World Journal of Stomatology

Submit a Manuscript: http://www.wjgnet.com/esps/
Help Desk: http://www.wjgnet.com/esps/helpdesk.aspx
DOI: 10.5321/wjs.v4.i2.103

World J Stomatol 2015 May 20; 4(2): 103-107
ISSN 2218-6263 (online)

Oral lichenoid lesion: A review of the literature

Soraya de Mattos Camargo Grossmann, Camila de Nazaré Alves de Oliveira, Giovanna Ribeiro Souto, Caroline Góes, Ricardo Alves Mesquita

Soraya de Mattos Camargo Grossmann, Universidade Vale do Rio Verde, Unincor, Barro Preto, 32600-416 Belo Hozironte/MG, Brazil
Camila de Nazaré Alves de Oliveira, Giovanna Ribeiro Souto, Ricardo Alves Mesquita, Department of the Oral Surgery and Pathology, School of Dentistry, Universidade Federal de Minas Gerais, Pampulha, 31270-010 Belo Horizonte/MG, Brazil
Caroline Góes, Private Dentistry, 73015-132 Brasília/DF, Brazil
Author contributions: de Oliveira C de NA, Souto GR and Góes C analyzed the literature and wrote the paper; Grossmann S de MC and Mesquita RA designed and prepared the paper.
Supported by Grants from National Council for Scientific Technological Development (CNPq, #309209/2010-2); Mesquita RA is research fellows of CNPq.
Conflict-of-interest: There is not conflict-of-interest.

Correspondence to: Ricardo Alves Mesquita, DDS, PhD, Professor, Department of Oral Surgery and Pathology, School of Dentistry, Universidade Federal de Minas Gerais, Faculdade de Odontologia, Av. Antônio Carlos, 6627 sala 3204 - Pampulha, 31270-010 Belo Horizonte/MG, Brazil. ramesquita@ufmg.br
Telephone: +55-31-34092499
Fax: +55-31-34092430
Received: December 3, 2014
Peer-review started: December 4, 2014
First decision: January 20, 2015
Revised: January 26, 2015
Accepted: February 10, 2015
Article in press: February 12, 2015
Published online: May 20, 2015

Abstract

The oral lichenoid lesion (OLL) is response that occurs on the oral mucosa. The OLL include allergic response to the dental materials, drugs, and on graft-*vs*-host disease (GVHD). OLL to dental material happen when restorative materials, most commonly amalgam, are in direct contact with the mucosa in sensitized individuals. Medications that produce OLL are oral hypoglycemic agents, angiotensin-converting enzyme inhibitors, and nonsteroidal anti-inflammatory agents. GVHD is a complication in bone marrow transplantation and OLL is a common lesion observed in this disease especially in chronic GVHD. The clinical and histological aspects of OLL are similar to oral lichen planus and turn it difficult to make a differential diagnosis. The purpose of this paper is review about OLL related to the dental materials, drug use and GVHD.

Key words: Oral lichenoid lesion; Lichenoid contact reaction; Lichenoid drug reaction; Lichenoid related to graft-*vs*-host disease

Core tip: There are various oral manifestations like oral lichen planus. These lesions are related to local or systemic factors and are important in oral diagnosis and patient's management. Considering the increased of number of these lesion in current moment, we investigated previous publications and aim to present a literature review.

Grossmann S de MC, de Oliveira C de NA, Souto GR, Góes C, Mesquita RA. Oral lichenoid lesion: A review of the literature. *World J Stomatol* 2015; 4(2): 103-107 Available from: URL: http://www.wjgnet.com/2218-6263/full/v4/i2/103.htm DOI: http://dx.doi.org/10.5321/wjs.v4.i2.103

INTRODUCTION

Oral lichenoid lesion (OLL) is a chronic inflammatory lesion of the oral mucosa that occurs as an allergic

response to dental materials, to use of certain medications, in patients with graft-*vs*-host disease (GVHD), in patients with systemic diseases, *e.g.*, chronic hepatitis C[1] and patients vaccinated against hepatitis B[2]. Various terminologies have been used to describe this condition, as oral lichenoid lesions, oral lichenoid reaction, oral lichenoid tissue reaction, lichenoid contact stomatitis or lichen-planus-like lesions, due the similar clinical and histological aspects of OLL and oral lichen planus[3].

The OLL is a frequent condition, with prevalence 2.4% in general population[4]. These lesions occurs generally in oral mucosa of adults[5], mostly in women with average age of 53 years old[6]. The lesions are mostly present in the buccal mucosa, lateral border of the tongue and oral mucosa of the lips, when associated with composite restorations. It is generally limited size and unilateral. This find can be important to distinguish OLL from the oral lichen planus (OLP) lesions, which occurred commonly bilateral in oral mucosa[2,3,6,7].

Clinically the lesions showed as white striations, plaques, erythema, ulcers or blisters, asymptomatic. The patients can complain of sensitivity to spicy foods or burning sensation. Histologically, observed a hyperkeratinization, liquefaction degeneration of the basal cell layer and band-like layer of lymphocytic infiltrate in the connective tissue[2,6-9]. OLP, OLL and GVHD could not be histopathologically discriminated[10,11], however some authors investigate possible histopathological aspects among the lesions[12]. Moreover, the clinical and histological aspects are not useful to distinguish OLL from OLP[13,14]. Some authors showed that the level of salivary IgA and IgG in OLP and OLL patients is higher than healthy controls, but they cannot be used as differential diagnosis of both alterations[15].

The purpose of this paper is review about OLL associated with dental materials, with medication use, with systemic diseases, in patients vaccinated against hepatitis B and in patients with GVHD. The methodology was a search of the literature, from 1966 through December 2014, about OLL related to the dental materials, drug use, GVHD listed on PubMed. The search was conducted in both English and Portuguese, and the keywords used were "oral lichenoid lesion", "oral lichenoid lesion and dental materials", "oral lichenoid lesion and drug" and "oral lichenoid lesion and GVHD". Additional studies were found in the reference lists of the selected articles.

ORAL LICHENOID LESION TO THE DENTAL MATERIALS

Resin-based composite, gold and the amalgam and its components can cause hypersensitivity reactions on the oral mucosa[6,13,16-19]. According to Lygre *et al*[20] the principal cause of OLL was associated with adverse reaction to dental materials, being amalgam fillings as responsible for 84% of the cases. The OLL associated with amalgam restorations can be observed in about 2% of population[7,20-22]. Although uncommon, the composite resin also can be associated with OLL[23].

The induction of OLL to the dental materials is probably the long-term. The contact of oral mucosa with dental material develops hypersensitivity reaction over a period of days and the clinical manifestations may present many years after initial contact with the dental material[7]. In case of the amalgam, reaction can occur the release of corrosion products from the restoration surface, and may result in lymphocyte activation and induction of a cell-mediated autoimmune response directed at basal keratinocytes[13]. The cell mediated type Ⅳ hypersensitivity response of amalgam restoration can result in immune-mediated damage of the basal keratinocytes[7,17,21,22]. In most cases of OLL the hypersensitivity is the mercury, however other components of amalgam fillings as copper, tin or zinc can be associated with the reaction[6,16,18]. Some authors suggested that this reaction occurs in susceptible individuals for long time of exposure[21,13], since OLR does not develop in all individuals with amalgam alloys in contact with oral mucosa. However, the levels of IL-6 and IL-8 in saliva of patients underwent to amalgam filling replacement showed significant reduction[8].

Although the use of patch test for OLL to dental material is controversial, showing limited value[16,24,25], Thornhill *et al*[13] showed that the combination of a positive patch test and the presence of oral lesions together to amalgam restoration were an important predictor of lesion improvement. An interesting observation made by these authors was that the desquamative gingivitis clinical aspect was not observed in any of the patch test positive patients or patients with a strong clinical association between the lesions and their fillings. Moreover, these patients did not demonstrated history of skin lesions.

Conflicted points related to skin patch test can be described: amalgam components to use in the test; distinguish sensitivity from irritant responses; how long the material should remain in contact with the skin; and the value of skin patch testing in identifying true oral lichenoid lesions. Furthermore, there is debate about the validity of extrapolating skin reactions to mucosal responses[17]. Nevertheless, the patch test may be helpful to determinate an alternative material to use when replacing amalgam[17,25].

The final diagnosis of OLL to dental material is confirmed by clinical and histological aspects associated with the resolution of the lesions after replacement of the restoration[6,16]. Most of the OLR associated to amalgam disappear in 3 to 15 mo after that the restoration was changed[6,8,13,17,18,24].

The study by Thornhill *et al*[14] confirmed the difficulty of histological distinction between OLL and OLP, showing that five oral pathologists were able to differentiate both conditions in just one-third of the cases. According to the authors, some features may be present in OLL and absent in OLP: an inflammatory infiltrate located

Table 1 Medications related to the induce oral lichenoid lesion described in literature

Type of medication	Example
Antibiotics and chemotherapeutic agents	Penicillin, tetracycline, streptomycin, pyrazinamide, sulfadoxin, ketoconazole, pyrimethamine, demeclocycline
Antidiabetic agents	Chlorpropamide, tolbutamide
Antiepileptic agents	Carbamazepine
Antihypertensive agents	Methyldopa, labetalol, propanolol, captopril
Antimalarials	Chloroquine, quinacrine
Antimaniac drugs	Lithium salts
Antiplatelet agent	Clopidogrel
Antirheumatic agents	Gold Salts
Antiulcer medication	Bismuth
Benzodiazepines	Lorazepam
Nonsteroidal anti-inflammatory drugs	Salicylates, indometacin, fenelofenac, isoxicam, piroxicam

Adapt from Guijarro Guijarro and López Sánchez[27].

deep to superficial infiltrate in some or all areas; focal perivascular infiltrate; plasma cells and neutrophils in the connective tissue. Juneja *et al*[3] found increased epithelial thickness in OLL compared to OLP, probably due to the release of inflammatory mediators from the cellular infiltrate, inducing the proliferation of basal keratinocytes. However, the number of mast cells, neutrophils and macrophages is significantly higher in OLP than in OLL, besides a continuous thin, linear band of basement membrane and numerous strands extending into the irregularity connective tissue. Thus, these parameters can be considered useful to differential diagnosis between OLP and OLL[3]. However, it is necessary to emphasize the importance of excluding the presence of Candida infection, which it is common in association with OLLs[26], principally in areas of ulceration since in both of them may result in accumulations of neutrophils and plasma cells[14].

ORAL LICHENOID LESION TO THE DRUG

Drugs are identified as inducers of oral lichenoid lesion (OLL-d), principally associated with prolongation use of the drugs[17]. When a drug is suspected to cause the OLL-d, the change of them should be considered[21]. In contrast to cutaneous lichenoid lesion to the drugs, the OLL-d is uncommon[17]. The Table 1 presented drugs that can induce OLL-d[27].

The final diagnosis of OLL-d is difficult, and the readministration of the medication can help to establish if the oral lesions are drug-induced, though this can be dangerous for the patient[17,28]. Generally the lesion disappears after suspension of the drug[3,17,28]. However, the complete resolution of the lesion can hold out several months.

In some cases, the medication is potentially indispensable to survival of the patients; thus its suspension or replace is not possible[17,28]. In these cases, the lesions must be treated conventionally as OLP. According some authors the patients with OLL-d related with drugs to cardiovascular diseases, have reported to decrease unstimulated whole saliva secretion[29], suggesting the hyposativation as a trigger to OLL-d in these patients[19].

Other medications have been related to OLL-d and OLP[1]. It has been extensively demonstrated that IFN may induce or worsen immunological diseases. With the advent of pegylated IFN-α, a causal link among the treatment of chronic hepatitis C with combination of pegylated IFN plus ribavirin and several autoimmune events have been suggested. The development or exacerbation of OLP has also been reported after the introduction of IFN-α to treat hepatitis C[2,3,9,28,30-32], and also contribute to the development of new lesions as OLL. It is quite plausible that IFN-α may induce or worsen previous lesions due to its interference with the cytokine cascade[31]. Grossmann *et al*[33] described three cases of exacerbation of OLP during the treatment of chronic hepatitis C with pegylated IFN plus ribavirin. However, it is difficult establish if the lesions were exacerbation of previous lesions of OLP or new lesions of OLL-d.

McCartan and Lamey[30] investigated the use of a lichen planus-specific antigen as a marker to distinguish idiopathic OLP from OLL-d and demonstrate that it is not a useful marker.

ORAL LICHENOID LESION ON GVHD

GVHD is a very frequent complication of allogeneic bone marrow transplantation (BMT), and it is associated with morbidity and mortality. It is characterized by dermatological, gastrointestinal and hepatic lesions[31,34,35].

GVHD occurs when the donor immune system recognizes the host tissue as foreign and attacks its cellular constituents. Donor's T-lymphocytes reacts against recipient of antigens[17,31,32,34-36]. Three conditions can be observed in patients with GVHD[37]: the graft must contain immunologically competent cells; the recipient must express tissue antigens that are sufficiently different from those of the donor; or the recipient must be incapable of rejecting the graft because of either tolerance, lack of recognition, or immunosuppression.

There are acute and chronic GVHD: acute GVHD (aGVHD) occurs within the first hundred days after

transplant, while chronic GVHD (cGVHD) in more than 100 d after BMT[17,34,31,35], and systemic organs and oral mucosa are involved[17].

The aGVHD presents as painful desquamative and ulcerative lesions in oral cavity. Clinical manifestations of cGVHD appear very similar to those of autoimmune connective tissue diseases: white papular eruptions or reticular lesions with areas of erythema, erosion, or ulceration. Generally are symmetrically distributed and the areas of involvement include the tongue, buccal and labial mucosa[31,32,35-36]. It is commonly seen to arise or worsen after an infectious insult or when immunosuppression is reduced[17,31], and they can influence quality of life of patients[36].

Erythema, mucosal atrophy, and lichenoid changes are common oral findings in patients with cGVHD, with lichenoid reactions having the highest positive predictive value[31]. According to a study of Nakamura *et al*[35], OLL were the only clinical sign that had a statistically significant relationship to the diagnosis of cGVHD[25]. Clinically the OLL on cGVHD appears as lacy white striations similar to the striae of Wickham in the OLP[31]. Histologically these reactions consist of a degeneration of the basal cell layer and lymphocytic infiltration in the sub-mucosa. In some cases intracellular edema of epithelial cells can be observed[35].

The OLL can be controlled when treating the systemic GVHD with immunosuppressive therapy. If the oral lesions persist or represent an isolated feature of GVHD, the management with potent topical corticosteroids is generally indicated[32]. Some medications used for the treatment of OLL associated with GVHD are diphenhydramine with kaolin and pectin or clobetasol gargles, topic fluocinonide, oral prednisone (20 to 50 mg/d) or thalidomide (50 to 200 mg/d)[34].

CONCLUSION

In summary, the OLL are a group of intriguing lesion principally when investigates their causal relationship. Moreover, determinate the differential diagnosis of OLL and OLP is important, considering that the different management of this lesions. Thus, epidemiological and laboratorial investigations including a larger number of patients are warranted to elucidate important aspects of OLL until obscures.

REFERENCES

1 **Grossmann Sde M**, Teixeira R, de Aguiar MC, de Moura MD, do Carmo MA. Oral mucosal conditions in chronic hepatitis C Brazilian patients: a cross-sectional study. *J Public Health Dent* 2009; **69**: 168-175 [PMID: 19486464 DOI: 10.1111/j.1752-7325.2009.00119.x]

2 **Pemberton MN**, Sloan P, Thakker NS. Oral lichenoid lesions after hepatitis B vaccination. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000; **89**: 717-719 [PMID: 10846126 DOI: 10.1067/moe.2000.104541]

3 **Juneja M**, Mahajan S, Rao NN, George T, Boaz K. Histochemical analysis of pathological alterations in oral lichen planus and oral lichenoid lesions. *J Oral Sci* 2006; **48**: 185-193 [PMID: 17220615 DOI: 10.2334/josnusd.48.185]

4 **Robledo-Sierra J**, Mattsson U, Svedensten T, Jontell M. The morbidity of oral mucosal lesions in an adult Swedish population. *Med Oral Patol Oral Cir Bucal* 2013; **18**: e766-e772 [PMID: 23792308 DOI: 10.4317/medoral.19286]

5 **Montebugnoli L**, Venturi M, Gissi DB, Cervellati F. Clinical and histologic healing of lichenoid oral lesions following amalgam removal: a prospective study. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2012; **113**: 766-772 [PMID: 22668704 DOI: 10.1016/j.oooo.2011.12.007]

6 **Bernardes VF**, Garcia BG, Souto GR, Novaes-Junior JB, Aguiar MCF, Mesquita RA. Lesão liquenóide oral relacionada ao amálgama. *An Bras Dermatol* 2007; **82**: 549-552 [DOI: 10.1590/S0365-05962007000600008]

7 **Mallo Pérez L**, Díaz Donado C. Intraoral contact allergy to materials used in dental practice. A critical review. *Med Oral* 2003; **8**: 334-347 [PMID: 14595258]

8 **Pezelj-Ribarić S**, Prpić J, Miletić I, Brumini G, Soskić MS, Anić I. Association between oral lichenoid reactions and amalgam restorations. *J Eur Acad Dermatol Venereol* 2008; **22**: 1163-1167 [PMID: 18393961 DOI: 10.1111/j.1468-3083.2008.02736.x]

9 **Karatsaidis A**, Schreurs O, Helgeland K, Axéll T, Schenck K. Erythematous and reticular forms of oral lichen planus and oral lichenoid reactions differ in pathological features related to disease activity. *J Oral Pathol Med* 2003; **32**: 275-281 [PMID: 12694351 DOI: 10.1034/j.1600-0714.2003.00134.x]

10 **Bratel J**, Dahlgren U, Simark Mattsson C, Jontell M. The frequency of different T-cell receptor V-families in oral lichen planus and lichenoid contact lesions: an immunohistochemical study. *J Oral Pathol Med* 1998; **27**: 415-419 [PMID: 9790094 DOI: 10.1111/j.1600-0714.1998.tb01978.x]

11 **Hasséus B**, Jontell M, Brune M, Johansson P, Dahlgren UI. Langerhans cells and T cells in oral graft versus host disease and oral lichen planus. *Scand J Immunol* 2001; **54**: 516-524 [PMID: 11696204 DOI: 10.1046/j.1365-3083.2001.00988.x]

12 **Aminzadeh A**, Jahanshahi G, Ahmadi M. A retrospective comparative study on clinico-pathologic features of oral lichen planus and oral lichenoid lesions. *Dent Res J* (Isfahan) 2013; **10**: 168-172 [PMID: 23946731 DOI: 10.4103/1735-3327.113328]

13 **Thornhill MH**, Pemberton MN, Simmons RK, Theaker ED. Amalgam-contact hypersensitivity lesions and oral lichen planus. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003; **95**: 291-299 [PMID: 12627099 DOI: 10.1067/moe.2003.115]

14 **Thornhill MH**, Sankar V, Xu XJ, Barrett AW, High AS, Odell EW, Speight PM, Farthing PM. The role of histopathological characteristics in distinguishing amalgam-associated oral lichenoid reactions and oral lichen planus. *J Oral Pathol Med* 2006; **35**: 233-240 [PMID: 16519771 DOI: 10.1111/j.1600-0714.2006.00406.x]

15 **Ghaleyani P**, Sardari F, Akbari M. Salivary IgA and IgG in oral lichen planus and oral lichenoid reactions diseases. *Adv Biomed Res* 2012; **1**: 73 [PMID: 23326803]

16 **Issa Y**, Brunton PA, Glenny AM, Duxbury AJ. Healing of oral lichenoid lesions after replacing amalgam restorations: a systematic review. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004; **98**: 553-565 [PMID: 15529127 DOI: 10.1016/j.tripleo.2003.12.027]

17 **Al-Hashimi I**, Schifter M, Lockhart PB, Wray D, Brennan M, Migliorati CA, Axéll T, Bruce AJ, Carpenter W, Eisenberg E, Epstein JB, Holmstrup P, Jontell M, Lozada-Nur F, Nair R, Silverman B, Thongprasom K, Thornhill M, Warnakulasuriya S, van der Waal I. Oral lichen planus and oral lichenoid lesions: diagnostic and therapeutic considerations. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007; **103** Suppl: S25.e1-S25.12 [PMID: 17261375 DOI: 10.1016/j.tripleo.2006.11.001]

18 **Segura-Egea JJ**, Bullón-Fernández P. Lichenoid reaction associated to amalgam restoration. *Med Oral Patol Oral Cir Bucal* 2004; **9**: 423-424 [PMID: 15580119]

19 **Bäckman K**, Jontell M. Microbial-associated oral lichenoid

reactions. *Oral Dis* 2007; **13**: 402-406 [PMID: 17577327 DOI: 10.1111/j.1601-0825.2006.01312.x]
20 **Lygre GB**, Gjerdet NR, Grønningsaeter AG, Björkman L. Reporting on adverse reactions to dental materials--intraoral observations at a clinical follow-up. *Community Dent Oral Epidemiol* 2003; **31**: 200-206 [PMID: 12752546 DOI: 10.1034/j.1600-0528.2003.00037.x]
21 **Ismail SB**, Kumar SK, Zain RB. Oral lichen planus and lichenoid reactions: etiopathogenesis, diagnosis, management and malignant transformation. *J Oral Sci* 2007; **49**: 89-106 [PMID: 17634721 DOI: 10.2334/josnusd.49.89]
22 **Holmstrup P**. Oral mucosa and skin reactions related to amalgam. *Adv Dent Res* 1992; **6**: 120-124 [PMID: 1292451]
23 **Blomgren J**, Axéll T, Sandahl O, Jontell M. Adverse reactions in the oral mucosa associated with anterior composite restorations. *J Oral Pathol Med* 1996; **25**: 311-313 [PMID: 8887075 DOI: 10.1111/j.1600-0714.1996.tb00268.x]
24 **Ostman PO**, Anneroth G, Skoglund A. Amalgam-associated oral lichenoid reactions. Clinical and histologic changes after removal of amalgam fillings. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996; **81**: 459-465 [PMID: 8705594 DOI: 10.1016/S1079-2104(96)80024-2]
25 **Pigatto PD**, Guzzi G, Persichini P, Barbadillo S. Recovery from mercury-induced burning mouth syndrome due to mercury allergy. *Dermatitis* 2004; **15**: 75-77 [PMID: 15473333 DOI: 10.2310/6620.2004.03021]
26 **Hebbar PB**, Pai A, D S. Mycological and histological associations of Candida in oral mucosal lesions. *J Oral Sci* 2013; **55**: 157-160 [PMID: 23748455 DOI: 10.2334/josnusd.55.157]
27 **Guijarro Guijarro B**, López Sánchez AF. Lichenoid reaction caused by Clopidogrel, a new anti-platelet drug. *Med Oral* 2003; **8**: 33-37 [PMID: 12556721]
28 **Abdollahi M**, Radfar M. A review of drug-induced oral reactions. *J Contemp Dent Pract* 2003; **4**: 10-31 [PMID: 12595930]
29 **Nederfors T**, Dahlöf C. Effects on salivary flow rate and composition of withdrawal of and re-exposure to the beta 1-selective antagonist metoprolol in a hypertensive patient population. *Eur J Oral Sci* 1996; **104**: 262-268 [PMID: 8831060 DOI: 10.1111/j.1600-0722.1996.tb00076.x]
30 **McCartan BE**, Lamey P. Lichen planus--specific antigen in oral lichen planus and oral lichenoid drug eruptions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000; **89**: 585-587 [PMID: 10807715 DOI: 10.1067/moe.2000.103885]
31 **Woo SB**, Lee SJ, Schubert MM. Graft-vs.-host disease. *Crit Rev Oral Biol Med* 1997; **8**: 201-216 [PMID: 9167093 DOI: 10.1177/10454411970080020701]
32 **Sedghizadeh PP**, Allen CM, Anderson KE, Kim DH, Kalmar JR, Lang JC. Oral graft-versus-host disease and programmed cell death: pathogenetic and clinical correlates. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004; **97**: 491-498 [PMID: 15088033 DOI: 10.1016/S1079-2104(03)00376-7]
33 **Grossmann Sde M**, Teixeira R, de Aguiar MC, do Carmo MA. Exacerbation of oral lichen planus lesions during treatment of chronic hepatitis C with pegylated interferon and ribavirin. *Eur J Gastroenterol Hepatol* 2008; **20**: 702-706 [PMID: 18679075 DOI: 10.1097/MEG.0b013e3282f1cc5d]
34 **de la Rosa García E**, Bologna Molina R, Vega González Tde J. Graft-versus-host disease, an eight case report and literature review. *Med Oral Patol Oral Cir Bucal* 2006; **11**: E486-E492 [PMID: 17072252]
35 **Nakamura S**, Hiroki A, Shinohara M, Gondo H, Ohyama Y, Mouri T, Sasaki M, Shirasuna K, Harada M, Niho Y. Oral involvement in chronic graft-versus-host disease after allogeneic bone marrow transplantation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996; **82**: 556-563 [PMID: 8936521 DOI: 10.1016/S1079-2104(96)80203-4]
36 **Sato M**, Tokuda N, Fukumoto T, Mano T, Sato T, Ueyama Y. Immunohistopathological study of the oral lichenoid lesions of chronic GVHD. *J Oral Pathol Med* 2006; **35**: 33-36 [PMID: 16393251 DOI: 10.1111/j.1600-0714.2005.00372.x]
37 **Billingham RE**. The biology of graft-versus-host reactions. *Harvey Lect* 1966-1967; **62**: 21-78 [PMID: 4875305]

P- Reviewer: Arabaci T, Bologna-Molina R, Lopez-Jornet P
S- Editor: Ji FF **L- Editor**: A **E- Editor**: Lu YJ