Original Research

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Beta-2 microglobulin levels and *Helicobacter pylori* in patients with portal gastropathy

Portal gastropatili hastalarda beta-2 mikroglobulin ve *Helikobakter pilori* ilişkisi

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Background/aims: *The relationship between Helicobacter pylori and beta-2 microglobulin in patients with portal gastropathy has not been clearly defined yet. In this study, we aimed to compare the levels of serum and tissue beta-2 microglobulin in patients with and without Helicobacter pylori infection and to examine the relationship between levels of serum and tissue beta-2 microglobulin in patients with portal gastropathy.* ***Methods:*** *Twenty-five patients with portal gastropathy and 22 healthy persons were enrolled in this study. Gastric biopsies were histologically analyzed and tissue and serum beta-2 microglobulin levels were measured.* ***Results:*** *Helicobacter pylori infection was detected in 15 of 25 portal gastropathy patients. Subendothelial beta-2 microglobulin was detected in 8 of 15 (53.3%) portal hypertensive gastropathy patients with Helicobacter pylori. Subendothelial beta-2 microglobulin was detected in 3 of 10 (30%) portal hypertensive gastropathy patients without Helicobacter pylori. The difference between groups was statistically significant (p<0.01). Five of the patients in the portal gastropathy group had moderate disease severity, and all 5 had Helicobacter pylori infection. However, all of the patients without Helicobacter pylori had mild portal gastropathy (p<0.001).* ***Conclusions:*** *There was no change in Helicobacter pylori infection frequency in portal gastropathy. Beta-2 microglobulin deposition in the gastric mucosa was more common in Helicobacter pylori-positive patients. There is an inverse relationship between portal gastropathy severity and beta-2 microglobulin deposition.*

Key words: *Portal gastropathy, Helicobacter pylori, beta-2 microglobulin*

Amaç: *Portal gastropatili hastalarda Helikobakter pilori ve beta-2 mikroglobulin ilişkisi net olarak tanımlanamamıştır. Bu çalışmada, Helikobakter pilori infeksiyonu olan ve olmayan hastaların serum ve doku beta-2 mikroglobulin düzeyleri ve bu düzeylerin portal gastropati ile ilişkisi incelenmiştir.* ***Gereç ve Yöntem:*** *Portal gastropatili 25 hasta ve 22 sağlıklı birey çalışmaya dahil edilmiştir. Mide biyopsileri incelenmiş, serum ve doku beta-2 mikroglobulin düzeyleri bakılmıştır.* ***Bulgular:*** *Yirmibeş portal hipertansif gastropatili olgunun 15'inde Helikobakter pilori infeksiyonu bulunmuştur. Bu 15 hastanın 8'inde (%53.3) subendotelial beta-2 mikroglobulin artışı görülmüştür. Helikobakter pilori olmayan 10 hastanın 3'ünde (%30) subendotelial beta-2 mikroglobulin bulunmuştur. Fark istatistiksel olarak anlamlıdır (p<0.01). Portal gastropatisi olan hastaların 5'inde gastropati orta derecede idi ve bu 5 olgunun hepsi de Helikobakter pilori pozitifti. Buna karşılık Helikobakter pilori olmayan hastaların hepsinde gastropati derecesi hafif derecede idi (p<0.001)* ***Sonuç:*** *Portal gastropatide Helikobakter pilori sıklığı değişmemektedir. Helikobakter pilori pozitif hastalarda gastrik mukozada beta-2 mikroglobulin depolanması daha yagındır.*

Anahtar kelimeler: *Portal gastropati, Helikobakter pilori, beta-2 mikroglobulin*

INTRODUCTION

Beta-2 microglobulin (β-2m) is a minor plasma protein, secreted from the plasma membranes as a result of the continuous regeneration of membrane proteins in the cell surface of all nucleated cells (1). The relationship between *Helicobacter pylori (H. pylori)* and β-2m in patients with portal gastropathy has not been clearly defined yet. In this study, we aimed to compare the levels of serum and tissue β-2m in patients with and without *H. pylori* infection and to examine the relationship between levels of serum and tissue β-2m in patients with portal gastropathy.

MATERIALS AND METHODS

Twenty-five patients with portal gastropathy and 22 healthy persons were enrolled in this study. Gastric biopsies were histologically analyzed, and tissue and serum β-2m levels were measured. Urease test of gastric biopsy tissue samples was performed.

RESULTS

The demographic features and etiologic distribution are shown in Tables 1 and 2, respectively. *H. pylori* infection was detected in 15 of 25 portal gastropathy patients. Subendothelial β-2m was detected in 8 of 15 (53.3%) portal hypertensive gastropathy patients with *H. pylori*. Table 3 shows the distribution of β-2m levels of portal gastropathy patients with respect to the patients' features. Tissue β-2m was detected in 3 of 10 (30%) portal hypertensive gastropathy patients without *H. pylori*. The difference between groups was statistically significant (p<0.01). Five of 13 healthy control cases with *H. pylori* but none of the healthy control cases without *H. pylori* were shown to have tissue β-2m (p<0.001). Five of the patients in the patient group had moderate severity of portal gastropathy, and all 5 had *H. pylori* infection. However, all of the patients without *H. pylori* had mild portal gastropathy (p<0.001).

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Table 1. Demographic features of study cases

	Patients (n: 25)	Healthy controls (n: 22)
Age (mean±SD)	49.4±11.9	40.5±14.1
Gender		
Male	19 (76%)	18 (82%)
Female	6 (24%)	4 (18%)

Table 2. Etiologic distribution of portal gastropathy

Etiology	N (%)	Male/Female
Hepatitis B	14 (56)	11/3
Hepatitis C	2 (8)	1/1
Hepatitis B+C	1 (4)	1/0
Alcoholic	3 (12)	3/0
Cryptogenic	4 (16)	1/3
Delta	1 (4)	1/0

DISCUSSION

Chronic liver disease was previously reported to be among the reasons for peptic ulcer disease (2). *H. pylori* is a very well-known etiologic agent of gastritis and peptic ulcer. On the other hand, the *H. pylori* and portal gastropathy relationship is still debatable. Foster et al.(3) showed a relatively low prevalence (17%) of *H. pylori* in patients with portal gastropathy and explained it by the unsuitable milieu of the gastric mucosa in portal gastropathy. However, in our study, 15 of 25 (60%) of our patients and 13 of 22 (59.1%) of the healthy controls had *H. pylori* infection. We attribute this to the high *H. pylori* prevalence in our country.

Portal gastropathy severity and the *H. pylori* relationship is unclear in the literature (4-6) (Table 4). *H. pylori* was shown to increase in mild portal gastropathy, but to decrease with severity of portal gastropathy. This was attributed to mucosal atrophy and mucus layer changes in severe portal gastropathy. Five of the study patients (20%) had moderate portal gastropathy, and all 5 patients had *H. pylori* infection.

Hematoxylin-eosin (HE) and Warthin-Starry staining might not disclose *H. pylori* in some specimens. However, foveolar lymphoid follicular presentation in these samples with *H. pylori* serology is compatible with *H. pylori* infection. This could explain urease-positive patients whose biopsy samples did not disclose *H. pylori*.

β-2m is a minor plasma protein, secreted from the plasma membranes as a result of the continuous regeneration of

Table 3. Distribution of gender, age and serum and tissue ß-2m levels in portal gastropathy patients

Patient	Gender	Age	Urease	*H. pylori* in biopsy	Congestion in biopsy	Serum β-2m (mg/dl)	Tissue β-2m
MG	M	61	+	+	Mild	0.33	+
MSC	M	47	+	+	Mild	0.27	+
YG	M	61	+	+	Moderate	0.21	-
YD	M	42	+	+	Mild	0.72	-
BI	M	63	+	+	Moderate	0.33	+
MK	M	57	+	+	Mild	0.23	-
BH	M	38	+	+	Moderate	0.17	-
MT	M	26	+	-	Mild	0.19	+
FO	M	49	+	-	Moderate	0.22	-
KT	F	61	+	-	Mild	0.10	+
YK	F	61	-	+	Mild	0.19	+
EB	F	38	-	+	Mild	0.15	-
KI	M	60	-	+	Mild	0.10	+
RT	M	45	-	+	Mild	0.30	+
HC	M	61	-	+	Moderate	0.33	-
MY	M	37	-	-	Mild	0.29	+
CA	F	59	-	-	Mild	0.22	+
SY	F	55	-	-	Mild	0.15	-
AY	M	64	-	-	Mild	0.16	+
MO	M	62	-	-	Mild	0.30	-
OE	M	40	-	-	Mild	0.22	-
YS	M	47	-	-	Mild	0.23	-
MC	M	36	-	-	Mild	0.14	-
SD	F	30	-	-	Mild	0.19	-
AP	M	34	-	-	Mild	0.21	-

M: Male F: Female

Table 4. *H. pylori* frequency and portal gastropathy severity relationship

	H. pylori frequency			
	None	**Mild**	**Moderate**	**Severe**
D'Amico (4)	50%	43%	-	28%
McCormick (5)	38%	50%	20%	22%
Ozdemir (6)	87% (none+mild)		-	45%

membrane proteins in the cell surface of all nucleated cells. Conz et al.(1) showed β-2m accumulation in *H. pylori*-positive gastric mucosa but not in *H. pylori*-negative gastric mucosa biopsies in uremic study patients with high serum β-2m. They also showed β-2m accumulation in *H. pylori*-positive gastric biopsy samples in healthy controls. We found significantly higher β-2m concentrations in our patient group (44%) than in healthy controls (22.7%) ($p<0.01$). On the other hand, we did not find a relationship between β-2m and severity of portal gastropathy ($p>0.05$).

In conclusion, there is no change in *H. pylori* infection frequency in portal gastropathy. β-2m deposition in gastric mucosa is more common in *H. pylori*-positive patients. There is an inverse relationship between portal gastropathy severity and β-2m deposition.

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