

# Traumatic Oral Mucosal Lesions: A Mini Review and Clinical Update

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## Abstract

Oral mucosal lesions are a common occurrence and very often dentists are called upon to see such patients. Among many causes trauma is one of the leading for oral mucosal diseases. Oral traumatic lesions are diverse in which some present as acute lesions while the majority are chronic lesions. Clinical presentation of traumatic lesions vary significantly and most of the occasions the cause and the effect can be established with thorough history and clinical examination. Although, biopsy of such lesions are not required in most of the occasions some may warrant histological investigations to exclude conditions which clinically mimic traumatic lesions. This paper provides an overview of common and some rare traumatic conditions of the oral mucosa.

## Introduction

Oral lesions associated with trauma are a relatively common finding in dental practice. A diverse array of oral mucosal disorders caused by acute and chronic trauma exists. They manifest in the oral mucosa as acute or chronic ulcers, white or red lesions, mucositis, and reactive hyperplasia or even as bone exposures with sequestration. Such lesions may also impair oral functions to a significant extent and also pose some difficulties in arriving at a diagnosis especially the chronic lesions. However, prompt diagnosis and elimination of the causative factor ensure cure. Injury of the oral mucosa could result from physical, chemical or thermal trauma. They could be originated from accidental dental biting, sharp or pointing food stuff, sharp edges of teeth, hot food or overzealous tooth brushing. Some injuries also could result from iatrogenic damage during dental treatment or other procedures involving oral cavity such as intubation during general anaesthesia. Similarly chemical or physical injury could result from undue or careless handling of chemicals and dental instruments during dental treatment procedures. This paper reviews some common causes of traumatic injuries, their diagnosis and management (*Table 1*).

## Linea Alba

Linea alba is a common finding which is described as a bilateral

raised white line on the buccal mucosae extending from the commissure to the last molar teeth along their occlusal line [1]. In a Turkish study among adolescents (13-16 years of age) linea alba was the second commonest finding which accounts for 5.3% of the total [2]. It is believed that parakeratosis occur along the line of the occlusal plane as the cheeks sucks in due to the negative pressure [1]. Linea alba is asymptomatic and generally considered a normal variation than pathological [3] and therefore, no treatment is required.

## Mucosal Biting

Accidental mucosal biting is a common occurrence which eventually leads to mucosal bleeding and painful ulceration. Such lesions generally heal in a few days' time with no complications. However, chronic habitual biting of the oral mucosa may lead to transient or persistent white patches.

Chronic biting (nibbling) of the buccal mucosa often leads to produce loose thread like keratin shreds, tissue tags or desquamative areas on the mucosal surface [4]. Such lesions have been referred to as "morsicatio buccarum" when occurs on the buccal mucosa (*Figure 1*) and "morsicatio labiorum" and morsicatio linguarum" when occur on labial mucosa and lateral borders of the tongue, respectively [5].

In a retrospective study of 584 patients Woo and Lin [4]

*Table 1. Summary of oral mucosal traumatic lesions and their management.*

Condition	Diagnosis	Common sites of occurrence	Management
Linea Alba	Clinical	Buccal mucosa	Explanation and reassurance, no specific treatment required
Mucosal Biting	Clinical	Buccal mucosa, lip and lateral border of the tongue	Explanation and reassurance, advice on habitual biting
Riga-Fede disease	Clinical	Tip or ventral surface of the tongue	Extraction of neonatal teeth
Eosinophilic ulcer	Biopsy	Gingiva	Spontaneous healing
Ulcers caused by self-mutilation or self-injury	Clinical	Gingiva	Psychological assessment and counselling
Ulcers due to oral trauma in patients with congenital insensitivity to pain	Clinical	Lips and tongue	Symptomatic treatment, prevent trauma from sharp edges of teeth
Electrical and Thermal Burns	Clinical	Lips and tongue	Surgical excision and reconstruction
Trauma associated with sexual practices	Clinical	Soft palate, lips	Symptomatic management
Denture associated hyper keratosis	Clinical	Alveolar ridge	Elimination of irritation from the denture
Denture associated ulcers	Clinical	Buccal and lingual sulci	Trim the denture to eliminate trauma
Inflammatory papillary hyperplasia	Clinical	Palate	Anti-fungal treatment, surgical excision
Epulis fissuratum	Clinical	Buccal sulcus	Surgical excision and vestibuloplasty

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reported a prevalence of 9.0% of such lesions where they refer as “morsicatio mucosa oris”. Although, buccal mucosa is the most commonly affected site [5], this study found lateral border of the tongue as the commonest site accounting for 53.6% and followed by buccal mucosa (32.1%) and labial mucosa (9%). These lesions can be found between 3<sup>rd</sup> to 6<sup>th</sup> decades of life and it is generally resulted from deliberate act of habitual biting or even subconsciously though many patients deny their habit [4,5].

In most cases clinical presentation is sufficient to arrive at the diagnosis and often does not require histological investigation. Oral hairy leukoplakia, uremic stomatitis and chewer’s mucosa are important differential diagnosis. Therefore, if the lesions are seen on the lateral border of the tongue in a patient with high risk of HIV infection further investigations are required to rule out HIV associated hairy leukoplakia [5]. In general no treatment is required and patient education and counseling may often resolve the problem. However, for some patients the use of acrylic shield to cover the facial surfaces of teeth may be used to eliminate the lesion by restricting the access to the lesion sites (lips and buccal mucosa) [5].



**Figure 1.** Cheek biting: A 30 year old male patient with a white patch on both sides of his cheek. Note irregular shaggy surface typical of cheek biting.



**Figure 2.** Traumatic ulcer: A 14 year old male patient with an ulcer on his right lower lip after 2 days of a tooth extraction. Traumatic ulcer due to lip biting after inferior dental nerve block was diagnosed on clinical grounds: Note the irregular margin with slough and exudate on flow of the ulcer.

## Traumatic Ulceration

Traumatic ulcers could result from physical, thermal or chemical injuries. Ulcers resulting from traumatic injuries are probably the most common types of ulcers encountered in clinical practice (*Figures 2 and 3*). Accidental biting during mastication, sharp pointy food may cause acute traumatic ulceration. Such ulcers generally heal within few days without complications. However, chronic trauma from sharp edges of teeth, restorations and appliances particularly ill-fitting dentures may cause chronic ulcers. The majority of such injuries are unintentional however, self-inflicted injuries also can frequently be found. In most occasions the cause and effect relationship can be established.

From two cohorts of patients in Thailand and Malaysia reported prevalence of traumatic ulcers 13.2% and 12.4% from Thailand and Malaysia, respectively [6]. Similarly in another study on elderly Thai patients also reported traumatic ulcers in 15.6% of their study population out of which the majority were due to poor dentures, fractured restorations and sharp edges of teeth [7]. Slightly lower prevalence of traumatic ulcers has been reported from Spain (7.1%) [8], Denmark (4.4%) [9] and Chile (3.5%) [10]. In a large group of Saudi dental patients over the age of 15 years Al-Mobeeriek and Al Dosari (2009) found a prevalence of 1.9% accounting for the third most common oral mucosal disease in that cohort of patients [11].

Chronic traumatic ulcers are commonly found on the mucosa that is subjected to trauma from dentition such as buccal mucosa, lateral border of the tongue or lips. Lesions on other areas including mucobuccal folds and gingivae are associated with other sources of irritation such as trauma from tooth brush or food.

Chronic traumatic ulcers generally present as a solitary shallow or deep discontinuation of epithelium with varying degrees of peripheral keratosis (*Figure 4*). Floor of the ulcer is covered by a white or yellowish fibrin clot. Ulcer resulted from repeated trauma may be symptomatic or asymptomatic, often exhibits an elevated border which is firm on palpation. Upon elimination of the causative factor often ulcer heals with or without scar depending on the extent of the damage occurred.



**Figure 3.** Traumatic ulcer: A 13 year old male patient with an ulcer on the ventral surface of the tongue for a period of 3 months. Traumatic ulcer on the ventral surface of the tongue was caused by lingually erupted mandibular central incisor.



**Figure 4.** Traumatic ulcer: A 60 year old man with a chronic ulcer on the left posterior lateral border of the tongue caused by lingually tilted mandibular 3rd molar. Note central ulceration with peripheral keratosis.

### Riga-Fede Disease

Riga-Fede disease is a reactive traumatic ulcerative disorder with benign course often seen on tip or ventral surface of the tongue [5,12,13]. Lesions tend to occur after the eruption of neonatal, natal or primary incisors. They are commonly observed in relation to the lower incisors believed to be caused by repetitive trauma associated with back and forward movement of the tongue against them. Although rare, they can also be reported on the dorsal surface of the tongue and the lower lip.

A number of terms have been used to describe these lesions including “traumatic lingual ulceration”, eosinophilic granuloma”, “traumatic eosinophilic ulceration of the tongue” and traumatic granuloma of the tongue [12,13].

van der Meij et al. [13] in a review summarized 34 cases (age range 0.3-24 months) that they found since 1902. Of them 82% of the lesions were found on the ventral surface of the tongue while rest of the lesions was equally distributed on the dorsum of the tongue and the lower lip. Only seven lesions (20.5%) were associated with neonatal teeth while the rest of the lesions were caused by primary teeth with a mean age of onset at 10 months. Overall, lesions were more commonly found among males with a male to female ratio of 1.8:1 [13].

Clinically these lesions appear as ulcerated area with fibrinous exudate with varying degrees of raised margins depending on the duration and frequency of trauma [12,13]. Generally the diagnosis is established clinically alone and rarely biopsy is required. Several treatment options have been described which principally aiming at elimination of the causative factor. The treatment of choice for lesions associated with neonatal teeth is extractions of such teeth. Contrary to this, more conservative approaches such as placement of a protective barrier or grinding of the sharp edges of teeth have also been attempted. However, in some intractable cases extraction of causative teeth is the only option [13].

### Eosinophilic Ulcer

Eosinophilic ulcer is a chronic, often self-limiting benign

lesion which is usually manifested on the lateral border of the tongue [14,15]. However, this has also been reported to occur in the gingiva and rarely on other sites [15,16]. Since its first description it has been described in a number of different names: traumatic granuloma, Traumatic Ulcerative Granuloma with Stromal Eosinophilia (TUGSE), atypical histiocytic granuloma [5,14-18]. When it occurs in infants and children in association with natal, neonatal or primary teeth it has been termed as Riga-Fede disease [12].

Lesions are commonly seen among individuals of 4th to 5th decades of life with equal sex distribution. Eosinophilic ulcer commonly presents as solitary indurated or granulomatous ulcer which may sometime mimic a malignant lesion [15]. Therefore, confirmation of the diagnosis may need an incisional biopsy. Generally ulcers tend to heal spontaneously but slowly over a period of 3-10 weeks. Some lesions heal promptly after carrying out a biopsy [15,19].

### Necrotizing Sialometaplasia

Necrotizing Sialometaplasia (NS) is first described by Abrams et al. as an uncommon inflammatory necrotizing process involving minor salivary glands predominantly affecting the hard palate [20]. Although the exact aetiology is not known, the mostly suspected and generally accepted underlying mechanism is ischaemia of minor salivary gland tissues [20-22]. Several predisposing factors which lead to ischaemia have been proposed by these authors. Brannon and coworkers reported that they observed after NS 8 days on average following a surgical procedure in 36% of their case series. Other traumatic episodes such as dental injection and wearing ill-fitting dentures were also been implicated in some cases [21]. In addition to trauma, smoking and alcohol consumption, allergy and upper respiratory tract infections were also considered as predisposing to NS [21].

Clinically NS presents as a solitary unilateral ulcer on the hard palate (*Figure 5*) [20-22]. However, a number of cases were also reported as non-ulcerated swelling or mass [21]. Lesions could accompany with pain or numbness while some may be asymptomatic [5,21-23]. Although a wide variation of



**Figure 5.** Necrotizing sialometaplasia: A 55 year old male with an ulcer on his left side of the palate. Clinical diagnosis of necrotizing sialometaplasia was made and the lesion healed in 14 days after the diagnosis. These ulcers may mimic squamous cell carcinoma clinically as well as histologically.

age of onset had been reported, NS is more commonly seen in adults with the mean age of 46 years and more commonly affecting males [21]. The most important clinical significance is that the lesion mimics malignancy clinically as well as histologically; squamous cell carcinoma and mucoepidermoid carcinoma in particular [5,23]. Although history of traumatic event could be of importance in arriving at diagnosis, due to the worrisome appearance of the lesion it is recommended to carry out an incisional biopsy. Lesions generally heal spontaneously in an average period of 5-6 weeks [5].

### **Factitious Disorders and Trauma**

Factitious mucosal lesions are not uncommon. Self-mutilation or self-injury is a behavioural disturbance that leads to deliberate harm to own body tissues and not associated with intention to commit suicide [24]. Although the exact prevalence in the world population is not known, it affects people regardless of their age, sex or ethnicity [25]. The aetiology can be of two fold; organic and functional. Organic self-mutilation injuries include injuries in comatose patients, patients with some syndromes such as Lesch-Nyhan syndrome, De Lange syndrome, Tourette syndrome, Leigh syndrome, patients with autism spectrum disorders and familial dysautonomia [26-30]. Functional self-mutilation refers to intentional self-injuring in physically healthy patients without detectable genetic disorders [25]. Oral self-mutilation injuries are commonly seen among children than adolescents or adults. In a recent review Hilderbrand et al. [25] reported 15 cases including their case among children with an age range of 3-13 years. Among them over 90% of the lesions were observed in the gingivae. Clinically such lesions appear as ulcers on the gingiva or as gingival recession [25]. Diagnosis and management of self-mutilation injuries are generally difficult and thorough medical history and physical examinations are of utmost importance in arriving at correct diagnosis. Patient's psychological assessment is mandatory and multidisciplinary approach helps to carry out overall management of the affected individual.

### **Oral Trauma due to Congenital Insensitivity to Pain**

Familial insensitivity to pain is a rare clinical syndrome in which affected individuals pain sensation is impaired. Individuals affected by this syndrome are generally normal in terms of their physical and mental health and with normal perception of other sensations [31]. This condition is generally present at birth and the majority of cases are diagnosed at early stages of life. Due to lack of pain sensation, self-inflicted and unintentional oral injuries are common especially to lips and tongue. Injuries to dental hard tissues are also common [32]. Oral mucosal lesions are generally observed when primary teeth erupt. Lips and tongue are the commonest sites to be injured. No specific management strategy is available to date however, preventive measures such as grinding and smoothening sharp edges of teeth may help prevent the injuries.

### **Electrical and Thermal Burns**

Oral electrical burns are relatively common and predominantly

occur in young children. Such injuries often lead to disfigurement and functional disabilities [5,33]. There are two types of electrical burns; contact and arc types. Contact type require a good ground and electrical current passes through the body from the site of contact to the ground where as in arc type burns saliva act as a conducting medium and current flows between the source and the mouth. The resultant high temperature causes electrical burn leading to severe destruction of oral tissues [33]. Most of the electrical injuries occur as a result of sucking or chewing of live electrical wires, extension cords, plugs or power outlets. Hence, they most commonly occur in toddlers or preschool children. Such injuries in adults are extremely rare [34]. Electrical trauma most commonly affects the lips, and present as painless, charred yellowish area with little or no bleeding followed by significant oedema within few hours [5]. Necrosed tissues slough and heal with scarring leading to significant disfigurement [33].

Thermal injuries to oral soft tissues most commonly occur due to accidental ingestion of hot food or beverages. Food associated thermal injuries generally occur in the palate or the posterior buccal mucosa. They appear as sloughy necrotic tissue surrounded by erythema [5].

Management of electrical burn injuries are difficult and often challenging due to extreme contractions leading to disfigurement and functional disability. Several methods have been attempted including excision and reconstruction and use of burn appliances [33]. Management of thermal burns is not as complicated as electrical burns and generally heals with fewer complications [5,35].

### **Chemical Injuries**

A wide array of chemicals and drugs come into contact with oral mucosa and some of them may cause direct mucosal trauma due to their caustic nature. Some people tend to apply certain chemicals as a remedy for their oral problems such as application of aspirin to ease toothache [19]. Health care practitioners are also responsible in applying certain chemicals in the mouth for treatment or investigative procedures. Some of such chemicals if used inadvertently can be potentially injurious. However, such injuries are not very common since the introduction of rubber dam in dental practice.

### **Sexual Practices and Oral Mucosal Trauma**

Oro-genital stimulation has become a popular practice during the last few decades and this is more common among homosexual males and females [5,36]. It has been reported that 90% of the married heterosexual couples under the age of 25 years also practice oro-genital sex [36]. However, the prevalence of trauma related to oro-genital stimulation is surprisingly low [5]. Despite this it is imperative for the dental practitioners to be familiar with the cause and the oral manifestations of lesions associated with oro-genital sex. Fellatio is the commonest among oro-genital sexual practices that causes oral trauma. This generally manifests as erythema, ecchymosis or petechiae in the soft palate [5]. These lesions may be noticed during routine oral examinations and the lesions are generally asymptomatic and heal within 7-10 days. It is also of utmost importance for the dentists to be vigilant of child abuse if such lesions are found in children.



**Figure 6.** Papillary hyperplasia of the palate: A 70 year old female with papillary hyperplasia of the palate; this lesion is localized to the area of retention device. However, in most of the cases lesion could be widespread in the palate.



**Figure 7.** Epulis fissuratum (inflammatory fibrous hyperplasia) in a 70 year old female denture wearer: Note the tumour like fibrous connective tissue on the alveolar ridge and extending to the labial sulcus. Lesion develops as a result of chronic irritation due to ill-fitting denture.

### Denture Associated Mucosal Trauma

Denture wearing can cause a number of acute or chronic oral mucosal problems ranging from histological to gross clinical changes [37]. Gross clinical changes include keratotic, hyperplastic, inflammatory and ulcerative lesions [38]. In a retrospective clinical study using 200 denture wearing patients Dorey et al. [38] reported a positive relationship of oral mucosal abnormalities and denture wearing in 60% of their cohort of patients. Of these, chronic trauma from denture was the most common cause accounting for 17% and presented in the form of stomatitis with or without candida infection. Other problems encountered were; hyperkeratosis (12%), ulceration (8%), inflammatory papillary hyperplasia (6%) and epulis fissurata (2%).

In response to chronic irritation from dentures particularly

exposure to masticatory forces oral mucosa shows acanthosis, rete hyperplasia and parakeratosis [37]. Hyperkeratosis is regarded as a normal adaptation to function and resolve once the irritation is removed. Traumatic ulcers generally occur soon after the insertion of new dentures. They often appear over the sharp bony ridges where the mucosa is sandwiched between the denture and bone, under spicules or high spots of dentures. Such trauma produces erythema, oedema and subsequently ulceration which generally produce soreness or pain preventing patients a comfortable mastication. Careful elimination of such trauma leads to complete resolution of the problem.

Inflammatory Papillary Hyperplasia (IPH) is a reactive form of tissue overgrowth clinically present as a nodular or papillary surface covering completely or partially the area of the palate covered by the denture (Figure 6) [37-39]. Although the exact aetiology is unknown, the condition is most often suspected to be related to; continuous wearing of ill-fitting dentures with poor hygiene that often causing trauma together with predisposition to Candidal infection [5,40]. Lesions are directly related to age of the patient and the length of denture usage [41]. IPH is usually asymptomatic and often identified during routine clinical examination. Very early lesions may subside once the irritation is eliminated together with the use of antifungal agents. However, more established lesions may need surgical removal, curettage, electrosurgery or cryotherapy [5].

Epulis fissuratum (inflammatory fibrous hyperplasia) is a tumour like fibrous connective tissue lesion that develops as a result of ill-fitting denture flange. Clinically, it presents as a sessile mass in the sulcus with smooth surface and normal mucosal colour and also may be erythematous and ulcerated (Figure 7) [5,42]. This is most often seen among middle aged and older adults occurring either in relation to the mandible or maxilla and shows a female predilection. Treatment is often involved with excision of the lesion with vestibuloplasty and correction of the denture.

### Summary

This paper gives an overview of traumatic lesions of the oral mucosa. The paper summarizes the common to rare disorders causing traumatic lesions. Diverse array of causative factors can be identified. Commonly encountered lesions are generally straightforward to diagnose and hence easy to manage by elimination of the causative factor and enhancing the healing of the lesion. If chronic ulcer persists after the elimination of the suspected causative factor within a reasonable time limit (2-3 weeks) a biopsy should be considered to confirm the diagnosis. Thorough clinical examination and attention to all possible aspects of causative factors are mandatory for complete resolution of the lesion.

### References

1. Seoane LJM, Aguado SA, Varela-Centelles PI, Vazquez GJ, Romero MA, et al. Oral Mucosa: variations from normalcy, part I. *Cutis*. 2002; **69**: 131-131.

2. Parlak AH, Koybasi S, Yavuz T, Yesildal N, Aydogan I, et al. Prevalence of oral lesions in 13 to 16 year old students in Duzce, Turkey. *Oral Diseases*. 2006; **12**: 552-558.

3. Bhattacharyya I, Chehal HK. White lesions. *Otolaryngologic Clinics of North America*. 2011; **44**: 109-131.

4. Woo SB, Lin D. Morsicatio Mucosae Oris-A chronic oral frictional Keratosis, Not a Leukoplakia. *Journal of Oral and Maxillofacial Surgery*. 2009; **67**: 140-142.
5. Neville BW, Damm DD, Allen CM, Bouquet JE. *Oral and Maxillofacial Pathology*. (3rd edn.) Philadelphia: Saunders. 2009; pp. 285-329.
6. Axéll T, Zain RB, Siwamogstham P, Tantiran D, Thampipit J. Prevalence of oral soft tissue lesions in out-patients at two Malaysian and Thai dental schools. *Community Dentistry and Oral Epidemiology*. 1990; **18**: 95-99.
7. Jainkittivosng A, Aneksuk V, Langlias RP. Oral mucosal conditions in elderly dental patients. *Oral Diseases*. 2002; **8**: 218-223.
8. Garcia-Pola VMJ, Martínez DCAI, García MJM, González GM. Risk factors for oral soft tissue lesions in and adult Spanish population. *Community Dentistry and Oral Epidemiology*. 2002; **30**: 277-285.
9. Vigild M. Oral mucosal lesions among institutionalized elderly in Denmark. *Community Dentistry and Oral Epidemiology* 1987; **15**: 309-313.
10. Espinoza I, Rojas R, Aranda W, Gamonal J. Prevalence of oral mucosal lesions in elderly people in Santiago, Chile. *Journal of Oral Pathology and Medicine*. 2003; **32**: 571-575.
11. Al-Mobeeriek A, Al-Dosari AM. Prevalence of oral lesions among Saudi Dental Patients. *Annals of Saudi Medicine*. 2009; **29**: 365-368.
12. Ceyhan AM, Yildirim M, Basak PY, Akkaya VB, Ayata A. Traumatic lingual ulcer in a child: Riga –Fede disease. *Clinical and Experimental Dermatology*. 2009; **34**: 186-188.
13. van der Meij E, de Vries, Eggink HF, de Visscher JGAM. Traumatic lingual ulceration in a new born: Riga-Fede disease. *Italian Journal of Pediatrics*. 2012; **38**: 20.
14. Compilato D, Cirillo N, Termine N, Kerr AR, Paderni C, et al. Long-standing oral ulcers: proposal for a new ‘S-C-D classification system’. *Journal of Oral Pathology and Medicine*. 2009; **38**: 241-253.
15. Horie N, Shimoyama T, Kato T, Ide F. Eosinophilic ulcer of the tongue: a case report with immune-histochemical study. *Oral Medicine and Pathology*. 1999; **4**: 25-29.
16. Tornes K, Bang G. Traumatic eosinophilic granuloma of gingiva. *Oral Surgery*. 1974; **38**: 99-102.
17. Regezi JA. Oral traumatic granuloma: characterization of the cellular infiltrate. *Oral Surgery Oral Medicine Oral Pathology*. 1993; **75**: 723-727.
18. Hirshberg A, Amariglio N, Akrisch S, Yahalom R, Rosenbaum H, et al. Traumatic ulcerative granuloma with stromal eosinophilia: A reactive lesion of the oral mucosa. *American Journal of Clinical Pathology*. 2006; **126**: 522-529.
19. Cawson RA, Odell EW. *Cawson's Essentials of Oral Pathology and Oral Medicine*. (8th edn) Edinburgh: Elsevier. 2008; pp. 237-238.
20. Abrams AM, Melrose RJ, Howell FV. Necrotizing sialometaplasia: a disease simulating malignancy. *Cancer*. 1973; **32**: 130-135.
21. Brannon RB, Fowler CB, Hartman KS. Necrotizing sialometaplasia: A clinicopathological study of sixty nine cases and review of literature. *Oral Surgery Oral Medicine Oral Pathology*. 1991; **72**: 317-325.
22. Anneroth G, Hansen L. Necrotizing sialometaplasia: the relationship of its pathogenesis to its clinical characteristics. *International Journal of Oral Surgery*. 1982; **11**: 283-291.
23. Carlson DL. Necrotizing sialometaplasia: A practical approach to diagnosis. *Archives of Pathology and Laboratory Medicine*. 2009; **133**: 692-698.
24. Favazza AR. Why patients mutilate themselves. *Hospital and Community Psychiatry*. 1989; **40**: 137-145.
25. Hilderbrand LC, Carvalho AL, da Rosa EM, Martins MD, Filho MS. Functional oral self-mutilation in physically healthy pediatric patients: Case report and analysis of 27 literature cases. *International Journal of Pediatric Otorhinolaryngology*. 2011; **75**: 880-883.
26. Kobayashi T, Ghanem H, Umezawa K, Mega J, Kawara M, et al. Treatment of self-inflicted oral trauma in a comatose patient: A case report. *Journal of Canadian Dental Association*. 2005; **71**: 661-664.
27. Shimoyama T, Horie N, Kato T, Nasu D, Kaneko T. Tourette's syndrome with rapid deterioration by self-mutilation of the upper lip. *Journal of Clinical Pediatric Dentistry*. 2003; **27**: 117-180.
28. Cauwels RGE, Martens LC. Self-mutilation behavior in Lesch-Nyhan Syndrome. *Journal of Oral Pathology and Medicine*. 2005; **34**: 573-575.
29. Medina AC, Sogbe R, Gomez-Rey AM, Mata M. Factitial oral lesions in an autistic pediatric patient. *International Journal of Pediatric Dentistry*. 2003; **13**: 130-137.
30. Mass E, Gadoth N. Oro-dental self-mutilation in familial dysautonomia. *Journal of Oral Pathology and Medicine*. 1994; **23**: 273-276.
31. Thompson CC, Park RI, Prescott GH. Oral manifestations of congenital insensitivity to pain syndrome. *Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontics*. 1980; **50**: 220-205.
32. Butler J, Felming P, Webb D. Congenital insensitivity to pain-review and report of a case with dental implications. *Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontics*. 2006; **101**: 58-62.
33. Linebaugh ML, Koka S. Oral electrical burns: Etiology, Histopathology and Prosthetic treatment. *Journal of Prosthodontics*. 1993; **2**: 136-141.
34. Shimoyama T, Kaneko T, Nasu D, Suzuki T, Horie N. A case of an electrical burn in the oral cavity of an adult. *Journal of Oral Science*. 1999; **41**: 127-128.
35. Baruchin AM, Lustig JP, Nahlieli O, Amos N. Burns of the oral mucosa: Report of 6 cases. *Journal of Cranio-Maxillofacial Surgery*. 1991; **19**: 94-96.
36. Damm DD, White DK, Brinker M. Variations of palatal erythema secondary to fellatio. *Oral Surgery Oral Medicine Oral Pathology*. 1981; **52**: 417-421.
37. Cook RJ. Response of the oral mucosa to denture wearing. *Journal of Dentistry*. 1991; **19**: 135-147.
38. Dorey JL, Blasberg B, McEntee MI, Conklin RJ. Oral mucosal disorders in denture wearers. *Journal of Prosthetic Dentistry*. 1985; **53**: 210-213.
39. Budtz-Jorgensen E. Oral mucosal lesions associated with the wearing of removable dentures. *Journal of Oral Pathology*. 1981; **10**: 65-80.
40. Pouloupoulos A, Belazi M, Epivatianos A, Velegraki A, Antoniadis D. The role of candida in inflammatory papillary hyperplasia of the palate. *Journal of Oral Rehabilitation*. 2007; **34**: 685-692.
41. Turker SB, Sener ID, Koçak A, Yılmaz S, Özkan YK. Factors triggering the oral mucosal lesions by complete dentures. *Archives of Gerontology and Geriatrics*. 2010; **51**: 100-104.
42. Niccolo-Filho W, Neves ACC, Penna LAP, Seraidarian PI, Riva R. Removal of epulis fissuratum associated to vestibuloplasty with carbon dioxide laser. *Lasers in Medical Science*. 1999; **14**: 203-206.