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Infectious nexus between oral and maxillofacial pathology and periodontology- a narrative review

(Nexo infeccioso entre la patología oral y maxilofacial y la periodontología: una revisión narrativa)

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Abstract(english)

The periodontium is the anatomical structure in which forces applied impinge upon the teeth which in turn is associated with the bacterial infection and plaque. The periodontium is associated with the gingivae and also the ligaments connected with the periodontal region and thus both the dentists and the periodontists can communicate with each other and together find the effects of the lesions and can detect and diagnose in the early stages. On various disease conditions, the classification and illustration is done as well some investigation is to be done whenever any abnormality is seen.

Keywords(english)

oral, periodontium, pathology, gingiva, lesion.

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Resumen(español)

El periodonto es la estructura anatómica donde las fuerzas aplicadas impactan los dientes, lo que a su vez se asocia con la infección bacteriana y la placa. El periodonto está conectado con la encía y los ligamentos que conectan con la región periodontal, por lo que tanto dentistas como periodoncistas pueden comunicarse entre sí y, juntos, identificar los efectos de las lesiones, así como detectarlas y diagnosticarlas en sus etapas iniciales. Se realiza la clasificación e ilustración de diversas enfermedades, y se debe realizar una investigación ante cualquier anomalía.

Palabras clave(español)

oral, periodonto, patología, encía, lesión.

Introduction

The gingival lesions are formed due to changes in the genetic origin, infectious origin, immunologic origin, traumatic origin, sometimes auto-immunity and auto-inflammatory disorders and also when drugs are induced in larger quantities and when proper care is not taken. Hence the dentist should do the early diagnosis to prevent the gingival lesions in the patients. Various gingival lesions are treated in different ways according to the severity and their origin. Based on the age, gender, pain, clinical representation the involvement of the tissue, the gingival lesions are classified into many disorders. The gingival lesions, if persists may be malignant and causes cancers like squamous cell carcinoma. So, both periodontists and the dentists must correlate, communicate and then follow the protocols so as to eradicate this type of above disorders by early detection and early diagnosis.

Periodontal lesions

The classification of the gingival lesions is named depending upon the genetic origin like hereditary gingival fibromatosis which is normal to slightly pale colour and very firm in their texture as well enlargement of the gingivae is seen in this condition. It is an autosomal dominant mutation which is mostly seen with son-of-sevenless gene that plays an important role in activation of Ras in which guanine-nucleotide exchange takes place and therefore proliferation of the cell takes place and thus causes fibrosis in the gingivae. In this syndrome, hypertrichosis is seen along with epileptic disorders and mental retardation is seen occasionally. Oral hygienic care is the ultimate treatment along with the surgery (1,2). The second gingival lesions named Ligneous gingivitis is a rarely seen disorder in which enlargement of the gingivae is seen because of no usage of the medicines and treatments. Some nodes are seen along with the ulcers on the gingival surface (3). Also in fewer patients with ligneous gingivitis, hypoplasminogenemia is seen in higher quantities as it is also associated with the inherited type 1 plasminogen deficiency (4). The third lesion called odontogenic gingival epithelial hamartoma is a benign hamartomatous lesion which is rarely seen in the epithelial remnants of dental lamina otherwise called as rests of Serres in most of the female adults. It is an asymptomatic gingival innocuous lesion diagnosed for the peripheral odontogenic neoplasms. The gingival lesions which are of traumatic origin are very common and can be easily detected by the dentists. These purplish or bluish peripheral giant cell lesions also called as giant cell epulis which are obtained from the periodontal ligament otherwise called periosteum in respond to the local trauma. There are several multinucleated giant cells which are of osteoclastic origin connected to vascular cellular tissue (5,6). Sometimes, brown tumours are seen which are related to hyperparathyroidism and they look alike the giant cell lesions which are to be excluded in the treatment process. Based on the infectious origin, gingival lesions are classified in various lesions such as herpes simplex virus infection, which is seen in young children with the painful reddened, swollen gums and intra-oral vesicles. The primary herpetic gingivostomatitis is an acute viral illness that is different from other viral infections which involves oral mucosa, herpangina, hand, foot and mouth disease. Treatment involves, antiviral agent such as acyclovir which is recommended in immunocompromised hosts as well it is also observed thoroughly that the patient is hydrated (7,8). Other herpes viruses like gingival herpes zoster virus and varicella zoster virus may rarely affect the peridontium. Based on the HIV infection, the linear gingival erythema and necrotizing periodontitis is seen but they are very rarely seen as highly active antiretroviral therapy is used to minimise the symptoms. Human papillomaviruses may cause gingival papillomas in the patients with HIV including focal epithelial hyperplasia and warts (9,10). Based on the immunologic origin, the

gingival lesions are classified as Lichen planus which is associated with lesions seen on the buccal mucosa, lateral margins of tongue and gingivae. These are asymptomatic, atrophic ulcers that affect the gingiva causing edema and desquamation. Biopsy is used in the diagnosis process. But there is a difficulty in perfect diagnosis as the cause of lichen planus is not totally known but it is immunologic process in which basal epithelial keratinocytes are destroyed by the cytotoxic T-cells which is triggered by the usage of the medications (11,12). Sometimes, the symptomatic gingival lichen planus may disturb the normal oral-hygiene routine of the patient leading to gingivitis which results in the oral lichen planus. In this condition, the dentist, and the periodontist should co-ordinate, communicate and diagnose in regular intervals so as there will be no occurrence of the oral squamous cell carcinoma which is pertaining to atrophic or ulcerative oral lichen planus (13,14). The mucous membrane pemphigoid is also a rare, chronic, auto immune gingival lesion seen in middle-aged and elderly females in which autoantibody like BP180 is formed in the basement membrane with the bulla formation or oral mucosal vesicle formation. Usually, desquamation is seen along with the painful erythema in the gingiva or minor trauma is also seen. Treatment involves biopsy followed by routine microscopic evaluation and immunofluorescence (15,16). Pemphigus vulgaris is another type of gingival lesions which is rare, auto immune disease which occurs in intraoral region. Antibody such as desmoglein-3 is formed when cell gets adhered to form molecules which lead to bulla formation, desquamation and ulceration in the oral mucosa. Biopsy is the ultimate treatment before doing therapies like topical and systemic immunosuppressive therapy. The tissue is very delicate, so, care has to be taken while taking the specimen and that too not directly from the gingival site but perilesional site is taken into consideration for the diagnosis. Therefore, the early diagnosis helps in minimising the severity of the oral lesions as well there is no requirement to induce desmoglein-1 antibody (17,18). Orofacial granulomatosis is also another type of gingival lesion in which the swelling in the lower part of the face is seen that includes lips, and the cheek mucosa. Diffused gingival erythema is also seen. The treatment includes biopsy that shows non-caseating granulomata. The oral lesions in the patient having this order may antedate gastrointestinal Crohn's disease otherwise called as sarcoidosis. Benzoates and cinnamonaldehyde are highly recommended to overcome this disorder (19,20). The lesions which are so common and that cause esthetic problems and interferes with mastication and which are associated with the enlargement of the gingiva when drugs such as phenytoin, calcineurin inhibitors like cyclosporine and tacrolimus, and calcium channel blockers like nifedipine, oxidipine, diltiazen, and amlodipine are called drug-induced gingival lesions (21,22). These drugs inhibits the cation flux, which ultimately leads to decreased uptake of folate by gingival fibroblasts and also lack of collagenase activation along with the changes in the matrix metalloproteinase metabolism are seen. The treatment involves care towards the oral hygiene. Xerostomia increases susceptibility towards the tooth-root dental caries and gingivitis and this in turn results in plaque formation at tooth-gingiva interface. So medications such as antidepressants and diuretics can be given to the patients. If the medication is not taken, symptomatic relief may occur with salivary substitutes or salivary stimulants or Sjogren's syndrome has to be taken into consideration. When the gingival involvement is peculiar with the cysts and neoplasms formation in the odontogenic tissues, then there is enlargement in the intra bony site and also expansion in the cortical plate is seen, this type of lesions are seen in the soft tissues of the gingiva like gingival cyst and are called as odontogenic cysts and neoplasms (23-25. When there is no bone involvement, there is predilection of lingual gingiva in the premolar area of the mandible and this condition is called as extra-osseous ameloblastoma otherwise called as peripheral ameloblastoma which has the histological features alike as the intra-osseous one which retains invasive growth (26,27). The benign neoplasms that produce cementum like ossifying fibroma and benign cementoblastoma and non-neoplastic bone lesions like osseous dysplasia and fibrous dysplasia may be present in the swelling of the alveolar bone (28). Another condition called as leukoplakia which may be homogenous or non-homogenous may develop in the gingiva and may give rise to malignant transformation due to more in-take of tobacco, alcohol and smoking. Biopsy is the best necessity to check the occurrence of the epithelial dysplasia (29). Squamous cell carcinoma is also the most prevailing oral malignancy in which the edentulous alveolar mucosa and the gingivae are mostly involved. The treatment involves biopsy which is to be done within 3 weeks as well local surgery can be done to eradicate gingival squamous cell carcinoma. If any lesions are not treated then they may cause more difficulty in the surgical process and as well it may be fatal (30). The heterogeneous group of malignant disorder which is derived from the lymphatic cells is

called lymphoma. It is the second most common intra-oral malignant disorder after squamous cell carcinoma. B-cell non-Hodgkin's lymphoma is the most common oral lymphoma. The extra-nodal Hodgkin's lymphoma is rarely seen. Immunodeficiency also causes gingival lesions in the people. Biopsy is the ultimate task for the diagnosis that shows atopical invasive growth. Immunohistochemical markers are also used for various classifications (31).

There are various disorders classified depending upon the prevalence, age group, gender, clinical representation, pain and other oral tissue involvement.

Erythroplakia is a disorder seen around 0.02%-0.83% of older adults. The clinical presentations are isolated fiery red patches which have a papillary or velvety surface observed on all mucosal surfaces which are painless.

Lymphoma is also one of the disorders which is seen in predominantly in males than in females in their 6th and 7th decades. Lymphadenopathy, bony expansion and oral mucosal swelling is seen on any oral mucosal surface, tonsils, bone and the palate which has variable pain.

Proliferative verrucous leukoplakia is seen mostly in 5th-7th decades of people predominantly in females than in males. Multifocal verrucous and papillary white plaque is seen with squamoproliferative lesions on all oral mucosal surfaces and are painless. Sometimes predilection of the gingiva is seen.

Acute myeloid leukemia is a disorder seen in males than in females of age group 85-89 years. Purpuric enlargement along with redness and bleeding is seen in the buccal mucosa with less or no pain.

Oral squamous cell carcinoma is disorder seen 3.1% in worldwide population. Seen predominantly in males than in females of age 70-74 years. Mobility of teeth, red and white exophytic or endophytic mass which is papillary, ulcerated and granular is seen on the lateral and ventral surface of tongue, buccal mucosa and the floor of the mouth with variable pain.

Scurvy is also one of the disorders seen equally in both males and females. Reddening, gingival enlargement, edema, spontaneous bleeding, shiny surface and necrosis with pseudomembrane formation are seen with variable pain.

Kaposi's sarcoma is seen in any age group but mostly seen in males when compared to females. The purpuric swelling of the oral mucosa including palate and the alveolar mucosa with no pain is observed in this disorder.

Oral tuberculosis varies accordingly with the region and the population. It is seen in any age group in both males and females equally. Mucous patches, ulceration, fiery redness and edema are seen on the floor of the mouth, tongue, trachea, larynx, trachea and the bone with variable pain.

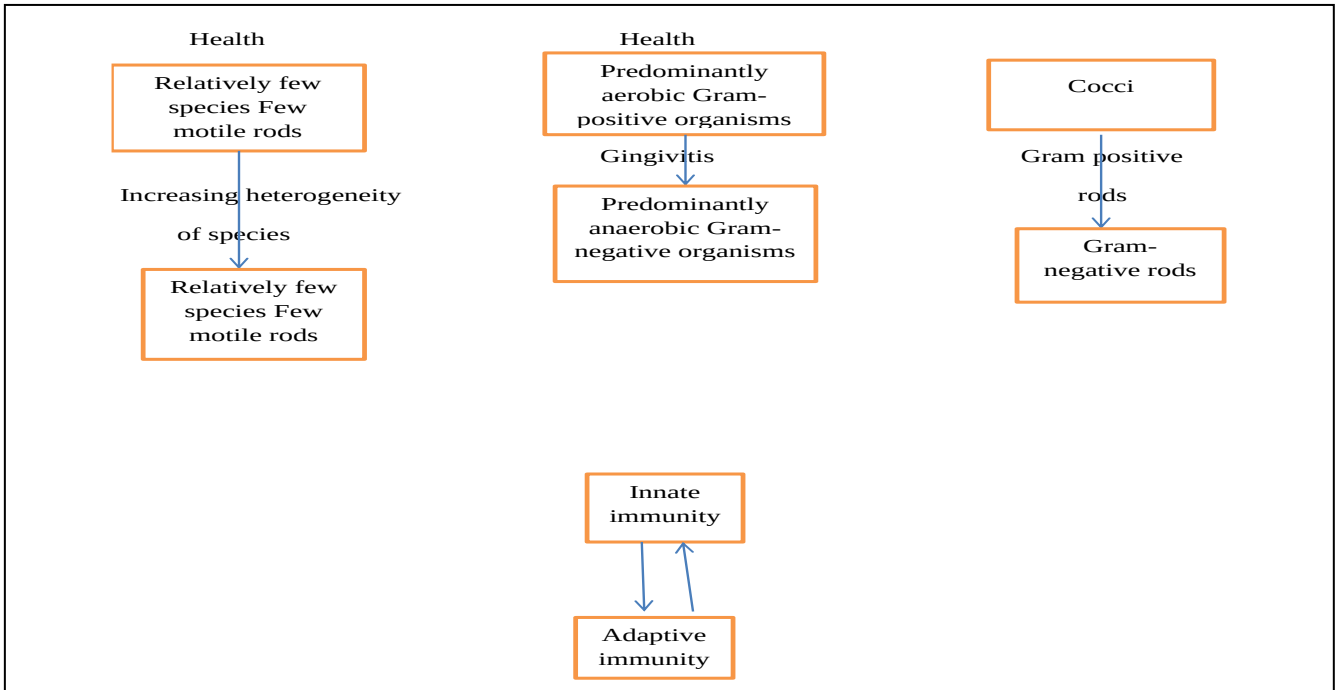


Figura 1. Flow chart

Erythematous candidiasis is disorder seen in any age group. Red lines are seen on tongue, lips, buccal mucosa, soft palate and hard palate with milder pain.

Drug induced gingival enlargement is seen in any age group who are exposed to the drugs. Gingival enlargement is seen with no pain.

Mouth breathing is one of the disorders in which reddening, shiny surface which are diffused and edema is also observed with no pain.

Gingival injuries may occur equally in both males and females. The coagulated surfaces, ulceration, erosion, vesicles, desquamation, petechiae and dilacerations are associated with this disorder and mild to severe pain is observed (32-34).

Discussion

The destruction of the tissue takes place by direct use of bacteria such as damage to cervical epithelium is activated by bacteria like Triponema denticola, Porphyromonas gingivalis. Impairment of leukotoxin is done by Aggregatibacter actinomycetemcomitans. Degradation of immunoglobulins, the cytokine networks and the fibrin are done by bacteria Capnocytophaga spp, and Porphyromonas gingivalis (35)**.** Degradation of collagen is done by fibroblastic collagenase by sulphur compounds from gram negative anaerobes like Tannerella forsythia, Prevotella intermedia. Bone resorption and complement activation by endotoxins. The bone resorption is stimulated by lipoteichoic acid which is obtained from cell walls of gram positive bacteria. Break down of periodontal tissues takes place by proteolytic enzymes to amino acids and peptides by providing nutrients to gram negative bacteria. Apart from the damage by the bacteria, indirect damage is also seen which is via the host response like polyclonal activation of B cells which prevents production of a specific antibody. Triggering immunity leads to activation of complement and more inflammation (Figure 1). Cellular immunity activated T-cells activate antigen-presenting cells that release so many cytokines such as IL-1β whose effects are bone-resorption, fever and are pro-inflammatory36. Another cytokine named TNF-α effects are bone-resorption, fever and are pro-inflammatory and these cytokines are synergistic with IL-1β cytokine. IL-6 is also one type of cytokine which effects B cells differentiation and production of the antibodies as well as differentiation in osteoclast are seen. Polymorphonuclear neutrophils (PMNs) release enzymes like collagenases, elastases, matrix metalloproteinases and stromelysins which destroy the effected tissues (37). In initial 24-48 hours, the lesions are localised to gingival sulcus and the periodontal tissue and the local vasodilation brings immunoglobulins, fibrin, complement and more PMNs into the tissues which in turn increases gingival crevicular fluid (38). The PMNs then migrate into gingival crevice through the junctional epithelium where few lymphocytes and macrophages are seen followed by the formation of the immune complexes, in turn increasing the permeability of PMNs. In the early 4-7 days, localised proliferation of junctional and sulcular epithelium is detected as well increment of gingival crevicular fluid is also seen (39). PMNs are still seen in the crevices and within the periodontal tissues, the accumulation of lymphocyte T-cells is seen. The lymphoid cells may give rise to gingival inflammation and the cytokines are released which proliferates invasion growth. In the period of 2-3 weeks, proliferation of sulcular and junctional epithelium and loss of collagen is seen. Plasma cells are found near to the gingival lesions and there is rapid increase in the gingival crevicular fluid. T-cells dominate the lesions where the PMNs are still present (40). Non-specific polyclonal B-cell mitogens are also present giving rise to gingival inflammation from which immunoglobulins and the plasma cells are produced. In the advanced weeks, the gingivitis may cause damage to the other parts by spreading to the soft tissues and the bones causing inflammation in peridontium which is called periodontitis. Loss of attachment, loss of collagen and bone loss is seen (41)**.** Destructive pathological reaction and imbalance in the host-microbial interaction is seen. The established lesion is with more immunoglobulins, and more gingival crevicular fluid is also seen where the PMNs are still present in large numbers. Due to invasion of the macrophages, plasma cells and lymphocytes, the epithelial wall will break and this is the gateway to direct access of the plaque antigens which activates immune cells further giving rise to tissue damage (42). The immunoglobulins, complement and the lymphocytes in turn give rise to formation of antibody-antigen reactions leading to the tissue wear and tear and the destruction (43).

Conclusion

The patients with the lesions on the periodontium should consult the periodontists as soon as possible so that they get a plan so as to follow the signs and the symptoms so as to access whether the lesions are simple to severe or to confirm whether they are local or systemic disorders**.**

Conflict of interest

None to declare.

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