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ETIOLOGICAL FACTORS IN URGENT GASTRODUODENAL ULCER

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Abstract

Aim: The goal of the study is to assess the impact of the etiological and risk factors in the cases of urgent gastroduodenal ulcer.

Methods: This is a prospective study which included 67 patients selected randomly, all with urgent, life-threatening complications of the gastroduodenal ulcer, treated at the University Clinic for Digestive Surgery and the University Clinic for Gastroenterohepatology. For the purpose of the study the titer of IgG antibodies to Helicobacter pylori in serum is examined, as well as the use of NSAID and aspirin. We also tested the use of nicotine and alcohol. Besides these factors, we took into consideration the sex and the age.

Results: The serological test of Helicobacter pylori was positive in 89.6% of the cases. 31.3% used NSAID and 16.4% used aspirin. Large number, 65.7% of the patients were smokers, while 25.4% used alcohol.

With regard to the sex, 83.6% were male and 16.4% were female. The most frequent age group was the one from 41 to 60 years with 40.3% and those between 61 and 80 years of age with 35.8%.

Conclusion: The presence of Helicobacter pylori with the urgent gastroduodenal ulcer was exceptionally high, in 89.6% of the duodenal and gastric ulcers. NSAID and aspirin are factors that also influence the etiology of this disease. Smoking is a significant risk factor. Alcohol is less present, but still an important factor.

Key words: Helicobacter pylori, nonsteroidal anti-inflammatory drugs (NSAID), aspirin

Introduction

The peptic ulcer is a break in the normal mucosal integrity of the stomach, duodenum, or both, that extends through the muscularis mucosa into the submucosa or deeper.

The peptic ulcer disease is a widely present health problem because of its high morbidity, mortality and economic costs [1, 2].

In normal conditions, the stomach mucosa maintains balance between the protective and the aggressive factors. Mucosal injury may occur when noxious factors "overwhelm" an intact mucosal defense or when the mucosal defense is impaired [3].

Mucosal defense is composed of three critical components: pre-epithelial, epithelial, and post-epithelial. Pre-epithelial defense is composed of the mucous gel containing mucin, bicarbonate, and surface-active phospholipids. The epithelial component includes the surface cells, their apical tight junctions, and membrane transporters. Post-epithelial defense is maintained by mucosal blood flow, which is essential for both defense and repair [3, 4].

Among the aggressive factors it is confirmed that the infection with Helicobacter pylori (H. pylori) is the most common etiological factor for gastric lesion, such as the chronic gastritis or the peptic ulcer.

Several interactive mechanisms, including the obstructions in the regulation of the gastrin, the secretion of the acid, the inflammatory res-

ponse and the nerve pathways, contribute to the development of the H. pylori induced lesions on the stomach with further consequences.

The use of nonsteroidal anti-inflamatory drugs (NSAID), including aspirin, are the second most common risk factor for these lesions on the gastroduodenum [5, 6].

NSAIDs interfere with mucosal defense via direct toxic effects in addition to cyclooxygenase inhibition and subsequent depletion of endogenous prostaglandins [7].

The infection with H. pylori and the use of NSAID or aspirin, independently one from another, increase the risk of peptic ulcer [8–10].

Although the infection and the NSAID are different risk factors and cause damage to the mucosis of the stomach through different mechanisms, they can also act through joint mechanisms which include some key molecular changes in the formation of the lesion on the mucosis [11–13].

Besides these two most common etiological factors, smoking, alcohol, advanced age, use of oral corticosteroids and salted food are considered as additional factors [14, 15].

Smoking increases gastric acid secretion, inhibits gastric mucous secretion, gastric prostaglandin generation, mucosal bicarbonate secretion and decreases gastric mucosal blood flow [16, 17].

Duodenal ulcers are most common in the age group from 45 to 64 years and are twice more common in men than in women, while the gastric ulcers are most common in the age group from 55 to 65 and are equally present in both men and women.

Materials and method

This is a prospective study which randomly involved 76 patients with perforated and bleeding ulcer, treated at the University Clinic for Digestive Surgery and at the University Clinic for Gastroenterohepatology in the course of 2014.

None of the patients involved in the study has previously received eradication therapy for H. pylori. None of the patients was tested by serological or other diagnostic methods for presence of an infection with H. pylori.

The following variables were analyzed: gender, age group, IgG titer antibodies to H. pylori, use of NSAID and aspirin, as well as consumption of cigarettes and alcohol.

The proportion of men vs women was also defined.

Regarding their age, the patients were divided in five groups: up to 20 years of age, 21 to 40 years of age, 41 to 60, 61 to 80 and above 80 years of age.

The presence of H. pylori as an etiological factor was determined serologically by defining the IgG antibodies to H. pylori, proven with immunofluorescent method with Immulite 2000xPi analyser at the University Clinic for Clinical Biochemistry. The normal values are from 0.4 to 1.1 U/ml (border limit from 0.9 to 1.1 U/ml). The study analyzed the level of increase of the titer of IgG antibodies form 1.1 to two or three times more.

Urea breath test and fecal antigen test for H. pylori, as well as the invasive test were excluded due to the risk of false results as a consequence of preceding use of antibiotics and/or inhibitors of proton pump before the admission to hospital institutions where the study was done, as well as because of the bleeding when bleeding ulcers are in question.

The use of NSAID and aspirin, as well as the use of cigarettes and alcohol were obtained from the anamnesis of the surveyed patients. The data gathered from the patients were recorded in questionnaires.

Percent of structure in this study is used. The difference among attributive series has been determined (Difference test).

Results

The duodenal ulcers are more common (68.7%). The percent difference between the registration of gastric and duodenal ulcer is statistically significant for $p < 0.05$ ($p = 0.000$. Difference test).

28 of the patients in the study had a perforated ulcer, i.e. 41.8%, while 39 had a bleeding ulcer, i.e. 58.2%. 19 of the patients with a bleeding ulcer, i.e. 48.7%, were operated, while 20, i.e. 51.3%, were treated conservatively (chart 1).

Chart 1

Presentation of patients according to localization, perforated and bleeding ulcer

Patients N = 69	Number	%
Localization		
Stomach (gaster)	21	31.3
Duodenum	46	68.7
Complications		
Perforations	28	41.8
Bleedings	39	58.2
Bleedings N = 39		
Operated	19	48.7
Non-operated	20	51.3

56 i.e 83.6% of the patients in the study were men, 11, i.e 16.4% were women. The percent difference between the gender registration is statistically significant (Difference test). The relation men-women was 5.1 to 1 (chart 2).

The most present adult group is over 61 years, i.e. 41.8%, followed by the adult group from 41–60 years, i.e. 40.3%, adult group below 40 years is present with 17.9% (chart 2). The percent difference is statistically significant between the age below 40 years vs the ages from 41–60, for $p < 0.05$ ($p = 0.005$, Difference test), as well as between the age below 40 years vs the age over 61 years, for $p < 0.05$ ($p = 0.0031$, Difference test).

44 were smokers, i.e. 65.7%; the percent difference in relation to those who are non-smokers is statistically significant for $p < 0.05$ ($p = 0.0000$, Difference test).

17 used alcohol, i.e. 25.4%. Most of the smokers have been smoking for over than 20 years, 28 of them, i.e. 63.6%, and most of them smoked 1 to 2 packs, 26 i.e. 59.1%. Of those who consumed alcohol, most of them drank 2 to 3 cups of brandy a day, 8, i.e. 47.1%, and most of them have been drinking in the course from 11 to 20 years, 7 of the patients, i.e. 41.2% (Chart 2).

Chart 2

Percent presentation of the risk factors

Gender	Number	%
men	56	83.6
women	11	16.4
Age from ... to		
< = 20 y.	2	3.0
21 to 40 y.	10	14.9
41 to 60 y.	27	40.3
61 to 80 y.	24	35.8
> 80 y.	4	6.0
Cigarettes		
Yes	44	65.7
No	23	34.3
Cigarettes/Period of consumption N = 44		
< = 5 y.	1	2.3
6 to 10 y.	4	9.1
11 to 20 y.	11	25.0
> 20 y.	28	63.6
Number of cigarettes		
<= 10 cigarettes	4	9.1
10 to 20 cigarettes	12	27.3
1 to 2 packs	26	59.1
> packs	2	4.5
Alcohol		
Yes	17	25.4
No	50	74.6
Alcohol/Period of consumption N = 17		
< = 5 y.	1	5.9
6 to 10 y.	3	17.6
11 to 20 y.	7	41.2
> 20 y.	6	35.3
Alcohol/Glass of brandy		
one	2	11.8
2 to 3	8	47.1
3 to 5	4	23.5
> 5 glasses	3	17.6

NSAID were used by 21 patient, i.e. 31.3%, while 11 used aspirin, i.e. 16.4% (chart 3).

The titer of IgG antibodies to H. pylori was increased in 60 patients, i.e. 89.6%, which were mostly three time above the normal values in 43.4%. The percent difference between the registration of increased titer of IgG antibodies vs not increased titer of IgG is statistically significant for $p < 0.05$ ($p = 0.0000$, Difference test).

In 28.3%, the increased titer of IgG antibodies to H. pylori was in combination with NSAID, in 15% in combination with aspirin, while in

6.7% all the three factors worked together (chart 3). The percent difference between the increased titer of IgG antibodies (89.6%) vs the use of NSAID (31.3%) is statistically signifycant for $p < 0.05$ ($p = 0.0000$, Difference test). The percent difference between the registration of the increased titer of IgG antibodies (89.6%) vs the use of aspirin (16.4%) is statistically significant for $p < 0.05$ ($p = 0.0000$, Difference test).

In the patients with perforated ulcer, 23, i.e. 82.1%, showed increased titer of IgG antibodies to H. pylori, while in the bleeding patients the IgG titer was increased in 37, i.e. 94.9% of the cases (Chart 3).

Chart 3

Percent presentation of etiological factors

Increased titer of IgG antibodies to H. pylori	**Number**	**%**
yes	60	89.6
no	7	10.4
Increased titer of antibodies to H. pylori in perforations N = 28		
yes	23	82.1
no	5	17.9
Increased titer of antibodies to H. pylori in bleedings N = 39		
yes	37	94.9
no	2	5.1
Increased titer of antibodies to H. pylori, stomach N = 21		
yes	19	90.5
no	2	9.5
Increased titer of antibodies to H. pylori, duodenum N = 46		
yes	41	89.1
no	6	10.9
Increased titer of IgG antibodies to H. pylori for		
1,1 to 2 times	23	38.3
2,1 to 3,0 times	11	18.3
> from 3,0 times	26	43.4
NSAID		
yes	21	31.3
no	46	68.7
NSAID perforations N = 28		
yes	7	25.0
no	21	75.0
NSAID bleedings N = 39		
yes	14	35.9
no	25	64.1
Aspirin		
yes	11	16.4
no	56	83.6
Aspirin perforations N = 28		
yes	2	7.1
no	26	92.9
Aspirin bleedings N = 39		
yes	9	23.1
no	30	76.9
Concomitantly present N = 60		
H. pylori + NSAID	17	28.3
H. pylori + Aspirin	9	15.0
H. pylori + NSAID + Aspirin	4	6.7

The percent difference between the registration of increased titer of IgG antibodies (82.1%) in perforated ulcer vs increased titer of IgG antibodies (94.9%) in patients with bleeding is statistically significant for $p < 0.05$ ($p = 0.0217$, Difference test).

In the duodenal ulcers, the titer of IgG antibodies to H. pylori was increased in 89.1%, while in the gastric ulcers in 90.5% (chart 3).

The percent difference between the registration of increased titer of IgG antibodies in stomach vs duodenum is not statistically significant for $p > 0.05$ ($p = 0.8720$, Difference test) (chart 3).

More common was the use of NSAID and aspirin by patients with bleeding ulcer compared to the patients with perforated ulcer, 35.9% to 25.0%, i.e. 23.1% to 7.1% (chart 3).

Discussion

H. pylori is a common human pathogen and health problem connected with the pathogenesis of gastritis and ulcer. The prevalence of infection with H. pylori varies from 40% to 60% in the developed countries and up to 80% to 90% in the developing countries.

H. pylori colonizes the gastric mucosa of more than 50% of human population. The chronic infection generates a state of inflammation, which however is asymptomatic in the majority of the subjects [18]. In 10–15% of the infected, the inflammation can lead to peptic ulcer.

According to McNeily, the prevalence of the infection with H. pylori is 80% for duodenal ulcers and 50% for the gastric ulcers, while in the study of Chen et al. it is 88.7% in duodenal ulcers and 90% in the gastric ulcers [19, 20].

According to Gisbert et al. the prevalence of the infection with Helicobacter pylori in patients with perforated peptic ulcer is about 60%, which is contrary to the 90–100% in non-complicated peptic ulcers. But, if the use of non-steroid anti-inflammatory medicines is excluded, the prevalence of the infection in the perforated ulcer is almost 90% [21].

According to the same author, if the use of non-steroid anti-inflamatory drugs is excluded in the bleeding duodenal ulcers, the prevalence of infection is almost 100%, i.e. 97%, similar to those with non-bleeding duodenal ulcers [22].

Among the participants in this study, in 89.6% the presence of H. pylori has been proven, which in the patients with perforated ulcer is 82.1%, and in those with bleeding ulcer it is 94.9%. In duodenal ulcers, the H. pylori was present in 89.1%, while in the gastric ulcers in 90.5%. The IgG antibody titer to H. pylori was in 43.4% increased for more than three times.

Another important factor directly related with the gastric lesion is the obstruction of the mucosal barrier through significant use of non-steroid anti-inflamatory medicines (NSAID). The research shows that the NSAID are one of the most commonly used medicines in the world. The use of NSAID is more common among women and increases with the age and is in correlation with the rheumatic diseases. More than 90% of the NSAID users are patients older than 65 years.

The main mechanism through which the NSAID causes an ulcer and gastric complications is considered the inhibition of cyclooxygenase (COX), with a deficit of prostaglandins. There are two identified isoforms of COX, COX1 and COX2.

COX1 isoform is present in a number of tissues, producing prostaglandins, which have an important protective role in the stomach by stimulating synthesis and secretion of mucus and bicarbonates, by increasing the mucosal integrity of the stomach at the basic line. On the other hand, COX2 is little or not at all present in the tissues, but it quickly induces as a response to inflammatory stimulus.

Some experimental studies have shown that inhibition of both Cox1 and COX2 is needed for the NSAID to cause lesion on the stomach [23].

Every fourth perforated ulcer is a result of the use of non-steroid anti-inflammatory drugs, an etiological factor that is particularly important among the elderly [24].

According to Ootani, the use of NSAID is associated with 28.4% in patients with bleeding gastroduodenal ulcers [25].

Low doses of aspirin can also lead to acute complications of gastroduodenal ulcers.

Concerning the participants in the study, NSAID was used by 31.3% of them, and 16.4% used aspirin. The use of NSAID and aspirin was more common in the patients with bleeding ulcer vs perforations, 35.9%, compared to 25.0%, i.e. 23.1% to 7.1%, respectively.

H. pylori was in combination with NSAID in 28.3%, in combination with aspirin in 15%, while in 6.7% in combination with all the three factors together.

Smoking is an independent risk factor for peptic ulcer disease [26]. The percentage of smokers with perforated gastroduodenal ulcer indicates that the smoking is a risk factor for ulcer and perforation.

The study of Swens et al. indicates that smoking is a risk factor for perforations in gastroduodenal ulcer and increases the risk for perforation 10 times in the age group from 15 to 74 [27].

Andersen et al. in their study assess the connection between smoking, the use of alcohol (including the type of alcohol) and the risk of perforated ulcer.

Smoking more than 15 cigarettes daily increases the risk of perforated ulcer for more than three times, in comparison with non-smokers.

Drinking of more than 42 dozes of alcohol a week increases the risk of bleeding to four times, compared with the drinking of less than one drink a week. The participants who drank more than 21 drinks a week, but not wine, were exposed to increased risk of bleeding from gastroduodenal ulcer, rather than the participants who drank the same amount of alcohol, but where 25% of their doze was wine [28].

Paracher et al. conclude that smoking is not an independent risk factor, but works through enhancing the negative effects of Helicobacter pylori and in combination it negatively impacts the gastrointestinal mucosal lining. Smoking also increases the risk of infection with H. pylori [29].

In this study 65.7% of the participants were smokers and 25.4% have used alcohol. Most of the smokers consumed cigarettes for more than 20 years, 63.6% and 59.1%, and smoked 1 to 2 packs a day. Most of those who consumed alcohol drank 2 to 3 dozes of brandy a day, 47.1%, and most of them were drinking in the past 11 to 20 years, 41.2%.

Gastroduodenal ulcer disease is common in the mid years from 40 to 60 and is more common in men.

In the study of Batra et al., the middle age of presentation was 40.9 ± 17.4 years. The maximum number of patients with gastrointestinal perforations were adult groups from 30 to 49 years, 34.4%, and between 50 and 69 years of age with 31.2%. Men were more present than women, i.e. 88.5% were men and 11.5% were women, or in correlation of 7.7 to 1 [30].

In this study, 83.6% were men and 16.4% were women, where the correlation was 5.1 to 1. The most present age group was between 41 and 60 years with 40.3%, and between 61 and 80 with 35.8%.

Conclusion

From the results of this study, we should highlight the high connection between the infection with H. pylori and the urgent, life-threatening gastroduodenal ulcer, which leads us to a conclusion that H. pylori is the main etiological factor for this clinical entity which is present in 90% of the patients tested.

It is equally present both in gastric and duodenal ulcer. H. pylori is more present in the cases of bleeding ulcer than in the cases of perforations.

Non-steriod anti-inflamatory drugs and aspirin are factors that also have influence in the etiology of this disease. More common is their connection with the bleeding ulcer disease in comparison with the perforations.

Approximately every third participant has used NSAID, and every sixth has used aspirin.

Very often Helicobacter pylori and one or two of these factors are present at the same time.

Smoking is an important risk factor for this disease and more than half (65.7%) of the patients were smokers. Alcohol is less present, every fourth patient, but it is also an important risk factor. Most of these patients have used alcohol or have smoked for a long period of time.

Men are far more present in this study, with correlation of 5.1 to 1.

Concerning the age groups, most present were the patients at the age from 41 to 60 (40.30%), and 61 to 80 (35.8%).

REFERENCE

1. Sonnenberg A, Everhart JE. Health impact of peptic ulcer in the United States. Am J Gastroenterol. 1997; 92: 614–620.
2. Dimaline R, Varro A. Attack and defence in the gastric epithelium – a delicate balance. Experimental Physiology. July 2007; Vol. 92, No. 4 pp. 591–601, ISSN 0958-0670.
3. Laine L, Takeuchi K, Tarnawski A. Gastric mucosal defense and cytoprotection: bench to bedside. Gastroenterology. 2008 Jul; 135(1): 41–60.
4. Scheiman JM. Pathogenesis of gastroduodenal injury due to nonsteroidal antiinflammatory drugs: implications for prevention and therapy. Semin Arthritis Rheum. 1992 Feb; 21(4): 201–10.
5. Lanas Á, Carrera-Lasfuentes P, Arguedas Y, et al. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. Clin Gastroenterol Hepatol. 2015 May; 13(5): 906–12.
6. Voutilainen M, Mäntynen T, Färkkilä M, et al. Impact of non-steroidal anti-inflammatory drug and aspirin use on the prevalence of dyspepsia and uncomplicated peptic ulcer disease. Scand J Gastroenterol. 2001 Aug; 36(8): 817–2.
7. Scheiman JM. NSAID-induced peptic ulcer disease: a critical review of pathogenesis and management. Dig Dis. 1994 Jul-Aug; 12(4): 210–22.
8. Nagata N, Niikura R, Sekine K, et all. Risk of peptic ulcer bleeding associated with Helicobacter pylori infection, nonsteroidal anti-inflammatory drugs, low-dose aspirin, and antihypertensive drugs: a case-control study. J Gastroenterol Hepatol. 2015 Feb; 30(2): 292–8.
9. Bazzoli F, De Luca L, Graham DY. Helicobacter pylori infection and the use of NSAIDs. Best Pract Res Clin Gastroenterol. 2001 Oct; 15(5): 775–85.
10. Huang JQ, Sridhar S, Hunt RH. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. Lancet. 2002 Jan 5; 359: 14–22.
11. Zapata-Colindres JC, Zepeda-Gómez S, Montaño-Loza A, et al. The association of Helicobacter pylori infection and nonsteroidal anti-inflammatory drugs in peptic ulcer disease. Can J Gastroenterol. 2006 Apr; 20(4): 277–280.
12. Aalykke C, Lauritsen JM, Hallas J, et al. Helicobacter pylori and risk of ulcer bleeding among users of nonsteroidal anti-inflammatory drugs: a case-control study. Gastroenterology. 1999 Jun; 116(6): 1305–9.
13. Zapata-Colindres JC., Zepeda-Gómez S, Montaño-Loza A, et all. The association of Helicobacter pylori infection and nonsteroidal anti-inflammatory drugs in peptic ulcer disease. Can J Gastroenterol. 2006 Apr; 20(4): 277–280.
14. Garcia Rodriguez LA, Jick H. Risk of upper gastrointestinal bleeding and perforation associated with individual non-steroidal anti-inflammatory drugs. Lancet. 1994; 343: 769–72.
15. Thorsen K, Søreide JA, Kvaløy JT. Epidemiology of perforated peptic ulcer: Age-and gender adjusted analysis of incidence and mortality. World J Gastroenterol 2013 January 21; 19(3): 347–354.
16. Maity P, Biswas K, Roy S, et al. Smoking and the pathogenesis of gastroduodenal ulcer--recent mechanistic update. Mol Cell Biochem. 2003 Nov; 253(1–2): 329–38.
17. Eastwood GL. Is smoking still important in the pathogenesis of peptic ulcer disease? J Clin Gastroenterol. 1997; 25 Suppl 1: S1-7.
18. Ruggiero P. Helicobacter pylori and inflammation. Curr Pharm Des. 2010; 16(38): 4225–36.
19. McNally P. UGI Bleeding: New and Old Risk Factors. Visible Human Journal of Endoscopy. 2010 March; Volume 9, Issue 1.
20. Chen TS, Luo JC, Chang FY. Prevalence of Helicobacter pylori infection in duodenal ulcer and gastroduodenal ulcer diseases in Taiwan. J Gastroenterol Hepatol. 2010 May; 25(5): 919–22.
21. Gisbert JP, Legido J, García-Sanz I, et al. Helicobacter pylori and perforated peptic ulcer prevalence of the infection and role of non-steroidal anti-inflammatory drugs. Dig Liver Dis. 2004 Feb; 36(2): 116–20.
22. Gisbert JP, Gonzalez L, de Pedro A, et al. Helicobacter pylori and bleeding duodenal ulcer: prevalence of the infection and role of non-steroidal anti-inflammatory drugs. Scand J Gastroenterol. 2001 Jul; 36(7): 717–24.
23. Matsui H, Shimokawa O, Kaneko T, et al. The pathophysiology of non-steroidal anti-inflammatory drug (NSAID)-induced mucosal injuries in stomach and small intestine. J Clin Biochem Nutr. Mar 2011; 48(2): 107–111.
24. Svanes C. Trends in perforated peptic ulcer: incidence, etiology, treatment, and prognosis. World J Surg. 2000 Mar; 24(3): 277–83.
25. Ootani H, Iwakiri R, Shimoda R, et al. Role of Helicobacter pylori infection and nonsteroidal anti-inflammatory drug use in bleeding peptic ulcers in Japan. J Gastroenterol. 2006 Jan; 41(1): 41–6.
26. Rosenstock S, Jørgensen T, Bonnevie O, et al. Risk factors for peptic ulcer disease: a population based prospective cohort study comprising 2416 Danish adults. Gut. 2003 Feb; 52(2): 186–193.
27. Svanes C, Søreide JA, Skarstein A, et al. Smoking and ulcer perforation. Gut. 1997 Aug; 41(2): 177–80.
28. Andersen IB, Jørgensen T, Bonnevie O, et al. Smoking and alcohol intake as risk factors for bleeding and perforated peptic ulcers: a population-based cohort study. Epidemiology. 2000 Jul; 11(4): 434–9.

29. Parasher G, Eastwood GL. Smoking and peptic ulcer in the Helicobacter pylori era. Eur J Gastroenterol Hepatol. 2000 Aug; 12(8): 843–53.
30. Batra P, Gupta D, Narang R, et al. Spectrum of gastro intestinal perforation. Peritonitis in rural central India. J MGIMS. 2013 March; Vol 18, No (i): 44–48.

Резиме

ЕТИОЛОШКИ ФАКТОРИ КАЈ УРГЕНТНИОТ ГАСТРОДУОДЕНАЛЕН УЛКУС

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Цел: Целта на студијата е да го процени влијанието на етиолошките и на ризик-факторите во појавата на ургентниот гастродуоденален улкус.

Metogu: Се работи за проспективна студија во која по случаен избор се вклучени 67 пациенти со ургентни, животнозагрозувачки компликации на гастродуоденалниот улкус, лекувани на Универзитетската клиника за дигестивна хирургија и Универзитетската клиника за гастроентерохепатологија.

За целта на студијата е испитуван титарот на IGg антитела насочени против Helicobacter pylori во серум, употребата на НСАИЛ и аспирин. Исто така, испитувано е и користењето никотин и алкохол како ризик-фактори.

Покрај нив, како ризик-фактори се испитувани и половата припадност и возраста.

Резултати: Серолошкиот тест за H. pylori беше позитивен во 89,6% од случаите, НСАИЛ употребувале 31,3%, а аспирин 16,4%.

Голем број од пациентите се пушачи 65,7%, додека 25,4% користеле алкохол.

Во однос на половата припадност, 83,6% беа мажи, а 16,4 од женскиот пол. Најчести возрасни групи беа од 41 до 60 години со 40,3% и од 61 до 80 години со 35,8%.

Заклучок: Присутноста на H. pylori кај ургентниот гастродуоденален улкус во студијата е исклучително висока, во 89,6% и при дуоденалните и при гастричните улкуси. НСАИЛ и аспирин се фактори што имаат, исто така, влијание во етилогијата за ова заболување.

Пушењето е значаен фактор на ризик за ова заболување. Алкохолот е помалку изразен, но е значаен фактор на ризик за ова заболување.

Особено е битно времетраењето на користење на никотин и консумација на алкохолот.

Клучни зборови: Helicobacter pylori, НСАИЛ, аспирин.