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Local cause of gingival overgrowth. Clinical and histological study

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Abstract

The overgrowth, depending on its extension, has multiple effects on the stomatognathic apparatus: functional disorders (impaired speech), difficulty in chewing and aesthetic problems but can cause significant psychological problems. We proposed this study, motivated by the relative increased frequency of the gum outgrowth, its multifactorial etiopathogeny, but especially from the point of view of the specialist practitioner, by the problems that this pathology raises not only for the functionality of the stomatognathic apparatus but also for the facial esthetics, and especially for future therapeutic attitudes needed to solve the existing pathology at this level. We conducted a clinical study and a histological one. For the clinical study, we selected 74 patients who experienced different degrees of gingival outgrowing associated with fillings, dental caries, fixed prostheses, mobile prostheses, orthodontic apparatus. Thirty gingival fragments from patients with gingival outgrowing were processed by paraffin-embedding histological technique and stained with Hematoxylin–Eosin. The morphological results obtained provide the necessary support for understanding the possibility of developing a therapeutic strategy to prevent or minimize the gum outgrowth by administering antibiotic and anti-inflammatory medications associated with medications, which shall cause the apoptosis of the fibroblasts.

Keywords: gingival outgrowing, oral hygiene index, gingival index, acanthosis, acantholysis.

Introduction

The extension or gingival overgrowth (GO) is the accepted terminology for increasing the size of gum and this is an important aspect of the gum diseases. This multifactorial disorder is developing due to interactions between the host and the various local stimuli [1]. The overgrowth, depending on its extension, has multiple effects on the stomatognathic apparatus: functional disorders (impaired speech), difficulty in chewing and aesthetic problems but can cause significant psychological problems. Despite the many studies conducted, it presents many unknowns, especially regarding the etiopatogenic issues.

Inflammation of the gums caused by specific microbial flora present in the oral cavity under the action of various physical, chemical and biological agents can exacerbate being a determinant of the local gum outgrowth. The morphological aspects of the gingival mucosa due to the action of the plaque, driven by local irritating factors represented by non-compliant or complicated endodontic treatments and under the action of mechanical factors represented by mobile and fixed prostheses or orthodontic apparatus are important for the dental clinician, for adopting a corresponding therapeutic behavior [2–4]. This information is provided by a thorough clinical examination associated with histological examination.

Aim

We proposed this study, motivated by the relative increased frequency of the gum outgrowth, its multifactorial etiopathogeny but especially from the point of view of the practitioner specialist, by the problems that this pathology raises not only for the functionality of the stomatognathic apparatus but also for the facial esthetics and especially for the future therapeutic attitudes needed to solve the existing pathology at this level.

Patients, Materials and Methods

For the clinical study, of the total of 426 patients that presented at the Clinic of Oral Rehabilitation, Faculty of Dental Medicine, University of Medicine and Pharmacy of Craiova, Romania, and private dental office between 2011–2015 were selected 74 patients, who presented gingival outgrowing with different degrees associated with fillings, dental caries, fixed prostheses, mobile prostheses, and orthodontic apparatus. These patients had no systemic diseases and were not under medication. For the patients under the study, there was compiled a personal statement in which there were specified the personal data and local and systemic clinical status and there were conducted:

- The examination of oral hygiene with determination of the simplified oral hygiene index (OHI-S) and O'Leary plaque index;
- The examination of periodontal coating with the determination of the Silness & Löe gingival inflammation index and papillary bleeding index in the survey (papillary bleeding index PBI or Mühlemann & Son index);

▪ Parodontometry with determining the depth of periodontal pockets (probing pocket depth or PPD), the level of gingival insertion (clinical attachment level or CAL), gingival outgrowing index.

For the histological study, the material used was represented by fragments of gingival mucosa from 30 patients who had gingival outgrowing selected from the group of patients selected for the clinical study. They had dental problems (fillings, carious processes) or were carriers of mobile or fixed prostheses or orthodontic appliances. Clinically, they have shown varying degrees of gingival hyperplasia without having systemic diseases (cardiovascular disease, diabetes, collagen diseases, kidney disease, etc.) or to be under medication. Also the selected patients for histological examination were not smokers.

For each patient there was prepared a record of the study, which contained, in addition to personal data and those on the pathology, an informed consent. There were excluded from the study the patients who had suggestive lesions and those who have not given their consent.

The processing of the material for the microscopic examination was done by paraffin-embedding histological technique, the sections obtained being stained with Hematoxylin–Eosin (HE) and Goldner–Szekely (GS) trichrome, within the Department of Histology, University of Medicine and Pharmacy of Craiova, Romania.

Results

The study was conducted on a group of 74 patients (30 men and 44 women), aged between 14 and 70 years. Of these, 73% are non-smokers and 92% from urban areas. Of the patients examined, 36 had grade 2 gingival outgrowing lesions (2.1–4 mm), 26 grade 3 gingival outgrowing lesions (4.1–7 mm) and 12 grade 1 gingival outgrowing lesions (0.5–2 mm) (Figure 1).

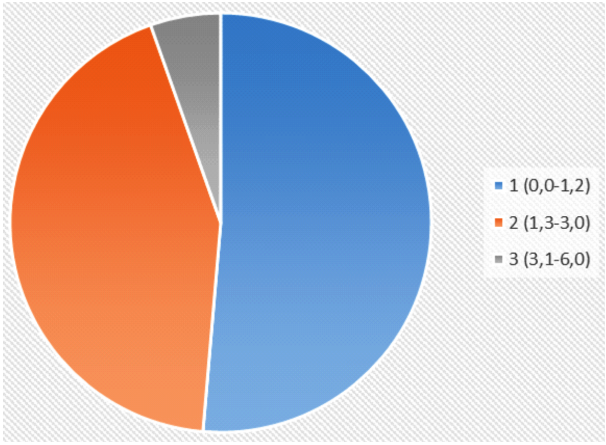

Figure 1 – ***Distribution of cases according to the GO degree.***

These lesions were associated particularly with fixed prostheses and dental caries approximately equal percentage cca. 45% in smaller percentages associated with overflowing fillings, dentures, root residues (Figure 2).

The oral hygiene of these patients is good or satisfactory in most cases (38 and 32 patients), and 14 patients had a mild gingivitis (grade 1 Silness & Löe), 42 show moderate gingivitis (grade 2 Silness & Löe) and 18 severe gingivitis (grade 3 Silness & Löe) (Figure 3).

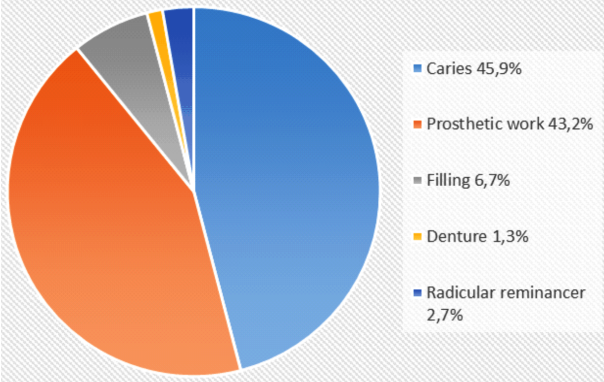

Figure 2 – ***Distribution of cases according to the local cause that determined GO.***

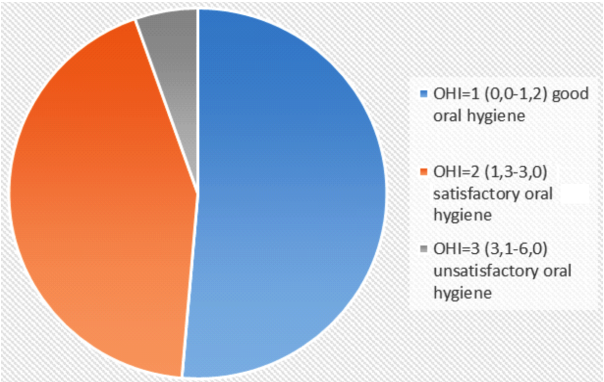

Figure 3 – ***Distribution of cases according to the oral hygiene index (OHI-S).***

Among the local factors that determine the gingival overgrowth for the patients with good or satisfactory oral hygiene, the approximated decay are present at 32 of the patients, the fixed prostheses at 30 patients, fillings at five patients, remains of the root occur at two patients and one patient presents a prosthesis over the implants that caused outgrowing injuries. For those with poor oral hygiene, the major coronary destructions are present in two cases and the fixed prostheses in two other cases.

Depending on the degree of gingival outgrowing, although the majority of patients, regardless of age or area of origin, the indications of oral hygiene emphasizes good or satisfactory hygiene, we found a gingival outgrowing, grade 2 and 3 in cases of important coronary destructions (proximal cavities and root residues) and in cases with fixed dentures and overflowing fillings and moderate and severe gingivitis (Figure 4, A–C).

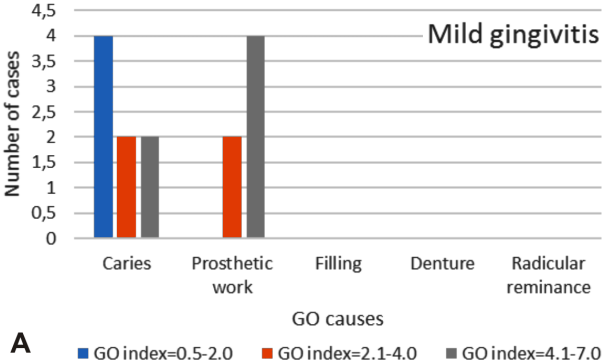

Figure 4 – ***(A) The distribution of the cases according to McGaw index, GO cause and Silness & Löe gingival index.***

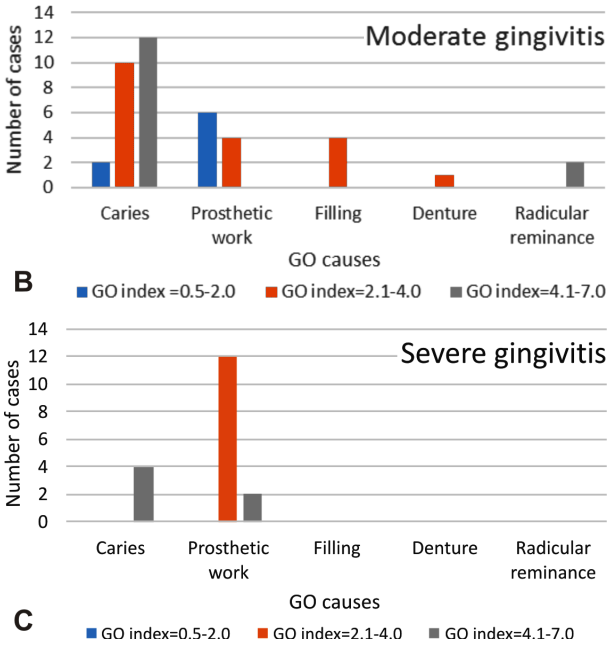

Figure 4 (continued) – ***(B and C) The distribution of the cases according to McGaw index, GO cause and Silness & Löe gingival index.***

The parodontometry conducted on areas affected by local irritation factors, highlights bleeding in line or drop or mass bleeding covering portions of the tooth, false gum pockets due to the outgrowth without significantly changing the level of the gum insertion.

Histological aspects of the local cause of GO

The gingival mucosa changes caused by plaque, under the action of various physical, chemical and biological agents is a key determinant of gum outgrowth of local cause.

The histopathological study revealed the presence the tissue substrate reshuffling. The sections from the batch of patients selected for the histological examination showed various microscopic issues that have concerned both the epithelium and the own lamina of the gingival mucosa. One of the responses to the action of local mechanic and microbial factors consists of changes in the epithelial layer of the gingival mucosa. We have observed, in most of the cases, the hyperplasia of the coating epithelium by its proliferation towards the papillary layer of its own lamina, with the increase in the amplitude of the epithelial ridges, sometimes in the form of digit extensions, sometimes branched (Figure 5A) and junctional each to other, which emphasizes a straight appearance (Figure 5B).

The squamous epithelium presents hyperkeratinization areas or parakeratinization (Figure 5C), quite extensive, sometimes, the keratinization areas are reduced or absent. Gingival epithelium presents acanthosis, because of the multiplication of cells in the intermediate layer, which increases the number of layers of the epithelium and, consequently, increases its thickness; this fact is triggering the epithelial projections at lamina propria, as profound increases. The presence of these epithelial fingerings that reach deep among the chorion palps are causing a papillo-mavirus appearance of the epithelium–corium interface, resulting in an interlocking of the epithelial ridges with the conjunctive palps of the superficial chorion.

The intense proliferation of epithelial cells of the basal layer and spinous is observed as thin cellular cords that deeply penetrate the gingival chorion. The epithelial hyperplasia produced by the proliferation of the epithelial basal layer cell associated with acanthosis, determines the penetration of the epithelial cordons in lamina propria, resulting in the formation, in the cross-section, of the conjunctival–vascular axes. The epithelial layer hyperplasia is not tested by its thickness but especially by the length of the epithelial ridges and the depth to which they penetrate the corium.

The acanthosis phenomenon was often found and it was associated with areas where the cytoplasm of epithelial cells experiences changes, putting on a balloon appearance due to the vacuolization process. There are noted vacuoles of various sizes, sometimes confluent, accompanied by nuclear degeneration small cores, hyperchromatic cells having a spongy aspect (Figure 5, D and E). These cell changes are caused by the prosthetic stress.

The increase in thickness of the epithelium has occurred both on the account of hyperplasia and hypertrophy. In the spinous layer, on some sections there is an acantholysis process due to the breaking of the intercellular thorns (Figure 5F).

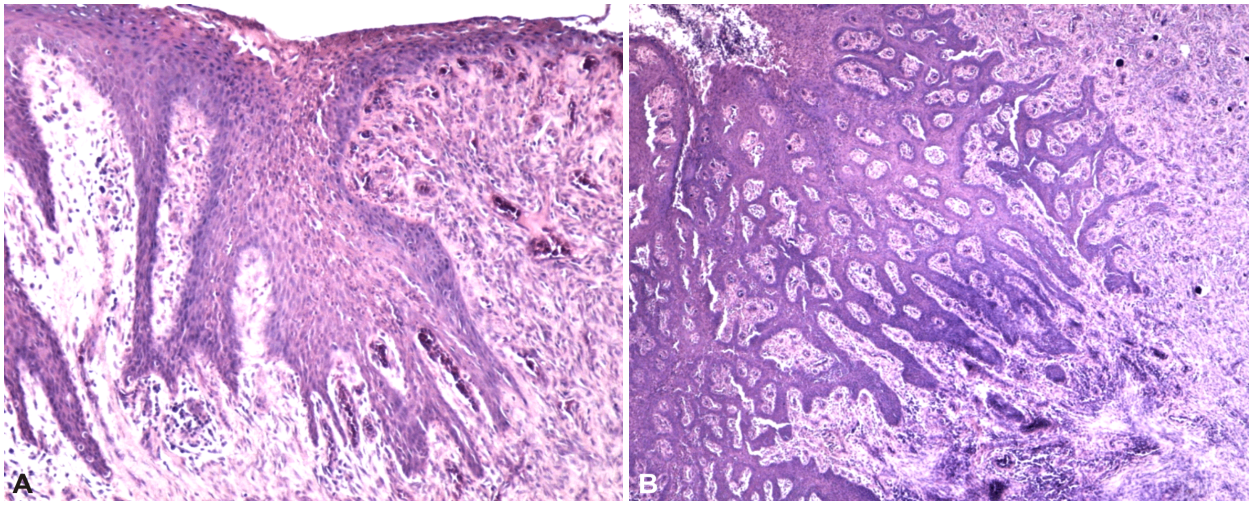

Figure 5 – ***(A) Acanthosis, long epithelial edges, branched at the extremities, HE staining, ×100.***

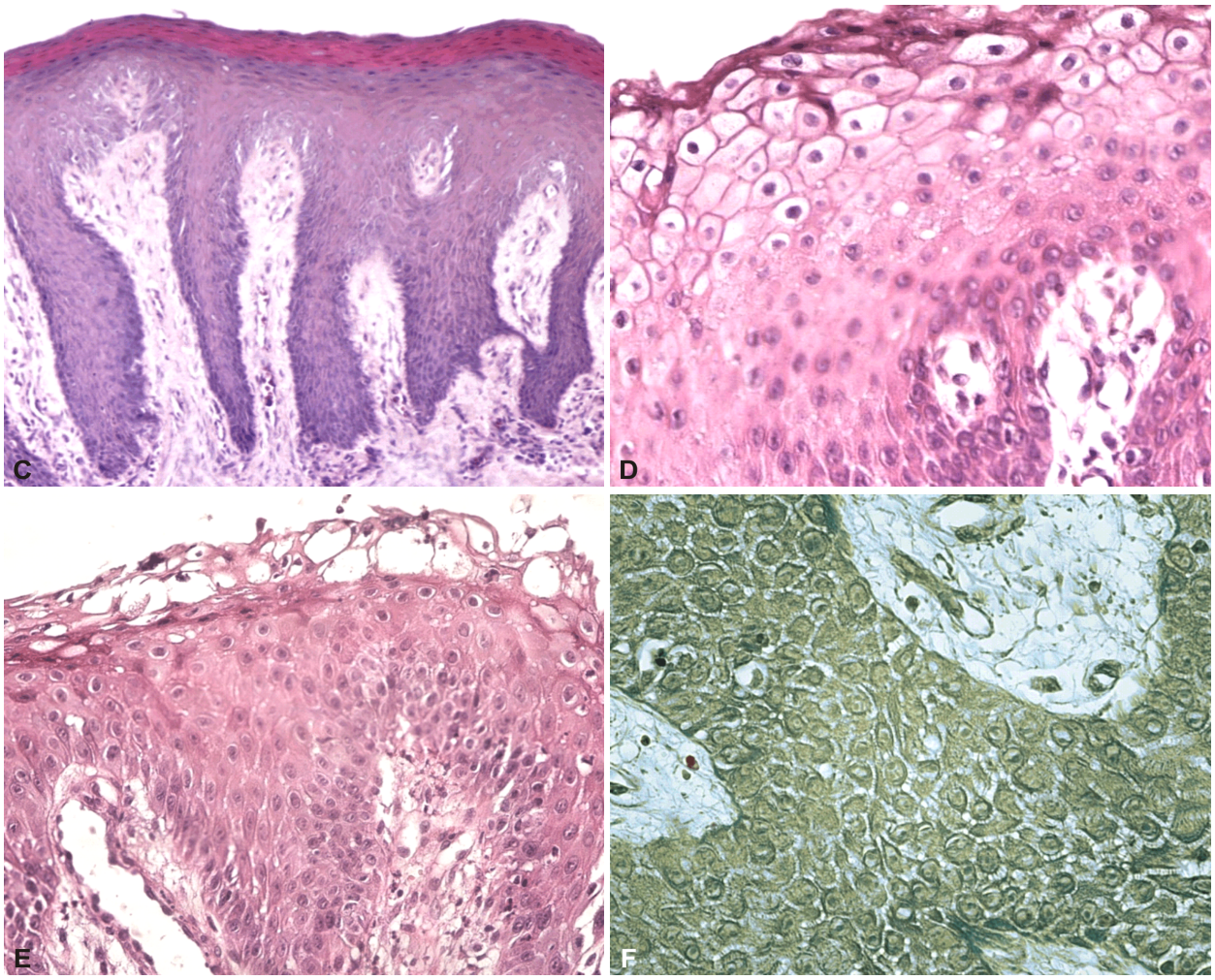

Figure 5 (continued) – ***(B) Epithelial ridges "glove finger" between the fingers with retiform aspect, HE staining, ×100; (C) Acanthosis with parakeratosis, HE staining, ×100; (D) Acanthosis and epithelial cell ballooning, HE staining, ×200; (E) Ballooning of superficial epithelial cells, HE staining, ×100; (F) Acantholysis. Conjunctival vascular islands among epithelial cords, GS trichrome staining, ×400.***

The corium of the gingival mucosa showed significant changes that have interested the cell populations, the blood vessels and the collagen fibrillary component. There is an increased cellularity, represented by fibroblasts, and inflammatory cells. In relatively frequent cases, the inflammatory elements in the gingival mucosa were, in large numbers, predominantly round mononuclear cells lymphoplasmacytic type and macrophage, which indicates the presence of a chronic inflammation, specific and non-specific, and in rare cases, these cellular elements of the chronic inflammation were associated with polymorphonuclear neutrophils, as a sign of activating the inflammation. The inflammatory infiltrate was present in varying degrees, in all the studied cases. The presence of the lymphoplasmacytic and macrophage inflammatory infiltrate indicates both the presence of an immune type defense process and a non-specific defense process, macrophage type.

The areas with a gingival epithelium marked hyperplasia were associated with the occurrence of an inflammatory infiltrate both in the superficial lamina propria and in the deep one. The chronic inflammatory infiltrate was sometimes diffuse (Figure 6A); sometimes, it presented a micronodular appearance (Figure 6B). The chronic inflammatory process had a variable intensity not only from one case to another, but also within the same case, varied from one area to another, suggesting that these changes are triggered by the action of plaque exacerbated by local aggressive factors.

The inflammatory process is associated with an intense proliferation of fibroblasts, which explains the existence of a well-represented fibrillary component, leading to a fibrous at the level of gingival lamina propria. The collagen fibers in the lamina propria are organized as conjunctive bands with an approximately parallel arrangement, perpendicular to the covering epithelium, among the fibrillar bundles being a chronic inflammatory infiltrate. On some sections, it was present a dense thick band of fibrous connective tissue arranged under the skin, demarcating an area of chronic lymphoplasmacytic infiltrated with fewer dilated blood vessels. Other times, the collagen fiber bundles were identified as packets in the gaps caused by them being found chronic inflammatory infiltrate and few blood vessels (Figure 6, D and E).

The number, layout and arrangement of collagen fibers, the inflammatory cellularity (lymphoplasmacytic and macrophage) and non-inflammatory (fibroblasts), the density of blood vessels, are the consequence of exacerbating inflammatory process caused by the plaque under the influence of local conditions created by inadequate treatments.

The density and the distribution of the blood vessels were different. The vascular congestion was present in the majority of the studied cases, especially in the superficial lamina propria and its papillae, justified by the support of the increased needs of an epithelium under proliferation. The vessels present on the sections were typical capillaries, but also neovascularization vessels with turgescent epithelium (Figure 6E). Sometimes, it has been found the presence of a superficial capillary networks, located immediately below the mucosal epithelium, with a few aspects of vessels expansion in the context of a lack of inflammatory elements in these areas. We have also encountered sections with low vascularization, especially where the fibrillar collagen component was more abundant (Figure 6F).

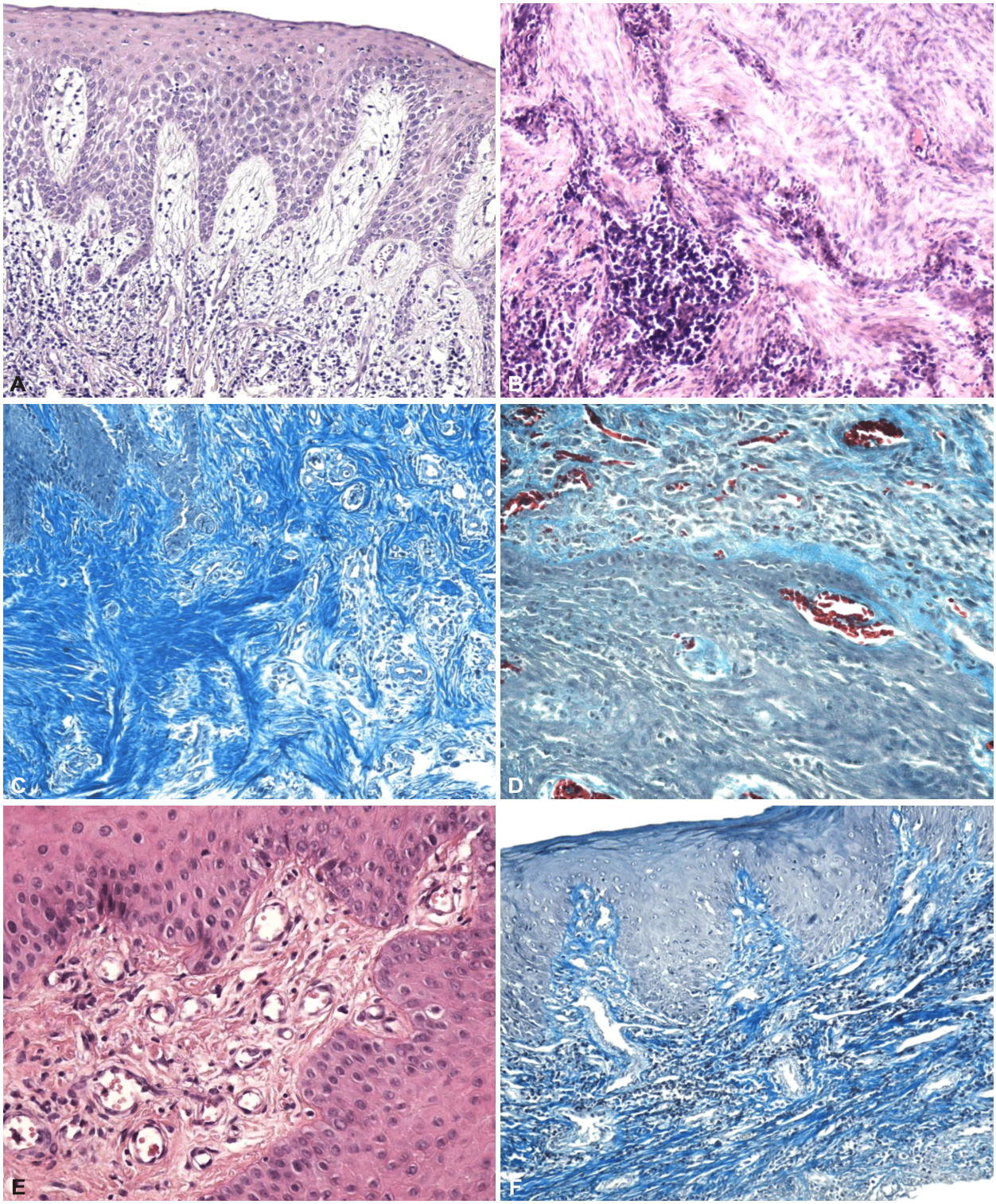

Figure 6 – ***(A) Diffuse chronic inflammatory infiltrate at lamina propria, HE staining, ×100; (B) Chronic inflammatory infiltrate with nodular appearance, HE staining, ×100l (C) Discreet chronic inflammatory infiltrate associated with an intense process of fibrillogenesis, Masson's trichrome staining, ×100; (D) Capillaries among collagen fiber bundles, Masson's trichrome staining, ×100; (E) Many neocapillaries and fibroblasts, subepitelial in the connective papillae, HE staining, ×100; (F) Collagen proliferation with the reduction of the chronic inflammatory process with very few vessels, Masson's trichrome staining, ×100.***

The outgrowing changes may predominantly affect the epithelial structures with reduced conjunctival reaction or may predominantly affect the fibro-conjunctive structures of chorion with minimal reaction from the epithelial component. In our study, we have met especially the involvement of conjunctive fibrillar structures but it was associated with an inflammatory process or diffuse and many vessels.

Discussion

The inflammatory hypertrophies are the most common forms of oral mucosal hypertrophies caused by the chronic inflammation determined by the local irritants. Plaque is the main local factor associated to the gingival hypertrophy, plus the secondary local factors: tartar, incorrect coronary restorations, carious processes, crowded teeth, dental alveolar disharmonies, lack of contact points between the teeth, oral breathing, prolonged use of orthodontic appliances [5–10].

When the adaptive capacity of maxillary dental device to the exaggerated requests is exceeded, it triggers the appearance of pathological phenomena [11, 12]. The periodontium participates in damping of the mechanical stresses from the maxillary dental device.

The gingival overgrowth varies from slight, isolated expansion of the interdental papillae, to the segmental one or to the even spread expansion and that affects one or both arches with diverse etiopatogenesis [13].

For the patients the in the study, at the clinical intraoral examination, we found the presence of some dental injuries ranging from simple approximated caries to deep decay with big coronary destruction or root moieties and the presence of dental restorations represented by overflowing fillings with rough surface, unfinished, fixed prosthetic works with raised edges or poor adaptation to gingival level and, also, in one case we noticed the presence of a mandibular overdenture supported by implants, that, used incorrectly, leaded to the damage of the periodontal tissue near the implant. In all cases, we found a outgrowing of the adjacent gingival tissue.

The overflowing dental restorations, the subgingival placement of fixed dentures margins, incorrectly adjusted dentures or the untreated cavity coronary lesions play an important role in the periodontal pathology. In general, the oral rehabilitation seeks to restore the integrity of a tooth or the entire dental arches, taking into consideration the form, function and aesthetics thereby preventing the periodontal and other dental injuries or the aggravation of the existing ones.

Numerous studies have focused on different aspects of the interaction between the dental restorations or dental caries lesions and the marginal periodontium. The interest was headed toward the edges of the restorations, whether or not they comply with marginal gum tissue, the presence of overflowing dental restorations or rough edges or restorations or caries with gingival edges. The main mechanism, by which these local factors cause the impairment of the marginal periodontium, is the retention of the plaque and food debris, and the alteration of the composition of subgingival microbiota [14–18].

It was used the determination of Silness & Löe gingival index (GI) on the areas corresponding to dental restorations and the contralateral areas with healthy periodontal. These measurements showed a significantly higher GI at the level of the restored areas compared to the contra lateral areas. Poor oral hygiene, favoring the accumulation of plaque is most often associated with gingival inflammation in periodontal disease. However, as evident from our study, the inflammation and the gingival overgrowth may be present under proper oral hygiene conditions or satisfactory, being associated with various local irritative factors that violate the biological space. The improper dental restorations cause the disruption of the ecological balance of the gingival sulcus favoring the development of anaerobic microorganisms responsible for triggering the gram negative periodontal disease and also impeding or hindering the cleaning and self-cleaning [19, 20]. The prevalence of periodontal impairment is according to the restoration type, placement of the edges, the used material [21].

The incorrect restorative treatments with overflowing margins are the iatrogens with different consequences on the gum tissue, long or short term, and the severity of the impairment varies from one country to another and from one practitioner to another [22, 23].

The periodontium, with the bacterial plaque antigens responds through structural changes and by altering the conjunctive tissue, causing inflammatory diseases, gingivitis or periodontitis. The lesions are inflammatory dystrophic degenerative, mixed (inflammatory and dystrophic) and hyperplastic lesions. Therefore, the gingival plaque generates the chronic inflammation, which can lead to gingival hypertrophy.

The primary local factor associated with the gingival overgrowth is the bacterial plaque. Maintaining the homeostasis between the plaque and the host's response is important for maintaining the health. Sickness occurs when the balance becomes unbalanced [24]. The secondary local factors may include gallstones, poor dental restorations, prolonged orthodontics orthodontic braces, cavities, open contacts with food, etc. Several authors are signaling a possible allergic reaction to the metals in the composition of the orthodontic appliances that may cause a gingival outgrowing. The allergic stomatitis in contact with the metals, particularly nickel, is the cause of gingival hyperplasia [25–27]. Other metals are rarely involved in gingival overgrowth, causing individual cases [28]. The fibrous growth associated to gingival fixed orthodontic appliances may be transient and resolves after the orthodontic treatment [29], but there are studies that show that this resolution is not complete [30].

A gingival expanding, which is inflammatory and fibrotic may be an outgrowing that was originally fibrotic and secondary with an inflammation or an extension, which was originally inflammatory, but, later, it became fibrotic [31].

Under certain conditions, GO can progress rapidly, leading to destructive periodontal affections, due to the alteration of the immune response of gum to bacterial plaque [32, 33].

Untreated, chronic gingivitis may evolve with periods of exacerbation and remission; ultimately, they can lead to tooth loss. The evolution of chronic gingivitis to peri-

odontitis depends on the exacerbation of the influence the local factors (bad habits, occlusal trauma, smoking) and the appearance of general diseases in the presence of bacterial plaque. After scaling and root planning in the initial stages, combined with a correct oral hygiene, gingivitis is reversible.

The histological changes found in GO are nonspecific, consisting, regardless of the etiological factor, of fibrosis present in varying degrees associated with an inflammatory process. The level of inflammation, those of fibrosis may vary depending on several factors, local, in particular in relation to the quality of oral hygiene (the presence bacterial plaque), or systemic as well as in relation to the individual susceptibility induced by the genetic factors [34–38].

The changes observed by us on histological sections showed interest in both the epithelium and lamina propria of the gingival mucosa. The epithelium was acanthotic presenting several occasions, especially in the spinous layer, but respected the stratification feature. The surface epithelium presented a parakeratinization process and hyperkeratinization, reflecting a defense mean. The epithelial proliferation determined the deep epithelial grows, penetrating the corium, causing papillomavirus characteristic appearance and coincided with those reported in the literature. The literature considers that the gingival overgrowth is composed of a conjunctive tissue or fibrotic tissue with different degrees of inflammation associating an extension of the epithelial tissue [39]. The quality of oral hygiene and the susceptibility of an individual influence the degree of inflammation, fibrosis and cellularity [33].

Inflammation is a local response of the host to the tissue injury as a response to microbial material invasion and chemical stimuli and/or physical. The inflammation caused by the dental plaque causes a proliferation of conjunctive tissue; the collagenase catabolic ability is overcomed, thus inhibiting the degradation of extracellular matrix, resulting its local accrual. The accumulation of the immune defense cells and of macrophage type is due to the local presence of bacterial antigens and triggers a cascade of events aimed to identify the cellular and humoral events in order to identify the antigenic structures for the purpose of annihilating them. On the examined sections, the most numerous were the lymphocytes. Plasma cells were also in great numbers, with their ability to synthesize and secrete antibodies. They had a random distribution from a preparation to another, depending on the intensity of the inflammatory process. Less and harder to highlight on the usual stained sections were the macrophages, being masked by the lymphoplasmacytic infiltrate. Through their own enzyme system, the macrophages remove the antigens by phagocytic process.

Some authors suggest that there is a definite association between gingivitis and gingival hyperplasia, and that mouth rinses with chlorhexidine can be effective in reducing the severity of gingival inflammation in 59% of the patients [40]. The results of the clinical studies indicate that the gingival inflammation increases the incidence and severity of gingival hypertrophy [41]. This we have also found in the cases we have studied.

The inflammatory process is followed by an intensive proliferation of fibroblasts. The fibroblast is the main cell type in the gingival connective tissue, responsible for forming and turnover of the extracellular matrix.

The presence of fibroblasts was constant in all sections examined with inflammatory cells present in a variable number. Fibroblasts were stimulated, on the one hand by the factors secreted by bacteria and, on the other hand by the immune system cells, such as fibroblast growth factor (FGF). The fibroblasts reaction at the patients in the study was present, associated with the inflammatory cell reaction and vascular reaction, because of local aggressions, which were different in type and intensity. This explains the numeric differences from one case to another and even from one area to another of the fibroblasts.

Lamina propria, on the examined sections consists of collagen fiber bundles hialinizated with a different distribution, dense or lax, associated with an inflammatory infiltrate, especially lymphoplasmacytic. We encountered an increased vascularity, located mostly under the skin with dilated blood vessels, indicating the influence of angiogenic factors associated with mediators of the inflammatory process [1]. Frequently, the periodontal blood vessels had parietal gaps, allowing the blood plasma and figurative elements extravasation, the periodontium being the headquarters of microbleeds.

At the studied casuistry, have been associated with changes in outgrowing prevailing interest for conjunctival–epithelial structures with reduced reaction or fibro-connective structures of the chorion with minimal reaction from the epithelial component. Most commonly, the changes in the lamina propria were very intense, associating the processes with inflammatory fibrillary proliferation in different proportions.

The hyperplasia and hypertrophy processes present in the studied cases, are an adaptation of the cells to mechanical stress caused by local aggressions that are also maintained by the microbial factor. The reactive epithelial hyperplasia at the local aggressions is due to an increased cell proliferation in the basal epithelial layer and deep parabasal. This explains why the process of gingival outgrowing was present at patients that after the clinical studies were found to show an index of oral hygiene and plaque index showing their optimum oral hygiene. In GO triggering, local mechanical action plays an important role in the plaque buildup, sustained by poor oral hygiene contributes to the worsening of the periodontal changes.

The regular professional oral prophylaxis and a good compliance from the patient to maintain a proper oral hygiene are necessary in managing such pathologies. They reflect the importance of the patient's education, motivation and adherence to, during and after the dental treatment.

Conclusions

The gingival overgrowth present the cases studied is an adaptation of cells to the mechanical stress caused by the local aggressions that are also maintained by the microbial factor. In GO triggering, the local mechanical action plays an important role in plaque buildup, sustained by poor oral hygiene contributes to the worsening of the

periodontal changes. At the studied casuistry, have been associated with changes in outgrowing prevailing interest for conjunctival epithelial structures with reduced reaction or fibro-connective structures of the chorion with minimal reaction from the epithelial component. Most commonly, the changes in the lamina propria were very intense, associating the processes with inflammatory fibrillary proliferation in different proportions. The clinical importance of this research is that, the morphological results obtained provide the necessary support to understand the possibility of developing a therapeutic strategy to prevent or minimize the gum outgrowth by administering antibiotic and anti-inflammatory medications associated with medications causing the apoptosis of the fibroblasts.

Conflict of interests

The authors declare that they have no conflict of interests.

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