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Oral Soft Tissue Lesions in Children

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ABSTRACT

Lips, teeth, gums, oral mucous membranes, palate, tongue, and the oral lymphoid system comprise the oral cavity. Oral cavity soft tissue lesions are extremely common in children, and they can present clinically as coloured lesions, mucosal ulceration, or Nodular lesions. The appearance of the oral mucosa alters to reflect the underlying health status, which could be local or systemic. While most oral diseases can be diagnosed visually, some disorders can be befuddling, and their diagnoses can be elusive. Pigmentation is defined as the process of pigment deposition in tissues. This article provides a brief overview of oral pigmented lesions in children

Keywords: *oral* soft tissue lesions, mucosal abnormalities, children, paediatric dentistry

INTRODUCTION

Soft tissue lesions in pediatric dentistry comprises of a wide variety of mucosal abnormalities. Soft tissue lesions of the oral cavity in children may be normal/developmental findings or indicative of gingivitis, periodontal disease, local or systemic infection, benign tumors, or life-threatening systemic conditions. Various mucosal abnormalities as well as proliferative and destructive lesions can occur in the oral cavity. Their causes include infectious agents, metabolic disorders, endocrinopathies, injuries, neoplasms, developmental anomalies, genetic syndromes, and immunologic disturbances. Soft tissue lesions of the oral cavity are common in children, have various clinical presentations as coloured lesions, mucosal ulceration or nodular lesions and so.

Some are a symptomatic and some are symptomatic or even disturb the daily activities of the child. When evaluating soft tissue lesions, it is important to distinguish between findings that are normal and that are indicative a pathological condition, and normal anatomical variations. Oral pathological conditions presented in children are various and often different from those of adults. The nature of many pediatric lesion changes with growth and development of the body. There is considerable variation in the prevalence of these lesions among different regions of the world as racial and environmental specificities, and the lifestyle of each population may influence the prevalence of these diseases. In spite of the World Health Organization (WHO) suggestions regarding the epidemiologic assessment of oral lesions, the majority of studies on oral conditions in children have been limited to investigation of caries, periodontal disease, malocclusion, and dental trauma.

The oral mucosa serves several functions. The major one is protection of the deeper tissues of the oral cavity; others include acting as a sensory organ and serving as the site of glandular activity and secretion. Oral cavity is lined by a mucous membrane that consists of two layers: an epithelium and subjacent connective tissue (the lamina propria). Although its major functions are lining and protecting, the mucosa also is

modified to serve as an exceptionally mobile tissue that permits free movement of the lip and cheek muscles. Histologically, the oral mucosa can be classified into three types: (i) masticatory (ii) lining (iii) specialized.

Soft tissue lesions can be classified into congenital lesions, mucosal lesions (white lesions and red lesions), nodules, benign tumors, cyst and infections. They can also be classified into ulcerative, vesico-bullous, and papillary. While diagnosis of the wide variety of mucosal lesions which occur in the oral cavity is an essential part of dental practice, there are relatively few systematic studies of the prevalence of such lesions in children and youths. This is a critical deficiency since appropriate diagnosis and treatment requires knowledge of the relative frequency or probability of possible lesions.

CLASSIFICATION

Table 1: Classification of Pigmented Oral Lesions in Children:-[1,2]

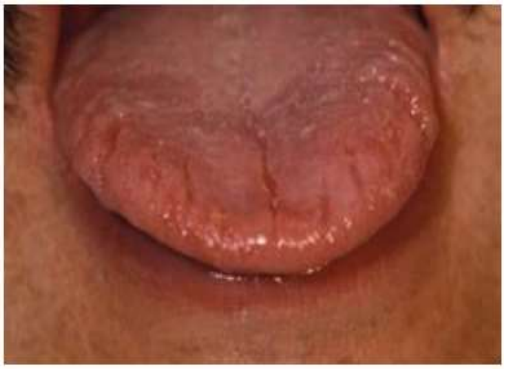
ORAL LESIONS IN CHILDREN	TYPE
DEVELOPMENTAL LESIONS	Geographic Tongue, Fissured Tongue, Retro Cuspid Papilla, Gingival Overgrowth.
MUCOSAL CHANGES(PIGMENTED)	**White lesions**: Linea alba Leukoedema, Pseudo Membranous - Candidiasis, White Sponge Nevus. **Red and/or white lesions**: Petechiae, Purpura, Ecchymosis, Erythematous Candidiasis, Angular Chelitis, Erythema Migrans (Benign Migratory Glossitis), Median Rhomboid Glossitis.
BROWN-BLACK LESIONS	Physiologic Pigmentation, Amalgam Tattoo/Graphite, Melanotic Nevus
SOFT TISSUE NODULES	**Inflammatory/reactive lesions**: Mucocele, Irritation Fibroma, Peripheral Ossifying Fibroma, Pyogenic Granuloma, Peripheral Giant Cell Granuloma
BENIGN TUMORS	Hemangioma, Lymphatic Malformations, Fibroma, Benign Neoplasms, Squamous papilloma.
CYSTS	Eruption Cyst
ULCERATIONS	Traumatic Ulcers, Aphthous Ulcers
INFECTIONS	Herpes Simplex Virus, Coxsackievirus, Herpangina, Hand-Foot Mouth Disease, Candida albicans, HIV Infection

DEVELOPMENTAL ANOMALIES

These lesions may be present at birth or become evident later in life[3]. Mucosal lesions may be discovered during routine dental examinations and vary depending on age, gender and /or race. Majority of oral diseases are confined to oral tissues, but numerous underlying systemic conditions may manifest with signs and symptoms within the oral cavity.[4] The tongue lesions fissured tongue, geographic tongue, median rhomboid glossitis and Retro cuspid papillae are classically considered as developmental lesions.

FISSURED TONGUE:

Also known as scrotal tongue.[5] May be seen located on the dorsal and lateral part of tongue.[1] Overall prevalence of geographic tongue is approximately 1-2.5% of the population.[2] Females are more commonly affected than males with the ratio of 1.5:1.4 Some studies have reported this condition to affect males more frequently.[3] Despite of many studies, the etiology of fissured tongue remains obscure, but several conditions associated with fissure tongue have been reported including psoriasis, orofacial granulomatous, pernicious anaemia, low serum levels of vitamin A, down syndrome, diabetes mellitus and certain autoimmune diseases.[4] Furthermore, fissure tongue is one of the three symptoms of Melkersson Rosenthal Syndrome (MRS). It may also be a normal variation of tongue architecture.[6]



Patients with fissured tongue exhibit multiple grooves, or furrows, on the surface

of the tongue, ranging from 2-6 mm in depth. In the most severe cases, numerous fissures cover the entire dorsal surface and divide the tongue papillae into multiple separate "islands."[7] Some patients have fissures that are located mostly on the dorsolateral areas of the tongue. Other patients exhibit a large central fissure, with smaller fissures branching outward at right angles.

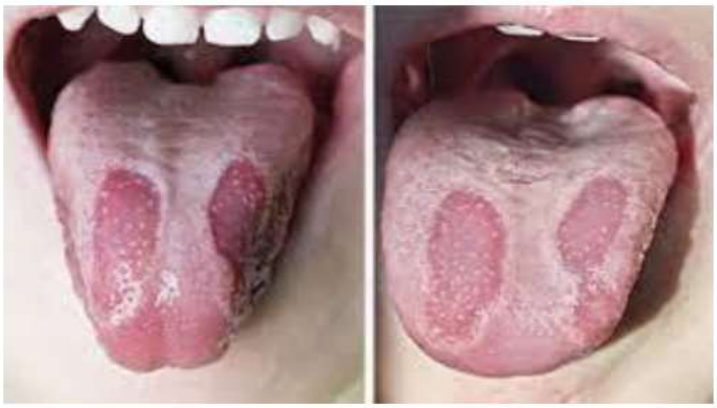
Condition is usually asymptomatic; some patients may complain of mild burning or soreness. Strong association has been found between fissured tongue and geographic tongue with many patients having both conditions. A hereditary basis has also been suggested for geographic tongue, and the same gene or genes may possibly be linked to both conditions. The pathophysiological characteristics of FT include intraepithelial land subepithelial infiltrates of polymorphonuclear leucocytes and lymphocytes. Clear histo-quantitative differences in the epithelium, connective tissue and uppermost muscles layers have been found between FT and healthy tongue samples, suggesting that the subepithelial connective tissues of FT may also differ from the healthy tongue.

Differential diagnosis may include erythema migrans, macroglossia with crenations, hemihyperplasia of tongue, orofacial granulomatosis. Treatment and Prognosis Fissured tongue is a benign condition, and no specific treatment is indicated. The patient should be encouraged to brush the tongue, because food or debris entrapped in the grooves may act as a source of irritation.

GEOGRAPHICAL TONGUE

Geographic tongue is a condition referred to by a variety of terms such as: benign migratory glossitis, erythema migrans, annulus migrans, or wandering rash of the tongue.[8] It is characterized by periods of remission and exacerbation of varying duration. Overall prevalence of geographic tongue is approximately 1-2.5% of the population. The prevalence of GT in the pediatric population ranges from 0.37% to 14.3% with unknown etiology. Several related etiologic factors have been proposed, however, none of the suggested causes provide clear-cut evidence of a causal relationship.[9]

Clinically lateral margins and tip of the tongue are the most commonly involved sites followed by dorsal and ventral surfaces. If the lesions occur in different sites other than tongue, then the term ectopic geographic tongue. Also, lesion of the geographic tongue may reveal erythematous atrophic areas with loss of filiform papillae surrounded by the white circinate borders. It can be either isolated or seen as multiple lesions, can be asymptomatic or present symptoms like pain, burning sensation, discomfort, dysgeusia, sensitivity to hot, spicy and sour food pain in ears or ipsilateral submandibular lymphadenopathy. presence of deep fissures infected with candida organisms could be responsible for symptoms.

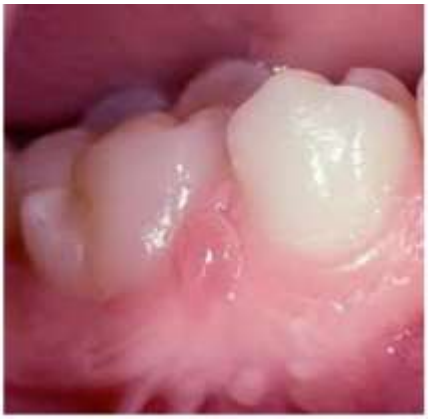


Differential diagnosis of GT includes: Atrophic candidiasis, Neutropenia, Psoriasis, Reiter's syndrome, Leukoplakia, Lichen planus, Systemic lupus erythematosus, Herpes simplex, Drug reaction. Geographic tongue typically does not require any treatment if asymptomatic. Periodic follow up to confirm diagnosis is required in case of fist visit and when history is unclear. If there is excessive pain and discomfort, medications like analgesics such as acetaminophen, anti- inflammatory drugs, antihistamines like diphenhydramine hydrochloride, mouth rinses with topical anaesthetics like lidocaine gel, topical corticosteroids like betamethasone, cyclosporine, vitamin A therapy like tretinoin, zinc and vitamin k supplements can be advised.

RETROCUSPID PAPILLA

Retrocuspid papilla is a slightly raised area of mandibular alveolar mucosa, which as the name implies, is commonly located lingual to the cuspids.[10] Occurs in first and second decade of life with more female predilection. Usually seen at lingual attached gingiva or adjacent to mandibular canines. A study revealed that 67% of children were having retrocuspid papillae in 3-5 years age group and 30% in 7-9 age groups. Earlier studies have indicated that prevalence of RCP decrease with increasing age [11].

It is asymptomatic, pink sessile papule or nodule and usually bilateral. This structure measures about 2 to 4 mm and is often present bilaterally between the marginal gingiva and the mucogingival junctions. Retrocuspid papilla is more common among children and it has structural resemblance to the incisive papilla.[12] Histologically, the papilla represents a focus of fibrovascular tissue with an ortho-keratinized or para-keratinized surface and it usually covers an osseous foramen of a nutrient blood vessel.[13]



DIFFERENTIAL DIAGNOSIS: Irritation fibroma, Giant cell fibroma, soft tissue abscess.

TREATMENT AND PROGNOSIS: No treatment required. It can also appear as a normal anatomic variation if left untreated.[14]

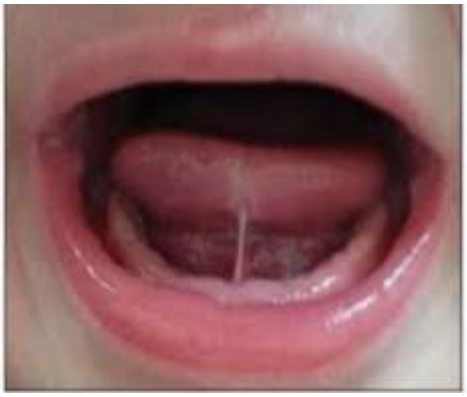
PARTIAL ANKYLOGLOSSIA

Ankyloglossia can be defined as a congenital developmental condition characterized by fixation of the tongue to the floor of the mouth; causing restricted tongue mobility. Also known as Tongue Tie. May be Present at Birth. With more Male Predilection that can be located on the Ventral surface of Tongue and Floor of the mouth. It occurs in 2% - 4% of children; rarely causes speech, feeding, swallowing or periodontal problems; multiple frenula associated with oral-facial digital syndrome. [15]

Incidence and prevalence - AG incidence varies from 0.02% to 5%, depending on the study, its definition of AG, and population examined. AG may occur with increased frequency in various congenital syndromes, including Opitz syndrome, orofaciodigital syndrome, Beckwith-Wiedemann syndrome, Simpson-Golabi-Behmel syndrome, and X-linked cleft palate. [16]

ETIOLOGY: Complete ankyloglossia is an extremely rare condition, however partial ankyloglossia, which is otherwise known as "tongue-tie" is a relatively common developmental anomaly of the tongue. It

occurs either due to a short and thick lingual frenulum (a membrane connecting the under surface of the tongue to the floor of the mouth) or due to a frenulum, which attaches too near to the tip of the tongue. According to few investigators this condition may sometimes develop as a result of trauma. Recent study of Harris EF et al indicate that incidence of tongue tie may increase with maternal use of cocaine during pregnancy. [17]



CLINICAL FEATURES: Although most affected individuals can perform tongue functions almost normally, the restricted tongue mobility in ankyloglossia may sometimes cause the following problems:

- Speech disorders
- patient cannot properly pronounce certain consonants and diphthongs such as L, R, T, D, N, TH, SH, Z, etc.
- Infants feel difficulty in sucking breast milk.
- Deformities in dental occlusion especially development of open bite and mandibular prognathism, etc.
- Difficulty in swallowing food and maintaining oral hygiene.
- Tension in the anterior lingual gingiva in tongue-tie may initiate some localized gingival or periodontal diseases near the site of frenal attachment (e.g.gingival recession and persistent gap between lower incisors).

Ankyloglossia may occur sometimes in association with the following syndromes: Ankyloglossum spurious syndrome, Vander Woudes syndrome, Fraser's syndrome, Rainbow's syndrome, Orofacial digital syndrome, etc. [18]

DIFFERENTIAL DIAGNOSIS • Complete ankyloglossia • Bifid tongue • Microglossia

TREATMENT AND PROGNOSIS: Because most cases of partial ankyloglossia result in few or no clinical problems, treatment is often unnecessary. For infants with specific breast-feeding problems, a frenotomy ("clipping" or simple release of the frenulum) can be performed. In children or adults with associated functional or periodontal difficulties, a frenuloplasty (release with plastic repair) may allow greater freedom of tongue movement. In young children it often is recommended that surgery be postponed until age 4 or 5, because the tongue is always short at birth, assessing the degree of tongue limitation caused by ankyloglossia is difficult in the infant's early life. As the infant grows, the tongue becomes longer and thinner at the tip, often decreasing the severity of the tongue-tie. The condition probably is self-correcting in many cases because it is less common in adults.

MUCOSAL LESIONS

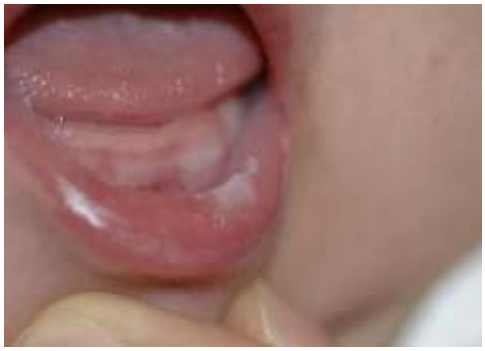
Mucosal lesions can be divided in following categories based on their color.

WHITE SURFACE LESIONS	RED LESIONS	BROWN AND BLACK LESIONS
Linea alba	Petechiae	Physiologic pigmentation
Leukoedema	Purpura	Amalgam tattoo
Pseudomembranous candidiasis	Ecchymosis	Melanotic nevus
White sponge nevus	Angular chelitis	
Frictional keratosis	Erythema migrans	
	Median rhomboid glossitis	
	Erythematous candidiasis	

FRICTIONAL KERATOSIS

It is a whitish lesion with a roughened keratotic surface that results from long-term mechanical irritation.[19] Located mainly on the buccal, labial, lateral, and gingival mucosa that are next to the occlusal plane. The first and second decade of childhood are mostly affected. It is caused by long-term habits of biting or sucking, discomfort from orthodontic appliances, broken teeth, and poor brushing techniques. The reported range of incidence is 0.26-5.3% in several published series in kids and teens.

CLINICAL FEATURES: At first, the patches are pale and translucent, but later become dense and white, sometimes with a rough surface. Habitual cheek biting causes an area of buccal mucosa to appear patchily red and white with a rough surface.[20]



DIFFERENTIAL DIAGNOSIS:

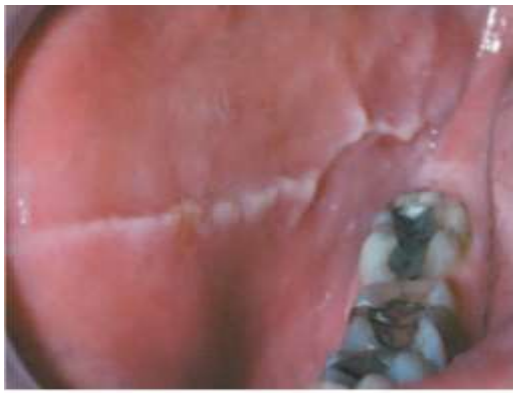
Leukoedema, Linea alba, Smokeless tobacco keratosis, Cinnamon contact stomatitis, Lupus erythematous.

TREATMENT AND PROGNOSIS: may include removal of the irritant that causes the patch quickly to disappear.[21] Biopsy is necessary only if the patch persists. Frictional keratosis is completely benign and there is no evidence that continued minor trauma alone has any carcinogenic potential.[22]

LINEA ALBA

The linea alba is a horizontal streak on the inner surface of the cheek, level with the biting plane. It usually extends from the commissure to the posterior teeth and can extend to the inner lip mucosa and corners of the mouth.[23] May be associated with biting irritation or sucking habit; may be associated with leukoedema. Linea alba was seen in 4.80%.[24] It was highly significantly more common among females (5.86%) than in males (3.64%).[25] It is most likely combines with pressure, frictional irritation, or sucking trauma from the facial surfaces of the teeth. [26]

CLINICAL FEATURES: Linea alba is asymptomatic and generally considered a normal variation than pathological.[27] It may be scalloped and likely to be located on the buccal mucosa at the level of the occlusal plane of the adjacent teeth. The line varies in prominence and usually is restricted to dentulous areas.[28] It often is more pronounced adjacent to the posterior teeth.[29]



DIFFERENTIAL DIAGNOSIS:

Cinnamon contact stomatitis, Scar formation, Cheek-biting keratosis.

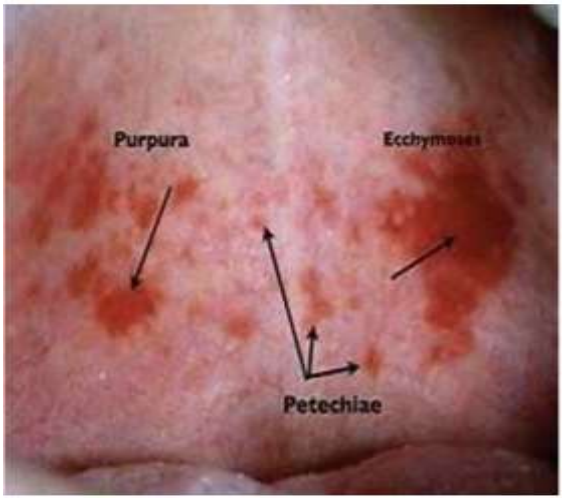
TREATMENT AND PROGNOSIS: No treatment is required for patients with linea alba, and no difficulties are documented as a result of its development. Spontaneous regression may occur.

PETECHIAE, ECCHYMOSIS, HEMATOMA

This occurs when a traumatic event results in hemorrhage and entrapment of blood within tissues. Different terms are used, depending on the size of the hemorrhage: ● Minute hemorrhages into skin, mucosa, or serosa are termed petechiae. ● If a slightly

larger area is affected, the hemorrhage is termed a purpura. ● Any accumulation greater than 2 cm is termed an ecchymosis. ● If the accumulation of blood within tissue produces a mass, this is termed a hematoma. Less well known are petechiae and purpura, which can arise from repeated or prolonged increased intrathoracic pressure (Valsalva maneuver) associated with such activities as repeated coughing, vomiting, convulsions, or giving birth.

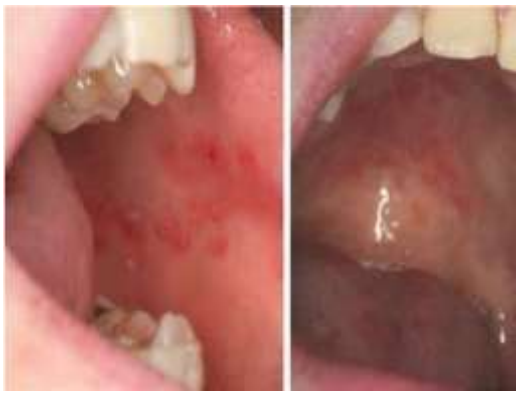
CLINICAL FEATURES: Blunt facial trauma often is responsible, but injuries such as minor as cheek biting may produce a hematoma or areas of purpura. Mild pain may be present. The hemorrhage associated with increased intrathoracic pressure usually is located on the skin of the face and neck and appears as widespread petechiae that clear within 24 to 72 hours. Patients with antral hematomas typically complain of frequent nasal bleeding but also may experience unilateral nasal obstruction, hyposmia, headache, and malar swelling. Although the hemorrhage is noticeable immediately in most patients, the problem may not become evident clinically for 4 to 6 hours.



DIFFERENTIAL DIAGNOSIS:
Amalgam/ graphite tattoo, blue nevus, Hemangioma, Erythematous candidiasis, Blood dyscrasias.

TREATMENT AND PROGNOSIS:
Often, no treatment is required if the hemorrhage is not associated with significant morbidity or related to systemic disease. If the hemorrhage occurs secondary to an underlying disorder, then treatment is directed toward control of the associated disease. Antral hematomas usually are removed via a Caldwell-Luc procedure for both therapeutic and diagnostic purposes.

ERYTHEMATOUS CANDIDIASIS
Caused by candida albicans and other species; contributing factors are antibiotics, immunosuppression, xerostomia, pacifier and palatal coverage appliances. Located on palate, buccal mucosa, dorsal tongue. with no gender predilection.
Clinically, there is widespread erythema (redness) and soreness of the oral mucosa. This can be concomitant with THRUSH. The tongue is most commonly affected, although any area of oral mucous membrane is susceptible. Patients may complain of tenderness, burning, and dysphagia, especially if oro-pharyngeal candidiasis is associated with œsophageal infections.[30] It may arise as a consequence of persistent acute pseudo-membranous candidiasis, when pseudo membranes are shed, may develop de novo, or, in HIV infection, may precede pseudo-membranous candidiasis.[31]



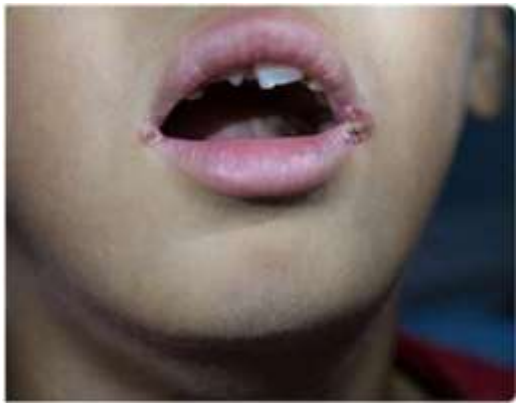
DIFFERENTIAL DIAGNOSIS: Contact allergy, Traumatic erythema, Erythema migrans, Thermal burn, Anemia.

TREATMENT AND PROGNOSIS: Cessation of treatment with the offending antibiotic / steroid medication usually leads to spontaneous resolution, however this may not be possible and topical antifungals may be necessary prophylactically if the causative therapy is to be continued. Systemic antifungals may be indicated. Patients using inhaled steroid prescriptions should be advised to rinse their mouth after inhalation to ensure speedy resolution of erythematous candidiasis.

ANGULAR CHELITIS

DEFINITION: Angular cheilitis also called perleche or angular cheilitis is a lesion marked with fissures, cracks on corner of lip, reddish, ulceration accompanied by burning sensation, pain and dryness on the corner of the mouth. In severe cases, these cracks can bleed when opening the mouth and cause shallow ulcer or krusta.[32]

ETIOLOGY: It is caused by children sensitivity against certain contact agents like toys, foods, sunlight, allergy against medicines, cosmetics, and long- term antibiotic treatment.[33] This disease can also be caused by vitamin B complex deficiency, blood iron deficiency, denture sore mouth and other factors such as breathing through mouth, wetting lips with tongue, and licking the corner of the mouth with tongue.[34]



CLINICAL FEATURES: Clinically, angular cheilitis can occur in chronic condition, where the corner of mouth becomes inflamed because of wound infection. Infection that caused this condition is a type of fungi or bacteria.[35] Affected area usually is painful and healing period depends on the treatment. All ages can be affected by this disease.[36]

DIFFERENTIAL DIAGNOSIS:

Secondary herpetic ulcer, Impetigo, Exfoliative cheilitis, Traumatic ulcers. [37]

TREATMENT AND PROGNOSIS: If it is caused by systemic disease, local treatment will not be successful if not accompanied by systemic treatment.[38] Angular cheilitis caused by vitamin B deficiency should be treated by providing vitamin B complex supplement or multivitamin that contain vitamin B.[39] However, deficiency of one type of vitamin is usually followed by nutritional deficiency, hence in the treatment, multivitamin administration is more effective than vitamin B complex alone.[40] Reported treatment of disease caused by vitamin B12 deficiency with vitamin therapy can be healed in 3 weeks.[41]

BROWN-BLACK LESIONS

The most prevalent type of widespread and bilateral pigmentation, known as "physiologic pigmentation," affects groups with dark complexion, such as those from the Middle East, Africa, and occasionally Asia. (Pediatric oral soft tissue lesions) race/ethnicity, raised age, smoking, pregnancy, endocrine syndromes, and hormonal changes are conditions that, generally, increase the prevalence of this pigment. There have been reports of unusual birth cases. Multiple intraoral and perioral pigmentations are linked to Peutz-Jeghers syndrome, an autosomal dominant characteristic; the majority of these pigmentations are not treatable and involute after the first ten years of life. An autoimmune condition known as Addison disease or adrenal insufficiency causes insufficient glucocorticoid and mineralocorticoid secretion. Diffuse bronzing of the skin and mucous

membranes is among the initial symptoms. The gingiva, tongue, buccal mucosa, and hard palate of the oral cavity are typical sites of pigmentation. Isolated macules may appear sometimes.[42]

CONCLUSION

An overall knowledge of oral soft tissue lesions in children aids in diagnosis and appropriate treatment planning of the same. A paediatric dentist should have an awareness and knowledge regarding the various lesions to be able to perform early detection.

Declaration by Authors

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REFERENCES

1. Shulman JD. Prevalence of oral mucosal lesions in children and youths in USA. Int J Paediatr Dent. 2005; 2:89-97.
2. Banoczy J, Szabo L, Csiba A. Migratory glossitis. A clinical histologic review of seventy cases. Oral Surg Oral Med Oral Pathol. 1975; 39:113-21.
3. Desai VD, Baghla P. Asymptomatic reversible lesion on tongue – case series in paediatric patients. J Adv Med Dent Scie. 2014;2(2):176-79.
4. Jainkittivong A, Lanlais RP. Geographic tongue: clinical characteristics of 188 cases. J Contemp Dent Pract. 2005; 1:123-35.
5. Casamassimo PS. Pediatric dentistry: infancy through adolescence. Elsevier/ Saunders, 2013.
6. Darwazeh AMG, Almelaih AA. Tongue lesions in a Jordanian population. Prevalence, symptoms, subject's knowledge and treatment provided. Med Oral Patol Oral Cir Bucal. 2011;16(6):e745-49.
7. Singh S, Nivash S, Mann BK. Matched case-control study to examine association of psoriasis and migratory glossitis in India. Indian J Dermatol Venereol Leprol. 2013; 79:59-64.
8. Purkait SK. Essentials of oral pathology. JP Medical Ltd; 2011.
9. Neville BW, Damm DD, Allen CM, Chi AC. Oral and maxillofacial pathology. Elsevier Health Sciences; 2015 May 13.
10. Casamassimo PS. Pediatric dentistry: infancy through adolescence. Elsevier/ Saunders, 2013.
11. Berman FR, Fay JT. Oral Surg Oral Med Oral Pathol 1976;42(1):80-5.
12. Easley JR, Weise RW. J Dent Child 1970; 37:523-526.
13. Gorsky M, Begleiter A, Buchner A, Harel-Raviv M. Oral Surg Oral Med Oral Pathol 1986; 62:240-243.
14. Purkait SK. Essentials of oral pathology. JP Medical Ltd; 2011.
15. Casamassimo PS. Pediatric dentistry: infancy through adolescence. Elsevier/ Saunders, 2013.
16. Kupietzky A, Botzer E. Ankyloglossia in the infant and young child: clinical suggestions for diagnosis and management. Pediatric dentistry. 2005 Jan 1;27(1):40-6.
17. Purkait SK. Essentials of oral pathology. JP Medical Ltd; 2011.
18. Neville BW, Damm DD, Allen CM, Chi AC. Oral and maxillofacial pathology. Elsevier Health Sciences; 2015 May 13.
19. Casamassimo PS. Pediatric dentistry: infancy through adolescence. Elsevier/ Saunders, 2013.
20. Pinto A, Haberland CM, Baker S. Pediatric soft tissue oral lesions. Dent Clin North Am. 2014;58(2):437–453. doi: 10.1016/j.cden.2013.12.003. [PubMed] [CrossRef] [Google Scholar].
21. Odell EW. Cawson's essentials of oral pathology and oral medicine e-book. Elsevier Health Sciences; 2017 May 2.
22. Woo SB, Lin D. Morsicatio mucosae oris–a chronic oral frictional keratosis, not a leukoplakia. J Oral Maxillofac Surg. 2009;67(1):140–6.
23. Casamassimo PS. Pediatric dentistry: infancy through adolescence. Elsevier/ Saunders, 2013.
24. Martínez AI, García-Pola MJ. Epidemiological study of oral mucosal pathology in patients of the Oviedo School of Stomatology. Med Oral 2002; 7(1): 4-16.
25. Neville BW, Damm DD, Allen CM, Chi AC. Oral and maxillofacial pathology. Elsevier Health Sciences; 2015 May 13.
26. Seoane Leston JM, Aguado Santos A, Varela-Centelles PI, Vazquez Garcia J, Romero MA, Pias Villamor L. Oral mucosa:

Variations from normalcy, partI. Cutis. 2002; 69: 131 - 134.
27. Parlak AH, Koybasi S, Yavuz T, Yesildal N, Anul H, Aydogan I, et al. Prevalence of oral lesions in 13- to 16-year-old students in Duzce, Turkey. Oral Dis. 2006; 12: 553 - 558. https://doi.org/10.1111/j.1601-0825.2006.01235.x PMid:17054767.
28. Cebeci AR İ, Gülşahı A, Kamburoğlu K, Orhan BK, Öztaş B. Prevalence and distribution of oral mucosal lesions in an adult Turkish population. Med Oral Patol Oral Cir Bucal 2009; Jun: 14 (6): E272-7.
29. Anisuzzaman M M, Khan M T I, Hasan, Adnan N, Afrin A. Prevalence of Linea Alba Buccalis in Bangladeshi Population. Update Dental College Journal. 2019 October; 9(2): 17-19.
30. Casamassimo PS. Pediatric dentistry: infancy through adolescence. Elsevier/ Saunders, 2013
31. Jump up to:a b Lyons, Faye (2014). Dermatology for the Advanced Practice Nurse. Springer Publishing Company. p. 95. ISBN 9780826136442. Archived from the original on 2016-08-16. Gayford JJ, Haskel R. Penyakit mulut (clinical oral medicine). Jakarta: EGC; 1990.
32. DowlW. Effect of angular cheilitis on children and teenagers. [internet]. Available at URL: http://www.EzineArticles/ childandac.html. Accesses 25December 2010.
33. Muray JJ, Nunn J. H.Steele J. The prevention of oral disease 4th ed. New York: oxford University Press; 2008.
34. Boedihardjo. Pemeliharaan Kesehatan Gigi Keluarga. Surabaya: Airlangga University Press; 1985.
35. Fajriani. 2017. Management of Angular Cheilitis in children. Journal of Dentomaxillofacial Science 2(1): 1-3. DOI:10.15562/jdmfs. v2i1.461.
36. . Lubis S. Hubungan status gizi dengan keilitisangularis pada anak umur 6-12 tahun di enam panti asuhan di Kota Madya Medan. Dentika J Dent 2006;11: 180-181.
37. Hari S. Angular cheilitis: Review of etiology and clinical management. K.D.J.[Internet] Available at: http://www. trivandrum.co.uk. Accessed 27 December 2010.
38. Langlais RP, Craig SM. Atlas Berwarna : Kelainan Rongga Mulut yang Lazim. 1st ed. Jakarta: Hipokrates; 2003. p. 34.
39. Morison MJ. Manajemen luka. Jakarta: EGC; 2003. p. 1-21.
40. Pinto, A., Haberland, C.M. and Baker, S. (2014) 'Pediatric soft tissue oral lesions', Dental Clinics of North America, 58(2), pp. 437–453. Pinto, A., Haberland, C.M. and Baker, S. (2014) 'Pediatric soft tissue oral lesions', Dental Clinics of North America, 58(2), pp. 437–453. doi: 10.1016/j.cden.2013.12.003.
41. Casamassimo PS. Pediatric dentistry: infancy through adolescence. Elsevier/ Saunders, 2013.
42. Pinto A, Haberland CM, Baker S. Paediatric soft tissue oral lesions. Dent Clin North Am. 2014; 58:437-53.

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