



Case report

DESQUAMATIVE GINGIVITIS: A CASE REPORT

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ABSTRACT

Desquamative gingivitis (DG) is the clinical term for the gingival manifestation of mucocutaneous diseases. It is characterized by erythematous, glazed, friable, and hemorrhagic gingiva, which pains can accompany. The three principal diseases at the origin of desquamative gingivitis are oral lichen planus, pemphigus vulgaris, and mucous membrane pemphigoid. Understanding these three diseases' clinical, histological, and immunohistochemical features is crucial for the diagnosis and appropriate therapy of DG. The present work reports a case of DG to describe the diagnostic procedures and treatment.

KEYWORDS: *mucosa, oral, gingivitis, inflammation, erosion*

INTRODUCTION

Desquamative gingivitis (DG) is a clinical oral manifestation of mucocutaneous disorders such as oral lichen planus, pemphigus vulgaris, and mucous membrane pemphigoid (MMP) (1). DG can also recognize different etiologies like allergic reactions, psoriasis, linear IgA disease, and Crohn's disease (2). DG appears as red, eroded, atrophic, glazed gingiva. It can be characterized by painful symptomatology, often unrelated primarily to plaque, and its disimmune nature is more commonly detected in middle-aged and older women (3). Discomfort and bleeding during daily oral hygiene manoeuvres lead to the accumulation of dental plaque (4). DG could represent a challenging condition when the attached gingiva is the only site involved. It is often misdiagnosed with the more common plaque-induced gingivitis, thus worsening the clinical manifestations leading to disease progression and involvement of several mucosal or cutaneous sites (5). Histological analysis with immunofluorescence is still the gold standard used to discriminate between the various vesiculobullous disease (6). DG manifestations are not significantly improved after conventional periodontal therapy, and the treatment includes topical or systemic immunosuppressive drugs such as corticosteroids and low laser therapy (7). Based on the different histological and immunohistochemical findings, the treatment can be further modulated: In oral lichen planus, topical retinoids can improve the clinical manifestations but cannot give clinical aid in other DG causes. During immunosuppressive therapy, caution must be paid to oral candidiasis (8).

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CASE REPORT

A 48-year-old female was visited at the Oral Pathology Unit of the University of Campania, “Luigi Vanvitelli”. The patient’s medical history highlighted an allergy to lincomycin and diabetes mellitus. The patient reported the occurrence of erosive-erythematous areas to both upper and lower attached gingiva and an itching sensation spread to all the gingiva (Fig. 1). The analysis requested included anti-desmoglein 1 antibody (DSG1), anti-desmoglein 3 antibodies (DSG3), bullous pemphigoid antigen 180 (BP180), and bullous pemphigoid antigen (BP230). The anti-BP180 tested positive at the control at 19.9 IU (negative < 9 IU). The patient underwent an incisional biopsy to perform histological analysis and direct immunofluorescence that revealed the dermal-epidermal split, eosinophil infiltration in the blister’s fluid, and IgG deposits upon the linear basement membrane with “band-like” distribution. Therefore the diagnosis of MMP was carried out, and the patient underwent topical medical therapy that consisted of clobetasol propionate twice a day and fluconazole once a day.

DISCUSSION

MMP is a chronic autoimmune disease characterized by the appearance of blisters on different mucosal surfaces, caused by the expression of autoantibodies (BP 180 and BP 230), one of the leading actors of cellular adhesion in the basement membrane zone that connect basal keratinocytes with the basal membrane (9). Blister development results from the linear deposition of IgG, IgA, or C3 along the mucosal and epithelial basement membranes. Several agents are described as triggering the predisposing factors, including drugs, internal malignancies, ultraviolet exposure, radiotherapy, infective agents such as Human Herpes Virus, cytomegalovirus, Epstein-Barr virus, and thermal or chemical burns (10).

Clinical manifestations of MMP comprise the appearance of blisters of varying size, varying from small vesicles corresponding to red erythematous areas to more giant vesicles (blisters <5 mm in diameter) and bullae (blisters >5 mm in size) (11). The lesions involve the mucosal surfaces lined by stratified epithelium (oral, nasal, conjunctival, laryngo-esophageal, anal, and genital mucosa). Skin lesions are infrequent and occur after mucosal manifestations that often include the upper body, head, and neck (12).

The oral cavity, especially the buccal mucosa, soft palate, and lips, is the first site affected in 90% of MMP patients (11). The oral blisters burst quickly, leaving painful erosions and ulcers, often the only clinical sign of the disease (12). The gingiva is the most commonly affected oral site in MMP. Given its autoimmune etiology, gingivitis in MMP does not improve with the elimination of plaque and tartar, unlike in common bacterial periodontal diseases associated with accumulating a microbial biofilm on tooth surfaces (13).

The treatment choices for MMP include corticosteroids (such as prednisone, clobetasol propionate, or methylprednisolone), immunosuppressive drugs (such as azathioprine, methotrexate, mofetil, and cyclophosphamide) and immunomodulators (such as tacrolimus) (11). Due to the epidemiological preference for the elderly, topical therapy seems preferable to limit the systemic adverse effect that could worsen the health of medically fragile patients (14,15). Further studies are needed to provide scientific information for safer and more reliable treatment options.



Fig. 1. Desquamative Gingivitis: erosive-erythematous areas to both upper and lower attached gingiva

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