J Istanbul Univ Fac Dent 2016;50(2):54-60.
http://dx.doi.org/10.17096/jiufd.57228 *REVIEW ARTICLE*

DESQUAMATIVE GINGIVITIS: A REVIEW

Deskuamatif Gingivitis: Derleme

Gizem KARAGÖZ, Kıvanç BEKTAŞ-KAYHAN, Meral ÜNÜR

Received: 15/03/2013
Accepted:19/06/2014

ABSTRACT

Desquamative gingivitis (DG) is characterized by the erythematous gingiva, desquamation and erosion of the gingival epithelium, and blister formation. It is a common clinical manifestation in several diseases. Contact allergic reactions to various oral hygiene products and chemical agents have also been reported to represent as DG. The management of DG has been a major problem, largely because the etiology of the disease has been elusive. In this paper, we aimed to review the current literature on the pathogenesis, diagnosis management and prognosis of DG.

ÖZ

Deskuamatif gingivitis (DG), vezikül oluşumu, atrofi, erozyon ve deskuamasyonla seyreden, marjinal ve keratinize dişetinin yaygın eritemi ile karakterize olan klinik bir bulgudur. Birçok önemli hastalığın ortak klinik belirtisidir. Çeşitli ağız bakım ürünleri ve kimyasal ajanların neden olduğu kontak allerjik reaksiyonlar sonucu da oluştuğu bildirilmiştir. DG'in etiyolojisinin belirlenmesi ve altta yatan hastalığın tanısı zor olduğundan hastalığın tedavisi ve kontrolü oldukça büyük bir problemdir. Bu yazıda, DG'in patogenezi, tanısı, tedavisi ve prognozu ile ilgili güncel kaynaklardan derleme yapılması amaçlanmıştır.

Keywords: *Desquamative gingivitis; Mucocutaneous diseases; Contact allergic reactions*

Anahtar kelimeler: *Deskuamatif gingivitis; Mükokütanöz hastalıklar; Kontak alerjik reaksiyonlar*

Department of Oral and Maxillofacial Surgery Faculty of Dentistry Istanbul University

Introduction

The term "desquamation" is derived from the Latin word 'Desquamare', which means scraping fish flakes. As a word, desquamation means 'loss of epithelial elements in small and large amounts, peeling of skin, and exfoliation' (1). Chronic desquamation of the gingiva is referred to as desquamative gingivitis (DG) (2). Chronic desquamative gingivitis was described for the first time by Tomes and Tomes in 1894 (3). In 1932, Prinz used the term 'chronic diffuse desquamate gingivitis' for chronic diffuse inflammation cases, which were characterized by severe epithelial desquamation in the marginal gingiva (4). In actual use, the term 'Desquamative gingivitis' is used for a specific clinical symptom and it is not a diagnosis alone (2, 3, 5-7). DG is a clinical finding, which progresses with vesicular formation, atrophy, erosion and desquamation, characterized with diffuse erythema of the marginal and keratinized gingiva (6, 8-10). Lesions start with diffuse erythema and minimal desquamation. The affected gingiva epithelium is very fragile and tends to exfoliate easily, even with the slightest trauma (3). Large ulceration areas can be observed in some cases (11). The desquamative gingivitis is seen after puberty, especially in individuals over 30 years of age (3). It is more common in women than in men. It has been reported that it can rarely be observed in children (2, 3, 8, 12).

Desquamation of epithelial tissue is generally seen in free and keratinized gingiva. Generally, the lesions that affect the buccal/labial surfaces of the gingiva, although not formed due to bacterial plaque, are exacerbated with plaque accumulation (13). Although they are generally observed in the anterior region, they can be seen in any gingival area (6). Similar lesions can be seen in the edentulous alveolar ridge. While only desquamations can be observed in the patients, there can also be associated vesicular-bullous lesions, in addition to ulcerative and lichenoid lesions. In severe cases, it can be seen generalized at the oral mucosa, and the alveolar mucosa can be affected together with the gingiva (13). The patient can either have no complaints or there can be a burning sensation or severe pain. In general, there is chronic pain, which especially increases with the intake of acidic foods. Limitation of oral function and speech difficulties due to pain can also be observed (2, 6). Nisengard and Levine (14) have mentioned some characteristics in order to diagnose the clinical findings as DG, since gingival erythema is not associated with plaque and presence of gingival desquamation. They also emphasized that the Nikolsky phenomenon is generally positive in DG patients. Only a single etiologic factor was considered in the first desquamate gingivitis (DG) cases. Initially, it was suggested that gingival desquamations were related to hormonal changes due to menopause on the basis that gingival desquamations were more common in the middle-aged and in women (13). In 1964, Glikman and Smulow (15) stated that DG could be the symptom of severe conditions, especially mucocutaneous diseases. Recently, it has been generally accepted that DG can be the initial symptom of vesicular bullous diseases and can emerge as a result of reactions against some chemicals and allergens, and that it is not related to hormones (2, 3, 6, 10, 12, 13).

Differential Diagnosis and Prognosis

The differential diagnosis of desquamative gingivitis (DG) includes a wide spectrum, such as chemical and electrical burns, allergic reactions, hormonal disorders and mucocutaneous diseases. Furthermore, a similar clinical pattern can be observed in reactions developing against mouthwashes (Figure 1), chewing gums, cosmetic products, drugs, cinnamon and dental materials (16). It is suggested that the disease may be observed when there is lack of estrogen or progesterone (17). Additionally, there are idiopathic gingival desquamative lesions without any etiologic factors (Figure 2) (3). There are conflicting arguments on whether it is a symptom of oral lichen planus (Figure 3), mucous membrane pemphigoid (Figure 4), or a clinical manifestation of these diseases (12, 18). In many articles, it has been reported that DG is related to lichen planus, mucous membrane pemphigoid and pemphigus vulgaris (88% - 98%) (Figure 5) (6, 9, 13, 16).

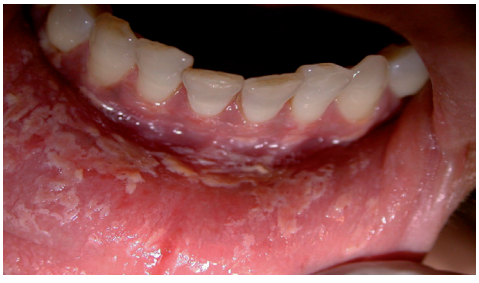

Figure 1. *Mucosal and gingival desquamation developing as a result of an allergic reaction against toothpaste.*

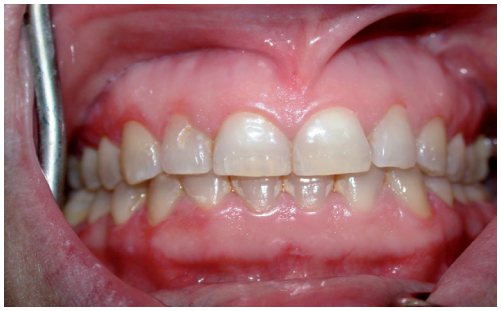

Figure 2. *Desquamative gingivitis not related to disease or allergic reaction.*

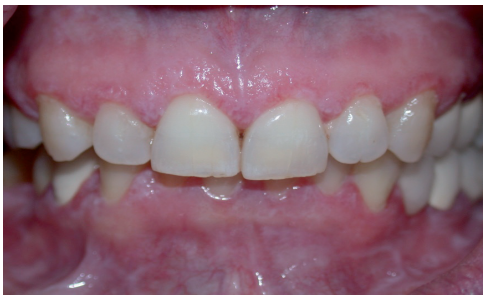

Figure 3. *Atrophic form of Lichen Planus creates a typical desquamative gingivitis appearance at the gingiva.*

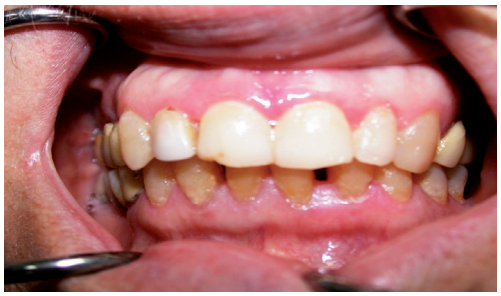

Figure 4. *The intraoral appearance of mucous membrane pemphigoid is generally similar to desquamative gingivitis.*

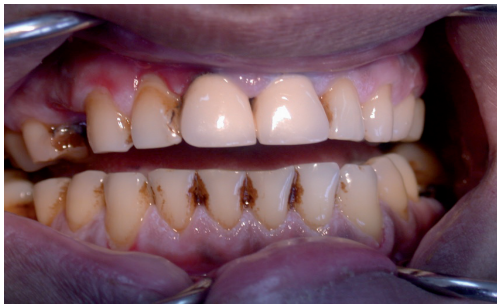

Figure 5. *Since the initial symptoms of pemphigus can begin as desquamative gingivitis, it is important to evaluate this clinical symptom to reach an early diagnosis.*

The definitive diagnosis of desquamative gingivitis is very difficult and complicated. Determination of the etiologic factors that cause the lesions or making the diagnosis of the underlying systemic disease can take a long time. Detailed history of the patient, systemic symptoms, presence of similar lesions at other sites of the body, medications used, contact with chemical materials and the family history should be questioned (3). If there is suspicion of allergy, a patch test against dental materials can be performed on the patient (16). The definitive diagnosis can be made by histopathological, direct (DIF) and indirect immune fluorescent (IIF) examinations of the tissues obtained from the lesions, in addition to examination of autoantibodies in the circulation (19). Several mucocutaneous diseases in which clinical desquamative gingivitis is observed have been reported in the literature (Table 1).

The clinical condition generally exacerbates with plaque accumulation, trauma or improper brushing. The clinical picture worsens with the disruption of oral hygiene practices due to pain and bleeding (3, 16). The disease continues chronically with periods of remission and exacerbation. Recovery of the gingiva may take months (11). Although the intraoral presence of desquamative gingival lesions differ, various durations from 2 months to 25 years have been reported (13).

Treatment

If there are previously determined etiologic factors (allergen materials, etc.) that cause DG, those should be eliminated and oral hygiene practices should be improved. Subgingival and supragingival plaques should be removed and proper teeth brushing with a soft brush in addition to flossing should be recommended (16). Besides, patients should be warned about mechanical and chemical trauma. Intraoral restorations or prosthesis should be removed (8). Systemic and topical corticosteroids are used for the medical treatment of DG. Topical corticosteroids are commonly used to treat DG. However, their effects are limited due to the saliva volume and the tongue movements which decreases the effectiveness of the treatment. Direct application of chlobetasole-17-propionate to the affected site is recommended (3, 11). Custom built silicone or acrylic carriers which provide long term contact of the drug with the gingival lesion can be prepared to increase the effectiveness of the topical treatment.

Table 1. *Disease in which DG is clinically observed.*

Lichen Planus	Scully *et al.* (2), 1997; Lo Russo *et al.* (20), 2008
Mucous membrane pemphigoid	Chan *et al.*(21), 2002; Alkan *et al.* (22), 2003
Pemphigus vulgaris	Navarro *et al.* (23), 1999; Scully *et al.* (24), 1999; Boy *et al.* (25), 2006; Scardina *et al.* (26), 2005
Bullous pemphigoid	Yih *et al.* (27), 1998;Sklavounou and Laskaris (28), 1983
Paraneoplastic pemphigus	Yih *et al.* (27) , 1998
Dermatitis herpetiformis	Chorzelski and Jablonska (29), 1975; Egan *et al.* (30), 1997
Chronic ulserative stomatit	Lorenzana *et al.* (31), 2000
Lineer Ig A disease	Porter *et al.* (32), 1992; del Valle *et al.* (33), 2003
Psoriazis	Jones and Dolby (34), 1972
Pyostomatitis vegetans	Wray (35),1984
Erythema multiforme	Arteaga and Eisenberg (36), 1990
Diskoid lupus erythematosus	Blanco *et al.* (37), 2000
Dyskeratosis congenita	Anil *et al.* (38), 1992
Epidermolysis bullosa	Kossard *et al.* (39),1979
Graft-versus-Host disease	Lo Russo *et al.* (20), 2008
Plasma cell gingivitis	Lo Russo *et al.* (6), 2009; Leao *et al.* (13), 2008
Foreign body gingivitis	Leao *et al.* (13), 2008
Kindler syndrome	Ricketts *et al.* (40), 1997
Ulserative colitis	Ricketts *et al.* (40), 1997
Hepatit C	Lo Russo *et al.* (6), 2009
Akut miyeloid lösemi (AML)	Lo Russo *et al.* (20), 2008
Dermatomitosis, mixed connective tissue disorders	Leao *et al.* (13), 2008
Crohn disease	Scully and Porter (2),1997
Sarcoidosis	Scully and Porter (2),1997
Drugs or chemicals implicated include various oral health care products	Corrocher *et al.* (11), 2006; Kuttan *et al.* (41), 2001
Sodium lauryl sulphate	Herlofson and Barkvoll (42),1993; Ahlfors and Lyberg (43), 2001
Magnesium monoperoxyphthalate	Scully *et al.* (44) ,1999

As for local lesions, beclomethasone dipropionate inhaler (50-100 microgram/spray), fluticasone propionate (50 microgram/spray) nasal spray can be directly applied onto the lesions four times a day. Furthermore, 0.1% triamcinolone orabase can also be used. For generalized lesions, prednisolone (5-10 mg), betamethasone (0.5-1 g) or fluticasone tablets dissolved in water can be used as a mouthwash for at least two minutes 2-3 times a day (16). Use of 0.15% benzydamine hydrochloride mouthwash is also recommended for its analgesic and anti-inflammatory effects. Topical use of sicatrizing drugs as supportive treatment accelerates regression of lesions (16). There are cases that have been reported concerning the successful use of topical tacrolimus (0.0%3, 0.1%, 0.3%). However, its use is not preferred, due to the necessity of controlling serum tacrolimus levels at certain intervals and because of the side effects in some

patients (11, 16). Use of drugs such as cyclosporine, azathioprine, and dapsone has also been mentioned in the literature (16). Estrogen support for the treatment of DG has been recommended based on the presence of estrogen-sensitive receptors in the human gingiva and estrogen destruction (17). The idea of estrogen therapy has been rejected since the estrogen receptors expressions in the gingival tissues are not related to the presence or absence of estrogen as well as the side effects of estrogen (45, 46).

Conclusion

Desquamative gingivitis can be the clinical symptom of some dermatitis and mucocutaneous diseases and the underlying primary cause should be evaluated meticulously. Taking detailed patient history, performing a careful intraoral examination and determining the presence or absence of similar lesions at other sites of the body are the most important steps in clinical practice. Definitive diagnosis of DG should be made by incisional biopsy, histopathological examination and DIF. Gingival lesions are controlled by improving oral hygiene and the use of topical corticosteroids. If there is an underlying systemic disease, the case should be consulted with the physician.

Source of funding
None declared

Conflict of interest
None declared

References

1. Desquamation. (Online) http://en.wikipedia.org/wiki/Desquamation, access date: October 6th, 2012.
2. Scully C, Porter SR. The clinical spectrum of desquamative gingivitis. Semin Cutan Med Surg 1997;16(4):308-313.
3. Guiglia R, Di Liberto C, Pizzo G, Picone L, Lo Muzio L, Gallo PD, Campisi G, D'Angelo M. A combined treatment regimen for desquamative gingivitis in patients with oral lichen planus. J Oral Pathol Med 2007;36(2):110-116.
4. Prinz H. Chronic diffuse desquamative gingivitis. Dental Cosmos 1932;74:331-333.
5. Endo H, Rees TD, Kuyama K, Matsue M, Yamamoto H. Use of oral exfoliative cytology to diagnose desquamative gingivitis: A pilot study. Quintessence Int 2008;39(4):e152-161.
6. Lo Russo L, Fierro G, Guiglia R, Compilato D, Testa NF, Lo Muzio L, Campisi G. Epidemiology of desquamative gingivitis: Evaluation of 125 patients and review of the literature. Int J Dermatol 2009;48(10):1049-1052.
7. Rees TD. Desquamative gingivitis/ mucocutaneous diseases commonly affecting the gingiva. In: Hall's Critical Decisions in Periodontology: People's Medical Publishing House, 2011.
8. Endo H, Rees TD. Diagnosis and management of desquamative gingivitis. Gingival diseases - their aetiology, prevention and treatment. InTech 2011, 171-188.
9. Rogers RS, 3rd, Sheridan PJ, Nightingale SH. Desquamative gingivitis: Clinical, histopathologic, immunopathologic, and therapeutic observations. J Am Acad Dermatol 1982;7(6):729-735.
10. Stoopler ET, Sollecito TP, DeRossi SS. Desquamative gingivitis: Early presenting symptom of mucocutaneous disease. Quintessence Int 2003;34(8):582-586.
11. Corrocher G, Di Lorenzo G, Mansueto P, Martinelli N, Esposito-Pellitteri M, Gelio S, Lombardo G, Pacor ML. Comparison of topical tacrolimus 0.1 % in pectin ointment with clobetasol 0.5% ointment in adults with moderate to severe desquamative gingivitis: A 4-week, randomized, double-blind clinical trial. Clin Ther 2006;28(9):1296-1302.
12. Robinson NA, Wray D. Desquamative gingivitis: A sign of mucocutaneous disorders--a review. Aust Dent J 2003;48(4):206-211.
13. Leao JC, Ingafou M, Khan A, Scully C, Porter S. Desquamative gingivitis: Retrospective analysis of disease associations of a large cohort. Oral Dis 2008;14(6):556-560.
14. Nisengard RJ, Levine RA. Diagnosis and management of desquamative gingivitis. Periodontal Insights 1995;2:4-10.
15. Glikman I, Smulow JB. Chronic desquamative gingivitis: Its nature and treatment. J Periodontol 1964;35:397-405.
16. Richards A. Desquamative gingivitis: Investigation, diagnosis and therapeutic management in practise. Perio 2005;2(3):183-190.
17. Yih WY, Richardson L, Kratochvil FJ, Avera SP,

Zieper MB. Expression of estrogen receptors in desquamative gingivitis. J Periodontol 2000;71(3):482-487.
18. Scully C, Laskaris G. Mucocutaneous disorders. Periodontol 2000 1998;18:81-94.
19. Challacombe SJ, Setterfield J, Shirlaw P, Harman K, Scully C, Black MM. Immunodiagnosis of pemphigus and mucous membrane pemphigoid. Acta Odontol Scand 2001;59(4):226-234.
20. Lo Russo L, Fedele S, Guiglia R, Ciavarella D, Lo Muzio L, Gallo P, Di Liberto C, Campisi G. Diagnostic pathways and clinical significance of desquamative gingivitis. J Periodontol 2008;79(1):4-24.
21. Chan LS, Ahmed AR, Anhalt GJ, Bernauer W, Cooper KD, Elder MJ, Fine JD, Foster CS, Ghohestani R, Hashimoto T, Hoang-Xuan T, Kirtschig G, Korman NJ, Lightman S, Lozada-Nur F, Marinkovich MP, Mondino BJ, Prost-Squarcioni C, Rogers RS, 3rd, Setterfield JF, West DP, Wojnarowska F, Woodley DT, Yancey KB, Zillikens D, Zone JJ. The first international consensus on mucous membrane pemphigoid: Definition, diagnostic criteria, pathogenic factors, medical treatment, and prognostic indicators. Arch Dermatol 2002;138(3):370-379.
22. Alkan A, Gunhan O, Alkan A, Otan F. A clinical study of oral mucous membrane pemphigoid. J Int Med Res 2003;31(4):340-344.
23. Navarro CM, Sposto MR, Onofre MA, Scully C. Gingival lesions diagnosed as pemphigus vulgaris in an adolescent. Case report. J Periodontol 1999;70(7):808-812.
24. Scully C, Paes De Almeida O, Porter SR, Gilkes JJ. Pemphigus vulgaris: The manifestations and long-term management of 55 patients with oral lesions. Br J Dermatol 1999;140(1):84-89.
25. Boy Z, Bektas Kayhan K, Ünür M. Gingivada pemfigus vulgarisin erken lezyonları: Olgu sunumu. İstanbul Üniv Diş Hekim Fak Derg 2006;40(3-4):37-43.
26. Scardina GA CF, Messina P, Valenza V, Leone A. Pemphigus vulgaris in a mediterranean young adult manifesting as desquamative gingivitis. Dent Med Probl 2005;42(2):367-369.
27. Yih WY, Maier T, Kratochvil FJ, Zieper MB. Analysis of desquamative gingivitis using direct immunofluorescence in conjunction with histology. J Periodontol 1998;69(6):678-685.
28. Sklavounou A, Laskaris G. Frequency of desquamative gingivitis in skin diseases. Oral Surg Oral Med Oral Pathol 1983;56(2):141-144.
29. Chorzelski TP, Jablonska S. Diagnostic significance of the immunofluorescent pattern in dermatitis herpetiformis. Int J Dermatol 1975;14(6):429-436.
30. Egan CA, O'Loughlin S, Gormally S, Powell FC. Dermatitis herpetiformis: A review of fifty-four patients. Ir J Med Sci 1997;166(4):241-244.
31. Lorenzana ER, Rees TD, Glass M, Detweiler JG. Chronic ulcerative stomatitis: A case report. J Periodontol 2000;71(1):104-111.
32. Porter SR, Bain SE, Scully CM. Linear iga disease manifesting as recalcitrant desquamative gingivitis. Oral Surg Oral Med Oral Pathol 1992;74(2):179-182.
33. del Valle AE, Martinez-Sahuquillo A, Padron JR, Urizar JM. Two cases of linear iga disease with clinical manifestations limited to the gingiva. J Periodontol 2003;74(6):879-882.
34. Jones LE, Dolby AE. Desquamative gingivitis associated with psoriasis. J Periodontol 1972;43(1):35-37.
35. Wray D. Pyostomatitis vegetans. Br Dent J 1984;157(9):316-318.
36. Arteaga S, Eisenberg E. Chronic desquamative gingivitis: A diagnostic approach. Conn Dent Stud J 1990;10:25-26.
37. Blanco A, Gallas MM, Gandara JM, Garcia A, Antunez J. Discoid lupus erythematosus with chronic desquamative gingivitis. A case report and. Med Oral 2000;5(4):231-237.
38. Anil S, Beena VT, Raji MA, Remani P, Ankathil R, Vijayakumar T. Oral squamous cell carcinoma in a case of dyskeratosis congenita. Ann Dent 1994;53(1):15-18.
39. Kossard S, Lofgren RK, Rogers RS, 3rd. Ultrastructural study of mechanobullous desquamative gingivitis: A case report. Acta Derm Venereol 1979;59(6):527-531.
40. Ricketts DN, Morgan CL, McGregor JM, Morgan PR. Kindler syndrome: A rare cause of desquamative lesions of the gingiva. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84(5):488-491.
41. Kuttan NA, Narayana N, Moghadam BK. Desquamative stomatitis associated with routine use of oral health care products. Gen Dent 2001;49(6):596-602.
42. Herlofson BB, Barkvoll P. Desquamative effect of sodium lauryl sulfate on oral mucosa. A preliminary study. Acta

Odontol Scand 1993;51(1):39-43.
43. Ahlfors EE, Lyberg T. Contact sensitivity reactions in the oral mucosa. Acta Odontol Scand 2001;59(4):248-254.
44. Scully C, El-Kabir M, Greenman J, Porter SR, Mutlu S, Barton I, Adair R. The effects of mouth rinses and dentifrice-containing magnesium monoperoxyphthalate (mmpp) on oral microflora, plaque reduction, and mucosa. J Clin Periodontol 1999;26(4):234-238.
45. Chlebowski RT, Wactawski-Wende J, Ritenbaugh C, Hubbell FA, Ascensao J, Rodabough RJ, Rosenberg CA, Taylor VM, Harris R, Chen C, Adams-Campbell LL, White E, Women's Health Initiative I. Estrogen plus progestin and colorectal cancer in postmenopausal women. N Engl J Med 2004;350(10):991-1004.
46. Stahlberg C, Pedersen AT, Lynge E, Andersen ZJ, Keiding N, Hundrup YA, Obel EB, Ottesen B. Increased risk of breast cancer following different regimens of hormone replacement therapy frequently used in europe. Int J Cancer 2004;109(5):721-727.

Corresponding Author:
Gizem KARAGÖZ
Department of Oral and Maxillofacial Surgery
Faculty of Dentistry Istanbul University
34093 - Çapa Fatih, Istanbul / Turkey
Phone: +90 212 414 20 20/30349
e-mail: gizem_karagoz@yahoo.com