg/dL)	Mean±SD	86.5±70.17	68.55±45.06	95.6±82.38	83.67±62.51	[c]**0.587**
	Median (Q1-Q3)	67.8 (42.5-107.1)	58.1(30.2-92.3)	75.7 (42.8-128.8)	65.5 (43.5-93.5)	
Haptoglobin (ng/m)	Mean±SD	146.76±354.09	99.35±395.15	103.75±236.21	224.45±443.84	[c]**0.003****
	Median (Q1-Q3)	7.4 (2-37)	2.4 (0.7-6.2)	7.2 (2.3-37)	9.3 (6.7-130.9)	
Haptoglobin/creatinine ratio (ng/mg)	Mean±SD	30.02±95.34	30.84±126.12	19.05±61.96	43.56±112.88	[c]**0.014***
	Median (Q1-Q3)	1.4 (0.3-7.4)	0.3 (0.1-1.1)	1.4 (0.3-7.2)	2 (0.6-26.3)	

[b]: One-way ANOVA test and Games-Howell test, [c]: Kruskal-Wallis test and Dunn-Bonferroni test, Q1: First quartile, Q3: Third quartile, SD: Standard deviation, IFG: Impaired fasting glucose, DM: Diabetes mellitus, NG: Normoglycemic, ANOVA: Analysis of variance, *: p<0.05, **: p<0.01

Table 5. Relationship between albumin/creatinine ratio and haptoglobin/creatinine ratio in groups

Groups	ACR-HCR	
	r	p-value
IFG	-0.074	**0.769**
DM	0.454**	**0.001****
NG	0.183	**0.278**

ACR: Albumin/creatinine ratio, HCR: Haptoglobin-to-creatinine ratio, IFG: Impaired fasting glucose, DM: Diabetes mellitus, NG: Normoglycemics, **: p<0.01

in other groups, suggesting that haptoglobinuria may be a more sensitive marker of obesity-related nephropathy than albuminuria. The influence of haptoglobin genotypes, not assessed in our study, could be a contributing factor.

Obesity, independent of cardiometabolic disease, is a risk factor for renal injury (6,7,22), with glomerular hyperfiltration as a potential mechanism (9). Previous studies have linked albuminuria to obesity (23,24). Our study contributes to this body of knowledge by investigating haptoglobinuria in non-diabetic obese individuals for the first time.

Study Limitations

Small sample size and female-dominant population may limit generalizability. Haptoglobin genotypes, which influence clearance and expression, were not assessed. As well as potential confounding factors, such as smoking and medication use.

CONCLUSION

In conclusion, while urinary did not differ between diabetic and non-diabetic obese individuals, its correlation with ACR in diabetic patients warrants further investigation. Prospective studies that consider haptoglobin genotypes are necessary to elucidate the role of haptoglobinuria in obesity among individuals with and without diabetes.

ETHICS

Ethics Committee Approval: The University of Health Sciences Türkiye, Bağcılar Training and Research Hospital Non-Interventional Clinical Research Ethics Committee (approval number: 2016-554, date: 27.12.2016) approved the study.

Informed Consent: All participants gave written informed consent.

FOOTNOTES

Authorship Contributions

Surgical and Medical Practices: M.G.A., A.Y., A.E.A., S.H.K., N.G., Concept: M.G.A., A.Y., A.E.A., S.H.K., N.G., Design: M.G.A., A.Y., A.E.A., S.H.K., N.G., Data Collection or Processing: M.G.A., A.Y., A.E.A., S.H.K., N.G., Analysis or Interpretation: M.G.A., A.Y., A.E.A., S.H.K., N.G., Literature Search: M.G.A., A.Y., A.E.A., S.H.K., N.G., Writing: M.G.A., A.Y., A.E.A., S.H.K., N.G.

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