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Original Article

Effect of *Helicobacter Pylori* Infection on Microalbuminuria and Renal Function Tests of Diabetic Patients with Endoscopy due to Dyspepsia

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ABSTRACT

Objective: In this study, we aimed to demonstrate the contribution of *Helicobacter pylori* (Hp) to microalbuminuria, which is an indicator of diabetic nephropathy and one of the chronic complications of diabetes, and to show that Hp eradication can be a therapeutic option in diabetic patients.

Methods: This study is a retrospective study conducted with type 2 diabetic patients who were followed up in the Gastroenterology and Diabetes Outpatient Clinics. The presence of Hp in pathology findings was investigated in patients who underwent gastroscopy for dyspepsia, and the relationship between microalbuminuria, laboratory findings, demographic characteristics, and clinical information was analyzed.

Results: A total of 156 cases meeting the inclusion criteria, 75 males (48.1%) and 81 females (51.9%) between the ages of 35 and 85 years with a mean age of 61.12±9.78 years, were included in the study. Hp was positive in 71 (45.5%) cases. Hp positivity and accompanying diseases, demographic characteristics, drug therapies used, biochemical parameters, albuminuria levels (micro–normo–macro), endoscopy, and pathology results were compared. The rate of Hp positivity in females was found to be statistically significantly higher than that in males ($p=0.048$, $p<0.05$). The effect of using acetyl salicylic acid (ASA) on Hp increased the Odds ratio by 2.105 (95% CI: 1.05–4.23) times. No significant findings were found during the evaluation of other variables ($p>0.05$).

Conclusion: There was no significant relationship between Hp positivity and microalbuminuria. Female gender and ASA use were identified to be risk factors for Hp infection. Studies with larger patient populations and requiring longer follow-ups are recommended.

INTRODUCTION

Diabetes mellitus (DM) is a disease which if not treated will result in high morbidity and mortality. Its incidence has been increasing in both Türkiye and around the world. Several factors were identified in the etiopathogenesis; however, it is basically due to insulin inadequacy or ineffectiveness.[1]

The aim of diabetes treatment is to prevent the development of toxic effects linked to the effect of chronic hyperglycemia and reduce morbidity and mortality. Complications of diabetes may be divided into two: acute and chronic.

Diabetic nephropathy is a chronic complication and plays an important role in the development of chronic kidney disease (CKD) and cardiovascular disease. Diabetic nephropathy comprises five stages, and the most important of these stages is the microalbuminuria stage. The presence of microalbuminuria indicates vascular and endothelial dysfunction and is the earliest clinical marker of nephropathy.[2] Protective treatments after early detection of microalbuminuria may prevent or delay the development of end-stage renal disease. For this reason, the target of treatment is to remove factors triggering microalbuminuria and reduce albuminuria.

Helicobacter pylori (Hp) is one of the most common infections in the world and has a role in the pathogenesis of many diseases.[3] For this reason, eradication indications may vary. The release of proinflammatory cytokine forms vascular endothelial injury, which may contribute to the development of microalbuminuria and may increase the severity of microalbuminuria in diabetics. Studies targeting

this effect in the recent period have brought the idea that ensuring the eradication of Hp may prevent the development of diabetic nephropathy to the agenda.

In our study, the target was to research the effect of the presence of Hp on microalbuminuria and other kidney function tests in diabetic patients.

MATERIALS AND METHODS

Study population

The study group included patients attending Gastroenterology and Diabetic Clinic from January 2019 to January 2020. Inclusion criteria were patients over 18 years of age, with a diagnosis of diabetes, and with gastroscopy performed. Exclusion criteria were patients with chronic renal failure undergoing hemodialysis, patients with malignancy related to Hp, patients with nephropathy developing linked to nondiabetic causes, pregnant patients, patients under 18 years of age, and patients with Hp eradication.

Laboratory findings

Hemoglobin, fasting blood sugar (FBS), HbA1c, lipid profile, microalbumin/creatinine ratio, and other biochemical analyses were performed in laboratories using standard kits and protocols. The gastroscopy procedure was performed by a single gastroenterologist with a Fujinon endoscopy device. The presence of Hp was assessed with endoscopic biopsy and pathological assessment was performed using Sydney protocol.

Statistical analysis

Statistical analyses used the Number Cruncher Statistical System (NCSS) program. When evaluating the study data, descriptive statistical methods (mean, standard deviation, median, frequency, percentage, minimum, and maximum) were used. Quantitative data were tested for normal distribution with the Shapiro–Wilk test and graphical investigations. Two-way comparisons of quantitative variables with normal distribution used Student's t-test, whereas two-way comparisons of quantitative variables without normal distribution used the Mann–Whitney U test. Comparisons of qualitative data used Pearson's Chi-squared and Fisher's exact tests. Logistic regression analysis was used to determine the effects on micro- and macroalbuminuria. Statistical significance was accepted as $p<0.05$.

RESULTS

The study was performed with a total of 156 cases, 48.1% male (n=75) and 51.9% female (n=81) from January 2019 to January 2020. The age of cases varied from 35 to 85 years, with a mean age of 61.12±9.78 years. Cases participating in the study had body mass index (BMI) measurements of 22–42 kg/m^2 with a mean value of 28.87±3.20 kg/m^2. Of cases, 73.1% had hypertension (n=114), 55.8% had hyperlipidemia (n=87), 25.6% had coronary artery disease (CAD) (n=40), 9% had chronic obstructive pulmonary disease/asthma (n=14), 12.8% had hypothyroidism (n=20), and 8.3% had CKD (n=13) (Table 1).

It was identified that 45.5% of participants in the study (n=71) were Hp positive and 54.5% were Hp negative (n=85). The incidence of Hp positivity among women was statistically significantly high compared with male cases ($p=0.048$, $p<0.05$). According to Hp positivity, there were no statistically significant differences identified based on age, BMI, medication use related to diabetes, and comorbid diseases ($p>0.05$) (Table 2).

There were no statistically significant differences for FBS, HbA1c, albuminuria, total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), non-HDL, hemoglobin, parathyroid hormone (PTH), phosphorus, urea, creatinine, glomerular filtration rate (GFR), and microalbumin in urine measurements according to Hp positivity of cases ($p>0.05$) (Table 3).

When the effects of age, gender, FBS, HbA1c, PTH, and acetyl salicylic acid (ASA) use on Hp were evaluated with logistic regression analysis, the model appeared to have a good explanatory coefficient (61.9%). The effect of being a woman increased the odds ratio for Hp by 2.090 (95% CI: 1.04–4.19) times. ASA use again had the effect of increasing the odds ratio for Hp by 2.105 (95% CI: 1.05–4.23) times (Table 4).

The explanatory coefficient for the logistic regression analysis for the effects of age, gender, FBS, HbA1c, HT, and Hp on macroalbuminuria appeared to be at a good level (78.8%). The effect of HT on macroalbuminuria increased the odds ratio by 4.387 (95% CI: 1.01–21.2) times. A one-unit increase in FBS measurement was observed to affect macroalbuminuria by increasing the odds ratio by 1.012 (95% CI: 1.00–1021) times (Table 5).

Table 1. Distribution of demographic characteristics

Age	Min–max (median)	35–85 (62)
	Mean±SD	61.12±9.78
Gender	Male	75 (48.1)
	Female	81 (51.9)
BMI (kg/m^2)	Min–max (median)	22–42 (29)
	Mean±SD	28.87±3.20
Comorbid diseases	Hypertension	114 (73.1)
	Hyperlipidemia	87 (55.8)
	CAD	40 (25.6)
	COPD/asthma	14 (9.0)
	Hypothyroidism	20 (12.8)
	CRD	13 (8.3)
Albuminuria	Microalbuminuria	57 (36.5)
	Normoalbuminuria	76 (48.7)
	Macroalbuminuria	23 (14.7)

More than one comorbid disease was observed. BMI: Body mass index; CAD: Coronary artery disease; COPD: Chronic obstructive pulmonary disease; CRD: Chronic respiratory disease; SD: Standard deviation.

Table 2. Assessment of demographic characteristics according to the presence of HP

	Hp		p
	Negative	Positive	
Age			
Min–max (median)	35–85 (62)	44–83 (62)	0.645[a]
Mean±SD	61.45±10.67	60.72±8.67	
Gender			
Male	47 (62.7)	28 (37.3)	0.048[b,*]
Female	38 (46.9)	43 (53.1)	
BMI (kg/m^2)			
Min–max (median)	22–40 (29)	23–42 (29)	0.208[a]
Mean±SD	28.58±3.16	29.23±3.23	
Hypertension			
No	24 (57.1)	18 (42.9)	0.686[b]
Yes	61 (53.5)	53 (46.5)	
Hyperlipidemia			
No	37 (53.6)	32 (46.4)	0.847[b]
Yes	48 (55.2)	39 (44.8)	
CAD			
No	63 (54.3)	53 (45.7)	0.940[a]
Yes	22 (55.0)	18 (45.0)	
COPD/asthma			
No	76 (53.5)	66 (46.5)	0.440[b]
Yes	9 (64.3)	5 (35.7)	
Hypothyroidism			
No	74 (54.4)	62 (45.6)	0.961[b]
Yes	11 (55.0)	9 (45.0)	
CRD			
No	80 (55.9)	63 (44.1)	0.226[b]
Yes	5 (38.5)	8 (61.5)	

*P<0.05. [a]Student's t test. [b]Pearson's Chi-squared test. Hp: Helicobacter pylori; BMI: Body mass index; CAD: Coronary artery disease; COPD: Chronic obstructive pulmonary disease; CRD: Chronic respiratory disease; SD: Standard deviation.

Table 3. Assessment of biochemistry results according to HP positivity

	Hp		p
	Negative	Positive	
FBS (mg/dL)			
Min–max (median)	51–322 (137)	74–282 (136)	0.096[a]
Mean±SD	156.52±57.01	142.8±45.13	
HbA1c (%)			
Min–max (median)	5.5–19 (7.9)%	5.4–19 (7.3)	0.063[b]
Mean±SD	8.18±2.04 %	7.81±2.07	
Total cholesterol (mg/dL)			
Min–max (median)	89–285 (173)	81–337 (165)	0.481[a]
Mean±SD	179.39±41.89	174.45±45.24	
Triglyceride (mg/dL)			
Min–max (median)	52–390 (135)	47–400 (143)	0.884[b]
Mean±SD	149.74±69.7	153.01±76.38	
HDL (mg/dL)			
Min–max (median)	12–81 (43)	18–82 (41)	0.960[a]
Mean±SD	43.75±11.64	43.65±13.18	
LDL (mg/dL)			
Min–max (median)	45–198 (101)	30–226 (97)	0.491[a]
Mean±SD	105.73±34.14	101.68±39.23	
Non-HDL (mg/dL)			
Min–max (median)	67–227 (132)	52–300 (130)	0.460[a]
Mean±SD	135.64±37.71	130.8±43.82	
Hgb (g/dL)			
Min–max (median)	8–16.6 (12.7)	8–15.6 (12.3)	0.442[a]
Mean±SD	12.48±1.8	12.26±1.57	
PTH (ng/L)			
Min–max (median)	1.2–148 (35.2)	7.5–180 (44)	0.071[b]
Mean±SD	42.04±25.19	49.25±31.24	
Phosphorus (mg/dL)			
Min–max (median)	1.9–5.4 (3.7)	2.4–4.9 (3.7)	0.854[a]
Mean±SD	3.63±0.58	3.64±0.53	
Urea (mg/dL)			
Min–max (median)	16–124 (33)	18–151 (34)	0.647[b]
Mean±SD	38.81±20.11	41.56±24.79	
Creatinine (mg/dL)			
Min–max (median)	0.4–2 (0.8)	0.4–3.1 (0.8)	0.829[b]
Mean±SD	0.85±0.33	0.98±0.6	
GFR (mL/min/1.73 m^2)			
Min–max (median)	20–149 (89)	19–114 (89)	0.267[a]
Mean±SD	82.79±24.55	78.17±27.17	
MAU (mg/g)			
Min–max (median)	2.6–2529 (39.8)	1.3–11 912 (25.1)	0.870[b]
Mean±SD	163.92±350.06	470.7±1724.94	
Albuminuria			
Microalbuminuria	35 (61.4)	22 (38.6)	0.403[c]
Normoalbuminuria	39 (51.3)	37 (48.7)	
Macroalbuminuria	11 (47.8)	12 (52.2)	

[a]Student's t test. [b]Mann–Whitney U test. [c]Pearson's Chi-squared test. Hp: Helicobacter pylori; FBS: Fasting blood sugar; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; Hgb: Hemoglobin; PTH: Parathyroid hormone; GFR: Glomerular filtration rate; MAU: Microalbumin in urine.

DISCUSSION

Hp is one of the most frequently seen infections around the world and is associated with the pathogenesis of many gastrointestinal system (GIS) or non-GIS diseases.[3] Studies showed a correlation between Hp with the release of proinflammatory cytokines.[4,5] It was stated that atherosclerosis and microalbuminuria may be caused as a result of this inflammation and vascular endothelial injury.[6] Hp infection was proposed to be associated with DM from many aspects and was explained by mechanisms such as the disrupted immune response and susceptibility to infection as a result of high blood glucose levels in diabetes.[7]

In type 2 DM, the target is to prevent or delay chronic complications. One of the chronic complications of diabetic nephropathy may be stopped in the microalbuminuria stage, which is a reversible stage. In the literature, there are studies showing that Hp infection increases microalbuminuria.[6,8–10] In line with these studies, eliminating

Table 4. Logistic regression results for factors affecting HP

	p	Odds	95% CI Odds	
			Lower	Upper
Age	0.394	0.984	0.949	1.021
Gender (F)	0.038*	2.090	1.042	4.191
FBS	0.059	0.993	0.986	1.000
HbA1c	0.819	0.979	0.819	1.171
PTH	0.195	1.008	0.996	1.021
ASA (yes)	0.037*	2.105	1.048	4.230

*P<0.05. CI: Confidence interval; FBS: Fasting blood sugar; PTH: Parathyroid hormone; ASA: Acetyl salicylic acid.

Table 5. Logistic regression results for factors affecting macroalbuminuria

	p	Odds	95% CI Odds	
			Lower	Upper
Age	0.585	1.016	0.959	1.076
Gender (F)	0.659	1.265	0.446	3.582
HbA1c	0.435	0.873	0.621	1.228
Hp (+)	0.759	1.177	0.415	3.337
HT (+)	0.046*	4.387	1.006	21.235
FBS	0.016*	1.012	1.002	1.021

*P<0.05. Hp: *Helicobacter pylori*; FBS: Fasting blood sugar; HT: Hypertension.

Hp infection is considered to reduce microalbuminuria in diabetic patients.

A meta-analysis study by Shi et al.[9] reported that patients with type 2 DM diagnosis were more susceptible to Hp, and as a result, Hp triggered the occurrence of proteinuria in diabetic patients. In T2DM patients, proteinuria shows glomerular filtration barrier injury along with vascular endothelial dysfunction. The development of proteinuria in type 2 DM is multifactorial and is an important marker for diabetic nephropathy diagnosis. As a result of Hp eradication in patients with membranous nephropathy, proteinuria was observed to reduce, and it was stated that eradication in the early period may be beneficial for treatment in diabetic nephropathy patients. In our study, no significant correlation was identified between microalbuminuria and Hp infection. This situation may be linked to the patients receiving renin–angiotensin aldosterone system blockers and SGLT-2 group medications effective on proteinuria as antidiabetic treatment in the early period. At the same time, this may be due to the insufficient patient numbers in the study and the epidemiological distribution of Hp.

A study by Chung et al.[8] concluded that Hp positivity has a positive correlation with the intensity of albumin/creatinine ratio in urine among diabetic individuals. Again, the same study found metabolic parameters such as glucose level, triglyceride level, and BMI associated with albuminuria. In our study, there was no significant difference in BMI and triglyceride level with a significant correlation found between high FBS and macroalbuminuria.

Hypertension, dyslipidemia, diabetic retinopathy, atherosclerosis, and cardiovascular complications are known to affect diabetic nephropathy. In the literature, there are studies showing that Hp infection is effective on atherosclerosis and lipid metabolism. Western individuals with Hp infection were shown to have high serum cholesterol and triglyceride levels. Adachi et al.[11] compared serum lipid levels before and after Hp eradication in their study and identified that the group with Hp eradication had higher HDL, lower LDL, total cholesterol, and triglyceride levels compared with those before eradication, different from the group with chronic infection. This was considered to be linked to inflammation in the stomach mucosa regressing after successful Hp eradication and reduced release of proinflammatory cytokines linked to this. Differently in our study, no significant correlation was identified between Hp and serum lipid levels. This difference may be linked to the study population receiving lipid-lowering treatment in the early period due to regular checkups.

There are studies showing that Hp is associated with insulin resistance, which is included in type 2 diabetes pathogenesis.[12,13] A study by Gunji et al.[12] showed there was a positive correlation between Hp infection and metabolic syndrome in the Japanese population, while another study showed that Hp infection in asymptomatic individuals increased insulin resistance. It is not fully known how Hp infection affects insulin resistance pathogenesis, but it is thought that it may be caused by reactive oxygen radicals and proinflammatory and vasoactive agents such as TNF-alpha, IL-6, and CRP linked to Hp infection.[8]

A few studies found a positive correlation between Hp with glycemic control in diabetic patients.[14,15] A study by Chen et al.[15] reported that there was a positive correlation between Hp seropositivity and HbA1c levels. Ibrahim et al.[7] stated diabetic patients infected with Hp positive strains for cytotoxin-associated gene A antigen led to weak glycemic control. Contrary to this, a meta-analysis by Horikawa et al.[16] stated that the group of Hp carriers was not identified to have significantly high levels of HbA1c. Whether eradication of Hp infection in diabetic patients will improve glycemic control or not is still controversial. In our study, there were no significant correlations identified between Hp infection with FBS and HbA1c levels. This situation may be linked to our patients being administered intensive antihyperglycemic treatment or control of nutrition and weight.

According to the Maastricht V/Florence consensus report, Hp treatment indications include patients with peptic ulcers with planned long-term ASA use and patients with GIS hemorrhage during ASA use. ASA use is thought to increase the risk of GIS hemorrhage after peptic ulcer development.[17,18] In our study, ASA use was identified as a risk factor for Hp infection. A study by Gajewski et al.[19] mentioned that ASA and Hp were associated with peptic ulcer pathogenesis and that ASA had dose-linked

cytotoxic effects. Changes induced in stomach epithelium by Hp may be intensified by ASA and simultaneously Hp may penetrate stomach epithelium to deeper levels with the effect of ASA. They mentioned that this may trigger a systemic inflammatory response. Eradication of bacteria is considered to reduce the risk of upper gastrointestinal complications in patients treated with ASA.

A study by Mossi et al.[20] stated that Hp prevalence increased with age, and there was no significant difference between male and female genders. On the contrary, in our study, there was no significant correlation between Hp infection and age; however, the female gender was identified to be a risk factor for Hp infection. This situation may be linked to the different epidemiological distribution of Hp and the effects of environmental factors.

In our study, we investigated the relationship between Hp and microalbuminuria in type 2 diabetic patients. There were no significant correlations found between Hp with microalbuminuria and the renal function markers of creatinine, parathormone, phosphorus, and GFR. While no parameter was identified to be effective on microalbuminuria, we found a positive correlation between the presence of hypertension and FBS levels with macroalbuminuria. We identified that female gender and ASA use were risk factors for Hp infection in our study.

Limitations of our study include that it was performed in a single center, with retrospective data and a low number of patients, along with the possibility that medications affecting microalbuminuria may have affected the results. Strong aspects of the study are that the presence of Hp was shown endoscopically and pathologically to prevent false positives and that cases comprised patients attending regular checkups.

CONCLUSION

In this study, we researched whether chronic infection with Hp triggered inflammation and caused vascular endothelial injury and the effect of this situation on microalbuminuria in diabetic patients. In conclusion, we did not find a relationship between microalbuminuria and Hp infection. Considering the strong and weak aspects of this study, there is a need for longer-duration multicenter studies with higher participant numbers.

Ethics Committee Approval

This study approved by the Kartal Dr. Lütfi Kırdar City Hospital Clinical Research Ethics Committee (Date: 21.10.2020, Decision No: 2020/514/188/6).

Informed Consent

Retrospective study.

Peer-review

Internally peer-reviewed.

Authorship Contributions

Concept: T.T.; Design: B.B.; Supervision: T.T.; Fundings: E.K.; Materials: T.T.; Data: S.A.; Analysis: Ö.K.; Literature search: B.B.; Writing: T.T.; Critical revision: S.A.

Conflict of Interest

None declared.

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Dispepsi Nedeniyle Endoskopi Yapılan Diyabetik Hastalarda *Helicobacter Pylori* Enfeksiyonunun Mikroalbuminüri ve Böbrek Fonksiyon Testleri Üzerine Etkisi

Amaç: Bu çalışmada, diyabetin kronik komplikasyonlarından diyabetik nefropatinin göstergesi olan mikroalbüminüri üzerine *Helicobacter pylori*'nin (Hp) katkısının gösterilmesini ve Hp eradikasyonun diyabetik hastalarda teröpatik bir seçenek olabileceğini göstermeyi amaçladık.

Gereç ve Yöntem: Bu çalışma, geriye dönüktür. Gastroenteroloji ve diyabet polikliniğinde takipli olan tip 2 diyabetik hastalar üzerinde yapıldı. Dispepsi nedeniyle gastroskopi yapılan hastalarda patoloji bulguları eşliğinde Hp varlığı karşılaştırıldı ve mikroalbüminüri, laboratuvar bulguları, demografik özellikleri ve klinik bilgileri arasındaki ilişki analiz edildi.

Bulgular: Çalışmaya, yaşları 35 ile 85 arasında, yaş ortalaması 61.12±9.78 yıl olan 75'i erkek (%48.1), 81'i kadın (%51.9) olmak üzere dışlama kriterlerini karşılayan toplam 156 olgu alındı. Olguların 71'inde (%45.5) Hp pozitif olarak saptandı. Hp varlığı ile eşlik eden hastalıklar, demografik özellikler, kullanılan ilaç tedavileri, biyokimyasal parametreler, albüminüri düzeyleri (mikro-normo-makro), endoskopi ve patoloji sonuçları karşılaştırıldı. Kadınlarda Hp pozitifliği görülme oranı, erkek olgulara göre istatistiksel olarak anlamlı düzeyde yüksek saptandı (p=0.048; p<0.05). Asetilsalisilikasit (ASA) kullanımının Hp üzerine etkisinin odds oranını 2.105 (%95 CI: 1.05–4.23) kat arttırdığı görüldü. Diğer değişkenlerin değerlendirilmesinde anlamlı bulgular saptanmadı (p>0.05).

Sonuç: Hp pozitifliği ile mikroalbüminüri arasında anlamlı bir ilişki görülmedi. Kadın cinsiyet ve ASA kullanımı, Hp enfeksiyonu için bağımsız risk faktörü olarak saptandı. Bu konuda daha geniş popülasyonda yapılacak daha uzun süreli yeni araştırmalara ihtiyaç olduğunu düşünüyoruz.

Anahtar Sözcükler: Diyabetik nefropati; *Helicobacter pylori*; mikroalbüminüri.