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Helicobacter Pylori and Gastroduodenal Lesions in Chronic Kidney Disease: Clinical Insights and Therapeutic Approaches

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Annotation: Recent decades have witnessed a growing interest in studying gastroduodenal pathology in patients with chronic kidney disease (CKD). The present paper summarizes the clinical, endoscopic, and morphological features, as well as diagnostic and therapeutic strategies for lesions of the esophagogastroduodenal zone in CKD. Chronic gastritis, erosive gastroduodenitis, and ulcerative lesions are commonly observed, especially in advanced CKD stages, often with mild or latent clinical manifestations. Endoscopic findings frequently include lymphocytic gastritis, antral mucosal erosions, and hemorrhagic changes. The prevalence and pathogenic role of Helicobacter pylori infection remain controversial among CKD patients, including those on hemodialysis. Despite variable eradication success rates, the Maastricht triple therapy regimen—clarithromycin, amoxicillin, and a proton pump inhibitor—remains the standard of care, with dosage adjustments required for renal dysfunction. Early eradication of H. pylori before initiating hemodialysis or kidney transplantation is essential to prevent gastrointestinal bleeding and perforation. The absence of CKD-specific treatment guidelines underscores the need for individualized therapeutic and preventive strategies. Further research is necessary to clarify etiopathogenic

mechanisms and optimize diagnostic and treatment algorithms in this high-risk patient population.

Keywords: Chronic kidney disease, gastroduodenal pathology, helicobacter pylori, endoscopy, chronic gastritis, peptic ulcer, maastricht consensus, eradication therapy, hemodialysis, gastrointestinal complications.

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. This method also enables the collection of biopsy material for histological, cytological, and bacteriological studies, allowing for accurate determination of the nature and severity of the pathological process and the selection of an appropriate treatment strategy.

The endoscopic evaluation of the gastric and duodenal mucosa is currently based on the Sydney classification (1996 modification). The preferred method for detecting *H. pylori* remains the combination of serological testing, urease testing, and polymerase chain reaction (PCR) with antibiotic sensitivity assessment [1–4]. Many researchers emphasize the role of nitrogenous metabolic waste, arterial hypertension, anemia-related hypoxia, electrolyte imbalance, and acid-base disturbances in the pathogenesis of multiorgan damage in CKD [7–9]. Cardiovascular diseases occupy a leading position among the causes of mortality in CKD patients. Long-term observations have shown that most CKD patients develop morphological features of chronic gastritis. According to the Sydney classification, chronic gastritis is categorized based on topographic, morphological, and etiological criteria. Its clinical symptoms are often masked by CKD manifestations, such as nausea, heaviness in the epigastrium, and loss of appetite. Endoscopically confirmed chronic gastritis is significantly more common in CKD patients than in the general population. Morphologically, mast cell degranulation, hyperemia, and inflammatory edema of the gastric mucosa are observed. In advanced CKD, progressive mucosal atrophy and intestinal metaplasia develop, while granulomatous gastritis is rare. Endoscopic and morphological features of chronic gastritis are detected in 71.5% of CKD patients, most commonly as lymphocytic chronic gastritis. Chronic gastritis with mucosal hemorrhages is frequent, while hemorrhagic duodenitis is rare and may lead to hematemesis or melena, worsening the patient's condition [10,11].

Acute erosions of the stomach and duodenum are also common, particularly in CKD stages III–V. The formation of acute erosions is linked to toxic, stressogenic, and psychotraumatic factors. In 75% of patients, duodenogastric reflux with bile acid reflux into the stomach is observed, which contributes to mucosal injury [12–14]. Clinically, acute erosions manifest as mild epigastric pain, heaviness, and nausea, but hemorrhagic complications occur in 3–5% of cases. Chronic erosions, mostly located in the antrum (93.7%), heal slowly—over 30 days [15–17]. *H. pylori* infection is detected in 60% of erosion cases in the general population, but its pathogenetic role in CKD remains controversial [18–22]. Epidemiological data show that *H. pylori* infection rates vary geographically—high in developing countries (up to 90%) and lower in developed regions (35–50%) [38]. Despite inconsistent findings, erosive and ulcerative lesions are most frequently reported in terminal CKD stages [24,25]. The clinical presentation of

gastroduodenal ulcerative lesions in CKD patients is generally latent, lacking the typical pain rhythm of peptic ulcer disease. Despite the subtle course, gastric and duodenal ulcers often present with acute bleeding or perforation [26,27]. NSAID-induced ulcers have been described as latent and predominantly gastric in location, with sudden hemorrhagic or perforative onset [28]. In CKD stages I–III, the use of corticosteroids and NSAIDs increases mucosal damage risk, particularly among women over 50 years old [29,30]. Symptoms such as heartburn, nausea, bloating, and weakness are common, whereas pain is less frequent [28]. Painless gastric and duodenal ulcers in terminal CKD often manifest through nausea, vomiting, melena, and anemia. Endoscopic examination reveals ulcer craters, hyperemia, and microbleeding [31–33]. These ulcers develop rapidly and heal within 7–21 days without scarring [33,34]. According to reports, ulcerative lesions are relatively rare in CKD patients, occurring in about 5.7% without hemodialysis and 6.67% with hemodialysis [35,36]. The influence of azotemia and hypoxia increases with disease progression, though direct correlation with CKD stage remains unclear [35,37]. Globally, *H. pylori* infection affects up to 60% of the population, with variable prevalence across regions [38–40]. Despite ongoing debate, eradication therapy remains the most effective approach. The **Maastricht Consensus** recommends a triple eradication regimen including **clarithromycin (500 mg twice daily), amoxicillin (1000 mg twice daily),** and **omeprazole (20 mg twice daily)** for seven days [41–43]. Treatment success is defined by at least 80% eradication confirmed four weeks post-therapy. Antibiotic resistance remains a challenge, with resistance rates of 59.7% for metronidazole and 23.1% for clarithromycin [44].

There are no CKD-specific standardized regimens, but clinical experience supports the safety and efficacy of adjusted triple therapy. Modified protocols using lower antibiotic doses have shown successful eradication in 78–86% of cases [45–47]. Eradication before hemodialysis or kidney transplantation is strongly advised to prevent gastrointestinal bleeding or perforation during anticoagulant or corticosteroid therapy. Given the high prevalence of gastroduodenal lesions in CKD—particularly in stages III–V—preventive measures, including routine use of antacids and proton pump inhibitors, are necessary [45]. However, unified preventive guidelines for pre-dialysis, dialysis, and post-transplant periods are still lacking. In conclusion, most authors agree that gastroduodenal lesions in CKD have an oligosymptomatic, latent course. Triple eradication therapy remains the most effective approach, while further studies are needed to develop CKD-specific diagnostic, therapeutic, and preventive standards.

Conclusion: New coronavirus infection COVID-19 continues to spread across the planet and New causing problems. The effects of the virus on the human body have not yet been sufficiently studied. Coronavirus damages the lungs, kidneys, vascular wall and digestive tract, and as a result the patient develops severe hypoxemia and polyorgan deficiency. Liver function in severe cases violations can also be observed. Fatal in severe acute liver failure cases have also been reported. To determine the degree and causes of liver damage in Covid-19, in this area to study the course of Covid-19 in patients with chronic liver diseases further research is required.

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