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# *The effect of chronic alcohol abuse on gastric and duodenal mucosa*

Ethyl alcohol is a protoplasmatic toxin which produces a multitude of biological effects in the human organism. Consumed in bigger quantities it directly harms each cell and tissue of the organism. The lipophyl character of alcohol causes that it is immediately and completely absorbed from the alimentary tract: in 20% from gastric mucosa and in 80% from intestinal mucosa. An adult's stomach contains at least three isoenzymes of alcohol dehydrogenase (7, 22). Delta dehydrogenase is an enzyme that is specific for the stomach. The level of this enzyme can vary in different people. Lower activity of alcohol dehydrogenase in the gastric mucosa is observed in women; therefore, higher absorption and a higher level of alcohol in the blood are found after consuming the same amount of alcohol (13). Alcohol is metabolized mainly in the liver with the participation of three independent metabolic systems, namely alcohol dehydrogenase, microsomal system of alcohol oxidation (MEOS) containing cytochrome P450 IIE, and catalasis (17, 19, 22). The main product of alcohol oxidation, independent of the metabolic tract is acetaldehyde, a compound which is ten times as toxic and responsible for organic damage.

A direct contact of alcohol with the mucosa of the alimentary tract causes the latter's damage. The effect of ethanol on gastric mucosa is a complex process, where a number of factors are involved. Alcohol has a negative influence on a number of elements of the mucous barrier. This effect is first of all related to the concentration of ethanol and the period of its abuse (23). Alcohol disturbs the physico-chemical properties of gastric mucus. In high concentrations it inhibits the synthesis and secretion of mucus glycoprotein; it also decreases the thickness of its layer considerably and limits the accumulation of mucus in intracellular spaces. As a result of these processes, the permeation of gastric mucosa for the regressive diffusion of hydrogen ions increases, causing its damage (5, 18).

Alcohol disturbs the structures of gastric surphactant, increases the permeation and fluidity of cell membrane, disturbs the distribution of electrolytes between the intercellular space and the inside of the cells, changes the activity of membrane enzymes and cellular metabolism (2, 14), as a consequence leading to the damage and death of the cells, which undergo desquamation from the surface layers of the epithelium (5). Concentrated ethanol penetrating into the inside of the mucosa damages the net of submucous vessels, which is followed by disturbances of the blood flow. The degree of the damage of the vessels and the inflammatory reaction accompanying it decides about how serious the damage of the mucosa is. Bleeding may occur in the course of alcoholic injury and the percent of hemorrhages taking place under the effect of ethanol can reach as much as 27–45% of cases of hemorrhages from the upper section of the alimentary tract. The changes found within the cells of the epithelium of the gastric mucosa, unless the vessels have been damaged, heal quickly. In the course of about an hour the migrating cells of the epithelium crawl up onto the mucosa injured by alcohol and although even a short exposition to ethanol damages 95% of the cells of the epithelial surface, after a few hours the whole damaged surface is covered by a new epithelium.

The effect of alcohol on the secretion of hydrochloric acid in gastric juice is related to the kind of consumed alcoholic drink and the concentration of ethanol in it (26). Alcoholic beverages with low concentration of ethanol, such as beer and wine, stimulate the secretion of gastric juice. This is a negative effect, especially when insufficient amount of food is consumed.

The changes observed in the gastric mucosa following the effect of alcohol are reversible if the causative factor – alcohol – is sporadically consumed. If, however, chronic consumption of alcohol takes place, especially in big quantities, then chronic alterations within the mucosa and impairment of the secretory function of this organ occur.

The purpose of the studies was to provide a morphological and functional estimation of the gastric and duodendal mucosa in persons who continue to abuse alcohol, and of the effect of the period of this abuse on the observed changes.

## MATERIAL AND METHODS

The studies covered a group of 79 people, including 61 who abuse alcohol chronically and 18 who do not abuse alcohol. The latter formed the control group. The group of addicted people was made up of men from 16 to 65 years of age (average age, 38) hospitalized in the Department of Disaccustoming Treatment of the Specialist Psychiatric Hospital in Lublin. The patients came to hospital on their own. The period

of the addiction ranged from 5 to 37 years. The control group was made up of 18 persons from 17 to 59 years of age (average age, 37). In this group, 13 people had never drunk alcohol, 5 people drank alcohol rarely in small quantities, the last consumption taking place a few months earlier. The program did not include the people who suffered from hepatocellular damage, chronic peptic ulcer disease, systemic disease or who were after an operation. The patients were divided into four groups depending on the duration of the addiction: studied group I – 17 male aged 17 to 43 (average age, 30) addicted to alcohol for 5–10 years; studied group II – 29 male aged 28–57 (average age, 38) addicted to alcohol for 11–20 years; studied group III – 15 male aged 36-64 (average age, 48) addicted to alcohol for over 20 years; control group IV – 18 people aged 17–59 (average age, 37) including 9 women and 9 men, who did not drink alcohol chronically.

All the patients from the four groups were submitted to gastroscopy with morphological estimation of gastric and duodendal mucosa. Besides, secretion of hydrochloric acid was tested using the Kay's method. Endoscopy of the mucous membrane of stomach and duodendum was performed using a gastrofibroscope OLYMPUS. During the examination, 2 biopsy specimens were taken from each, the prepyloric area, the body of the stomach and the duodendal bulb. The histological estimation of the specimens was performed on the basis of the examination of colored paraffin preparations H + E, PAS and mucicarmine.

The results were analyzed statistically. An independence test $Chi^2$ for multi-field contingency tables (with Yartes' correction) was applied. The mean values were compared with t-Student's test. The results are presented in tables and figures. Probability of $p<0.05$ was accepted as statistically significant.

## RESULTS

A morphological examination of the gastric mucosa showed chronic inflammatory changes in all the patients addicted to alcohol and in 72% of those who did not drink alcohol (Table 1). Both groups considerably differed from each other with the progression of the inflammatory changes. Chronic inflammatory changes of atrophic inflammation type were observed in 14 out of 61 patients (22.9%) addicted to alcohol, including four people, where those changes occurred in both segments of the stomach (Table 1). In four cases atrophic changes were accompanied by intestinal metaplasia. The focuses of metaplasia were observed in the body and the pyloric area. Among the examined patients from the control group and the group of patients addicted to alcohol for over 10 years, the percent of duodendal mucosa inflammation was similar and it was 64.7% and 66.7%, respectively. The highest percent of mucous inflammation within the duodendal bulb was observed in the group of patients who had been drinking alcohol

Tab.1. Extension and character of inflammatory changes in gastric and duodendal mucosa

| Examined area | | | Studied group | | Control group | |
|---|---|---|---|---|---|---|
| Body | Pylorus | Duodenum | N | % | n | % |
| – | a.i. | a.i. | 1 | 1.6 | 0 | 0.0 |
| a.i. | a.i. | a.i. | 21 | 34.4 | 6 | 33.3 |
| a.i. | s.i. | a.i. | 5 | 8.2 | 0 | 0.0 |
| a.i. | s.i. | s.i. | 1 | 1.6 | 0 | 0.0 |
| a.i. | a.i. | n.c. | 6 | 9.8 | 2 | 11.1 |
| a.i. | s.i. | n.c. | 3 | 4.9 | 0 | 0.0 |
| a.i. | n.c. | a.i. | 2 | 3.3 | 0 | 0.0 |
| z.z. | s.i. | a.i. | 4 | 6.6 | 0 | 0.0 |
| z.z. | a.i. | n.c. | 1 | 1.6 | 0 | 0.0 |
| n.c. | n.c. | n.c. | 0 | 0.0 | 3 | 16.7 |
| n.c. | n.c. | a.i. | 3 | 4.9 | 2 | 11.1 |
| n.c. | a.i. | n.c. | 2 | 3.3 | 1 | 5.6 |
| n.c. | a.i. | a.i. | 12 | 19.7 | 4 | 22.2 |

a.i. – atrophic inflammation s.i. – surface inflammation n.c. – no changes

Tab.2. Kind of inflammation and a singular dose of ethanol

| Cunsumed dose of alcohol | Surface Inflammation | | Atrophic inflammation | | Total | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| < 200 g | 6 | 66.7 | 3 | 33.3 | 9 | 100.0 |
| 200 g | 29 | 80.6 | 7 | 19.4 | 36 | 100.0 |
| > 200 g | 12 | 75.0 | 4 | 25.0 | 16 | 100.0 |

$\chi^2$=0.185; df = 2; n.i.

for over 20 years (89.7%). The studies did not point to any relation between the amount of alcohol (converted into pure ethanol) consumed once and the percentage of cases with atrophic inflammation of the gastric mucosa (Table 2). No effect of alcohol concentration in the consumed alcoholic drinks on the kind (progression) of inflammatory changes of gastric mucosa was found out, either. Within the groups of people preferring weaker drinks and those who preferred beverages with higher content of ethanol a similar percent of cases with chronic atrophic inflammation of the

gastric mucous membrane was observed (Table 3). On the other hand, the studies showed a relation existing between the progression of inflammatory changes in the gastric mucous membrane and the period of alcohol abuse (Table 4).

Tab.3. Character of inflammatory changes of gastric mucosa in relation to the model of drinking

| | Surface inflammation | | Atrophic inflammation | | Total | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| Soft drinks | 22 | 78.6 | 6 | 21.4 | 28 | 100.0 |
| Strong drinks | 25 | 75.8 | 8 | 24.2 | 33 | 100.0 |

$\chi^2$=0.068; df = 1; n.i.

Tab.4. Character of gastric mucous membrane inflammation in relation to the period of alcohol abuse

| Period of addiction | Surface inflammation | | Atrophic inflammation | | Total | |
|---|---|---|---|---|---|---|
| | n | % | N | % | N | % |
| < 10 years | 16 | 94.1 | 1 | 5.9 | 17 | 100.0 |
| 10-20 years | 21 | 72.4 | 8 | 27.6 | 29 | 100.0 |
| > 20 years | 10 | 66.7 | 5 | 33.3 | 15 | 100.0 |

$\chi^2$=0.938; df = 2; n.i.

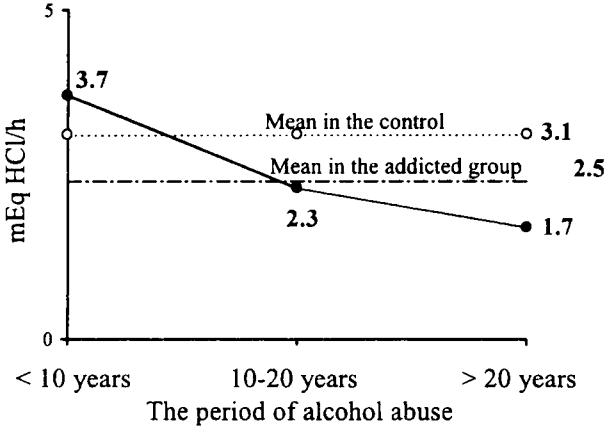


Fig.1. Average secretion of hydrochloric acid (BAO) in basic conditions in the studied and control groups

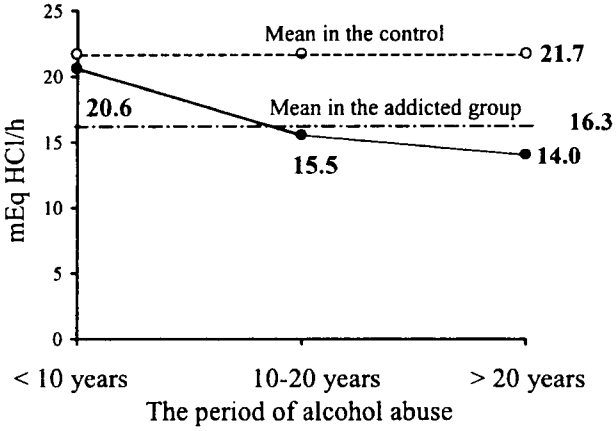


Fig. 2. Average maximum secretion of hydrochloric acid (MAO) in the studied and control groups

Examination of the secretory function of the stomach showed lower mean values of hydrochloric acid secretion with an empty stomach (BAO) and after stimulation with pentagastrin (MAO) in the group of people addicted to alcohol as compared to analogous values obtained in the group of patients who did not drink alcohol (Figs. 1, 2). The values of gastric secretion got lower with the time of alcohol abuse. The mean values of BAO obtained in the patients who had been drinking alcohol for over 20 years were twice as high in comparison to the patients who had been alcohol addicts for 10 years (Fig. 1). The mean values of MAO observed in the group of patients drinking alcohol for over 20 years were by 32% lower than in the group of patients addicted up to 10 years, and by 35% lower as compared to the control (Fig. 2). The studies also observed a relation existing between hydrochloric acid secretion and the character of inflammatory changes in the gastric mucous membrane. The secretion of hydrogen ions in the basic conditions and after stimulation with pentagastrin was most limited in patients with atrophic inflammation of the mucosa (Tabs. 5, 6).

Tab.5. Basic secretion of hydrochloric acid (BAO) – relation to the kind of inflammatory changes in gastric mucous membrane

| Character of inflammatory changes | Mean mEq HCl/h | n |
|---|---|---|
| Surface inflammation | 2.7 | 40 |
| Atrophic inflammation | 1.8 | 13 |
| Total | 2.5 | 53 |

$t^2$=0.938; df = 2; n.i.

Tab.6. Maximum secretion of hydrochloric acid (MAO) – relation to the kind of inflammatory changes in the gastric mucous membrane

| Character of inflammatory changes | Mean mEq HCl/h | n |
|---|---|---|
| Surface inflammation | 17.5 | 40 |
| Atrophic inflammation | 12.7 | 13 |
| Total | 16.3 | 53 |

t = 2.006; df = 51; n.i. ($p < 0.10$) ($p < 0.05$ for one-side test)

## DISCUSSION

Within the group of 79 examined patients the studies found out chronic inflammation of the gastric mucosa in all the patients chronically abusing alcohol but also in 72% of people who constituted the control group. Both compared groups of patients differed with the degree of progression of the inflammatory process of the gastric mucosa, which was tested with a histological examination. Atrophic inflammation was observed exclusively in the people addicted to alcohol. Atrophic changes were closely related to the duration of addiction.

The studies did not show any relation between the percent of cases with atrophic inflammation and the kind of alcoholic drinks or the content of ethanol in them.

The views on the effect of alcohol on the stomach in medical literature are differentiated. Though the damaging effect of ethanol on gastric mucous membrane, i.e. the appearance of acute gastritis is well based in documents, the role of alcohol in the etiology of chronic mucous inflammation of the stomach and duodendum is still a controversial matter.

The causative relation between chronic abuse of alcohol and chronic mucous inflammation is negated by a number of researchers (6, 16, 29). On the other hand, however, numerous observations confirm frequent occurrence of this disease in alcoholics. Chronic inflammation of mucosa in cases of chronic alcoholism was observed by Altman et al. (1), Hermandez-Munoz and Montiel-Ruiz (14), Glass and Pitchumoni (12), Roberts (24), Segawa et al. (25), Daniluk (8). Both chronic superficial and chronic atrophic inflammation of mucous membrane were observed in alcohol addicts.

Endoscopy in 69 patients who had been drinking alcohol chronically for at least 5 years showed changes in all the patients with different degrees of chronic or chronic aggravated inflammation of the gastric mucous membrane, and in 80% those changes also referred to the duodendum.

In physiological conditions, the cells of the gastric mucous membrane epithelium are covered with a layer of mucous gel, which protects them from the effect of exo-

and endo-genous damaging factors. Destruction and removal of the mucous layer lowers the resistance of the mucous membrane of the stomach to damaging factors and clearly makes the time of its reconstruction longer. Alcohol causes considerable quality changes in its structure (4, 15). The alcohol drunk by a person directly injures the surface cells of the mucous epithelium, which fulfil the function of the first defense line. The studies by Bailey et al. (2) showed that already alcohol in 1% concentration affects the phospholipids of cellular membranes causing their liquefaction. These disturbances are accompanied by changes in the activity of enzymes associated with cellular membrane (system ATP Na-K). The main effect of increased liquefaction is greater fragility of the cell membrane and greater susceptibility to damage.

Higher concentrations of ethanol (10–15%) cause injury of the surface cells of the mucous epithelium, which is visible in histological examination. It also results in cell dehydration; it disturbs oxidation processes, oxygen phosphorylation, active transport and secretory processes of the cells. The disturbances caused by ethanol are made greater by its toxic metabolite – acetaldehyde, which is formed in the stomach wall.

A characteristic syndrome of mucous changes in histopatological examination was observed in alcoholics by Lubczyńska-Kowalska et al. (21). They found out that chronic alcohol abuse causes the damage and excessive desquamation of the cells of the surface epithelium and stomach pits. Within a few hours reconstruction processes take place, but their effect is clear restructuring of the mucous membrane. As a consequence, the number of secretory cells is decreased and they are replaced by immature cells secreting mucus. Inhibition of maturation and differentiation of mucous membrane cells under the effect of ethanol is confirmed in the studies by Tarnawski et al. (27) and Tonlibilir and Leevy (28).

The major role in the processes of regeneration of the gastric mucous membrane is played by prostaglandins. Prostaglandins intensify the secretion of mucus, they increase the content of mucin and sialic acid in it, they cause thickening of its layer, owing to which they inhibit the permeation of alcohol into the mucosa and make its damage less serious.

Prostaglandins do not prevent the damage of surface epithelium cells but thanks to better mucous circulation (10, 27) and intensification of DNA and RNA synthesis they protect the deeper parts of the stomach mucosa from injury. They limit the damage of regeneration zone of the epithelium and contribute to faster epithelialisation of the injured surface of the mucous membrane (9, 27). Chronic abuse of alcohol is associated with considerable inhibition of prostaglandins synthesis in gastric mucosa. Bode et al. (3) studied prostaglandins synthesis in alcoholic patients and in patients who did not drink alcohol. The former ones showed remarkable decrease of prostagladins synthesis related to the progression of the inflammatory process.

Singer and Leffenmann (26) found out that ethanol in concentrations 1.4% and 4% increases the secretion of gastric juice up to 22 and 23% of MAO value. Higher concentrations of alcohol (5–10%) stimulate the secretion only slightly, while

20–40% ethanol lowers the acidity of gastric juice. According to the authors, stimulation of hydrochloric acid secretion by low-grade alcohol can be related to the release of acetylcholine and histamine. A possible explanation of the lack of stimulating effect of alcohol concentrations higher than 4% is the fact that ethanol in this concentration causes the injury of the mucous barrier and increases back diffusion of hydrogen ions from the stomach lumen inside the mucosa.

The effect of ethanol on the acidity of gastric juice can also be related to the changes in bicarbonate secretion. Low concentrations of ethanol (3.5%) inhibit gastric transport of bicarbonates, while alcohol with higher concentrations of ethanol (14%) intensifies the passive transport of bicarbonates into the stomach lumen, which is due to the damage of the mucosa (11).

Chronic exposition to ethanol leads to disturbances in the maturation and differentiation of gastric mucosa membrane cells, which results in reduction of parietal and chief cells (28). These disturbances in cell renewal with simultaneous ultra structural changes taking place in these cells (25) can be the cause of lower values of basic and maximum secretion in patients who chronically abuse alcohol.

## CONCLUSIONS

1. Chronic abuse of alcohol predisposes to atrophic inflammation of gastric mucous membrane.
2. The progression of inflammatory changes in the gastric mucous membrane points to the relation with the period of alcohol abuse, while showing no connection with the kind of alcohol or the concentration of ethanol in it.
3. Hydrochloric acid secretion gets lower with the period of alcohol abuse.

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## SUMMARY

Alcohol consumed in small quantities is not dangerous for health but if it is drunk in big amounts it has a negative effect on somatic and psychical health. A number of studies have been published on the harmful effect of ethanol on the nervous system, circulation, endocrine and immune systems. Ethyl alcohol can directly damage the mucosa of the alimentary tract. The studies conducted and published so far have estimated acute ethanol damage of gastric mucosa in experimental animals. The views found in literature on the effect of continuous consumption of ethanol on the stomach are differentiated. The purpose of the paper was to provide a morphological and functional analysis of stomach and duodendum in patients who continuously abuse alcohol. Besides, at attempt was made to find the answer to the question whether the changes observed in gastric mucosa point to any connection with the period of alcohol abuse. The study referred to 79 persons: 61 male aged 17-65 who had been chronically drinking alcohol for the period of 5–37 years and 18 persons aged 17-59 (9 male and 9 female) who had never drunk alcohol or who had drunk it only rarely and in small quantities. The latter ones were the control group. The patients addicted to alcohol

were divided into three groups differing with the period of addiction (I – 5–10 years; II – 10–20 years; III – over 20 years). Hydrochloric acid secretion in gastric juice was marked and gastroscopy was performed in all the examined patients. Gastroscopy estimated the appearance of the gastric and duodenal mucosa, while biopsy specimens were taken for histological examination from the pyloric area, the body of the stomach and the duodendal bulb. Hydrochloric acid secretion was determined using the Kay's aspiration method. Pentagastrin was used to stimulate gastric secretion. Within the group of 79 patients, gastric mucosa inflammation was observed in all patients chronically drinking alcohol and in 72% patients who were the control. Both groups differed considerably with the degree of progression of the inflammatory process, which was estimated with histological examination. Atrophic gastritis was observed only in patients addicted to alcohol. The appearance of atrophic changes pointed to a close relation with the period of addiction. 13 out of 14 people with this type of inflammation had been drinking alcohol for at least 10 years. The studies did not show any relation existing between the percentage of cases with atrophic inflammation and the kind of drinks or the content of ethanol in them. Examinations concerning the secretory function of the stomach showed lower values of hydrochloric acid secretion, both in basic conditions and after pentagastrin stimulation, in patients addicted to alcohol as compared to the control. The results prove that continuous abuse of alcohol predisposes to atrophic inflammation of the gastric mucosa, and the appearance of this type of inflammatory changes is related to the duration of addiction. The longer the addiction, the lower the secretion of hydrochloric acid is.

Wpływ przewlekłego nadużywania alkoholu na błonę śluzową żołądka i dwunastnicy

Alkohol spożywany w niewielkich ilościach nie zagraża zdrowiu, wypijany w dużych dawkach wywiera ujemny wpływ na zdrowie somatyczne i psychiczne. Opublikowano szereg prac dotyczących szkodliwego oddziaływania etanolu na układ nerwowy, krążenia, endokrynny i odpornościowy. Alkohol etylowy może również bezpośrednio uszkadzać śluzówkę przewodu pokarmowego. W dotychczas prowadzonych, publikowanych badaniach oceniano przede wszystkim ostre etanolowe uszkodzenie błony śluzowej żołądka u zwierząt doświadczalnych. Poglądy odnośnie do wpływu przewlekłego spożywania etanolu na żołądek, prezentowane w piśmiennictwie, są bardzo zróżnicowane. Celem pracy była ocena morfologiczna i czynnościowa błony śluzowej żołądka i dwunastnicy u osób przewlekle nadużywających alkoholu. Podjęto również próbę odpowiedzi na pytanie, czy stwierdzone w śluzówce żołądka zmiany wykazują związek z czasem nadużywania alkoholu. Badaniem objęto 79 osób: 61 mężczyzn przewlekle pijących alkohol przez okres 5-37 lat w wieku 17-65 lat oraz 18 osób (9 mężczyzn i 9 kobiet) niepijących nigdy lub bardzo rzadko i w niewielkich ilościach, w wieku 17-59 lat, stanowiących

grupę kontrolną. Pacjentów uzależnionych podzielono na trzy grupy różniące się czasem trwania nałogu (1-5-10 lat; 11-10-20 lat; III-powyżej 20 lat). U wszystkich badanych osób oznaczono wydzielanie kwasu solnego w soku żołądkowym oraz wykonano badania gastroskopowe. Podczas badania gastroskopowego oceniano wygląd śluzówki żołądka i dwunastnicy oraz drogą biopsji pobierano wycinki do badania histologicznego z okolicy odźwiernikowej i trzonu żołądka oraz opuszki dwunastnicy. Wydzielanie kwasu solnego oznaczano metodą aspiracyjną Kaya. Do pobudzenia wydzielania żołądkowego stosowano pentagastrynę. W grupie 79 pacjentów objętych analizą przewlekłe zapalenia śluzówki żołądka stwierdzono u wszystkich pijących alkohol przewlekle i u 72% osób stanowiących grupę kontrolną. Obie porównane grupy pacjentów różnił zasadniczo stopień zaawansowania procesu zapalnego, oceniany badaniem histologicznym. Zapalenie zanikowe obserwowano wyłącznie u osób uzależnionych od alkoholu. Występowanie zmian zanikowych wykazywało ścisły związek z czasem trwania uzależnienia. 13 z 14 osób, u których stwierdzono ten typ zapalenia, piło alkohol co najmniej 10 lat. Przeprowadzone badania nie wykazały zależności pomiędzy odsetkiem przypadków zapalenia zanikowego a rodzajem wypijanych trunków, jak również zawartością w nich etanolu. Wykonane badania czynności wydzielniczej żołądka wykazały niższe wartości wydzielania kwasu solnego, zarówno w warunkach podstawowych jak również po stymulacji pentagastryną, w grupie osób uzależnionych od alkoholu w porównaniu z grupą kontrolną. Wyniki niniejszej pracy dowodzą, że przewlekłe nadużywanie alkoholu predysponuje do rozwoju zapalenia zanikowego błony śluzowej żołądka, a pojawienie się tego typu zmian zapalnych śluzówki wykazuje związek z czasem trwania nałogu. Wraz z wydłużeniem okresu uzależnienia ulega również obniżeniu wydzielanie kwasu solnego.