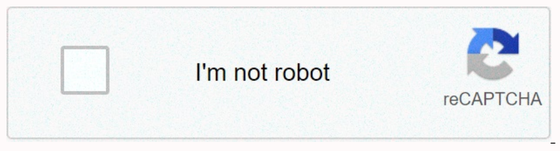

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Oral lichen planus case report pdf

Case Report Vol.5 No.2 Department of Internal Medicine and Radiology, Institute of Medicine, Bangalore School of Dentistry, India: Goraf, School of Pharmacy and Radiology, Bangalore School of Dentistry, India Accepted: October 31, 2016 |Release: November 28, 2016 2016 Citation: Gauraf, Naik V, Sody A. Oral erosion lichen planus - Case report with highlights on the etiology and immunomodulation effects of tacrolimus in the management of OELP. MOJ Crimmed Case Rep 2016; 5 (2): 209-212. DOI: 10.15406/mojcr.2016.05.00130 Download PDF Abstract Oral Lichen Planus (OLP) is one of the most common diseases of the oral mucosa. Clinically, bilateral symmetrical presentations that have certain clearly identifiable features and show a network (known as Wickham's line) such as fine white line lace are essential elements of OLP even when lesions mainly show atrophy and bedrid patterns. Both antigen-specific and nonspecific mechanisms can be involved in the etiology of oral lichen planus (OLP), normal oral mucosa has a healthy immune system overall, and changes in the immune system can result in OLP and possibly other autoimmune oral mucosal diseases. Recent findings in mucosal graft-versus-host disease are clinical and histological correlations of lichen planus, suggesting the involvement of TNF-a, CD40, Fas, MMMPs, and mast cell degranulation in disease etiology. To treat this lesion using multifactorous etiology, various treatment methods are available today. 2 keywords: oral lichenus, licinoid dissoplifying, malignant conversion, OLP etiology, conjunctival, rheumatic heart disease Intraoral flatitis is a common chronic inflammatory mucosal cutworm disorder that generally affects the oral mucosa and additional cases. Lichen planus can affect other parenteral mucosal surfaces such as genitals, anus and pharynx. The involvement of conjunctival and eating paths rarely occurs. Oral lichen planus (OLP) is a disease of unknown etiology that is A T-cell-mediated chronic inflammatory oral mucosal disease. It most commonly affects middle-age adults of both sexes, with a slight advantage in women. The prevalence of skin LP is estimated to be 2,2 in unknown Carozzo and others, but <1% of the population. LP is considered significantly less than exclusive oral LP (OLP), which affects about 1-2% of the population. While the incidence of malignant conversion of OLP remains controversial, careful, regular and long-term follow-up of OLP patients is necessary for early detection of malignant conversion from OLP. A .50-year-old female patient (Figure 1), who has follow-up intervals ranged from two months to each year, reported to lumen medicine and radiology for the past two to three months complaining of severe pain and burning sensation in her mouth. History of her presentationRevealing severe pain and burning sensation throughout the oral cavity, which began three years ago as low-intensity pain, it was intermittent in nature and had gradually progressed to its current state for the past two months. She underwent a hysterectomy 20 years ago and then a disc replacement procedure 11 years ago. She had insomnia, which she had been treating for the past 15 years. She was diagnosed three years ago with RHD (rheumatic heart disease) and hypercholesterolemia and received penicillin injections (once every 20 days) that worked for a year longer. She had unders received little tooth repair using silver amalgam. She also undert received root canal treatment with a crown arrangement performed on the post-tooth. The patient was not known of any known drug allergies. The patient had a mixed diet plan and showed up well nourished for her age. Her oral hygiene was moderate. The patient had no harmful habits. Fig. 1 The patient's profile picture lesions were coarse, non-scrap, and non-persistent were not noted. Oral examinations showed diffuse lesions in the form of white corneal regions scattered on the face of the gums (FIG. 2) and more backwardly on both sides of the mucosa of the cheeks (FIG. 3). The left cheek mucosa showed about 2x2.5 cm of white erythema extending superior postgraduately from the occlusal line to the upper and lower vestiligo from the area adjacent to the mandible first molar to the retro molar pad area. The white radial striation (Wickhamstria) had scattered erythema. The right cheek mucosa showed extensive lesions of about 2.5x3 cm extending backwards from the area in the back veins against the baccarus and alveoli mucosa of the lower ginguum against the lower gingual baccarus and alveoli mucosa. The boundaries of the lesions appear irregular and are not defined. In palpation, the lesion appeared to be soft. The surface of the lesion was rough, non-scrap, and no nonvolatile was not noted. FIG. 3 Lesions of facial gums 2 Lesions of the right and left cheek mucosa In a lesion hard tissue examination, all complements of teeth were present in all four quadrants except 46, with generalized wear and tear and a thick band of local deposits of plaque and d'ealation. Mobility did not exist, and the blockage was a class I mole relationship of angles. There were also few cali teeth that showed no symptoms such as pain, swelling or tenderness. Based on the patient's history and clinical findings, the lesion was tentatively diagnosed bilaterally with oral corrosive lichenus planus in the facial gums and cheek mucosa. Differential diagnosis lysenoid drug reaction. Investigation Complete blood imaging and blood body grams – found to be within normal range..An open biopsy (Figure 4)Micrograph of 4 lesions. Histopathological examination of tissue biopsies was revealed to be the case of lichen planus. Hematoxylin and eosin staining showed epithelium and connective tissue. The epithelium was parakeratinized squamous and superplified in some areas. Also, some areas showed atrophic epithelium. The underlying connective tissue showed a dense band of chronic inflammatory cells in lymphocytes, mainly with plasma cells. Prominent blood vessels were seen in the section. The tooth retepeg, a hallmark of lichen pranus, was evident in micrographs. Final diagnosis of facial gingiosa and both sides of the cheek mucosa the final diagnosis of oral lichen pranus. Treatment prescribed 0.1% of topical loritactums three times daily for a month, with antifungal candid ointment as the basic regime, and then tapered the dose twice daily for two weeks after 30 days. This continued for two months and the patient continued to be triggered continuously for six months. Since the lesion was completely healed, the patient showed drastic improvement. She has been placed on a regular recall so that her condition can be monitored and the recurrence of lesions can be prevented. White and red lesions occur in the oral mucosa, including white protrusions, red blood cells, candidiasis, geographical tongue, lichen planus, lichenoid lesions, etc. Oral gluctivosis and oral red blood cell prakia are well known to be precancerous lesions, but the malignant potential for oral lichen planus (OLP) and/oraltícenoid lesions (OLLs) has been the subject of much debate in the past few decades. The clinical and histological features of these white-red lesions are similar, so their differential diagnosis is important. Oral lichen planus characteristically has a bilateral distribution and typically includes the cheekbone mucosa, ointment and abdominal surface of the tongue and / or gums, often presented as dehydrated gingiitis. Often asymptic, but if there is an area of ulcer, the patient experiences varying degrees of discomfort and is exacerbated by eating spicy or acidic foods. There are many antigen-specific mechanisms that can be involved in the etiology of OLP, including MHC class I- and MHC class II restriction antigen presentation by lesioned square cells, activation of antigen-specific CD4+ helper T cells and CD8+ cytology T cells, clonal dilation of antigen-specific T cells, and annodized apoptosis induced by antigen-specific CD8+ cytogenic T cells. In addition to the large number of nonspecific mechanisms in the etiology of OLP, (Fig. 5) heat shock protein (HsMP), reactive oxygen species (ROS), stress, mogranulation stimulated by mast cell chemotaxis and T-cell RANTES, mast cell kimase including endothelitis cell adhesion molecular expression, mast cell TNF-a by epithelial base membrane, activation by T cell MMP-9 activationBy mast cell protease or T cell MPP-9, it was caused by keratinocyte apoptosis caused by epithelial base destruction, in-epithelial CD8 + T cell free due to base membrane destruction, inflammatory cell survival by T cell RANTES, nonspecific T cell recruitment with keratinocyte-derived chemokine. OLP can be divided into six types (reticul, tail, plaque, atrophic, eroding, and bullfighting types), while the two types of white and red are most commonly classified as three types: reticul, atrophic, and erosive. Lesions are not homogeneous, and some cases can exist as a mixture of these clinical subtypes. The World Health Organization (WHO) devised diagnostic criteria for OLP in 1978, revised in 2003. The modified WHO diagnostic criteria are defined by these modified WHO criteria with the distinction between OLP and OLLs.7,8, whereas the essential clinical feature of OLP is the presence of bilateral lesions indicating a network like a race of white lines (reticulous patterns), but not the presence of plaque, atrophic, erosive, and bull lesions. In the presence of bilateral reticulation lesions, it is designated as clinically compatible with OLP. For example, biopsy and histopathological examination of the affected area is required to eliminate other diseases that mimic erythema and identify the possibility of epithelial dysoplasty. The need for biopsy in all suspected cases of lichen planus will be discussed, but appropriate if it is atypical in presentation, atrophic or ulcerative. Skin tests for allergies to mercury amalgam may be performed when there may be a lysenoid reaction in response to this dental material. However, there is a debate about the value of such investigations. The histopathology of OLP was first described by Dubreuill in 1906 and revised in 1972 by Shklar, which described three features: keratinization, liquid denedation of the basal cell layer, and dense subcutaneous subcutaneous bands of lymphocytes. One of the most important issues with OLP is the possibility of malignant conversion to OSCC. Who classifies OLP as a precancerous condition, but the risk of malignant conversion of OLP remains the subject of literature debate. It is unclear what mechanism may cause the malignant trans formation of OLP. The preferred site of oral squamous cell carcinoma (OSCC) developing from OLP lesions is the tongue and cheek mucosa, which in the former has a higher incidence than the latter, and epithelial hyperpl formation of OLP is more common in the cheek mucosa. You need to take a step-by-step approach. Topical corticosteroid therapy is the mainstay of treatmentDisease. There is limited evidence from randomized controlled trials on the exact effectiveness of various formulations, which are common uses. As an aid to treatment, patients should also be advised that the need to maintain a high level of oral hygiene and the causes of mucosal trauma, such as inconsistent dentures, sharp cusp, poor dental repair, should be eliminated. Patients should be informed that the risk of malignant tumors associated with oral lichen planus is very small and long-term monitoring is appropriate. 10 In our case, the systematic and systematic use of tacrolimus (immunomodule) showed a drastic improvement in the healing of persistent lesions. Tacrolimus is a macrolide calcinulin inhibitor. Calcinulin is a calcium and carmodulin-dependent serine/threonine protein phosphatase that activates T cells in the immune system. When activation of T cell receptors occurs, there is an increase in intracellular calcium, which exists using carmodulin as a catalyst, to activate calcinulin. This step is followed by dephosphorylation, which stimulates the transfer of the transcription factor of the nuclear factor of the activated T cell to the T cell nucleus, increasing the activity of il-2 and other genes that code cytokines. This was found to be one of the mechanisms that results in OLP, such as lesions. In the mechanism of the disease, TAKROLIMUS acts in the dephosphorylation stage (FIG. 6), thus interfering with it, resulting in phosphorylation. This ultimately reduces the activity of genes that code various OL's, so progress to OLP, such as lesions, stops. This showed the mechanism of action of tacrolimus as an immunomodulant in the treatment of oral eroding lichen below. Various studies have found topical tacrolimus to be effective in treating OLP, and some have also reported better initial treatment responses than other drugs, including corticosteroids. However, few studies have evaluated the effectiveness of topical tacrolimus in the Indian population, especially over a long period of time. Tacrolimus (0.1%w/v) is reported to be effective and safe for the treatment of OLP. This was found to be an effective means of controlling the symptoms and signs of erodib or ulcerative oral lichen, and did not have a noticeable adverse effect over the average duration of the application of 19.8 months. Currently, the cause of lichen planus remains unknown, so there is no specific preventive regime for the disease. The etiology of OLP can be accompanied by both antigen-specific and nonspecific mechanisms. However, regular clinical reviews are considered prudent in terms of controversy over the possible malignancy of the condition.The early days of oral lichen planus are based on the clinical presentation of bilateral white patches with or without ulcers or blisters, typically affecting the cheek and side surfaces of the tongue and gums. Nice Guidelines 27 [Referral Guidelines for Suspected Cancer] clearly state that patients with oral lichen planus should monitor for oral cancer as part of regular dental checkups. 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