AN OVERVIEW OF DIFFERENT AUTOIMMUNE DISORDERS LINKED TO DENTAL ENAMEL HYPOPLASIA

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Abstract:
Autoimmune illnesses typically present themselves with oral symptoms as their initial manifestation. As a result of this, dentists play a significant role in the identification of newly emerging autoimmune disorders. As a matter of fact, a timely diagnosis has the potential to significantly contribute to the enhancement of both the quality of treatment options and the quality of life. Having a specialized understanding of the oral symptoms of autoimmune disorders is necessary in order to acquire this expertise. The purpose of this article is to provide a description of the oral manifestations of many autoimmune disorders, including systemic lupus erythematosus, celiac disease, Sjogren syndrome, pemphigus vulgaris, and Behcet disease, among others

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Introduction:

A growing body of research suggests that the incidence of autoimmune illnesses has been steadily increasing over the past few decades [1]. In point of fact, the rise in autoimmune disorders is equivalent to the rise in allergy and cancer pathologies; on the other hand, it has been demonstrated that infections are less common in Western countries [2]. There are several instances in which oral symptoms of autoimmune disease constitute the primary indicator of autoimmune diseases. Dental practitioners are consequently in a position to play a crucial part in the detection process as well as in the subsequent comprehensive therapy. The effectiveness and efficiency of treatment strategies are both improved when they are based on a precise and early diagnosis [3]. As a result, the purpose of this review is to provide an overview of the most prevalent autoimmune illnesses that exhibit themselves through the initial clinical signs and symptoms expressed in the oral cavity. These symptoms are a manifestation of the overall clinical condition.

Review:

Systemic lupus erythematosus, sometimes known as SLE, is a severe and persistent autoimmune inflammatory disease that has a variety of clinical manifestations and an etiopathogenesis that is being investigated. When compared to men, women are eight times more likely to be affected by SLE. It is estimated that the prevalence of SLE varies from 12 to 50 per 100,000 people around the globe, depending on factors such as region and ethnicity [4].

In most cases, systemic lupus erythematosus (SLE) is a chronic and progressive disease, with a dormancy and progression that are mostly consistent and sequential. Despite the fact that it has been hypothesized that the primary involvement is mostly due to cell-mediated immunity and subsequent humoral involvement [5], there are cellular and cell-mediated mechanisms that are engaged in the systemic lupus erythematosus. Through the accumulation of immunological complexes in many organs, an inflammatory response is triggered, which ultimately results in the impairment of organ function that is characteristic of the disease. Activation of type I interferon pathways, malfunction of B and T cells, and the development of antinuclear antibodies were all established to have a role in the pathogenesis of systemic lupus erythematosus (SLE). Anti-DNA antibodies, also known as antinuclear antibodies and deoxyribonucleic acid antibodies, can be discovered in the serum of the patients. Oestrogens are responsible for the maintenance of the proliferation of these antibodies. There have been instances where antilymphocyte antibodies have been observed in some patients. Etiopathogenesis of systemic lupus erythematosus (SLE) takes into account genetic factors as well [5].

Skin damage is the characteristic clinical indication of **systemic lupus erythematosus (SLE)**, and it has been documented in 85 percent of patient cases [5]. Lesions on the skin can range from being simple circle lesions to being multiorgan impairments that could potentially be lethal. Oral discoid lesions are one of the more common manifestations of the disease, and severe erythema on the surface of the skin that is exposed to light is the skin lesion that occurs the most frequently. The so-called malar rash, also known as butterfly rush, is found on the nose and cheeks, and the erythema can even be observed on the tips of the fingers. There is a high incidence of recrudescence throughout the healing process of these lesions, which are characterized by a core scar and the surrounding area. In systemic lupus erythematosus, the joints, skin, muscles, eyes, lungs, central nervous system, and kidneys are all affected by the disease. The progression of systemic lupus erythematosus is commonly linked to the development of joint conditions such as arthralgia and arthritis. An asymmetric appearance and a migratory pattern of behavior are characteristics of the arthralgia. There is a great deal of variety in the geography of joint presentations. In point of fact, it has the potential to be of interest to any articular surface that mimics rheumatoid arthritis. Inflammatory processes of the tendons, as opposed to degenerative processes, are responsible for the development of deformities. In addition, purpuric symptoms and vitiligo might be seen on the surface of the skin [4,5]. The nerve fibers in the retina can be damaged by lesions of the retina, such as vasculitis, which can result in vision impairment or loss. Lupus nephritis, often known as renal illness, is a serious consequence of lupus that affects thirty percent of individuals. The traditional clinical manifestation is characterized by a region that is either circular or somewhat uneven and slightly raised in color. Both atrophy and the appearance of ulceration are possible characteristics of this condition. The red region is distinguished by the presence of telangiectasia and the distinctive white radiating striae. In spite of the lack of symmetry, these symptoms might be similar to those of lichen planus. Petechial lesion and gingival bleeding, including desquamative gingivitis, marginal gingivitis, or erosive mucosal lesions, have been observed in as many as forty percent of patients, and they may be an indication of significant

thrombocytopenia. This is despite the fact that the oral disease is not considered to be a chronic condition. Sjogren syndrome can be present in a significant number of SLE patients at the same time [6].

It is possible to define **celiac disease (CD)**, which is also known as celiac sprue or gluten-sensitive entheropathy, as a chronic inflammatory intestinal disease that is characterized by nutritional malabsorption and improvement following the removal of gluten (which is found in wheat and barley) from the diet. Across Western nations, the prevalence of CD has been found to range anywhere from 1:85 to 1:300 [7]. Additionally, CD can produce minor intestinal damage and weak or absent systemic symptomatology, which is commonly referred to as the "silent form." This is in addition to the conventional gastrointestinal presentation, which includes symptoms such as diarrhea, abdominal distension, vomiting, weight loss, and pallor. In some patients, the absence of symptoms may continue for an extended period of time, despite the fact that the biopsy of the colon reveals the characteristic atrophy of the intestinal mucosa [7]. It is also common knowledge that CD is linked to a number of problems, including lymphomas, autoimmune illnesses, and degenerative diseases of the nervous system [8].

It is also possible for people with CD to experience a number of problems that affect the oral cavity, which is a component of the gastrointestinal system. As the mouth is very easy to examine, oral lesions can provide a valuable clinical clue for early diagnosis of CD; in fact among the atypical aspects of CD (extra-intestinals), in the international literature has been reported some affections interesting the oral cavity, the most common are recurrent aphthous stomatitis (RAS) and dental enamel defects, in addition have been described the association between CD and unspecific forms of atrophic glossitis, oral manifestations of dermatitis herpetiformis, Sjögren's syndrome and oral lichen planus. Even in the absence of a typical intestinal symptomatology, these illnesses can provide helpful signals that can lead to a quick diagnosis [9].

The inflammatory disease known as **Sjogren syndrome** is characterized by the presence of lymphocytic infiltration and subsequent destruction of the exocrine glands [9]. This condition affects the salivary and lacrimal glands and results in a decrease in the secretory activity of the glands. Dryness in the mouth, also known as xerostomia, is caused by hyposalivation, which includes a decrease in saliva production. Xerophthalmia, on the other hand, is caused by a lack of tears. Despite the fact that the etiopathology of Sjogren syndrome is yet unknown, humoral and cell-mediated immunity phenomena are engaged in the process. In point of fact, increased activation of B cells, which is then followed by the formation of immunological complexes and the production of autoantibodies, plays crucial roles [9]. Both genetic and environmental factors have the potential to play a role in the development of the syndrome according to [9].

It is estimated that between 0.5 and 3% of the whole population is affected by Sjogren syndrome, with women being more likely to be affected than men (9:1 ratio). The normal age at which Sjogren syndrome is identified is approximately fifty years of age. It is essential to emphasize that there are two typical surges: the first one occurs shortly after the onset of menarche, and the second one occurs after menopause [10].

There are some people who exhibit clinical indications that are just present in the mouth and eyes, while there are others who have a more significant autoimmune damage. A separate autoimmune illness, such as rheumatoid arthritis or systemic lupus erythematosus, is present in fifty percent of the cases [5]. The condition known as primary Sjogren syndrome is characterized by damage to the glands that does not exhibit any other signs of autoimmune problems. The condition known as secondary Sjogren syndrome is characterized by the presence of an additional autoimmune illness. There are a number of symptoms associated with the oral cavity that are the most prominent [5]. At the level of the oral cavity, the xerostomia is the factor that is responsible for the development of various forms of SS. Patients are more likely to acquire tooth cavities if they do not produce enough saliva. The absence of saliva makes it easier for plaque to accumulate and for it to be removed after elimination. Clinical symptoms that are frequently observed include gingival inflammation and gingival edema. Additionally, a decrease in salivary flow might lead to the development of opportunistic infections. The absence of lysozyme and immunoglobulins provides a favorable environment for the development of Candida, which is why it is frequently found. An relationship between Candida and a lower stimulated salivary flow rate was demonstrated by Radfar et al. as well as Bayetto and Logan [11]. In patients with Sjogren syndrome, both the major and minor salivary glands are affected. An increase in volume of the parotid glands, which is symmetrical on both sides, is observed in fifty percent of the cases. [12] The histological appearance of hypertrophic glands is defined by the presence of epimyoepithelial islands

and the replacement of the gland tissue by lymphocytes. This appears to be the case.

Patients also exhibit irritation and dryness of the eyes, which are caused by xerophthalmia, in addition to oral symptoms. Photophobia is also a contributing factor in these patients' symptoms. There are approximately twenty percent of people who are affected by Sjogren syndrome who exhibit symptoms of the Raynaud phenomenon, which is a disorder that affects the fingers and toes [8]. Myalgia, arthralgia, and asthenia are among symptoms that may be experienced by patients who are afflicted with this disease.

Pemphigus vulgaris is a persistent condition that is caused by the immune system. The mucosa and the skin are both affected by this disease. Patients who are afflicted with pemphigus have an immune globulin G autoantibody that produces antibodies against desmosomal components such as desmoglein-1 and desmoglein-3. By doing so, the characteristics of adhesion cell molecules are altered, which results in the formation of intraepithelial blisters between the Malpighian epitheliocytes respectively. This condition is referred to as acantolysis of suprabasilar cheratynocites [12].

Despite the fact that there is no evidence suggestive of gender preference from an epidemiological standpoint, a few research have indicated a minor frequency among females. Despite the fact that patients in their 40s and 50s are the ones who experience the biggest number of cases, the condition can affect people of any age [8].

It would appear that genetic and ethnic factors are connected to the etiology of the condition. It appears that the lesions are brought on by a variety of stimuli, including physical agents, viruses, hormones, medications, and stress [8, 13]. In more than half of all cases, the oral mucosa is the site where the initial symptoms of the disease appear. The buccal mucosae, soft palate, lower lip, and tongue, as well as the gingiva, which is less common, are all possible locations for the lesions, despite the fact that there is no definite preference for any one place [13]. Lesions that occur in the mouth can range from ulcers that are relatively superficial to blisters or tiny vesicles. Within the oral cavity, the bubbles burst in a short amount of time, leaving behind a painful erosion that causes a burning feeling [13]. There is a wide range of variation in the size of the ulcers. It has been observed that applying a very light amount of pressure on the epithelium of these patients might result in the detachment of a significant portion of the surface, which is accompanied by the formation of blisters. The term "Nikolsky phenomenon" is currently being used to describe this occurrence [5].

For unexplained reasons, Behcet syndrome is a multisystemic, autoimmune disease that affects multiple organs. The presence of at least two of the three primary typical factors—oral ulcers, vaginal ulcers, and eye inflammation—generally serves as a defining characteristic of this condition. In spite of the fact that its initial description was associated with dermatological pathology, Behcet disease is frequently characterized by involvement of the nervous system and the cardiovascular system. Typically, it affects people in their 30s, and there is no evidence that it is more prevalent in one gender than the other. It is noted that populations from the Mediterranean and Asia had the highest incidence of the condition, with Turkey having a significant prevalence of the disease. The existence of antimucous autoantibodies, in conjunction with the relationship of the disease with the HLA configurations B5 and B51, is evidence that leads to the conclusion that the disease has an autoimmune origin [14].

In many cases, the mucocutaneous lesions are the initial indication that the patient is suffering from Behcet syndrome. The detection of these symptoms is an essential component in the process of early diagnosis, and they allow for a more favorable prognosis [14]. Ulcers of the oral mucosae are the oral lesions, and they are indistinguishable from the typical aphthae of the oral mucosa. They cause excruciating agony and are marked by recurrent manifestations. The lips, buccal mucosa, soft palate, and tongue are the areas where they are most typically found. An erythematous lesion represents the initial manifestation of the lesion, which is then followed by the development of ulcers. A few millimeters to a few centimeters is the range of possible dimensions for them [15].

Conclusion:

Regarding the alterations that occur in dental tissues, we did not discover that individuals with CD were more likely to suffer from enamel abnormalities that were both systematic and symmetric. There is, in fact, a wide range of frequencies of enamel abnormalities in patients with CD that have been described in various investigations. It is necessary to conduct additional research in order to have a better understanding of the pathophysiology of this deficiency, as it has been speculated that dietary, immunologic, or genetic variables (associated with the HLA DR3 allele) may play a role. There is a correlation between enamel abnormalities and an altered phosphate-calcium metabolism and/or the production of antibodies against the matrix of the

enamel organ in people who have celiac disease. It is possible that the antigen that is associated with class II molecules of the MHC could initiate an immune response directed against the enamel organ, which could then lead to a mineralization issue coming about. When it comes to addiction, there is no substantial evidence to suggest that these anomalies are associated with the nutritional status, a lack of vitamin D, or an excessive amount of fluoride incorporation.

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