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Relationship of Gastric Metaplasia and Age, Sex, Smoking and Helicobacter pylori Infection in Patients with Duodenal Ulcer and Duodenitis

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A B S T R A C T

Gastric metaplasia is one of the factors in duodenal ulcer appearance. The aim of this study was to investigate the frequency of gastric metaplasia and its connection with age, sex, cigarette smoking and H. pylori infection. In the study 216 patients were included. There were 98 patients with duodenal ulcer, 60 with duodenitis, and 58 healthy control subjects. There was no statistically significant difference in gastric metaplasia frequency according to age and sex. Gastric metaplasia was statistically more significant in patients with duodenal ulcer ($p < 0.01$). In all the subjects cigarette smoking did not significantly influence gastric metaplasia. In smokers with duodenal ulcer, and those who besides duodenal ulcer and smoking had H. pylori infection gastric metaplasia was more frequent ($p < 0.01$). However, in patients with duodenal ulcer, there was no statistically significant difference of gastric metaplasia related to H. pylori presence. It may be suggested that H. pylori infection is not of indispensable significance for gastric metaplasia appearance.

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Introduction

Gastric metaplasia or the appearance of gastric type mucous cells within the duodenal epithelium is a common finding of biopsy specimens from the patients with duodenitis or duodenal ulcer[1,2]. It can be found in healthy individuals, but in lesser extent[3]. An assumption is that the gastric metaplasia is a protective mechanism against persistently high concentrations of gastric acid in the duodenal bulb. Generally, it is assumed that gastric metaplasia arises from the neck of Brunner´s glands as a result of persistently increased concentration of acidity in the duodenal bulb. However, gastric metaplasia can also develop as a non-specific response to mucosal injury unrelated to acid peptic damage[4].

As the colonization of Helicobacter pylori is limited to a gastric type of epithelium[5,6] the presence of gastric metaplasia in duodenum allows H. pylori to colonize the duodenum and to cause active inflammation. Inflammed duodenal mucosa becomes susceptible to acid damage, which leads to ulceration[7]. Prevalence of gastric metaplasia is not influenced by H. pylori[7–9], but its extent is higher in Helicobacter pylori positive individuals[10], assuming that H. pylori may be responsible for spreading of preexisting gastric metaplasia.

The aim of this study was to determine the prevalence of gastric metaplasia and Helicobacter pylori, especially eventual relationship of Helicobacter and gastric metaplasia in the patients with duodenal ulcer and duodenitis.

Materials and Methods

Subjects

Three groups of patients were included in this study: 98 with duodenal ulcer, 60 with duodenitis and 58 controls. Duodenal ulcer and duodenitis were diagnosed by endoscopy of upper gastrointestinal tract in patients with positive anamnesis of dyspeptic complaints, and confirmed histopathologically.

Patients with anamnesis of hemorrhagic diathesis or coagulation defects, malignancy of esophagus, gastric or duodenal malignancy as well as patients who underwent gastric surgery, or those having insulin dependent diabetes mellitus, were excluded from the study. None of the control subjects had any significant medical history or any gastrointestinal symptoms. They had a completely normal endoscopy. No aspirin or other NSAID, antacid or H2 receptor antagonist had been taken at least four weeks before, or during the study. All subjects gave an informed consent to the study that was approved by the Hospital Ethical Committee.

Endoscopy

Endoscopy was performed in the morning after an overnight fast with an Olympus GIF, type Q20 (Olympus Optical Co., Ltd., Tokyo, Japan). Smooth, clear and intact mucosa was considered a normal finding. Duodenal mucosa injury of over 0.5x0.5 cm was considered a duodenal ulcer. The endoscopically visible changes in duodenal mucosa pertinent to the nonspecific chronic duodenitis were classified according to Stephen et al.[11] as: 0 = normal appearance; 1 = mucosal hyperemia; 2 = edematous hyperemic mucosa that bleeds to the touch; and 3 = same as previous, but including »pepper and salt« changes. The positive findings included degrees 1, 2 and 3 of the changes.

Biopsy specimens

The determination of H. pylori presence required taking three biopsy specimens from the antrum of the gastric mucosa, 2 cm from the pylorus by biopsy forceps (Olympus FB 24-K). One specimen was intended for a quick urease test

(CLO-test), and two for culture using standard methods. H. pylori was indentified by morphological and biochemical tests for oxidase, catalase and urease activity (Merck, Darmstadt, Germany). The presence of H. pylori in the antral mucosa was confirmed only if both tests were positive. Four specimens from the duodenal mucosa were used to determine the degree of duodenitis by somewhat modified method of Witehead et al.[12]. The duodenal histological findings were reported as follows: degree 1: normal appearance of the surface epithelium, mild edema, cellular infiltrate, predominantly into the cellular plasma; degree 2: generally normal occurrence of the surface epithelium, randomly distributed and thick inflammatory infiltrate of the lamina propria spreading deep bellow to Bruner's glands; degree 3: ulceration of the surface epithelium, considerable inflammatory infiltrate of the lamina propria, some crypts and parts of the surface epithelium permeated with neutrophils. The positive findings referred to changes of the degrees 2 and 3. Duodenal biopsy specimens were stained with periodic acid Schiff (PAS) for the detection of metaplastic gastric epithelium. The presence and degree of duodenal inflammation were graded according to Wyatt´s classification.

Statistical analysis

The results are expressed as mean SD. Statistical evaluation of the data was based on Student's t-test for unpaired data and Schee's test for the analysis of variance to determine respective differences between smokers and nonsmokers, and between H. pylori positive and negative patients. Differences associated with probability values of $p < 0.05$ were considered statistically significant.

Results

In the group of 216 patients (132 men and 84 women) aged 19 to 65 years (mean age 45.42 years) there were 98 patients with duodenal ulcer, 60 with duodenitis and 58 healthy control subjects. Helicobacter pylori infection was confirmed in 136 out of 216 patients (63%). Gastric metaplasia was present in 120 patients (56%). In 136 Helicobacter positive patients gastric metaplasia was present in 100 patients (74%). In the patients with duodenal ulcer, the incidence of Helicobacter pylori infection was significantly higher (86%) in relation to the patients with duodenitis (60%) and healthy subjects (28%) (Table 1).

Presence of gastric metaplasia was significantly higher in the patients with duodenal ulcer (90%) than in the healthy subjects (24%) and the patients with duodenitis (30%), ($p < 0.01$), (Table 2).

Prevalence of concomitant occurrence of GM and HP was significantly higher in the group of patients with duodenal ulcer (78%) in relation to other two groups (Table 3), ($p < 0.01$).

H. pylori patients with duodenal ulcer had significantly higher incidence of GM

TABLE 1
PREVALENCE OF H. PYLORI ACCORDING TO GASTRIC METAPLASIA

Group of patients	N	GM present	GM absent	Total
Control	58	10/14 (71.4%)*	6/44 (13.6%)*	16/58 (28%)
Duodenitis	60	14/18 (77.7%)**	22/42 (52.9%)**	36/60 (60%)
Duodenal ulcer	98	76/88 (86.3%)	8/10 (80%)	84/98 (86%)

Student's test: *$p < 0.01$; **$p < 0.05$.

TABLE 2
PREVALENCE OF GASTRIC METAPLASIA ACCORDING TO H. PYLORI

Group of patients	N	H. pylori present	H. pylori absent	Total
Control	60	6/16 (37.5%)	8/42 (19%)	14/58 (24%)
Duodenitis	58	14/36 (39%)*	4/24 (16.6%)	18/60 (30%)
Duodenal ulcer	98	76/84 (90.5%)	12/14 (85.7%)	88/98 (90%)

Student's t-test: *p < 0.01.

TABLE 3
PREVALENCE OF GASTRIC METAPLASIA AND HELICOBACTER PYLORI

Group of patients	N	GM+ HP+	GM+ HP–	GM– HP+	GM–HP–
Control	58	10 (17%)	4	6	38 (65%)**
Duodenitis	60	14 (23%)	4	22 (37%)**	20
Duodenal ulcer	98	76 (78%)*	12	8	2
Total	216	100	20	36	60

HP: Helicobacter pylori; GM: gastric metaplasia

Student's t-test, *p < 0.01 in relation to the control and dudenitis; **p < 0.05 in relation to the control and duodenal ulcer; **p < 0.05 in relation to duodenitis and duodenal ulcer.

TABLE 4
PREVALENCE OF GASTRIC METAPLASIA IN RELATION TO AGE, SEX, AND H. PYLORI

	Prevalence				
Age (y)	HP+ve male	HP+ve female	HP-ve male	HP-ve female	Total
20–39	18/28	4/6	2/14	6/16	30/64 (47%)
40–59	42/52	26/36	4/28	8/20	80/136 (59%)
>60	6/8	4/6	0/2	0	10/16 (63%)
Total	66/88 (75%)	34/48 (71%)	6/44 (14 %)	14/36 (39%)	

HP: Helicobacter pylori

(90%), than H. pylori patients with duodenitis (30%), (Table 2), (p < 0.01).

There were no statistically significant differences in occurrence of gastric metaplasia in men (55%) and women (57%). There were no significant differences in presence of gastric metaplasia in different age groups (Table 4) ($X^2 = 4.25$, p > 0.05). In Helicobacter pylori positive subjects there is no statistically significant difference in occurrence of gastric metaplasia regarding the sex while in Helicobacter negative subjects prevalence of gastric metaplasia was higher in women ($X^2 = 0.14$; p > 0.05), (Table 4).

There is no statistically significant difference in presence of gastric metaplasia among Helicobacter pylori positive and Helicobacter pylori negative patients with duodenal ulcer ($X^2 = 0.17$, p > 0.05), (Table 2). In the patients with duodenal ulcer, gastric metaplasia was present in 76 out of 84 Helicobacter positive patients (90%), while in 14 Helicobacter negative patients with duodenal ulcer gastric metaplasia was present in 12 (86%),

TABLE 5
PREVALENCE OF GASTRIC METAPLASIA IN RELATION TO H. PYLORI AND SMOKING OF CIGARETTES

Smoking	H. pylori present			H. pylori absent			Total
	Duodenal ulcer			Duodenal ulcer			
	Absent	Present	Total	Absent	Present	Total	
Non-smoking	10/20	28/36 (77%)**	38/56 (68%)	2/26	4/6	6/32 (19%)	44/88 (50%)
Smoking							
<15 cig/d	10/22	34/34 (100%)	44/76 (58%)	2/18	2/2	4/20 (20%)	48/96 (50%)
>15 cig/d	6/10	14/14 (100%)	20/24 (83%)*	4/22	6/6	10/28 (36%)	30/52 (57%)

Variance analisis: Schefee's test, **$p < 0.05$ in relation to smoking;
*$p < 0.05$ in relation to smoking >15 cig/d.

TABLE 6
PREVALENCE OF GASTRIC METAPLASIA IN ASYMPTOMATIC SUBJECTS AND DUODENAL ULCER [13]

Author	Prevalence	
	Asymptomatic subjects	Duodenal ulcer
James		90%
Patrick et al.		75%
Kreuning et al.	64%	
Greenlaw et al.		60%
Shousha et al.		39%
Wyatt et al.		65%
Fizgibbons et al.	22%	
Carrick et al.		92%
Tucci et al.	30%	40-90%
Offerhaus et al.		88%
Noach et al.		90%
Bago et al.	28%	90%

(Table 2). In the population without duodenal ulcer there was statistically significant difference between Helicobacter pylori positive and Helicobacter pylori negative subjects and appearance of gastric metaplasia ($X^2 = 12.17$, $p < 0.001$), (Table 2).

There is no statistically significant difference in occurrence of gastric metaplasia in all patients regarding smoking status ($X^2 = 0.048$, DF = 2; $p > 0.05$) as well as in Helicobacter positive patients ($X^2 = 2.72$, DF = 2; $p > 0.05$), (Table 6). Prevalence of gastric metaplasia is higher in smokers with duodenal ulcer than in non-smokers with the same disease. Gastric metaplasia was statistically significantly higher in Helicobacter positive smokers than in Helicobacter negative non-smokers ($p < 0.05$, Table 6).

Discussion

It is known that duodenal ulcer disease is related to the excess secretion of gastric acid[13,14]. But, other factors, including Helicobacter pylori play an important role in the pathogenesis of duodenal ulcer disease[7,15–18]. In our study we found significantly higher incidence of H. pylori as well as gastric metaplasia in the patients with duodenal ulcer than in the healthy subjects (controls), which is in accordance to the literature[19,20].

It appears, however that only the minority of the patients infected with Helicobacter pylori develop duodenal ulcer while the majority of the patients with gastric metaplasia belong in a group with duodenal ulcer[21–25]. In our study we found that the prevalence of gastric metaplasia is significantly higher in the pa-

tients with duodenal ulcer than in the healthy subjects and the patients with duodenitis. There is still an open question whether the appearance of gastric metaplasia is a consequence of high acid secretion[4,7,26,27] (that is proved by the low incidence of gastric metaplasia in hyposecretors of gastric acid, lack of gastric metaplasia in patients with atrophic gastritis and low incidence of gastric metaplasia two years following vagotomy[2,28]) or gastric metaplasia appears primarily rendering the mucosa more susceptible to gastric acid[29].

On the other hand some authors claim that gastric metaplasia presents even a protective factor against gastric acid (since it is more common on the edges of duodenal ulcer than in other areas of duodenum)[30–32].

The results of our study have shown no direct relationship between occurrence of gastric metaplasia and Helicobacter pylori in the patients with duodenal ulcer, which is in concordance with some authors[8,9,33], but renders open a possibility of Helicobacter's indirect (together with other factors) influence on the appearance of gastric metaplasia through inducement the gastric acid hypersecretion. The finding that the prevalence of gastric metaplasia is higher in Helicobacter positive patients suggests that Helicobacter infection could be responsible for spreading of gastric metaplasia[4] by causing hypersecretion of gastric acid, thereby increasing duodenal acidity[34–36]. The results of some studies in which eradication of Helicobacter resulted in regression of inflammation and extent of gastric metaplasia[37] are in the accordance to the previos statement.

In our study Helicobacter pylori appears more commonly in the areas of gastric metaplasia. In the patients with duodenal ulcer and Helicobacter, gastric metaplasia was present in 90%, but gastric metaplasia was present in 86% of the Helicobacter negative patients with duodenal ulcer as well. It seems that Helicobacter pylori and gastric metaplasia are two (independent?) etiological factors in breaking the duodenal mucosas resistance on the way toward the duodenal ulcer disease.

Some authors consider gastric metaplasia as a primary event (protective against lesion caused by gastric acid) which makes a susceptible environment for Helicobacter colonization leading to the inflammation[15,38]. The prevalence of Helicobacter pylori was significantly higher than the prevalence of gastric metaplasia in the patients with duodenitis (36 against 18), which was not case in the patients with duodenal ulcer disease, where the incidence of gastric metaplasia was even somewhat higher than the incidence of Helicobacter (88 against 84 of 98 patients with duodenal ulcer).

The increased prevalence of Helicobacter pylori is evident in the patients with duodenal ulcer group in comparison with groups with duodenitis and healthy subjects. The prevalence of gastric metaplasia is, however, more increased in the patients with duodenal ulcer, than in other two groups. Similar results were found by other authors[19,20,24,39,40]. According to some studies, a capacity of gastric hypersecretion is unchanged following the eradication of Helicobacter pylori, so it can be concluded that gastric metaplasia is a more serious risk factor for the development of duodenal ulcer disease than Helicobacter pylori, or Helicobacter is only »following« gastric metaplasia (colonizing mucosa like »gastric one«) causing by itself no duodenal ulcer disease[21]. We could say that gastric metaplasia is an original event that arises as a protection mechanism to the increased secretion of gastric acid, while the second event is colonization of Helicobacter that causes the inflammation[16,38]. It is supported by the constantly (in all three

groups) high correlation between the appearance of H. pylori and the presence of gastric metaplasia.

Gastric metaplasia is an almost constant finding in the patients with duodenal ulcer[4,14,28,38,40]. A finding that a high percent of healthy subjects is infected with Helicobacter pylori, while the prevalence of gastric metaplasia in healthy subjects is low, provides another piece of evidence for a hypothesis that gastric metaplasia is more important factor in pathogenesis of duodenal ulcer disease. On the other hand, gastric metaplasia in the patients with duodenitis and healthy subjects appeared statistically more often in Helicobacter positive than in Helicobacter negative patients (this was not the case in the patients with duodenal ulcer). One of the reasons why we observed this pattern was a fact that a small group of patients with duodenal ulcer disease and Helicobacter negativity was observed, and the second one was caused by the independence of gastric metaplasia and Helicobacter, but rather its dependance on hyperacidity[41].

In our study we have shown no significant difference in the prevalence of gastric metaplasia according to sex in Helicobacter positive patients. There was also no statistically significant difference in the incidence of Helicobacter in different age groups. Similar results were reported in the literature[14]. We could also conclude that gastric metaplasia is a primary factor (protective against lesions caused by gastric acid) that makes a susceptible environment for Helicobacter colonization which leads to the inflammation[16,38]. Results of our study show significantly higher prevalence of gastric metaplasia in the patients with duodenal ulcer in comparison to those with duodenitis (compatible with the results of other authors[2,22,24,39,40].

The finding of significantly higher prevalence of gastric metaplasia in the patients with duodenitis found in the study of Noach et al.[14] can be explained by the utilised methodology and a place of sample taking. In the patients with duodenitis we took samples from the areas of the most clear changes of the anterior wall of bulb and from the edge of ulcus in the duodenal ulcer patients. It is clear that this could cause such result discrepancies, as mentioned in the study of Marshall et al.[41], where the appearance of gastric metaplasia was higher in samples taken from the edge of duodenal ulcer in comparison with the other areas of ulcer (63%).

According to our results there is a relationship between the occurrence of gastric metaplasia and Helicobacter infection, especially in the patients with duodenal ulcer. The same suggests that gastric metaplasia arises as a response to high output of gastric acid[42] with disturbance of gastroantral segment motility, and/or rapid emptying and consequent colonization of duodenum[21]. It should certainly be that at the same level, there is consequent duodenitis, which in fact is just a step toward ulceration process. This theory is supported by the results of this study, where the prevalence of gastric metaplasia is higher in the patients with duodenitis compared with the healthy subjects. As a proof of this statement there is also a high concordance of duodenal ulcer disease and duodenitis in the same patients[43]. However, the role and the importance of duodenitis in the development of duodenal ulcer is not yet clear. The basic question is whether the duodenitis appears before duodenal ulcer disease, in the same time, or is it the consequence (residua) of ulcer disease. A high percent of our patients with duodenitis had positive H. pylori (60%) as well as gastric metaplasia, thus implying similar pathogenetic events if duodenitis is to be assumed as one step in duodenal ulcer development[43]. Smoking is a risk factor

in development[44] and recurrence of peptic ulcer[45] with influences badly upon ulcer healing[46]. By decreasing cytoprotective prostaglandins[47], smoking can cause duodenal mucosa damage. In our study in all patients there was no significant difference in the appearance of gastric metaplasia with respect to smoking. Significant increase of prevalence of gastric metaplasia in the patients with duodenal ulcer, however, implies that smoking has an important role in pathogenesis of duodenal ulcer disease. We have also found that smokers with duodenal ulcer and Helicobacter pylori infection had higher prevalence of gastric metaplasia that speaks in favour of possible synergistic activity of these two etiopathogenic factors upon duodenal ulcer disease appearance.

REFERENCES

1. MARSHALL, B. J., C. S. GOODWIN, J. R. WARREN, R. MURRAY, E. D. BLINCOW, S. J. BLACKBOURN, Lancet, II (1988)1437. — 2. WYATT, J. I., B. J. RATHBONE, M. F. DIXON, R. V. HEATLEY, J. Clin. Pathol., 40 (1987) 841. — 3. KREUNING, J., F. T. BOSMAN, G. KUIPER, A. M. vd WAL, J. LINDENMAN, J. Clin. Pathol., 31 (1978) 69. — 4. WYATT, J. I., B. J. RATHBONE, G. M. SOBALA, T. SHALLCROSS, R. V. HEATLEY, A. T. R. AXON, J. Clin. Pathol., 43 (1990) 981. — 5. JOHAN, G., A. OFFERHAUS, E. N. MOLYVAS, P. J. HOEDEMAEKER, J. Pathol., 162 (1990) 239. — 6. MADSEN, J. E., K. VETVIK, S. AASE, APMIS, 99 (1991) 997. — 7. WALKER, M. M., J. E. CRABTREE, Ann. NY Acad. Sci., 859 (1998) 96. — 8. FRIERSON, H. J., S. H. CALDWELL, B. J. MARSHALL, Mod. Pathol., 3 (1990) 271. — 9. KREUNING, J., A. M. WALL, G. KUIPER, J. LINDENMAN, Scand. J. Gastroenterol., 167 (1989) 16. — 10. WITEHEAD, R., R. ROCA, D. D. MIKLE, J. DKINER, S. C. TRUELOVE, Dig. Dis., 13 (1975) 129. — 11. STEPHEN, J. G., M. LESNA, C. W. WENEBLES, Br. J. Surg., 65 (1978) 438. — 12. NOACH, L. A., T. M. ROLF, N. B. BOSMA, M. P. SCHWARTZ, J. OOSTING, E. J. A. RAUWS, G. N. J. TYGTAT, Gut, 34 (1993) 1510. — 13. SAVARINO, V., G. S. MELA, P. ZENTILIN, M. R. MELE, L. LAPERTOSA, R. PATETTA, E. DALLORTO, A. VASSALO, C. MANSI, S. VIGNERI, G. CELLE, Gut, 39 (1996) 508. — 14. WORMSLEY, K. G., Gut, 15 (1974) 59. — 15. RAUWS, E. A. J., W. LANGENBERG, H. J. HOUTHOFF, H. C. ZANEN, G. N. J. TYGTAT, Gastroenterology, 94 (1988) 33. — 16. MARSHALL, B. J., J. A. ARMSTRONG, D. B. Mc GECHIE, R. J. GLANCY, Med. J. Aust., 142 (1985) 436. — 17. FUTAMI, H., M. TAKASHIMA, T. FURUTA, H. HANAI, E. KANEKO, J. Gastroenterol. Hepatol., 14 (1999) 114. — 18. PEURA, D. A., Am. J. Gastroenterol., 92 (1997) 8S. — 19. SOLCIA, E., L. VILLANI, R. FIOCCA, O. LUINETTI, R. BOLDORINI, E. TRESPI, M. PEREGO, C. ALVISI, M. LAZZARONI, G. BIANCHI PORRO, Scand. J. Gastroenterol., 201 (1994) 28. — 20. HSU, T. C., C. YEH, H. H. CHENG, Journal of Formosan Medical Association, 91 (1992) 81. — 21. WYATT, J. I., B. J. RATHBONE, M. F. DIXON, R. V. HEATLEY, A. T. R. AXON, Lancet, (1988) 118. — 22. TUCCI, A., D. SUSI, I. BOVANI, Gastroenterology, 98 (1990) A 141. — 23. JAMES, A. J., Gut, 5 (1964) 285-94. — 24. GREENLAW, R., D. G. SHEAHAN, V. De LUCA, D. MILLER, D. MYRESON, P. MYRESON, Dig. Dis. Sci., 25 (1980) 660. — 25. PATRICK, W. J. A., D. DENHAM, A. P. M. FORREST, Gut, 15 (1974) 767. — 26. CASELLI, M., L. TREVISANI, A. ALEOTTI, M. R. BOVOLENTA, G. STABELLINI, Dig. Dis. Sci., 34 (1989) 1374. — 27. CARRICK, J., A. LEE, S. HAZELL, M. RALSTON, G. DASKALOPOULOS, Gut, 30 (1989) 790. — 28. WYATT, J. I., B. J. RATHBONE, Gastroenterol. Clin. Biol., 13 (1989) 78B. — 29. BODE, G., P. MALFERTHEINER, C. KULLMAN, H. DITSCHUNEIT, Gastritis and duodenal ulcer: lecitin-binding characteristics of Campylobacter pylori infected and non-infected surface mucosu cells. In: MEDGRAUD, F., H, LAMOULIATTE (Eds.): Gastroduodenal pathology and Campylobacter pylori. (Elsevier, New York, 1989). — 30. FULLMAN, H., G. Van DEVENTER, D. SCHEIDMAN, J. WALSH, J. ELASHOFF, W. WEINSTEIN, Gastroenterology, 88 (1985) 1390. — 31. GRAHAM, D. J., Gastroenterology, 96 (1989) 615. — 32. URAKAMI, Y., M. KIMURA, H. SEKI, Jap. J. Gastroenterol., 93 (1996) 893. — 33. KIM, N., S. H. LIM, K. H. LEE, S. E. CHOI, J. Clin. Gastroenterol., 27 (1998) 246. — 34. MOSS, S., J. CALAM, Gut, 34 (1993) 888. — 35. LEVI, S., K. BEARDSHALL, G. HADDAD, R. PLAYFORD, P. GHOSH, J. CALAM, Lancet, 1 (1989) 1167. — 36. EL-OMAR, E., I. PENMAN, C. A. DIORRIAN, J. E. S. ARDILL, K. E. L. Mc COLL, Gut, 34 (1993) 1060. — 37. KHULUSI, S., S. BADVE, P. PATEL, R. LLOYD, J. M. MARRERO, C. FINLAYSON, M. A. MENDALL, T. C. NORTHFIELD, Gastroenterology, 110 (1996) 455. — 38. DIXON, M. F., J. I. WYATT, BMJ, 302 (1991) 1535. — 39. YANG, H., M. F. DIXON, J. ZUO, F. FONG, D. ZHOU, I. CORTHESY, A. BLUM, J. Clin. Gastroenterol., 20 (1995) 110. — 40. FITZGIBONS, P. L., C. P. DOOLEY, H. COHEN, M. D. APPLEMAN, Am. J. Clin. Pathol., 90 (1988) 711. — 41. RAUWS, E. A. J., G. N. J. TYGTAT, Lancet, I

(1990) 1233. — 42. SHOUSHA, S., I. G. BARRISON, W. EL-SAYED, S. KHAN, R. A. PARKINS, Dug. Dis. Sci., 29 (1984) 311. — 43. BAGO, J., M. RENIĆ, N. KUČIŠEC, Coll. Antropol., 21 (1997) 507. — 44. FRIEDMAN, G. D., A. B. SIEGLANB, C. C. SELTZER, N. Eng. J. Med., 290 (1974) 469. — 45. SONNENBERG, A., S. A. MULLER, E. VOGEL, Gastroenterology, 81 (1981) 1061. — 46. IPPOLITI, A., J. ELASHOF, J. VALENZUELA, Gastroenterology, 85 (1983) 875. — 47. BAGO, J., M. KLJAJIĆ TURKELJ, N. KUČIŠEC, Croat. J. Gastroenterol. Hepatol., 4 (1995) 59.

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POVEZANOST ŽELUČANE METAPLAZIJE S DOBI, SPOLOM, PUŠENJEM I HELICOBACTER PYLORI INFEKCIJOM U BOLESNIKA S DUODENALNIM ULKUSOM I DUODENITISOM

S A Ž E T A K

Želučana metaplazija je jedan od čimbenika u nastanku ulkusa dvanaesnika. Namjera ove studije bila je istražiti učestalost želučane metaplazije, te njenu povezanost s dobi, spolom, pušenjem i H. pylori infekcijom. U istraživanje je bilo uključeno 216 ispitanika, od čega 98 s ulkusom dvanaesnika, 60 s duodenitisom i 58 zdravih ispitanika. Nije bilo statistički značajne razlike kod učestalosti želučane metaplazije po dobi i spolu. Želučana metaplazija je bila statistički značajno veća u bolesnika s ulkusom dvanaesnika ($p < 0,01$). Kod svih ispitanika pušenje nije imalo značajnijeg utjecaja na prisustvo želučane metaplazije. Bolesnici s vrijedom dvanaesnika, koji su bili pušači i bolesnici koji su bili pušači te imali vrijed dvanaesnika i H. pylori infekciju imali su značajno veću učestalost želučane metaplazije ($p < 0,01$). Međutim, u bolesnika s ulkusom dvanaesnika nije bilo statistički značajne razlike u prisutnosti želučane metaplazije s obzirom na prisustvo Helicobacter pylori. Čini se da Helicobacter pylori infekcija nije od presudnog značenja u nastanku želučane metaplazije.