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Chronic Desquamative Gingivitis: A Case Report

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ABSTRACT

Desquamative gingivitis is a type of gingival keratinization disorder, characterized by chronic ulceration, desquamative erosions and edematous erythema of the free and attached gingiva. Desquamative gingivitis has been known to be refractory in treatment and only a few cases have been reported. Various etiologic factors are present for the appearance of such lesions. Despite of considering etiology, treatment is often provided by systemic or topical corticosteroids. Since the cause of this condition is still unclear, symptomatic therapy remains the mainstay of treatment. Here, presenting the case of a patient who had moderate periodontitis with chronic desquamative gingivitis.

Keywords: Corticosteroids, Desquamative, Erythema, Periodontitis

INTRODUCTION

Although first recognized and reported in 1894 by Tomes and Tomes, the term *chronic desquamative gingivitis* was coined in 1932 by Prinz[1] to describe a peculiar condition characterized by intense erythema, desquamation, and ulceration of the free and attached gingiva.[1] It is seen mainly in adults, especially women, although rare cases have been observed in children[2]. Oral lichen planus, mucous membrane pemphigoid and pemphigus vulgaris have emerged as most common causes for desquamative gingivitis. It may occur due to aging, abnormal response to bacterial plaque, allergy, idiopathic, chronic infections, autoimmune diseases or idiopathic[1]

Patients may be asymptomatic, however, when symptomatic, their complaint ranges from mild burning sensation to an intense pain. Approximately 50% of desquamative gingivitis cases are localized to gingiva, although involvement of the gingiva and intraoral/extraoral sites are not uncommon.

Initially, the cause of this condition was not clear with a variety of possibilities that had been suggested. Etiologic factors have been considered which includes estrogen deficiency, hypothyroidism and nutritional deficiencies. However, Foss, Glickman and Smulow[3] considered the disease to be primarily a degenerative process. In 1953 Glickman, speculated that chronic desquamative gingivitis may represent a manifestation of several disease processes. While he favored an endocrine etiology, he thought that the condition might be a variant of chronic mucous membrane pemphigoid or bullous lichen planus. Because most of the cases were diagnosed in women in the fourth- fifth decades of life (but desquamative gingivitis may occur as early as puberty or as late as the seventh or eighth decades), a hormonal derangement was suspected. However, in 1960 McCarthy

and colleagues suggested that desquamative gingivitis was not a specific disease entity, but a gingival response associated with a variety of conditions. This concept was further supported by numerous immunopathologic studies. They also proposed a classification of desquamative gingivitis based on etiologic considerations: Dermatoses, Hormonal influences, Abnormal responses to irritation, Chronic infections and Idiopathic causes. Among dermatoses manifest as desquamative gingivitis, the authors included benign mucous membrane pemphigoid (now often called cicatricial pemphigoid), pemphigus and lichen planus. Some reports have suggested that psoriasis also may manifest as desquamative gingival lesions.

The diseases that can cause desquamative gingivitis can be classified into 2 categories: *immunological* (autoimmune or auto-immune like) and *idiopathic*. Immunological diseases causing desquamative gingivitis include erosive lichen planus, lichenoid mucositis, benign mucous membrane pemphigoid, pemphigus vulgaris, paraneoplastic pemphigus, linear IgA disease, and bullous pemphigoid. Idiopathic lesions are probably not autoimmune mediated, and may be caused by chronic bacterial, fungal, and viral infections, or other factors capable of causing chronic irritation and inflammation.
The current classification system for periodontal diseases and conditions includes non plaque induced gingival disorders and gingival manifestations of systemic conditions. The mucocutaneous disorders e.g., lichen planus, pemphigoid, pemphigus vulgaris, erythema multiforme, lupus erythematosus, drug induced lesions and others are listed in this subgroup together with allergic reactions to dental materials, foods, and other substances for topical application.

The advent of clinical and laboratory parameters revealed that approximately 75% of desquamative gingivitis cases have a dermatologic genesis. Cicatricial pemphigoid and lichen planus account for over 95% of the dermatologic cases. However, other mucocutaneous autoimmune conditions such as lupus erythematosus, bullous pemphigoid, dermatitis herpetiformis, pemphigus vulgaris, linear immunoglobulin A (IgA), and chronic ulcerative stomatitis can clinically manifest as desquamative gingivitis.

Other conditions considered in the differential diagnosis of desquamative gingivitis are chronic bacterial, fungal, and viral infections, as well as reactions to medications, mouthwashes, and chewing gum. Although less common, Crohn's disease, sarcoidosis, some leukemias, and even factitious lesions have also been reported to clinically present as desquamative gingivitis.

Therefore it is of paramount importance to ascertain the identity of disease responsible for desquamative gingivitis to establish an appropriate therapeutic approach and management. To achieve this goal, the clinical examination has to be coupled with a thorough history, and routine histologic and immunofluorescent studies. It should be mentioned, however, that despite this diagnostic approach, the cause of desquamative gingivitis cannot be elucidated in up to one third of the cases.

CASE REPORT

A 51-year-old female patient reported to the Department of Periodontology, BRS Dental College & Hospital, Panchkula, having a chief complaint of burning sensation in lower posterior gingiva since 9 months which aggravates on having spicy & hot beverages. On examination, red, linear, diffused patches were noticed on the buccal marginal gingiva with involvement of attached gingival showing signs of desquamation. (Fig 1) Limitation of oral function and speech difficulties due to pain was also reported by the patient. Non-significant dental and medical history was given was given by the patient. Patient had a

habit of tobacco chewing, 3-4 times in a day since last 10 years but she had quitted her habit 3 yrs back. She has also given a history of change in tooth paste a year back which was continued by her for 6 months and again she shifted to her regular tooth paste. Due to the discontinuation of the tooth-paste, the appeared desquamated lesion didn't healed. Exact etiology for the appearance of desquamated lesions in this patient was not identified. After obtaining written consent, an incisional gingival biopsy was taken from mandibular posterior region for the histopathological examination. The hematoxylin & eosin (H & E) stained tissue showed clefts at the sub-epithelium with underlying connective tissue showing bundles of collagen fibers arranged haphazardly along with fibroblast & small foci of inflammatory cells. (Fig.2). epithelium clefts were suggestive of desquamative lesions. Thorough oral prophylaxis (scaling & polishing) was done. Patient was advised to rinse her mouth with 0.2% chlorhexidine mouthwash, enforced to do modified bass technique for brushing her teeth & also was asked to maintain good oral hygiene. Topical application of steroid (0.1% Triamcinolone acetonide) twice in a day for three weeks was prescribed. Lesions did not show any improvement clinically with topical steroids application in the follow up visits (Figure:3)

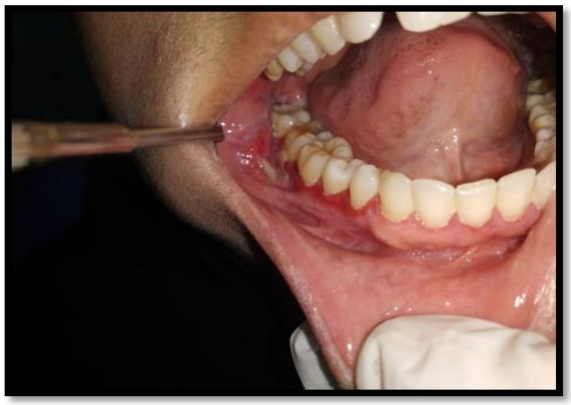

Figure:1 Desquamative gingivitis in mandibular posterior region

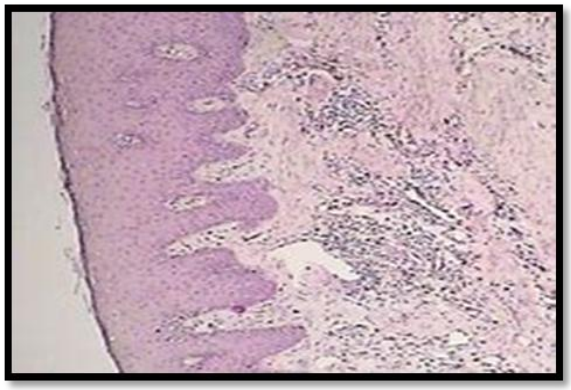

Figure:2 H & E stained tissue for histopathological examination

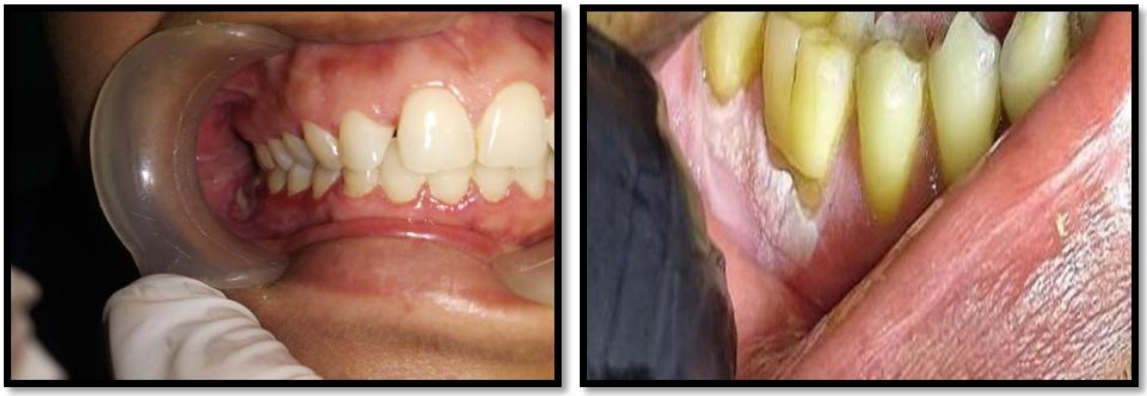

Figure:3 Appearance of desquamative gingival lesions in mandibular region after steroid therapy

DISCUSSION

Desquamative gingivitis is only a clinical term that describes a peculiar clinical picture. This term is not a diagnosis per se and once it is rendered, a series of laboratory procedures should be used to arrive to a final diagnosis. Thus the success of any given therapeutic approach depends on the re-inforcement of an appropriate final diagnosis. The following represents a systematic approach to elucidate the disease triggering desquamative gingivitis. Differential diagnosis of multiple oral ulcers may include aphthous ulcers major, erosive

lichen planus, mucous membrane pemphigoid, pemphigus vulgaris, acute necrotizing ulcerative gingivitis, allergies, acute herpetic gingivostomatitis, and erythema multiforme.[4]

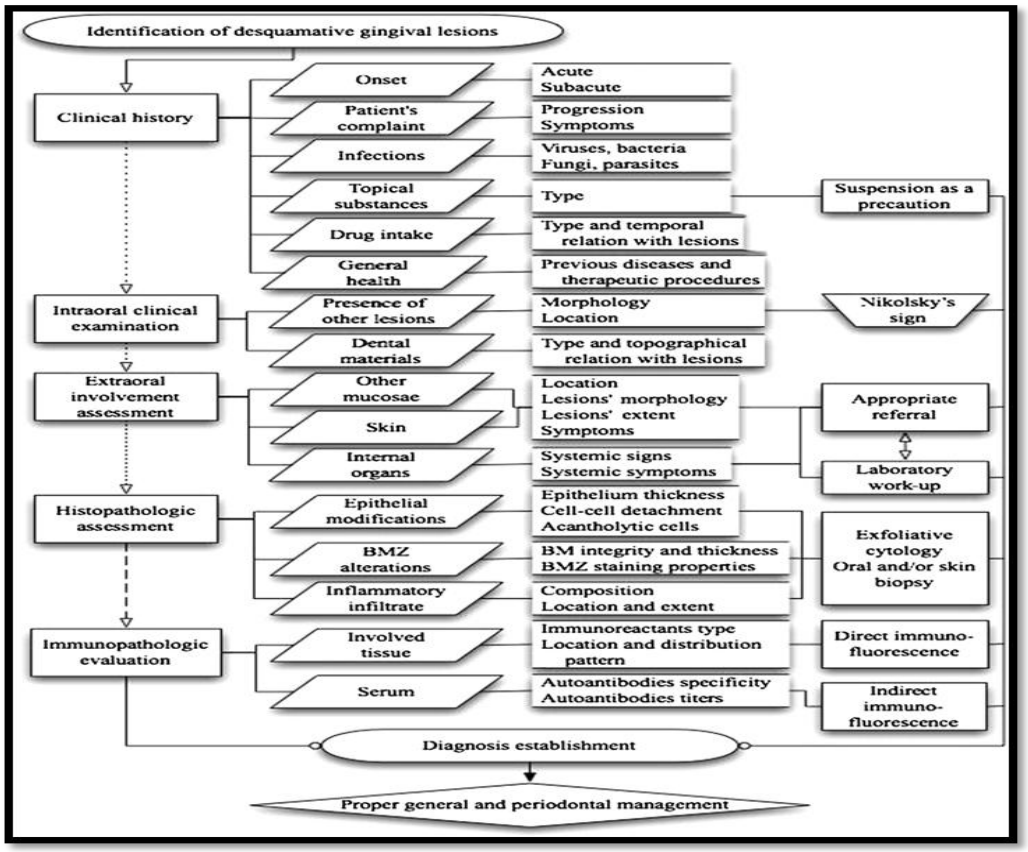

Figure:4 Representing identification of gingival lesions

Desquamative gingivitis is entirely a common disorder in which the gingiva is desquamated. Chronic soreness is commonly seen and intake of spicy foods may further worsen the condition. Erythematous gingiva with loss of stippling, extends apically from the gingival margins to the alveolar mucosa is frequent. Severity may range from mild, almost insignificant small patches to widespread erythema with glazed appearance. Occasionally, such lesions may occur in the absence of bacterial plaque[1]. Sometimes contact allergic reactions to various oral hygiene products have also been reported in desquamative gingivitis patients[2]. But oral mucosa is less commonly prone to contact allergic reactions, when compared to skin, though the latter is exposed to a wide variety of antigenic stimuli[4]. The therapeutic approaches to desquamative gingivits are based on expert opinion rather than empirical evidence. Several treatment methods have been reported (Carrozzo & Gandolfo, 1999; Chan et al., 2002; Endo et al., 2008b; Fatahzadeh et al., 2006; Kirtschig et al., 2003; Lamey et al., 1992; Motta et al., 2009; Nisengard, 1996; Nisengard & Levine, 1995). Treatments are available in the form of systemic or topical steroids, antimetabolites (cyclophosphamide, azathioprine, mycophenolate mofetil, methotrexate), antibiotics (tetracyclines), and dapsone intravenous immunoglobulins, plasmapheresis, and Low level laser therapy which causes pain relief and accelerates regeneration of damaged tissues.. Treatment of desquamative gingivitis requires elimination or control of local irritants. Rough restorations, illfitting dentures, traumatic oral hygiene procedures, and

dysfunctional oral habits should be corrected[5]. In most of the cases, such lesions are successfully managed with topical corticosteroids combined with effective plaque control[6,7]. Plaque accumulation itself acts as a stimulus for worsening the condition of desquamative gingivitis, but the plaque itself does not cause such lesions. Patient with desquamative gingivitis are unable to maintain a proper oral hygiene due to unbearable pain. Therefore, their oral hygiene is likely to be ineffective, making it difficult to treat this condition. Lack of correct oral hygiene and the accumulation of plaque may increase the long-term risk for plaque-induced periodontal diseases along with desquamative gingivitis.[8]

Clinical Examination:

Recognition of pattern of distribution of the lesions (i.e., focal or multifocal, with or without confinement to the gingival tissues) provides leading information to begin the formulation of a differential diagnosis. A clinical maneuver such as Nikolsky's sign offers insight into the plausibility for the presence of any vesiculobullous lesions.

Biopsy:

Given the extent and number of lesions that may be present in a given individual, an incisional biopsy is the best alternative to begin the microscopic and immunological evaluation. An important consideration is selection of the biopsy site. Once the tissue is excised from the oral cavity, the specimen can be bisected and then submitted for microscopic examination. Buffered formalin (10%) should be used to fix the tissue for conventional hematoxylin and eosin (H&E) evaluation. Michell's buffer (ammonium sulfate buffer, pH 7.0) is used as transport solution for immunofluorescent assessment.. However, there are notable exceptions such as in lichen planus and subacute lupus erythematosus where only the lesional tissue will exhibit the corresponding immunologic markers.

Microscopic Examination:

Approximately 5-µm sections of formalin-fixed, paraffin embedded, tissue stained with conventional H&E are obtained for light microscopy examination.

Immunofluorescence:

For direct immunofluorescence, unfixed frozen sections are incubated with a variety of fluorescein-labeled, anti-human serum (anti-IgG, anti-IgA, anti-IgM, anti-fibrin and anti-C3). With indirect immunofluorescence, unfixed frozen sections of oral or esophageal mucosa from an animal such as a monkey are first incubated with the patient's serum to allow attachment of any serum antibodies to the mucosal tissue. The tissue is then incubated with fluorescein-labeled antihuman serum (anti-IgG, anti-IgA, anti-IgM, anti-fibrin, and anti-C3). Immunofluorescence tests are positive if a fluorescent signal is observed either in the epithelium, its associated basement membrane or in the underlying connective tissue.

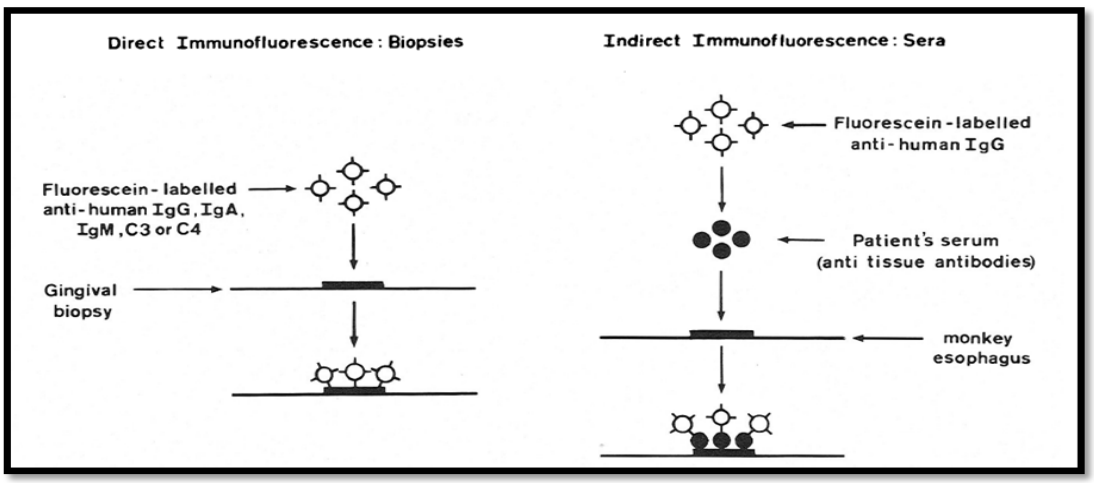

Figure:5 Direct/indirect immunofluorescence

CONCLUSION

The management of desquamative gingivitis is challenging because lesions reoccur after it goes into remission.

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