MORPHOFUNCTIONAL CHANGES OF THE GASTROINTESTINAL TRACT DURING CHRONIC ALCOHOLISM

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Abstract: *chronic alcoholism is a universal risk factor for the occurrence and severe course of chronic diseases of the digestive system, as well as one of the causes of early disability of the young and most able-bodied population. Dangerously high prevalence of somatic alcohol-induced pathology depends on the level of alcohol consumption in the population. With chronic alcohol intoxication, the whole body suffers, and the severity of organ damage may depend on the duration of alcohol abuse.*
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It has been established that in the structure of mortality in recent years, death from chronic alcoholism and acute alcohol intoxication, as well as associated complications, occupies a leading position, second only to mortality from cardiovascular pathologies and malignant neoplasms [1, 9, 14, 17].

Alcoholic beverages have a negative effect on the mucous membranes of human organs [2, 4, 10, 15]. The gastrointestinal tract (gastrointestinal tract) is most at risk, as a result of which the patient experiences constant heartburn acute alcohol poisoning. Often dependent people suffer from disorder and dysbiosis. This is due to the disinfecting properties of alcohol, which disrupts the balance of microflora inside the digestive tract.

Ethyl alcohol (ethanol) belongs, from the point of view of pharmacology, to a group of depressants that depress the central nervous system. Acute ethanol poisoning is usually associated with the intake of ethyl alcohol or beverages containing more than 12% ethyl alcohol. Ethanol is well absorbed in the gastrointestinal tract. Its absorption begins in the oral cavity and esophagus, about 20% is absorbed in the stomach and 80% in the duodenum. The speed of absorption depends on the strength of the drink: weak 10% solutions are absorbed faster, and stronger ones (about 40%) are absorbed more slowly due to the tanning effect of ethanol on the mucous membrane, local vasoconstriction and evacuation disorders. Food masses in the stomach slow down the absorption of alcohol due to its adsorption properties. The main manifestation of liver damage with systematic alcohol consumption is alcoholic liver disease. Alcoholic liver disease (ABP) is a set of morphogenetic manifestations of changes in the structure and functions of the liver that occur when hepatotoxic doses of alcohol are consumed. There are three main forms of ABP - fatty degeneration, alcoholic hepatitis and cirrhosis of the liver. The most frequent liver injury in alcoholic disease is fatty degeneration - a pathological process characterized by a violation of lipid metabolism in liver cells, which eventually leads to the accumulation of fat in the cytoplasm of hepatocytes, mainly triacylglycerides. Fatty liver dystrophy occurs as liver steatosis, hepatosteatosis, fatty hepatosis, fatty liver. Microscopic examination reveals hepatocytes in the pathological material, in the cytoplasm of which fatty inclusions of various diameters are determined. In accordance with this, fatty dystrophy is divided into large-drop and small-drop. With large-drop steatosis, the size of fat vacuoles exceeds or corresponds to the diameter of the cell nucleus, while the cell nucleus itself is displaced, that is, it is located eccentrically. According to the increasing degree of fatty infiltration of hepatocytes, LDP is divided into minor, moderate and pronounced. With small-drop steatosis, a large number of small fat droplets are detected in the hepatocyte, the nucleus remains in the center. It is assumed that the accumulation of fatty inclusions is associated with damage to the mitochondrial apparatus of the cell and activated lipid synthesis. Steatosis is characterized by anisocariosis, expansion of the sinuses, in the lumen of which single neutrophils are detected, however, inflammatory infiltration of the portal tracts is not observed. In pathoanatomic examination, the liver is enlarged in size, the anterior edge is smooth, the surface is smooth, yellowish-brown color on the incision. However, fatty liver dystrophy is not an irreversible process. Stopping alcohol intake without exposure to other toxic factors leads to complete morphological normalization of liver cells with restoration of functional activity. With continued alcoholism, alcoholic hepatitis is the next stage in the progression of alcoholic liver damage. This is a serious disease that develops against the background of a prolonged alcoholic excess, while very often the occurrence of the disease is preceded by fatty degeneration. The main mechanism of damage leading to the development of pathology is the toxic effect of acetaldehyde, in particular, its participation in the launch of lipid peroxidation of cell membranes, which further leads to hypoxia and necrosis. Necrotized elements will provoke the development of the inflammatory process that underlies hepatitis. This pathology is characterized by pronounced microscopic and macroscopic pictures that allow you to correctly diagnose. Microscopic examination of liver tissues reveals small-nodular monolobular cirrhosis of the liver, characterized by variability in the size of hepatocytes, individual necrosis, inflammatory infiltration by neutrophils, mononuclears, pericellular fibrosis, as well as large-drop and small-drop steatosis [2, 8, 16].

A distinctive feature of alcoholic hepatitis is the presence of a large amount of alcoholic hyaline, or Mallory bodies, in the cytoplasm of hepatocytes. Alcoholic hyaline has not only a cytotoxic effect, but also stimulates leukotaxis, has antigenic properties, which leads to the formation of circulating immune complexes [5, 7, 17].

Alcohol contributes to the production of gastric juice and hydrochloric acid, which corrode the walls of the digestive organs and cause ulcers and may eventually cause gastrointestinal cancer. Alcohol clogs the capillaries and thereby disrupts the absorption of vitamins, which is detrimental to the human body. Alcohol affects the gastric mucosa, which can lead to the development of atrophic gastritis. It can cause the development of stomach ulcers, duodenitis (inflammation of the duodenum), the appearance of malignant neoplasms, disruption of the biliary tract. The gastric mucosa contains numerous cells of the diffuse endocrine system (DES), which regulate the processes of digestion and absorption, the severity of inflammation and regeneration, synchronize the work of the entire gastrointestinal tract and change their activity in various pathological conditions [10].

Ethyl alcohol and its main metabolite acetaldehyde have a toxic effect on the intestines. The production of mucin, the main component of mucus covering the inner wall of the small and large intestines, protecting them from damage, decreases. The permeability of biological membranes is disrupted, intestinal mucosa becomes inflamed, edema forms, moisture absorption, absorption of vitamins, micro- and macroelements is upset. Clinically, inflammation is manifested by pain in the lower abdomen, burning, and sometimes intestinal colic [9, 13].

The small intestine performs a mechanical function - pushes the food lump towards the anus. In this organ, all types of nutrients are chemically processed: proteins, fats and carbohydrates. The use of small portions of alcohol can disrupt both peristalsis and digestive processes. The absorption of nutrients slows down and then stops, which leads to exhaustion of the body; digestion of food is disrupted, bloating occurs as a result of excessive gas formation due to digestive disorders; the production of the most important water-soluble vitamins is blocked [9]. The problem of the influence of alcohol on the development of pathology of the gastroduodenal zone is of particular relevance, because the organs of the gastrointestinal tract function as the first barrier to the penetration of alcohol into the body and are the first to experience its negative effects [10].

Clinicians have repeatedly paid attention to the peculiarities of the course of diseases of the gastroduodenal zone in alcoholic illness, their resistance to traditional therapies, a tendency to frequent development of gastrointestinal bleeding and other complications that become fatal for the patient [1, 9, 10].

At the same time, many clinical features and mechanisms of damage to the gastroduodenal zone are not fully understood [8].

The above can be noted the lack of data on the relationship of morphometric indicators of the components of the diffuse endocrine system of the esophagogastroduodenal zone and the stage of alcoholic illness. In connection with the above, the study of this problem seems relevant. The aim of the study is to determine the clinical and morphological features and mechanisms of the development of gastropathies in alcoholic illness.

Materials and methods of research. 69 patients (men) were included in the study aged from 28 to 55 years (average age - 41 ± 0.5 years) with stage II alcohol disease verified by a narcologist. The experience of alcohol consumption averaged 12.24 ± 0.67 years. Based on the data of ultrasound examination of the liver and biochemical parameters of liver function, the patients were divided into 2 groups: group I included 48 patients without liver pathology, group II consisted of 56 patients with alcoholic steatohepatitis (ASG). Exclusion criteria from the study: acute and chronic viral and autoimmune liver diseases; alcoholic psychoses; alcoholic dementia; diabetes mellitus; oncological diseases; cholelithiasis; inflammatory bowel diseases; diseases of the cardiovascular, bronchopulmonary and urinary systems in the decompensation phase; refusal of the patient from examination. The comparison group consisted of 49 patients aged 24 to 47 years (average age – 34 ± 0.5 years), of which 40 men and 9 women who practically do not drink alcohol, have chronic digestive diseases: gastroesophageal reflux disease, endoscopically positive variant – 7 people (14.3%); chronic multifocal atrophic gastritis – 18 patients (36.7%); antral gastritis (superficial and erosive) – 16 patients (32.6%); duodenitis – 4 person (8.2%); gastric and duodenal ulcer in the acute phase – 3 patients (16.2%), in the remission phase - 1 patient (2.0%). All patients underwent a comprehensive examination, including a questionnaire and clinical examination, fibroesophagogastroduodenoscopy (FEGDS) with targeted biopsy of the mucosa of the distal esophagus, antral stomach and duodenal bulb, followed by histological examination of biopsies according to the generally accepted technique with hematoxylin-eosin staining. The degree of contamination of the antral gastric mucosa with Helicobacter pylori was determined histobacterioscopically and using a urease test (CLO test).

Results and discussion. The analysis of the clinical picture of the pathology of the gastroduodenal zone revealed low symptoms and "erasure" of clinical manifestations. As the liver was involved in the pathological process, the clinical symptoms were further minimized, the intensity of the pain syndrome decreased. The syndrome of gastric dyspepsia also varied depending on the presence of alcoholic liver damage: with alcoholic disease without liver pathology - 51.6%, with ASG - 41.7%. It should be noted that patients with alcoholic disease are characterized by the presence of spontaneous vomiting and anorexia. The nature of the pathology of the mucous membrane of the gastroduodenal zone in patients with alcoholic disease According to the results of endoscopic examination in patients with alcoholic disease, depending on the presence or absence of liver

pathology, various variants of damage to the gastroduodenal zone were identified. Acute erosion of the esophagus was not recorded. Signs of chronic gastritis were more often detected in ASG (91.7%) than in alcoholic illness without liver pathology (90.3%). At the same time, signs of atrophy of the gastric mucosa prevailed in patients with ASG, with focal atrophic gastritis accounting for 27.8% of those examined, diffuse (multifocal) atrophic gastritis - 34.9% of patients, chronic catarrhal gastritis was poorly expressed and amounted to only 9.5% of those examined, and signs of hemorrhagic gastritis were also detected – 14 (19.4%) of those examined. In patients with alcoholic disease without liver pathology, signs of catarrhal gastritis prevailed – 18.1% of the examined, focal atrophic gastritis - 23.8%, diffuse atrophic gastritis was detected in 30.3% of the examined, signs of hemorrhagic gastritis - 16.1% of the examined. Due to the fact that duodenogastric reflux is an independent factor of ulceration, it can be assumed that one of the reasons for the more frequent detection of erosive and ulcerative defects of the gastric mucosa in patients with ASG is a higher frequency of registration of duodenogastric reflux. When comparing the clinical manifestations of the gastroduodenal zone lesion with morphological data in alcoholic disease, it was noted that, in general, among patients with ASG, the frequency of asymptomatic variants of the pathology of the gastroduodenal zone was higher than in alcoholic disease without liver pathology. Thus, when the liver is involved in the pathological process, the frequency of subjective manifestations of pathology of the mucous membrane of the gastroduodenal zone decreases. When alcohol is broken down, acetaldehyde is formed, which has a damaging effect on the mucous membrane as a result of the formation of free radicals and the intensification of lipid peroxidation processes.

Conclusions. 1. Clinical signs of gastroduodenal zone lesion in patients with stage II alcoholic disease were largely determined by the degree of involvement of the liver in the pathological process and were characterized by the presence of atypical pain syndrome, as well as spontaneous vomiting without previous nausea and anorexia. 2. In alcoholic steatohepatitis, the endoscopic picture of the gastroduodenal zone is characterized by a predominance of atrophic changes in the gastric mucosa, against which chronic erosions or peptic ulcers are noted without perifocal inflammation.

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