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Clinical and Endoscopic Features and Treatment Aspects of Gastroduodenal Pathology in Patients with Chronic Kidney Disease

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Annotation: In recent years, growing attention has been given to the study of gastroduodenal pathology in patients with chronic kidney disease (CKD). This review highlights the prevalence, morphology, diagnosis, and treatment of these disorders. Chronic gastritis, erosive gastroduodenitis, and ulcerative lesions are frequently observed in CKD, particularly in advanced stages, often with few or nonspecific symptoms. Endoscopic findings commonly reveal lymphocytic chronic gastritis and multiple antral erosions. The role of Helicobacter pylori infection remains debated, with variable detection rates among dialysis and pre-dialysis patients. The Maastricht triple-therapy regimen—clarithromycin, amoxicillin, and a proton pump inhibitor—remains effective, though dosing should be adjusted for renal function. Given the risk of complications such as bleeding and perforation, individualized therapeutic and preventive approaches are essential. Further research is needed to refine diagnostic and treatment strategies for gastroduodenal pathology in CKD patients.

Keywords: Chronic kidney disease, gastroduodenal pathology, chronic gastritis, gastric erosion, Helicobacter pylori, esophagogastroduodenoscopy, proton pump inhibitors, gastrointestinal bleeding.

Since the late 20th and early 21st centuries, there has been a significant increase in research on the etiology, pathogenesis, clinical and endoscopic, morphological diagnosis, treatment, and prevention of diseases of the esophagogastroduodenal zone in relation to various internal pathologies [1,2]. At the current stage of gastroenterology development, esophagogastroduodenofibroscopy remains the leading screening method for diagnosing gastroduodenal pathologies. Through direct visual examination, it allows for highly reliable assessment of the mucosal condition of the esophagus, stomach, and duodenum. In addition to the visual determination of the pathological process, this method enables the collection of biopsy material for histological, cytological, and bacteriological studies, which provide accurate determination of the nature, severity, and prognosis of the pathological process, as well as the choice of an appropriate comprehensive treatment strategy.Currently, the universally accepted method for assessing the condition of the gastric and duodenal mucosa remains the endoscopic criteria of the Sydney classification, taking into account its 1996 modification. The method of choice for detecting H. pylori infection remains the serological test combined with the urease test and polymerase chain reaction (PCR), allowing for simultaneous determination of strain sensitivity to antibiotics [1–4]. In addition, several authors, such as A.N. Okorokov (2002), B.I. Shulutko, and S.V. Makarenko (2003), consider the study of gastric secretory function mandatory, as it provides a more comprehensive assessment of the condition and characteristics of the pathological process [5,6]. Many modern researchers recognize the etiopathogenetic significance of factors such as nitrogenous metabolic waste, arterial hypertension, oxygen deficiency associated with anemia, electrolyte imbalance, and acid-base disturbances in the development of multiple organ damage in patients with chronic kidney disease (CKD) [7–9]. Among the internal organ disorders in CKD patients, cardiovascular pathologies hold a leading position and are most often the direct cause of mortality.

As a result of long-term observation of patients with chronic renal failure (CRF), most authors note that the gastric mucosa undergoes changes consistent with chronic gastritis. In such cases, the use of the Sydney classification of chronic gastritis is recommended, which is based on three main criteria — topographic, morphological, and etiological. The clinical presentation of chronic gastritis in patients with CRF does not have specific features; it is usually masked by the symptoms of the underlying disease and manifests as a feeling of heaviness in the epigastric region, nausea, loss of appetite, and occasionally vomiting. Clinicians often interpret these symptoms as typical manifestations of CRF itself, associating them with general disturbances in homeostasis, acid-base balance, and azotemia. Endoscopically and morphologically confirmed chronic gastritis occurs relatively more frequently in patients with CRF than in the general population. Morphologically, degranulation of mast cells, hyperemia, and inflammatory edema of the gastric mucosa are commonly observed.

In patients with advanced CRF, particularly in the terminal stage, there is progressive atrophy with the development of areas of intestinal metaplasia. Less frequently, a picture of granulomatous gastritis with small granuloma formation in the lamina propria of the gastric mucosa is observed. The morphological features of chronic gastritis in *H. pylori*-associated and non-associated forms in the antral region of the stomach are found to be identical. Endoscopic and morphological signs of chronic gastritis were observed in 71.5% of CRF patients, predominantly of the lymphocytic chronic gastritis type, characterized by inflammatory lymphoplasmacytic infiltration of the gastric mucosa. The prevalence of various forms of chronic gastritis in CRF patients reaches 30%. Chronic gastritis with mucosal hemorrhages is frequently noted, whereas hemorrhagic duodenitis is relatively rare. In such cases, episodes of hematemesis and melena may occur, leading to a sharp deterioration of the general somatic condition in patients with chronic kidney disease (CKD) [10,11]. Acute erosions of the stomach and duodenum are often observed in CKD patients, particularly in stages III, IV, and V. The pathogenesis of acute erosion formation is associated with toxic-chemical effects, stressogenic, and psychotraumatic factors, which are common among patients with different stages of CKD.

Many authors have reported that in 75% of patients with acute erosions, duodenogastric reflux was detected, linking it to the reflux of bile acids into the stomach [10,11]. This has been experimentally confirmed in animal studies [12], where the introduction of bile acids into the stomach induced acute erosions. In the acidic gastric environment—especially in patients with CRF—bile acids penetrate deeply into the mucosal layers, causing tissue destruction. Additionally, duodenogastric reflux leads to microcirculatory disturbances with impaired terminal blood flow in the antral mucosa [13,14].Clinically, acute erosions of the stomach and duodenum often present with mild symptoms—moderate epigastric pain, a feeling of heaviness, heartburn, and nausea. However, in 3–5% of patients, hemorrhagic complications such as hematemesis may occur. More often, occult bleeding leads to increasing weakness, fatigue, and anemia, especially in patients with advanced and terminal stages of CKD. The clinical course of chronic erosions of the stomach and duodenum in CKD patients is also generally latent, although some may experience moderate pain, bloating, heartburn, and constipation. Chronic erosions are more frequently found in the stomach, particularly in the antral region (93.7%), and less commonly (6.3%) simultaneously in both the antral and fundic regions. In 77.9% of cases, multiple erosions (more than 2–3) are observed in the mucosa [15,16].

In some cases, endoscopic examination reveals chronic erosions in the pyloroantral region, numbering from 5 to 15, arranged in a chain directed toward the pylorus. Histologically, chronic erosions are characterized by areas of necrosis and desquamation of the surface epithelium, inflammatory lymphoplasmacytic infiltration with neutrophil admixture in the perilesional region, areas of epithelial hyperplasia with dysregulation of proliferative processes, and intense mucus secretion. An increase in subepithelial vascular networks with fibrotic and cystic changes is also noted. Such chronic erosions of the stomach and duodenum heal slowly, sometimes taking over 30 days [15,17]. The prevalence of *H. pylori*-associated chronic erosions in CKD patients remains a subject of debate. In the general population without CKD, *H. pylori* is detected in 60% of patients with chronic erosions. Some authors argue that *H. pylori* is not the main pathogenetic factor in the development of gastroduodenal erosions but may contribute to their chronicity and progression [18,19]. Others emphasize the importance of the local immune response of the gastric mucosa to *H. pylori* infection, particularly to its cytotoxic strains, in the formation of gastroduodenal erosions. Most studies have shown that *H. pylori*-associated erosions are more common in CKD patients undergoing hemodialysis [20–22]. Other studies indicate that *H. pylori*-associated gastric and duodenal erosions occur less frequently (22.6%) in patients with advanced CKD but are more common (37%) in those with preserved renal excretory function. Japanese researchers, studying antibody titers to *H. pylori*, reported that *H. pylori* infection in patients with terminal renal failure was 62.3%, whereas in the early stages of CKD it was slightly lower (53.3%), and among hemodialysis patients it was 39.9% [23]. Despite these conflicting data, most recent studies suggest that erosive and ulcerative lesions of the stomach and duodenum are more frequently detected in patients with terminal CKD [24,25]. The clinical presentation of gastroduodenal ulcerative lesions of the gastric and duodenal mucosa in CKD patients is generally more latent compared to classical peptic ulcer disease. The seasonal and rhythmic characteristics of pain are usually absent, and there are no age or sex differences. Despite the subtler course, gastric and duodenal ulcers—including peptic ulcers—often manifest with sudden acute bleeding or perforation [26,27].

Symptomatic gastroduodenal ulcerations associated with long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs) have been described in detail by Ya.S. Zimmerman et al. (2013). The authors note the latent course of such ulcers, which are more frequently localized in the stomach than in the duodenum and often present suddenly with acute bleeding or perforation. The etiology of these ulcers, unlike that of peptic ulcer disease, is usually known and depends on the dosage and duration of the medication [28]. In CIS countries, nonspecific anti-inflammatory drugs and corticosteroids are widely used in the treatment of CKD patients, especially in stages I–III, which contributes to mucosal damage in the stomach and duodenum, and their prevalence

remains high [29,30]. Gastric and duodenal ulcerations are more common in women, particularly those over 50 years of age. These ulcers often develop without significant pain, though some patients report epigastric discomfort and dyspeptic symptoms. Upper abdominal pain occurs in 20%, nausea and occasional vomiting in 22%, heartburn and belching in 24%, bloating in 21%, and constipation or anorexia in 19% of patients [28]. Painless forms of gastric and duodenal ulcers in CKD patients—especially in severe and terminal stages—are mainly accompanied by nausea, vomiting, heartburn, anorexia, and sometimes melena, along with progressive weakness and anemia. Hidden or acute bleeding symptoms are most often seen in patients with gastric ulcers (68%) and duodenal ulcers (20%) [31,32]. Endoscopic examination of ulcerative lesions reveals marked hyperemia, ulcer craters, microbleeding, and sometimes submucosal hemorrhages. Symptomatic gastric ulcers are observed in 12–30% of cases, while duodenal ulcer localization occurs in 2–19%. There are no definitive distinguishing features between symptomatic ulcers and those of classical peptic ulcer disease, although symptomatic ulcers tend to be multiple and paired. They develop relatively rapidly and heal within 7 to 21 days, without the formation of coarse scars or deformities [33,34]. According to several authors, ulcerative lesions of the gastroduodenal zone are relatively rare in patients with chronic kidney disease (CKD). More commonly, pathology presents as chronic gastritis with erosive changes in the gastric and duodenal mucosa, or their combination. The incidence of gastric and duodenal ulceration among patients with renal insufficiency not receiving hemodialysis therapy was reported at 5.7%, whereas among those undergoing programmed hemodialysis, ulcerative defects of the stomach and duodenum were observed slightly more frequently—**6.67% of cases [35,36].

It should also be noted that the negative influence of uremic symptoms—particularly **azotemia and tissue hypoxia**—on the development of gastric and duodenal mucosal pathology has been demonstrated. With the progression of CKD stages, the frequency of erosive and ulcerative lesions increases. This trend was confirmed in the studies of **S.I. Ryabov (1997) and E.I. Sazonova (2005), who also noted the absence of a direct correlation between CKD stage and the severity of erosive and ulcerative changes in the gastric and duodenal mucosa. Reports on the detection rate of *H. pylori* in patients with CKD and ulcerative lesions of the gastroduodenal zone are inconsistent: some authors found it infrequently, while others reported high prevalence rates [35,37].

The frequency of *H. pylori* detection among CKD patients compared with those suffering from classical peptic ulcer disease also varies considerably. Moreover, several studies question the independent and decisive role of *H. pylori* in the pathogenesis of gastric and duodenal ulcer formation. Recent epidemiological studies on *H. pylori* infection have shown its global distribution, affecting up to 60% of the world’s population. Its prevalence is particularly high in developing countries across Asia, Africa, and Latin America, where by the age of ten, 80% of children are already colonized with *H. pylori*, and infection rates among adults reach 90–95%. In contrast, in developed regions such as Western Europe, the United States, and Japan, *H. pylori* prevalence ranges between 35–50%, while in Russia it is 44–50% among children and 73–91% among adults [38]. Environmental reservoirs for *H. pylori* have not yet been clearly identified, and there is no consensus regarding their existence. Most researchers agree that humans are the primary reservoir, and infection occurs predominantly through fecal–oral or oral–oral transmission. Furthermore, the majority of *H. pylori*-infected individuals—approximately 70%**—remain lifelong asymptomatic carriers [39]. According to some studies, only **30% of infected individuals eventually develop *H. pylori*-associated diseases such as chronic gastritis, chronic gastroduodenitis, and, less frequently, peptic ulcer disease or gastric cancer [40]. The mechanisms behind such diverse clinical manifestations induced by the same bacterium remain a subject of ongoing debate.

Epidemiological studies conducted by V.V. Tsukanov (2004) in Europe, Southeast Asia, and Siberia demonstrated no direct correlation between the prevalence of peptic ulcer disease and the

frequency of *H. pylori* infection [28,38]. Some researchers suggest an ethno-ecological dependence of *H. pylori*-associated gastrointestinal disorders. A review of the literature indicates a high frequency and diversity of gastroduodenal pathologies among CKD patients, particularly during stages of impaired nitrogen excretion accompanied by symptoms of suburemia and uremia. Currently, the most widely accepted and effective treatment strategy for gastroduodenal mucosal pathology is based on the "Maastricht Consensus", developed and recommended by a group of European gastroenterologists [41]. According to this consensus, a strategy of total eradication of *H. pylori*** is recommended. The **standard eradication therapy for treating gastroduodenal mucosal lesions, including erosive and ulcerative changes, is a triple therapy regimen, which includes two antibiotics—**clarithromycin** and amoxicillin**—combined with a **proton pump inhibitor (PPI) such as omeprazole or its analogues (lansoprazole, pantoprazole). The Maastricht Consensus also established a minimum success threshold for eradication therapy of 80%, which should be confirmed by a negative test result four weeks after treatment completion. The optimal treatment duration recommended is 7 days.

The triple eradication regimen includes the following dosages:

- ✓ Clarithromycin – 500 mg twice daily
- ✓ Amoxicillin – 1000 mg twice daily
- ✓ Omeprazole – 20 mg twice daily (morning and evening) for 7 days

To improve the effectiveness of eradication therapy, it is not recommended to exceed these dosages to avoid adverse side effects. Similarly, extending treatment duration to 10 or 14 days does not significantly increase eradication efficacy in most cases. Conversely, shortening the course from 14 to 7 days, while maintaining comparable *H. pylori* eradication rates, reduces the likelihood of side effects and lowers treatment costs. The authors believe that the 7-day treatment regimens are sufficiently effective and economically advantageous. The Maastricht Consensus recommendations suggest a three-week follow-up course with continued administration of omeprazole at a dosage of 20 mg twice daily. These recommendations are intended to consolidate the remission phase following the 7-day course of H. pylori eradication therapy [42, 43]. It should also be noted that the effectiveness of eradication therapy clearly depends on the antibiotic resistance of H. pylori, which leads to a significant reduction in sensitivity with repeated treatment courses. According to various authors, H. pylori resistance rates reach 59.7% for metronidazole, 23.1% for clarithromycin, 26% for amoxicillin, 14% for tetracycline, and 33.3% for doxycycline. These variations are most likely related to the geographical and climatic distribution of H. pylori strains, the duration and frequency of antibiotic use in eradication protocols, as well as the diagnostic methods employed for bacterial detection [44]. Thus, when analyzing widely recognized and highly effective therapeutic methods for gastric and duodenal pathologies, including erosive and ulcerative lesions, it should be emphasized that these recommendations primarily apply to the general principles of treatment for chronic gastritis, erosive gastroduodenitis, and peptic ulcer disease of the stomach and duodenum in the broader sense. Currently, there are no specifically adapted standardized treatment regimens for gastroduodenal pathology in patients with chronic kidney disease (CKD), including those with chronic renal failure.

Practical experience and long-term clinical observations in patients with CKD at various stages demonstrate the feasibility and effectiveness of the aforementioned regimens in treating gastric and duodenal disorders. The literature contains isolated reports emphasizing the necessity and efficacy of basic anti-ulcer drugs in patients with erosions, ulcerative defects, and peptic ulcers associated with CKD [37, 45]. Some authors have reported successful treatment of erosive and ulcerative lesions of the stomach and duodenum in patients with end-stage renal failure using the Maastricht triple-therapy regimen. They administered metronidazole, clarithromycin, and omeprazole in standard dosages for seven days, achieving an eradication success rate of 86.8%

[46]. Other publications describe simplified combination therapy protocols in patients with end-stage renal disease, involving reduced daily doses of amoxicillin, lansoprazole, and plaunatol. Patients with H. pylori–associated gastroduodenal diseases were prescribed amoxicillin (500 mg), lansoprazole (30 mg), and plaunatol (80 mg) once daily for three weeks. Follow-up endoscopic evaluation confirmed complete eradication in 78.6% of patients. Most authors [47] emphasize the need to adjust the dosage, dosing interval, and duration of therapy in CKD patients with H. pylori–associated gastroduodenal disease, taking into account their reduced glomerular filtration rate. In cases of eradication failure, a repeated treatment course is recommended, substituting the antibiotic with metronidazole. Several researchers suggest that H. pylori eradication should be achieved before initiating maintenance hemodialysis or kidney transplantation, particularly in patients with erosive or ulcerative lesions of the stomach and duodenum. This approach is fully justified, since hemodialysis involves the administration of heparin, and post-transplant management requires multiple medications, including corticosteroids. These factors can provoke serious, life-threatening complications such as acute gastrointestinal bleeding or perforation. The high frequency of gastroduodenal lesions and their potential for severe complications in patients with CKD—especially in stages III–IV and terminal stages—necessitate a more detailed consideration of preventive strategies for upper gastrointestinal tract disorders and their complications [45]. Despite the importance of this issue, there are still no clearly defined, unified recommendations for the prevention of gastrointestinal pathology in pre-dialysis, maintenance dialysis, or post-transplant periods. Practical experience and long-term observations confirm the need for the regular inclusion of antacid agents in the complex treatment of CKD patients, as these medications can effectively prevent the development of erosive and particularly ulcerative lesions and their complications in the upper gastrointestinal tract [35, 45].

Thus, summarizing the analysis of clinical and endoscopic features of gastroduodenal lesions in chronic kidney disease, it can be concluded that most authors recognize their oligosymptomatic and latent course, lacking pronounced manifestations of gastritis, gastroduodenitis, erosions, and ulcers of the stomach and duodenum. Data regarding the incidence of gastroduodenal pathology, including H. pylori–associated forms, remain contradictory. In terms of treatment, the majority of authors give preference to the triple-therapy regimen. Further investigation of gastroduodenal pathology in chronic kidney disease will provide an opportunity for a more comprehensive evaluation of the conflicting data available in the literature and will enhance our understanding of the processes occurring in the upper gastrointestinal tract. Moreover, the identification of new theoretical and practical findings will undoubtedly improve the accuracy of establishing detailed clinical diagnoses, contribute to a better understanding of the regional characteristics of disease progression, and simultaneously allow for the optimization of treatment and preventive strategies.

Conclusion: Gastroduodenal pathology in chronic kidney disease is characterized by a predominantly latent clinical course and a high frequency of chronic and erosive changes in the gastric and duodenal mucosa. The variability in *H. pylori* detection rates and the inconsistent correlation between CKD stage and lesion severity highlight the multifactorial nature of these disorders. Current evidence supports the efficacy of the Maastricht triple-therapy regimen for *H. pylori* eradication, provided that drug dosages and treatment duration are adjusted according to renal function. Preventive use of antacid agents and proton pump inhibitors can significantly reduce the risk of erosive and ulcerative complications. Continued investigation into the mechanisms of mucosal injury, microbial factors, and systemic influences in CKD will enhance diagnostic accuracy, refine treatment strategies, and improve patient outcomes.

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