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Duodenogastric Reflux Cause Occurrence of Gastritis and Stomach Ulcers

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Abstract: The purpose of the study is to look into the problem of duodenogastric reflux (DGR) among different categories of patients. And also to identify consequences of the disease. Material and methods. The literature review shows different existing positions on path physiological mechanisms of the DGR and development of reflux gastritis. The main attention is paid on of the articles pointing on the link between DGR and the development of precancerous changes in the gastric mucosa. Comparison of different surveys on how biliary reflux affects H. Pylori displays controversy in their data. Results. Mainly it's because the clinical picture of DGR has no specific symptoms, so generally the diagnosis is based on instrumental methods of research. Conclusion. It is known than different groups of drugs are used in the treatment of the DGR and reflux gastritis. Nevertheless their efficiency has not been studied uniformly. And the generally accepted regimen for this illness seems has not been found yet. It is shown the validity of UDCA prescription for DGR treatment.

Keywords: duodenal reflux, reflux gastritis, intestinal metaplasia, UDCA.

Introductions. Path physiological aspects of duodenogastric reflux. Duodenal contents are a frequent companion and component of gastric juice. It is detected in 20-25% of subjects [1], and in patients suffering from pathology of the upper gastrointestinal tract (GIT), retrograde casting of duodenal contents through the pyloric sphincter becomes a constant phenomenon and is detected in 45-100% of cases [2]. Some authors consider it as a protective reaction of the body in response to the intake of gastric juice with high acid-peptic activity into the duodenum, and emphasize its important role in regulating gastric secretion [8, 9]. Other researchers assign the role of reflux of duodenal contents into the stomach as a pathological agent in the development of diseases of the stomach and duodenum [3]. During the measurement of the concentration of bile acids in the stomach by using a fiber-optic spectrophotometry was shown that the bile in the stomach in healthy patients is present in 37% of the time record: 28% in the daytime and 47% at night [18]. H. Sjövall (2011), exploring the peculiarities of the work of the migrating motor complex in the area of the antral part of the stomach and duodenum, highlights the presence of reflux in the contents of the duodenum, where the bile is missing, but contains bicarbonate and secretory Ig A. The author assigns such reflux a major physiological role in regulating the acidity of the antroduodenal zone [1-2]. Based on the data of daily - intragastric pH - metry, V. S. Volkovetal. (2010) conclude that duodenogastric reflux (DHR) is constantly present in healthy people, occupies about 40% of the time of day, and increases at night. The total number of DGR in healthy people is on average 60-68 times, while in patients with duodenal ulcer (UD) — 27-32 times. It was also found that in patients with duodenal UD, the average duration of reflux is half as long as in healthy people, they are characterized by the absence of its nocturnal activation and restriction mainly to the antral region [8]. In such patients, increased acidity in the body of the stomach is combined with a normally functioning DHR,

which helps to compensate for antral alkalinization. At the same time, patients with duodenal UD have significantly increased acidity in the body, stomach and its antrum, lower frequency and duration of DHR, and no normal circadian fluctuations. According to the authors, the role of DHR in the pathogenesis of such acid-dependent diseases is considered important. diseases such as chronic gastritis and duodenal ulcer are not fully understood and are the subject of further research [9]. Pathogenesis of reflux gastritis. Reflux gastritis (RG) (alkaline reflux gastritis) is considered as a peculiar form of the disease associated with the throwing of the contents of the duodenum into the stomach, which has a damaging effect due to the constant traumatization of the gastric mucosa by refluxate components. Currently, the following mechanisms of development of bile reflux leading to pathological changes in the gastric mucosa have been identified: failureof the sphincter apparatus: 1) duodenal contents can freely reach the stomach and esophagus through the pyloric and lower esophageal sphincters; antroduodenaldysmotor disorder 2)coordination between the antrum, pyloric parts of the stomach and duodenum that control the direction of the flow of duodenal contents; elimination of the natural anti-reflux 3) barrier (after partial gastrectomy and other surgical interventions) [5]. Along with the ant peristaltic mechanism, the factor of increased duodenal pressure and the subsequent development of chronic duodenal obstruction (CDN) are involved in the development of DHR [14]. There are compensated, sub compensated and decompensated stages in the course of CDN syndrome. At the compensated (or latent) stage of CDN, the closing function of the gatekeeper is still preserved. In the sub compensated stage of CDN, hypertension in the duodenum increases, ant peristaltic contractions appear, and stagnation of bile and pancreatic secretions occurs. The duodenum gradually expands, its neuromuscular apparatus is depleted, which leads to relaxation of the pyloric pulp and throwing duodenal contents into the stomach, DHR develops. The stomach expands, and as a result of damage to its mucous membrane by bile acids and lysolecithin, reflux gastritis (RG) is formed. In the stage of decompensation of CDN, hypertension and increased peristalsis of the duodenum are replaced by hypotension and atony, its significant dilation, which leads to the development of a vicious circle: the contents of the duodenum move freely through the gaping pylorus to the stomach and back. Prolonged stasis in the duodenum leads to infection of the contents and causes the development of already chronic duodenitis with progressive atrophy of the mucous membrane, the appearance of erosions and ulceration in it. Among the components of refluxate, the mechanism of damaging action of bile acids is most well studied; they seem to play a major role in the pathogenesis of esophageal and gastric damage in duodenogastroesophageal reflux (DHER). Bile acids contained in refluxhave detergent properties and contribute to the solubilization of lipids in the membranes of the surface epithelium. This effect depends on the concentration, level of conjugation and hydroxylation of bile acids and, very importantly, on the pH of the gastric contents. At low values of the latter, only taurine conjugates and other conjugates damage the mucous membrane., in such conditions precipitate. On the contrary, at high pH values, which is especially typical for the stump of a resected stomach, unconjugated and dihydroxylic bile acids have significantly greater damaging properties than conjugated and trihydroxylic ones [5]. Components of the duodenal contents that damage the esophageal and gastric mucosa are represented not only by bile acids, but also by lysolecithinand trypsin. Conjugated bile acids (primarily taurine conjugates) and lysolecithin have a more pronounced damaging effect on the esophageal and gastric mucosa at acidic pH, which determines their synergy with hydrochloric acid in the pathogenesis of esophagitis and reflux gastritis. Unconjugated bile acids and trypsin are more toxic at neutral and slightly alkaline pH, i.e. their damaging effect in the presence of DHER is enhanced against the background of drug suppression of acid reflux. The toxicity of unconjugated bile acids is mainly due to their ionized forms, which are easier to penetrate through the esophageal mucosa. These data may explain the fact that there is no adequate

clinical response to monotherapy with antisecretory drugs in 15-20% of patients with inflammatory diseases of the esophageal and gastric mucosa [12]. The result of permanent damage to the gastric mucosa by intestinal contents is dystrophic and necrobiotic changes in the gastric epithelium. Morphological changes are stereotyped: foveolar hyperplasia, edema and proliferation of smooth muscle cells in the lamina propria against the background of moderate inflammation. Foveolar hyperplasia is defined as the expansion of mucosal cells. In RG, it covers exclusively the superficial epithelium and this differs from type B gastritis (helicobacter), which is characterized by hyperplasia of not only the superficial, but also the pit epithelium of the gastric mucosa. The epithelium becomes sharply flattened, basophilic, saturated with RNA and almost free of mucus. In epithelial cells, rough vacuolization of the cytoplasm, pycnosis of the nuclei, necrobiosis and necrosis are noted, which is considered the beginning of the formation of erosions. Over time, atrophic changes increase, accompanied by the progression of proliferative processes and the development of dysplasia of varying severity, which increases the risk of malignancy [6]. The information content of morphological diagnosis of changes in the gastric mucosa in DGR is very high. Due to the stereotypical changes in the gastric mucosa, it became possible to develop a DHR index based on histological data. This index was introduced by G. M. Sobalaetal. in 1993, based on a biopsy of the antral part of the stomach or distal part of the stump of the stomach. The index is derived based on the presence and severity of certain histological parameters: edema in the proper plate of the gastric mucosa (indicated as E in the formula below), intestinal metaplasia (IM), chronic inflammation (CI in the formula below) , and colonization with Helicobactor pylori (H. p.) in the stomach. The pathologist assigns a score from 0 to 3 to each histological parameter, which corresponds to the following levels: no parameter, mild, moderate, or high degree of presence, respectively. The formula for determining the biliary reflux index (BRI) was derived from a stepwise logistic regression analysis: According to Sobalaetal., a BRI above 14 indicates DHR (which corresponds to a bile acid level greater than 1 mmol/ L, i.e. above the upper limit of the physiological norm) with 70% sensitivity and 85% specificity. Based on these data, as well as on the fact that other methods of diagnosing DHR do not have high accuracy today, this index is used by a number of authors as a diagnostic criterionin clinical studies [10,1-5, etc.]. Secondary biliary reflux-gastritis. Probably the largest group of patients who develop secondary reflux gastritis (RG) are patients who have undergone cholecystectomy (CE). More and more evidence is emerging that CE, which is the gold standard for treating cholelithiasis (GI), is associated with a high risk of developing various pathologies of the gastric mucosa. Patients after CE havean increased risk of developing RG [1-6], intestinal metaplasia, and evengastric cancer [1-3] as the time after surgery is prolonged. Previous studies [1-5] have shown that between 51 and 89% of patients are after. At the same time, the level of H. pylori infection in such patients remained unchanged both before and after surgery, despite the high level of DHR indicators [45]. Another type of surgery, in which the percentage of changes in the gastric mucosa due to bile reflux is high — is CE resections have a pathological DHR, there is a significant correlation between the level of intragastric bile acids and the severity of DHR. G. Aprea etal. (2012) studied patients after CE during the 6-month postoperative period. It was found that 58% of these patients develop RG, for the diagnosis of which histological BRI was used. Moreover, clinical symptoms in the postoperative period do not correspond to histological data: while BRI was positive in 58% of patients after CE, clinical symptoms in the form of dyspepsia were found only in 41.9% of patients with gastric or small bowel surgery [14, 21]. S. Kuranetal. (2008) studied patients who underwent various manipulations on the biliary tract, such as endoscopic papillosphincterotomy, endoscopic stenting, or choledohoduodenostomy for pathologies not associated with malignant neoplasms. It turned out that all the above procedures are associated with an increased risk of developing DHR. The highest percentage of DGR occurs

in patients after choledohoduodenostomy. At the same time, if in patients who have not been operated on, DHR mainly affects the antral part of the stomach, then in patients who have undergone the above operations on the biliary tract, including after CE, DHR affects both the antral part and the stomach body [16]. Thus, as new studies are conducted, there is more and more data on the occurrence of postoperative complications caused by DHR. Duodenogastric reflux and precancerous changes in the gastric mucosa. Due to the general position that chronic inflammation is a predisposing factor in the development of cancer, as well as the proven fact that in the stomach, inflammation caused by Helicobacter pyloriis associated with the development of stomach cancer, the question arises about the relationship of gastritis caused by DHR with carcinogenesis. To date, there is no direct evidence that DHR is a cause of stomach cancer. The Scandinavian study examined the relationship between DHR and gastric cancer in patients after CE. To study whether long-term non-infectious inflammation, such as DHR, is a predictor of carcinogenesis, K. Fall etal. (2007)conducted a large-scale study of a large cohort of the population after CE. As you know, after such an operation, DHR and reactive gastritis occur with a high frequency. Cancer incidence rates among the entire Swedish population were used as a comparison. Standardized morbidity rates were calculated for both groups. As a result, it was found that patients after CE have an 11% increased risk of distal gastric cancer. Moreover, this relationship was found only in men and within 10 years after surgery. There was no change in the risk of morbidity in women, in addition, in men with an operation duration of more than 10 years, such an association was also not observed. The fact that the relationship changed depending on gender and affected a relatively short period of time, and weakened over time, then, according to the authors, it weakens the causal relationship between CE and gastric cancerover a long period of time [1-5]. A number of studies have investigated the pathogenetic mechanisms of biliary reflux on the gastric mucosa and determined whether DHR can be a factor of carcinogenesis. Since increased proliferation of mucosal cells is observed in DGR, this increases the likelihood of a tumor clone of epithelial cells. Such clones occur, in particular, in cases where chronic damage tothe epithelium is associated with bile reflux [1-6]. A number of other studies have investigated factors that contribute to the development of intestinal metaplasia of the gastric mucosa, since intestinal metaplasia can also be a factor of carcinogenesis. Previously, it has been shown that such metaplasia is associated with chronic inflammation caused by either gastro-esophageal reflux, gastro esophageal reflux disease (GERD), or Helicobacter pylori infection. Another etiological factor is bile reflux. It has been shown that bile reflux is an independent risk factor in the development of intestinal metaplasia both at the level of the cardiac section of the gastric mucosa [1-1] and in other parts of the stomach.

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