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Persistent desquamative gingivitis without dental bacterial plaque: What is the diagnosis? What approaches should be used?

Abstract: Gingival lichen planus, lichenoid lesions and gingival pemphigoid are difficult to diagnose because they are confused with chronic inflammatory periodontal disease associated with dentobacterial plaque, especially when there is painful sensitivity that makes oral hygiene difficult. The patient searches, successively, several professionals, in search of a definitive solution. There has to be confidence in the Gingival lichen planus diagnosis and control because it is an autoimmune disease associated with chronic and intense stress. It requires treatment and control of the oral and systemic conditions, besides a coherent interaction of conducts and information between the dental surgeon and the dermatologist. There is a great possibility that it is not classic gingival lichen planus, but a lichenoid lesion of local causes, which may be associated with electrogalvanism. The patient should be clarified about the real dimension of the cancerous potential of these changes, without there being unrealistic exacerbation or negligence of this possibility. The diagnosis and control in the cases of these diseases require, by the dental surgeon, an intense and active participation as protagonist.
Keywords: Gingival lichen planus, Lichen planus. Pemphigoid. Lichenoid lesion. Lichenoid reaction. Gingivitis. Periodontal disease.

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INTRODUCTION

A relatively frequent clinical picture on the mucosa has long been described as "Chronic Desquamative Gingivitis". Its etiology was considered idiopathic or associated with systemic causes such as endocrinopathies. Its therapeutics was hampered by the lack of knowledge of its nature and causes. Over time, cases got more complicated due to the difficulty in maintaining a minimally reasonable oral hygiene.

The name "Chronic Desquamative Gingivitis" was gradually abandoned, as the cause and nature of this disease were, in most cases, a gingival manifestation of lichen planus, or lichenoid lesion, or pemphigoid, which may not affect other areas of the mouth or body.

Lichen planus is a very common mucocutaneous disease and, when gingival, it manifests in an atrophic, erosive and desquamative form. The attached gingiva has reddish or eroded areas, randomly permeated by white micro-areas in the shape of discrete plaques, papules or striae, which very much eventually extend to the alveolar mucosa.

The carrier of gingival lichen planus looks for the professional for aesthetic and functional reasons. The gingiva becomes reddish, sore and bleeding, making oral hygiene difficult, overlapping with chronic inflammatory periodontal disease.

In cases where the final diagnosis is pemphigoid, the delicate superficial detachment of parts of the gingiva is more easily perceived, as layers are detached, especially if the patient pulls them. In other words, the scaly aspect is more evident when it comes to pemphigoid, but, in the vast majority of gingival cases, it is indistinguishable from gingival lichen planus.

In order to highlight the differences and, especially, the lichenoid lesions that can be confused with gingival lichen planus and with pemphigoid, we set out to discuss the subject, highlighting an exemplary clinical case.

WHAT DOES GINGIVAL LIQUEN PLANUS REPRESENT?

Lichen planus is a common autoimmune mucocutaneous disease[2,6] that represents, in general, 20% of the cases seen in stomatology services. The cells of the basal layer of the epithelia are the structures attacked by the immunopathological mechanisms in lichen planus, and they accumulate water in the vacuoles inside, that is, a vacuolization of the basal cells occurs. The lesions in the membrane of the basal epithelial cells lead them to lose control of the entrance of ions and water.

The increasing accumulation of water leads these cells to necrosis by rupturing the membranes. Simultaneously, these cells are induced to apoptosis by the mediators of the autoimmune response, which determines the appearance of corpuscles or apoptotic fragments between the cells of the disorganized basal layer. Also, among the cells of the basal layer are eosinophilic foci of keratin fibrillary proteins, known as hyaline Civatte bodies.

The described disorganization of the basal layer has been known as lichenification, and may separate the epithelium of the oral mucosa with the underlying conjunctive, upon any mechanical action in feeding or hygiene, causing it to detach in small epithelial scales. Hence, in the past, the condition was called chronic desquamative gingivitis.

Below the oral mucosa epithelium - at the interface with the underlying connective tissue, known as lamina propria, and in continuity with the most superficial part of the oral submucosa -, the mononuclear inflammatory infiltrate,

predominantly lymphocytic and macrophagic , is positioned in band and juxtaepithelial . This cellular infiltrate performs the autoimmune phenomena against the epithelium, from which the Langerhans cells (as they are called the intraepithelial macrophages) also participate, and increase their number during the process.

In the epithelium above these areas of attack to the oral mucosa, speed is lost or epithelial desquamation is accelerated, which would renew it around 24 days, changing the cells continuously. These changes promote, in the superficial layer, an increase of the layers of keratin and of cells in the granulosa layer.

When analyzing the white areas with papules, striae and plaques, it may be suggested that, just below, these autoimmune phenomena are occurring. In the reddish and eroded areas, the epithelium is very thin or detached from the underlying connective tissue, exposing it to the acidic mouth environment and full of aggressive components to the directly exposed conjunctiva, promoting stinging and burning. The profile of the patient with oral lichen planus is middle-aged, between 30 and 50 years of age, affecting slightly more women, in the proportion of 3: 2 in relation to men.[2,6]

DIFFERENTIAL DIAGNOSIS OF GINGIVAL LICHEN PLANUS WITH GINGIVAL PENFIGOID

Gingival pemphigoid is an autoimmune mucocutaneous disease much less frequent than lichen planus, and the structure attacked by the immunopathological mechanisms are glycoproteins of the hemidesmosomes of the basal cells of the mucosa. These structures attach epithelial cells to the basement membrane that binds them firmly to the underlying connective tissue of the submucosa. The gingiva is the most affected site by the oral manifestations of pemphigoid.

The fragility of the epithelium-basement membrane junction can disconnect or disengage the epithelium of the oral mucosa with the conjunctiva, promoting an accumulation of liquid between both, with formation of subepithelial bubbles. Any mechanical action, in food or hygiene, may give off small scales of this epithelium or break the subepithelial blisters, exposing the gingival connective tissue with much pain. The clinical picture is very similar and is often indistinguishable from gingival lichen planus.

Below the oral mucosa epithelium, at the interface with the underlying connective tissue, there is a mononuclear inflammatory infiltrate, permeated by eosinophils, which are positioned, at random, in a diffuse or focal form. In the reddish and eroded areas, the epithelium is absent because it has detached from the underlying connective tissue. In the mouth acidic and full of aggressive components, directly exposed connective tissue promotes and justifies so much stinging and burning.

Pemphigoid therapy requires the use of anti-inflammatory drugs and immunoregulators, associated with extreme care with oral hygiene and elimination of parafunctional habits and addictions. Other mucous membranes are affected and/or may be affected, requiring periodic visits to the dermatologist for patient control. The other most affected sites are the genital and respiratory mucosa and the eyes. The integration of the therapy between dentist and dermatologist is fundamental for the patient's control and comfort.

The profile of the patient affected by pemphigoid, as for the age at which it begins, varies between 50 and 60 years old being, therefore, a disease of the elderly; it affects women at a ratio of 2: 1, compared to men. In its etiopathogenesis there is, explicitly, no other component as a cause other than autoimmunity triggered by some still unknown factor.

CAUSES OF GINGIVAL LICHEN PLANUS

The nature of lichen planus is autoimmunity associated with/induced by chronic long-term psychosomatic stress. It is the personal, family, social and financial problems that induce a constant state of alertness in the patient, which promotes discharges and more frequent peaks of suprarenal gland hormones in the blood. Hormones, thus produced in excess in the cortical of the suprarenal glands, promote immunoderegulation.

This imbalance associated with psychosomatic stress almost always promotes disturbances in the functioning of the immune system, greatly increasing the possibility that some of the body's own proteins are mistakenly recognized as strange in different places. In the case of lichen planus, proteins that may be recognized as foreign are on the surface of the basal cells of the skin and/or mucosal coating epithelia, with the consequences already described above.

WHAT IF IT IS A LICHENOID REACTION/ INJURY? WHAT'S THAT?

Out of ten cases diagnosed as lichen planus, seven have oral manifestations and, of these, four have only oral manifestations. Why do many cases have only oral manifestations? Probably some facts may help explain the frequency of oral lichen planus.

The oral mucosa has a great ability to absorb products, ranging from medicines to ions and food components. When drugs or any other product are retained as they pass through the oral mucosa, they may be incorporated into the surfaces of epithelial cells. Within the epithelia, we have the dendritic cells known as Langerhans cells - what are recognizing macrophages and foreign protein processors - which present them to T lymphocytes to trigger and control immunopathological response.

Non-protein chemicals would not be recognized as foreign proteins since they are not of that chemical nature, but may be incorporated into the proteins of epithelial cell surfaces. This incorporation of ions and/or other products into a protein structure may attribute a strangeness to it. In this case, located on the epithelial cell surfaces, membrane proteins are now recognized and fought by the immune system as being foreign.

As described, one may induce autoimmunity against the oral mucosal epithelial cells in the same way as it would occur in lichen planus; when this occurs, this biological and immunopathological reaction should be called the lichenoid reaction. The term "lichenoid " means, literally, similar to lichen planus, and this similarity is clinical, microscopic and immunocytochemical. The lesions produced by this reaction are called lichenoid lesions and are indistinguishable the lesions of oral lichen planus.

LICHENOID REACTION/INJURY: HOW TO DIFFERENTIATE? WHAT TO DO?

The only possible way to differentiate a lichenoid lesion from lesions of lichen planus is from the identification of the causes that induced this type of reaction in the oral mucosa. If they are removed and the case recedes, it is a lichenoid lesion, not lichen planus. These causes may be local and/or systemic.[14]

The metals in the oral cavity suffer a lot of corrosion, with the release of many ions and products that, when entering the oral mucosa, may incorporate, in some patients, the surfaces of the epithelial cells, giving them a protein nature of strangeness, to be recognized by the immune system in the induced autoimmunity process, as described above.

Among these metals, the amalgam of silver and mercury stand out. However, there are many other non-noble metallic alloys that are exposed to the oral environment, in parts of restorative or prosthetic components.[4,11] Many chemical reactions occur in the mouth on the surface of these metals in contact with food components, hygiene products, medicines and others. There is intense release of products into the oral environment, which can be absorbed by the oral mucosa.

There is a way to exacerbate this reaction with other products resulting from superficial corrosion of metals exposed to the oral environment: electrogalvanism. This occurs when electrical microcurrents, imperceptible to patients, are generated when metals with different compositions are united or bound by a chemical medium - the saliva - generating energy that corrodes their surfaces and releases many products that can enter the oral mucosa and be retained in the epithelial cells.

Electrogalvanism resembles the energy generated by the batteries of electronics, which are constituted by several metal cells in its interior, united by a chemical means - that in the case of the mouth, is the saliva. That process was initially reported by the Italian Luigi Galvani and then applied by Alessandro Volta in the form of the electric battery.

For this reason, when a patient has lichen planus or lichenoid lesion, one must analyze the existence of different metals in the oral environment[11]. In general, polishing, repairing restorations and proper oral hygiene will cause the condition to improve considerably or disappear. However, after weeks or a few months, the process restarts. That is why the ideal is to replace these metals with resinous or ceramic materials. When such control is possible, the clinical diagnosis should be of lichenoid lesion, rather than lichen planus.

Lesions induced by lichenoid reactions were identified and associated with restorations and metal crowns,[11] but also with oral hygiene products, such as dentifrices and rinses. Some chemicals used at work, such as photographic developers, induce lichenoid reactions that take on the appearance of contact dermatitis. In cases where metal exposure has been eliminated in the mouth or another cause has been identified and eliminated, the oral mucosa, including the gingival one, usually returns to normal.

Similarly, some medications taken orally may induce such reactions and lichenoid lesions and, in general, this appears in their package inserts as one of the side effects. Among these medicines are drugs like gold, chlorothiazide (antihypertensive and diuretic) and chloroquine (antimalarial). Likewise, may induce lichenoid lesions: methyldopa, allopurinol and non-steroidal anti-inflammatory drugs. Suspension or exchange of the drug, if possible, is very important in the resolution of the clinical picture.

THE CASE REPORT

A 59-year-old white patient sought dental treatment with no symptoms in the oral mucosa and without knowing that he had lesions on the gingival tissues. The attached gingiva was white and interspersed with erythematous areas with a scaly appearance (Fig 1). Some discrete striae and white plaques were prominent and contrasted with the reddish areas (Fig 2 and 3). At the biopsy, the clinician collected material between teeth # 45 and # 46, and the microscopic report was lichen planus, with the recommendation that clinical and etiopathogenic differentiation be made with lichenoid lesions - since, microscopically, they are indistinguishable.

In medical history, the patient reported mercury poisoning and hypertension treated with the con-

tinuous use of “hydrochlorothiazide” and “condesartan cilexethyl”. Hydrochlorothiazide represents one of the systemically administered drugs that may induce lichenoid reactions in the oral mucosa. Mercury represents one of the metal ions that induces lichenoid reactions in its users or in patients who were poisoned by it, as is often the case in gold diggers and miners.

The patient reported, in his history, mercury poisoning. Numerous metallic restorations with very variable composition and age were observed in the patient’s teeth (Fig 4), strongly suggesting the occurrence of electrogalvanism.

In the present case, there were three factors strongly associated with the induction of lichenoid lesions in the oral mucosa: the drug for hypertension, mercury intoxication and an oral situation that strongly suggested the existence of electrogalvanism. The clinical aspects, the patient’s medical history and the microscopic findings make it possible to safely affirm that this was a case of a gingival lichenoid lesion.

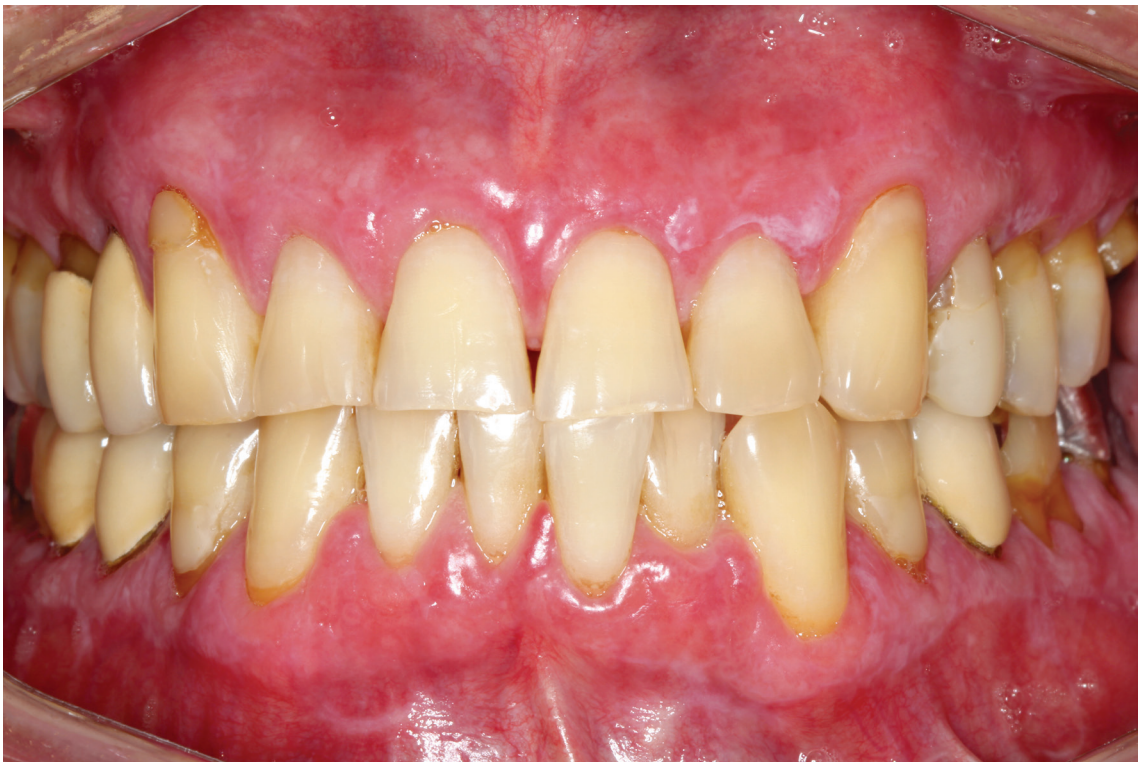

Figure 1: Gingival lichenoid lesions, clinically indistinguishable from gingival lichen planus, with papules, small plaques and delicate striae in the attached gingiva, permeated by erythematous and erosive areas, providing a clinical picture that has been called chronic desquamative gingivitis.

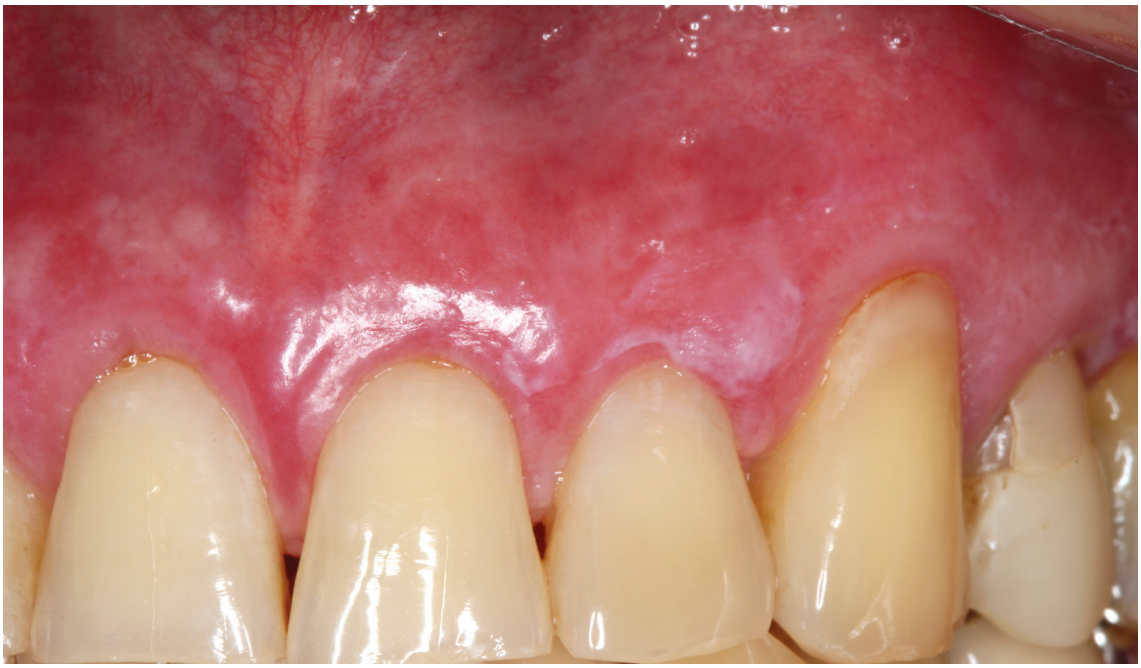

Figure 2: The papules, small plaques and delicate striae in the attached gingiva, permeated by erythematous and erosive areas may be associated with the sensation described as stinging and burning, making oral hygiene difficult.

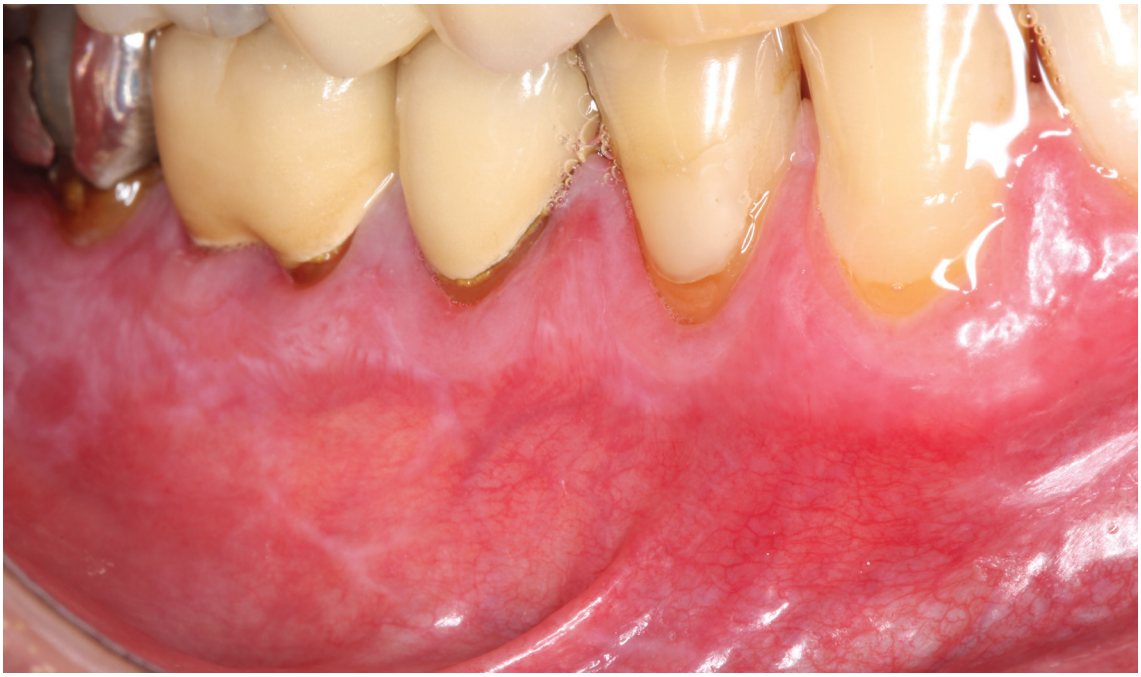

Figure 3: The white streaks are very delicate and, subtly, extend into the alveolar mucosa. Prosthetic components and metallic restorations stand out.

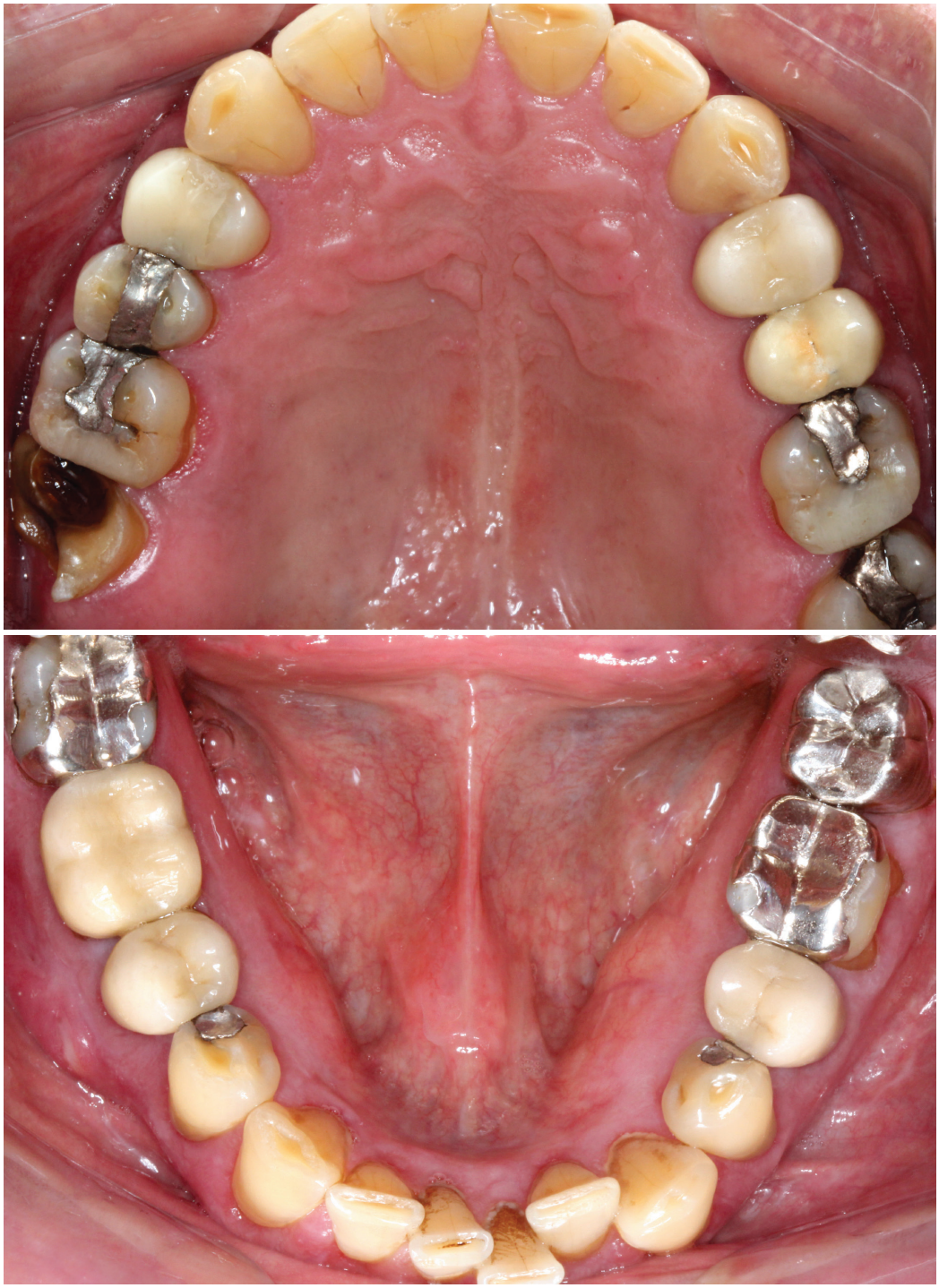

Figure 4: The current state of teeth denotes sharp attrition with bruxism, and many metal restorations and prostheses with different ages and components. This condition is compatible with the oral condition of electrogalvanism, one of the etiological factors associated with lichenoid lesions.

AND IF IT IS LICHEN PLANUS: WHAT IS THE CONDUCT?

Once the possibility of diagnosis of lichenoid lesion has been eliminated, one of the main benefits will be obtained with the control of psychosomatic stress. This goes through the awareness of the patient and family, because it requires changes in lifestyle, the way of facing life and relating to the world. These changes are possible, but very difficult. In general, we should recommend the assistance of professionals such as psychologists, psychiatrists, relatives and even religious. As for lichen planus, it can be said that it represents a somatization of chronic intense mental/psychological stress. Medication control in this setting sometimes requires medications to be prescribed by psychiatrists.

The use of systemic anti-inflammatory drugs - including corticosteroids and non- steroids - has effects on oral and skin lesions while administered.[5,13] The most important will be the control of chronic psychosomatic stress. Alternative treatments can be adopted, emphasizing their limited and specific effects [3,7,12,15]. All such clarifications and information should be thoroughly passed on to the patient and his/her companions.

Oral hygiene should be careful and supervised by the dental surgeon,[10] preferably monthly, and after a few months, it may be done quarterly and then every six months. Lesions are painful and hamper habitual oral hygiene.

LICHEN PLANUS AND LICHENOID LESION: ARE THEY CANCERABLE?

A cancerable condition is a systemic condition that increases the risk of the patient having a malignant neoplasm somewhere in their body. Lichen planus represents a disease that affects the body as a whole, and should be regarded as a cancerous condition; although the increased risk of the carrier is non-measurable, it actually exists.[1,4,9] The patient should be under the professional's control, with periodic visits to both the dental surgeon and the dermatologist.

On the other hand, a cancerable lesion is when, at the site affected by a disease, the risk of establishing a malignant neoplasia, especially a carcinoma - such as in leukoplakia and actinic cheilitis. In lichenoid lesions, the suggested risk is increased, but much less than in a leukoplakia or actinic cheilitis .

These clarifications and information should be provided to patients and their families, as they favor changes and necessary controls.

FINAL CONSIDERATIONS

Gingival lichen planus, lichenoid lesions and gingival pemphigoid are difficult to diagnose because they are confused with chronic inflammatory periodontal disease associated with dentobacterial plaque, especially when there is a painful sensitivity that makes oral hygiene difficult.

In addition to clinically simulating gingivitis and/or periodontitis associated with dentobacterial plaque accumulation, the symptoms of these diseases make oral hygiene difficult, which may overlap the two pictures, making it even more difficult to diagnose accurately and early.

In many cases, the professional tries to control the clinical picture from the maintenance of oral hygiene and palliative measures, causing the patient to search successively several professionals, in search of a definitive solution. For a long time, in the past, gingival lichen planus, lichenoid lesions and gingival pemphigoid were described as chronic desquamative gingivitis. These diseases are almost always indistinguishable, requiring incisional biopsy for a definitive diagnosis.

The professional should be confident in the diagnosis and control of gingival lichen planus, as it is an autoimmune disease associated with chronic and intense stress. It requires treatment and control of the oral and systemic conditions, besides an interaction and coherence of conducts and information between the dental surgeon and the dermatologist.

Among this information, it is possible that the case is not a classic gingival lichen planus, but a lichenoid lesion with local causes, associated or not with electrogalvanism. Regarding the information and conduct of the professionals who control the patient, there is the clarification to the patient about the real dimension of the cancerous potential of these alterations, without there being an unrealistic exacerbation or negligence of this possibility.

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