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Morphological Changes in the Mucous Membrane of the Oral Cavity in Patients with Gastric and Duodenal Ulcers

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Abstract According to the World Health Organization, 5-10% of the world's population is affected by gastric and duodenal ulcers. Every year, more than three hundred thousand (300,000) peptic ulcer patients undergo emergency surgical procedures. This, in turn, causes complications of patients with stomach ulcers to undergo surgical procedures in the hospital. According to the World Health Organization, the incidence of duodenal ulcer disease is 4 times higher than the incidence of peptic ulcer disease. Duodenal ulcers are more common in men than in women. Changes in the mucous membrane of the stomach and duodenum, including ulcers, are caused by the harmful effects of hydrochloric acid, which is part of gastric juice and aids in digestion. This, in turn, does not affect the initial parts of the digestive organs, that is, the oral cavity. Because it includes changes in the stomach environment, starting from the violation of the diet.

Keywords Peptic ulcer disease, Oral cavity, Gum epithelium, Chronic gingivitis

1. Introduction

Gastric and duodenal ulcers affect individuals of various ages within the local population. Prevalence is linked to lifestyle, environment, diet, and drinking water. Consequently, these gastrointestinal ulcers, like their effects on other organs, impact the oral cavity (the initial organ of digestion), causing various pathological changes. These oral changes may stem from salivary composition, physical and chemical factors, local immunity, the use of hard objects for oral hygiene, dental appliances, and other factors. Teeth and their gingivae are heavily involved in eating, making the gingival epithelium prone to pathological processes. Currently, Helicobacter pylori (H. pylori) is a leading cause of peptic ulcers. H. pylori colonization of the gastric mucosa begins in the oral epithelium. Studies have shown H. pylori presence in the tongue, gingiva, hard palate, and soft palate. The highest concentration of this microorganism is found in gingival tissue. Treatment of H. pylori-associated periodontitis requires a targeted approach, considering its etiology, pathogenesis, and key reaction mechanisms. Therefore, this study investigates the association between gastric/duodenal ulcers and oral conditions.

Purpose of the study:

Through this study, the changes in the mucous membrane of the oral cavity, that is, the histopathological changes in the epithelium of the gums, in peptic ulcer disease.

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2. Materials and Methods

The study comprised 27 patients; 11 with chronic gastric ulcers and 16 with duodenal ulcers. Tissue samples were obtained from the gingiva via biopsy. Histomorphological changes were observed in the gingival epithelium of these samples.

For morphological analysis, gingival biopsies were fixed in 12% formalin, processed through 96% ethanol and xylene, and embedded in paraffin. Sections of 5-6 microns thickness were prepared and stained with hematoxylin and eosin, and using the Van Gieson method.

Histological analysis was performed using an NLCD-307 B Android microscope with a digital screen. Statistical analysis was conducted using Excel-2016.

3. Results and Discussions

Biopsies from the 27 patients with gastric and duodenal ulcers revealed 15 cases of chronic gingivitis and 12 cases of chronic periodontitis.

Diagram showing the prevalence of chronic gingivitis and chronic periodontitis in the 27 studied patients.

Of the 27 patients studied with gastric and duodenal ulcers, 15 showed histopathological changes consistent with chronic gingivitis, while the remaining 12 showed changes consistent with chronic periodontitis. This indicates that 56% of the studied patients had chronic gingivitis and 44% had chronic periodontitis (Diagram 1).

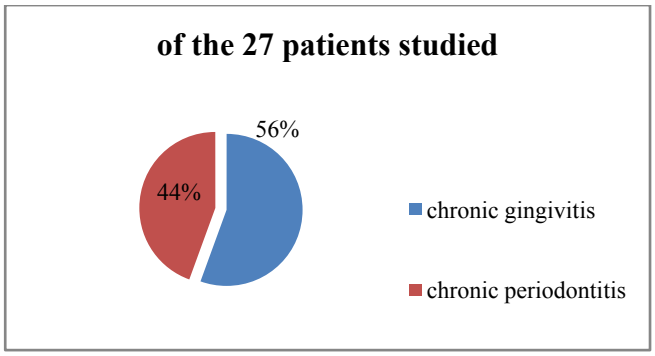

Diagram 1.

Analysis of the age of the 15 patients with chronic gingivitis revealed: 3 in the young age group (14-30 years), 8 in the middle-aged group (31-59 years), and 4 in the elderly group (60 years and older). Analysis of the age of the 12 patients with chronic periodontitis revealed: 3 in the young age group (14-30 years), 7 in the middle-aged group (31-59 years), and 2 in the elderly group (60 years and older).

The study results include an age-based analysis of the identified cases of chronic gingivitis and chronic periodontitis.

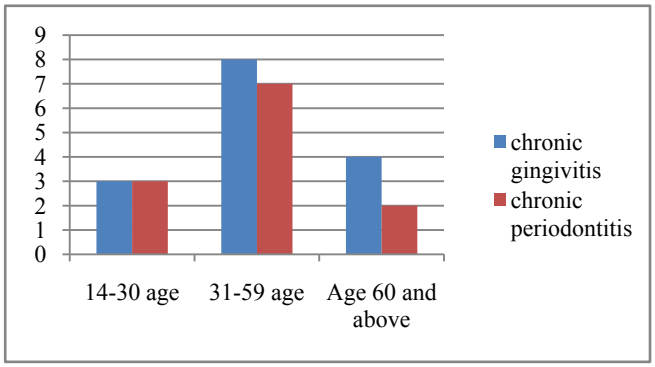

Diagram 2

Analysis results show that the prevalence of chronic gingivitis and chronic periodontitis is similar in the 14-30 age group. In the 31-59 age group, the prevalence of chronic gingivitis is higher than that of chronic periodontitis, and this trend continues in the 60+ age group, where chronic gingivitis is also more prevalent than chronic periodontitis.

Studies are crucial for determining the presence or absence of dentoalveolar deformities and the development of dental defects in patients.

Examination of the obtained biopsies reveals histomorphological changes in the gingival epithelium. In chronic periodontitis, irregular thickening of the granular and spiny layers of the gingival epithelium is observed, along with penetration of the surface epithelium into the underlying connective tissue. In some cases, disrupted cytoarchitecture of the epithelial layer is visible. In patients diagnosed with chronic gingivitis, a higher degree of acanthosis of the gingival surface epithelium was observed. Histomorphological analysis showed that the average thickness of the overlying surface epithelium layer was 748.5 ± 5.0 microns in chronic gingivitis and 341.5 ± 5.0 microns in chronic periodontitis ($P \leq 0.01$).

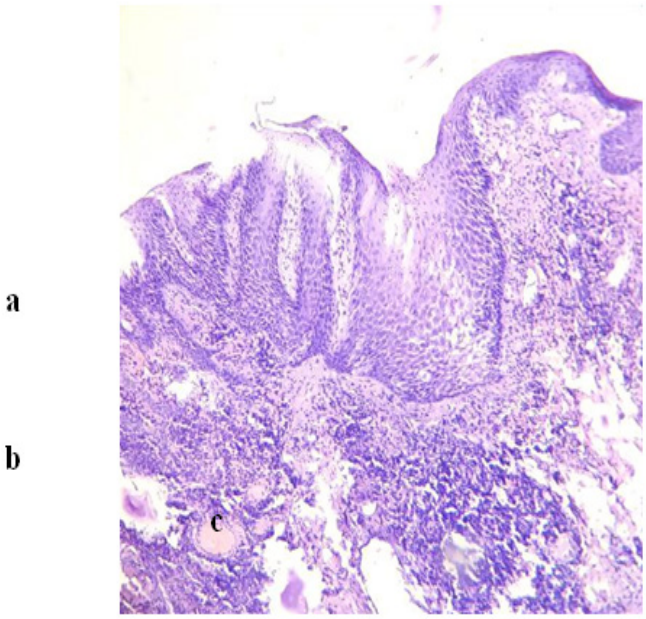

Figure 1. Microscopic view of chronic gingivitis. a- acanthosis of the surface epithelium. b- inflammatory infiltrate. c- marked vascular congestion. Hematoxylin-eosin stain. (NLCD -307 B 10x/0.25)

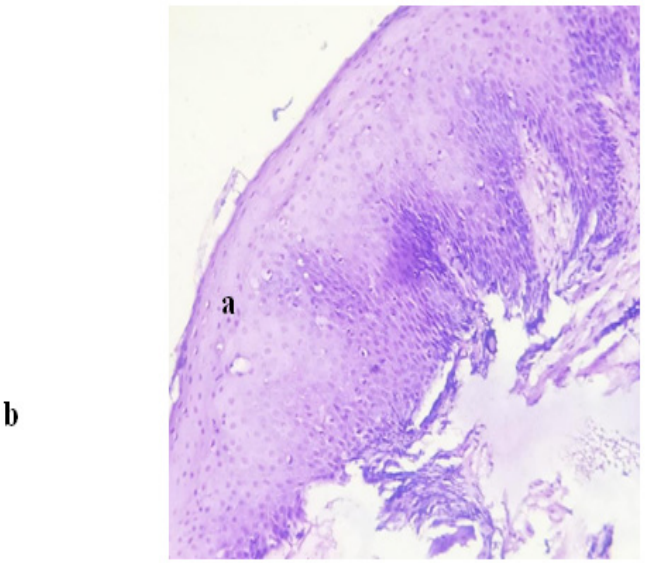

Figure 2. Microscopic view of chronic gingivitis. a- acanthosis of the surface epithelium. b- subepithelial connective tissue with edema. Hematoxylin-eosin stain. (NLCD -307 B 10x/0.25)

The milk's lining epithelial cells exhibit variations in size and irregular arrangement. Widening of intercellular spaces, edema, and the formation of spongy zones are observed in the intermediate area between the basal and suprabasal layers of the epithelium. In the upper regions of the epithelial tissue, zones of hydropic dystrophy are manifested by the appearance of optically bright cells.)

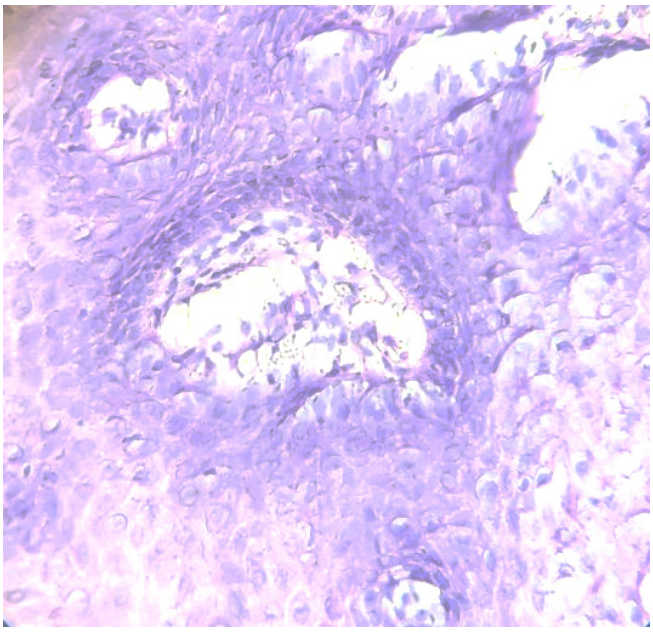

Figure 3. Microscopic view of chronic gingivitis. Intercellular edema, widening of intercellular spaces. Hematoxylin-eosin stain. (NLCD -307 B 40x/0.65)

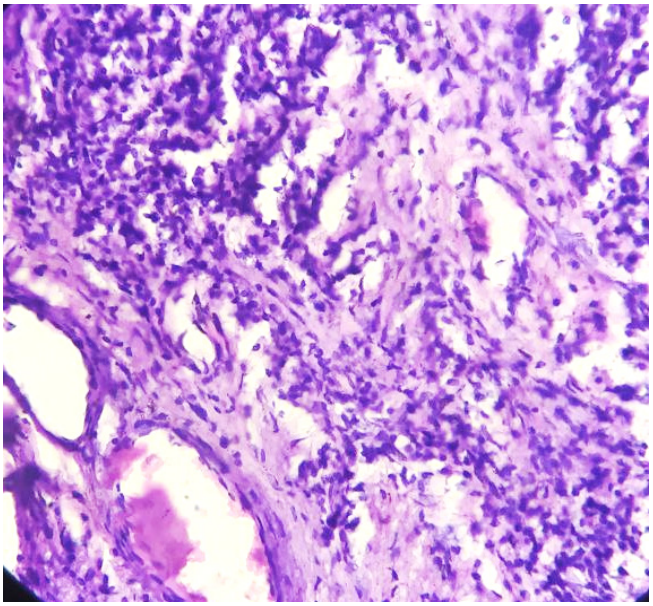

Figure 4. Chronic Periodontitis. Inflammatory infiltrate with lymphocytes and plasma cells. Hematoxylin-eosin stain (NLCD-307B 10x/0.25). Spongiosis (intercellular edema) and widening of the intercellular spaces were observed in the basal and parabasal layers of the gingival epithelium. The oral cavity epithelium is an anatomically significant area. Due to its frequent exposure to mechanical, physical, chemical, and bacterial insults, it is essential to consider morphological changes

4. Conclusions

1. The study results indicate a need for further immunologic and morphometric analysis of the observed changes in the gingival tissue.
2. Morphologic examinations reveal a high incidence of chronic gingivitis and chronic periodontitis in patients with gastric and duodenal ulcers, predominantly affecting the middle-aged population (31-59 years).
3. Among the studied patients with gastric and duodenal ulcers, chronic gingivitis was diagnosed in 56%, while chronic periodontitis was diagnosed in the remaining 44%. This indicates a higher prevalence of chronic gingivitis compared to chronic periodontitis.
4. Almost all examined micropreparations showed inflammatory infiltration, with substrates including plasma, lymphocytes, and macrophages. Inflammation was observed intraepithelially, perivascularly, and in the connective tissue adjacent to the basal lamina and blood vessels. Inflammatory zones were characterized by plasmatic swelling of cells, and in some cases, thinning and spasm of microvascular walls.

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