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Case Report

Periodontal Treatment Improves Dyspepsia Caused by Erosive Gastritis: A Case Report

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Abstract

Dyspepsia is a condition resulting from problems related to poor digestion and changes in the sensitivity of the stomach mucosa. It is usually related to diseases such as gastritis, gastroesophageal reflux disease, peptic ulcer and neoplasms of the upper gastrointestinal tract. Dyspepsia is strongly related to the presence of *Helicobacter pylori* bacteria. Dyspepsia is characterised by pain, pyrosis (heartburn or a burning sensation), anorexia (loss of appetite), abdominal distention, gastric bloating, eructation, flatulence, nausea and a feeling of satiety. Fantogeusia (bitter taste) and halitosis can also be observed. The purpose of this article is to present the case of a patient with dyspepsia caused by erosive gastritis. After periodontal treatment and control of the periodontal disease, an improvement in the symptoms of dyspepsia was reported.

***Keywords:** Dyspepsia; Stomach Diseases; Periodontal Diseases; Periodontal Treatment.*

Introduction

Dyspepsia is a condition resulting from problems related to poor digestion and changes in the sensitivity of the stomach mucosa. It is usually related to diseases such as gastritis, gastroesophageal reflux disease, peptic and duodenal ulcers, and neoplasms of the upper gastrointestinal tract. Dyspepsia is strongly related to the presence of *Helicobacter pylori* bacteria [1-14].

Dyspepsia is characterized by pain, pyrosis (heartburn or burning sensation), anorexia (loss of appetite), abdominal distention, gastric bloating, eructation, flatulence, nausea and a feeling of satiety. Fantogeusia (bitter taste) and halitosis can be observed [1,8,15].

Erosive gastritis is an inflammatory process of the gastric mucosa that causes sores or erosions. The condition can be chronic or acute, and the lesions can affect different areas of the stomach. Erosive gastritis can develop into ulcers, cause bleeding and present symptoms such as dyspepsia [1,15].

Erosive gastritis can also be caused by infection with the bacterium *Helicobacter pylori*, excessive consumption of alcoholic beverages, administration of non-steroidal anti-inflammatory drugs and chronic stress [1].

Erosive gastritis is diagnosed using upper digestive endoscopy with biopsy to determine the presence of *Helicobacter pylori* [1-3,5,10,13,15]. Other procedures can be used, such as microbial culture, PCR, Real Time PCR or urease testing, with varying sensitivity and specificity [3,5,9,11-13,16].

The purpose of this article is to present the case of a patient with dyspepsia caused by erosive gastritis. After periodontal treatment and control of the periodontal disease, an improvement in the symptoms of dyspepsia was reported.

Case Report

A Caucasian male patient, 62-years-old, attended the clinic complaining of periodontal disease.

Clinically, generalized supragingival deposits of dental calculus and plaque were observed. Incisal tooth wear was widely observed, particularly on the maxillary teeth (Figure 1).

Radiographically, bone loss was observed in the alveolar bone crests. Teeth 37 and 48 showed the greatest bone loss, characterized by radiolucent areas adjacent to the tooth roots (Figures 2 and 3).

Regarding medical history, the patient reported dyspepsia caused by erosive gastritis (Figure 4), with a previous diagnosis of *Helicobacter pylori*. The patient is given omeprazole daily to treat the symptoms: pain, heartburn, bloating.

Periodontal treatment was recommended and began with oral hygiene instruction (Figure 5). Subsequent appointments were made for scaling and root planing.

At the supportive periodontal therapy (periodontal maintenance) appointment, a 70% improvement in dyspepsia symptoms was reported, with the withdrawal of omeprazole, indicated by the gastroenterologist. The patient has been under periodontal control for 3 years, with no signs of recurrence of periodontal disease or dyspepsia symptoms (Figure 6).

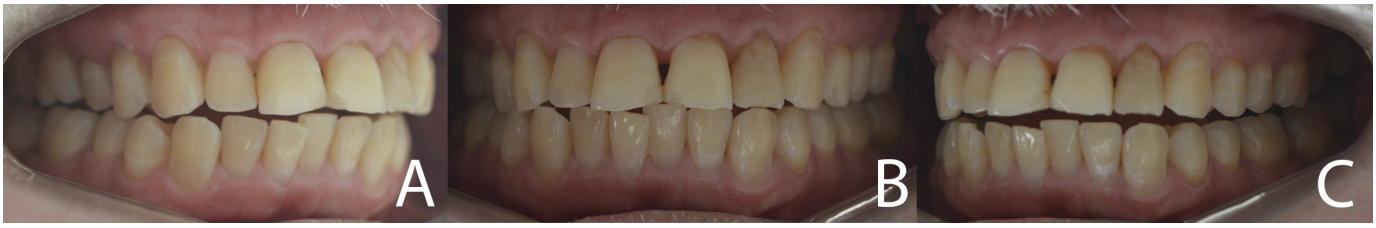

Figure 1: *Generalized supragingival deposits of dental calculus and plaque.*

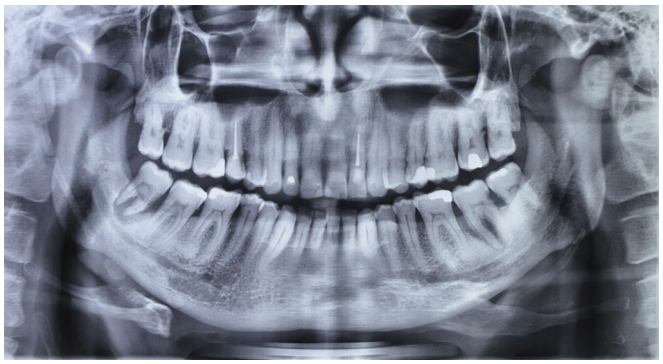

Figure 2: *Bone loss observed on the panoramic radiograph.*

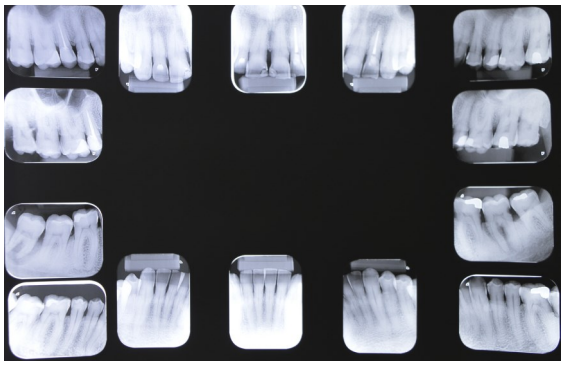

Figure 3: *Generalized bone loss observed observed in the alveolar bone crests (periapical radiograph).*

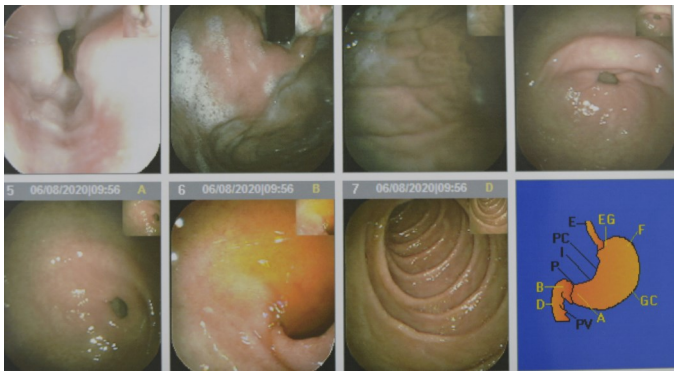

Figure 4: *Upper digestive endoscopy with diagnosis of erosive gastritis.*

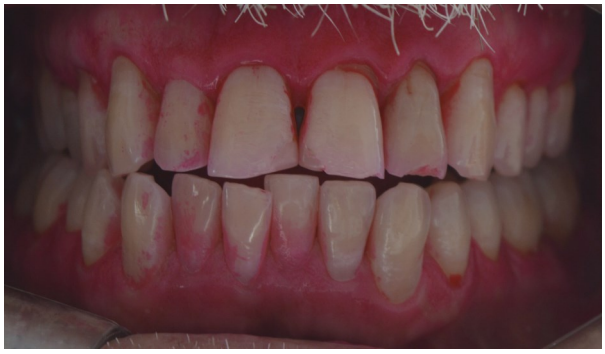

Figure 5: *Oral hygiene instruction.*

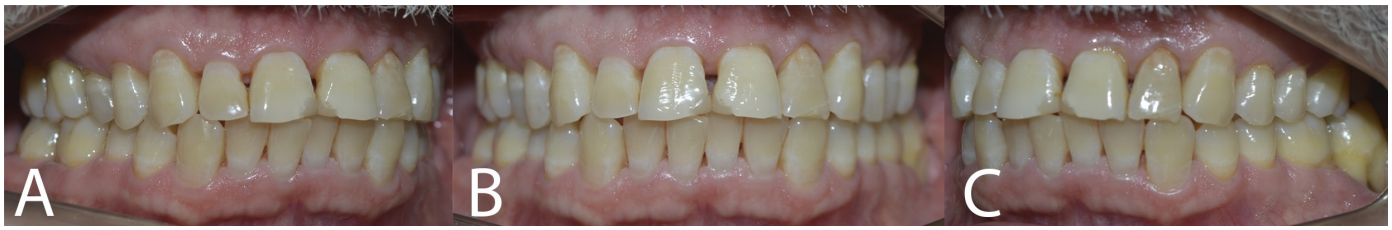

Figure 6: *Supportive periodontal therapy (periodontal maintenance), after periodontal treatment.*

Discussion

The cure of gastric diseases and the prevention of complications from gastric ulcers depend on the successful eradication of *Helicobacter pylori*. However, in the last decade, a reduction in eradication rates has been observed [12]. Considering the combination of antibiotics and other drugs (proton pump inhibitors) recommended to combat *Helicobacter pylori* [1-7,10-15], a reduction in eradication rates is also estimated, since the possibility of selecting resistance to antimicrobials must be assumed [12].

Helicobacter pylori can be present in the oral cavity, in saliva, on the back of the tongue and in the supra- and subgingival dental biofilm. From this perspective, the oral cavity becomes a suitable reservoir for establishing the recurrence of gastric infection [1-3,5-7,9-16].

Dental biofilm is a satisfactory ecosystem in which *Helicobacter pylori* can be protected from the immune response of the host and the action of antimicrobials [1-3,5-7,9-16]. From a microbiological perspective, an association has been observed between *Helicobacter pylori* and *Tannerella forsythia* and other periodontopathogens microorganisms in dental biofilm, which can stimulate their development and favour the course of periodontal diseases [2,5,7,12]. From this perspective, the biofilm must be mechanically destructured in order for antimicrobial therapy to work properly. This occurs through periodontal treatment and mechanical control of dental biofilm, established through oral hygiene [12].

Clinically, unsatisfactory and negligent oral hygiene can also be a risk factor for the presence of *Helicobacter pylori* in the oral cavity [1-3,5-7,9-16].

In the oral cavity, the presence of *Helicobacter pylori* leads to a series of pathological changes: a reduction in lysozyme activity (an indicator of immunity); changes in salivary biochemical parameters, particularly a reduction in oral pH and an increase in salivation, but associated with sialic acid, which increases viscosity and impairs the rheological properties of saliva, and which subsequently promotes xerostomia and the development of some oral pathologies, such as mucositis, caries and periodontal diseases [5].

Periodontal diseases are associated with high levels of inflammatory cytokines that induce systemic diseases and chronic inflammation [7,15]. With regard to diseases of the gastrointestinal tract in particular, the severity of *Helicobacter pylori* infection depends on specific attachment sites in the biofilm, colonization and the genetic of the host and environmental factors. *Helicobacter pylori* colonization also causes the diffusion of neutrophils and mononuclear cells into the lining of the gastrointestinal mucosa, which develops chronic gastritis. The acidic pH of the stomach favours the growth of *Helicobacter pylori*, producing an adequate amount of urease for its survival. *Helicobacter pylori* metabolises urea into carbon dioxide and ammonia, which favours its establishment in the gastrointestinal mucosa. *Helicobacter pylori* colonization also depends on adhesive molecules, particularly to exceptional carbohydrates [1]. H. pylori infection promotes delayed healing of gastric ulcers, inhibition of re-epithelialisation and worsening of the quality of mucosal healing [11].

Patients with periodontitis showed a significantly increased risk of gastric events related to *Helicobacter pylori*, particularly men, the elderly, with or without comorbidities [7,11].

The relationship between chronic gastritis caused by *Helicobacter pylori* infection and periodontal diseases has been widely reported. *Helicobacter pylori* is a Gram-negative, spiral, microaerophilic and opportunistic bacterium that is an aetiological factor in some gastrointestinal diseases [1-7,10-15]. Advanced periodontal disease, characterized by deep periodontal pockets, showed a greater association with *Helicobacter pylori* infection [2-4,6,7,12].

The prevalence of periodontal and gastric co-infection ranged from 1% to 100%. This wide variation in co-infection prevalence rates can be observed by the estimated difference between diagnostic tests, such as microbial culture, urease test or positive biopsy, for example [2,3,11].

The eradication of *Helicobacter pylori* from the oral cavity, residing in the dental biofilm, should be part of the management of gastrointestinal diseases associated with *Helicobacter pylori* [1,6,9-16]. Some studies have observed that gastric eradication coincided with the eradication of *Helicobacter pylori* in the oral cavity, thanks to associated periodontal treatment [2,5,9-13]. On the other hand, other studies have determined that *Helicobacter pylori* is rarely eradicated from the dental biofilm by systemic antibiotic eradication therapy [3,7]. It has also been observed that *Helicobacter pylori* positive patients in the oral cavity have a lower success rate of gastric *Helicobacter pylori* eradication after triple therapy [12]. Thus, the oral cavity and dental biofilm can become a source of future reinfections [1-3,5-7,9-16]. Bohatu et al. (2023) reported an improvement in clinical periodontal parameters after anti-*Helicobacter pylori* therapy, possibly aided by the administration of antibiotics.

In addition, the greatest reduction in gastric *Helicobacter pylori* was observed in patients who maintained satisfactory oral hygiene after receiving combined triple and periodontal therapy [9,11]. In the present report, despite being based on the subjective free expression of the patient, it is also important to consider the adherence to dental biofilm control of the patient after periodontal treatment.

Tsimpiris et al. [8] (2022) did not observe a direct association between periodontitis and *Helicobacter pylori* gastric infection. However, the association between gastric *Helicobacter pylori* infection and the evolution of periodontal disease was observed when they coexisted. The association between gastric and periodontal eradication treatments was better than in patients who received only gastric eradication treatment.

A relationship was observed between poor oral hygiene and gastric precancerous lesions, as well as the presence of *Helicobacter pylori* in gastric histopathology and its association with periodontal disease. Considering the recurrence of gastric infection due to reinfection of *Helicobacter pylori* from the oral cavity, the institution of a multidisciplinary clinical management protocol, associating triple therapy of gastric involvement with mechanical periodontal treatment and chemical antiseptic disinfection [1,2,6,7,11,15].

Gladyshev et al. [16] (2022) assessed the presence of distinct morphological forms of *Helicobacter pylori* in the gingival sulcus and antrum of the stomach using an immunocytochemical assay. The spiral (bacillary) forms of *Helicobacter pylori* were dominant over the coccoid forms in the gastric mucosa, while the coccoid forms were frequently found in the oral cavity, particularly in the gingival sulcus. The colonization of vegetating *Helicobacter pylori* cells, also found in periodontitis, was also observed. The methodology proposed by the authors was valuable in diagnosing *Helicobacter pylori* in the oral cavity, allowing the various morphological forms to be visualised. It was possible to postulate that the coccoid forms in the oral cavity are not the natural site of *Helicobacter pylori*. The spiral forms were related to chronic inflammation. Persistent coccoid forms in periodontal pockets could repopulate and reinfect the gastric mucosa after eradication therapy.

Persistent infection with *Helicobacter pylori* can cause chronic gastritis, and subsequently evolve into atrophic gastritis, metaplasia, dysplasia and, finally, gastric adenocarcinoma [1-7,10-15].

It is important to emphasise that the recommendation for periodontal treatment and stomatological manifestations should be associated with the gastric treatment proposed by the gastroenterologist [9,14,16].

In short, using associated treatment to increase periodontal and systemic (gastric) immunity, as well as improving the ecology of the oral cavity, restoring the microbial and acid-base balance of the oral cavity could reduce cases of recurrence. The aim is to increase the remission time of periodontal disease by eliminating the microbiological reservoir of *Helicobacter pylori* in the oral cavity and subsequently preventing reinfection of the oral and gastric mucosa [16].

Gastritis, gastroesophageal reflux disease and ulcerative colitis can develop symptoms including dyspepsia. Patients with gastrointestinal diseases can be affected by various stomatological changes. Periodontal diseases (gingivitis and periodontitis), caries, tooth erosion (tooth wear), particularly on the palatal and lingual surfaces of the upper and lower teeth, predominantly in the anterior region, mucosal alterations (ulcers, gingival erythema, glossitis, burning mouth) are commonly observed (88%) [17]. Tooth wear resulting from gastric alterations has been widely studied by our research group [18,19], with particular emphasis on the management of these alterations [20,21].

Conclusions

The symptoms caused by gastric diseases can cause discomfort to the patient. Stomatological changes can be caused by these diseases, particularly those related to *Helicobacter pylori* infection. This microorganism can be present in the oral cavity, especially in the dental biofilm, and can be a co-participant in periodontal infection.

Periodontal treatment and the control of dental biofilm through oral hygiene are of the utmost importance, making it possible to prevent and control oral and gastric reinfection with *Helicobacter pylori*. Logically, the patient must be seen by a gastroenterologist. Periodontal treatment can be carried out before or during systemic antibiotic therapy. Associated periodontal treatment can help reduce the administration of antibiotics, reducing costs for the patient, possibly avoiding the chances of selecting antimicrobial resistance and eliminating a source of reinfection.

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