



What Could Cause Gingival Discoloration and Pigmentation in Children?

Karimi M**Department of Pediatric, Sepideh Dental Clinic, Iran***Corresponding author:* Karimi M, Department of Pediatric, Sepideh Dental Clinic, Iran**Received:** August 18, 2022**Published:** August 29, 2022

Abstract

The gums are hard connective tissues that give a remarkable beauty to the teeth. Therefore, any color changes in the gums worry parents in this regard. Normal gingiva should be pink; hence, discoloration of the gums can indicate an early health problem. The underlying reason may be a source of systemic diseases, genetics, use of medication, hormonal changes, smoking, acute gingivitis, and amalgam tattoo. The attached gingiva is the most common site of such pigmentation.

Keywords: Gum discoloration; genetics; health problem; systemic diseases; gingivitis; medication; smoking; hormonal changes; amalgam

Introduction

One of the reasons for having dark gums is the natural balance of skin pigments in a person's body. Just as the skin color of different people is different, the color of the gums can be naturally different. In many people who are not completely white, the gums may have dark spots or may not be light pink at all. This is due to the high level of melanin in the skin and tissues of the body, and in this case, the dark color of the gums, just like the dark color of the skin, is normal. The natural color of the gums is pink and sometimes the color of the gums changes and darkens. Discoloration of the gums can be a natural phenomenon or indicate oral disease or severe health problems such as Peutz-Jeghers syndrome and Addison's disease [1].

Pigmentation of the gums has various causes. The most common cause of gum discoloration is the accumulation of melanin (dark pigments) in the gingival tissue [2], which causes healthy,

pink gums to turn brown or even black. People with darker skin are more likely to experience changes in the color of their gums due to the accumulation of melanin [3]. Genetic factors can also play a role in darkening the gums [4]. Smoking and the use of tobacco are also the factors that change the color of the gums [4,5] or aggravate its unpleasant appearance that leading to the formation of dark spots on the gums. Gum disease and certain medications are also effective in changing the color of the gums. This problem is sometimes caused by the amalgam restoration he area beneath the gingiva means that the gums turn blue or gray in the vicinity of the amalgam filling due to the movement of silver amalgam particles into the gum tissue. Physiological pigmentation is generally symmetric, and persistent, and it does not change the normal gingival structure. It is more relevant in women than in men [6]. Below is the suggested table provided by Peeran et al. for a new improved classification of gingival pigmentation and pigmented lesions [7].

Class	Criteria of Classifications
I	Coral Pink/salmon pink colored gingiva
II	Localized/Isolated spots/areas of gingival melanin pigmentation which does not involve all the three parts of gingiva, that is, attached, free, and papillary gingiva Mild to moderate pigmentation severe/intense pigmentation
III	Localized/Isolated units of melanin pigmentation which involve all the three parts of gingiva, that is, attached, free, and papillary gingiva Mild to moderate pigmentation severe/intense pigmentation
IV	Generalized diffuse pigmentation which involve all the three parts of gingiva, that is, attached, free, and papillary gingiva Mild to moderate pigmentation severe/intense pigmentation
V	Tobacco associated pigmentation like smoker's melanosis and chewing tobacco
VI	Gingival pigmentation due to exogenous pigments e.g., Amalgam tattoos, Cultural gingival tattooing, Drinks, Food colors, Habitual betelnut/khat chewing, Lead-Burtonian line, Mercury, Silver, Arsenic, Bismuth, Graphite, Other foreign bodies, Topical medications, Idiopathic
VII	Gingival pigmentation due to endogenous pigments like Bilirubin, Blood breakdown products, Ecchymosis, Petechiae, Hemochromatosis, Hemosiderin
VIII	Gingival pigmentation association with systemic diseases and syndromes like Addison's Diseases, Albright's syndrome, Basilar melanosis with incontinence, Beta thalassemia; Healed mucocutaneous lesions-Lichen planus, Pemphigus, Pemphigoid; Hereditary hemorrhagic telangiectasia; HIV-associated melanosis, Neurofibromatosis, Peutz-Jeghers and other familial hamartoma syndromes, Pyogenic granuloma/Granulomatous epulis
IX	
X	

Causes

Gingival pigmentation may range from physiologic reasons (such as racial pigmentation) to the development of systemic illnesses (e.g. Addison's disease) to malignant neoplasm (melanoma and Kaposi's sarcoma). There is a range of conditions that can cause black spots on a patient's gums, including:

Melanin activity

The body naturally produces melanin, a substance that colors the skin, hair, and eyes. The darker hair, skin, or eyes indicate more melanin is produced in the human body. Brown or black gums may be due to having more melanin in the body. In case of color changes or appearing of dark spots on the gum over a short period, we should consider this condition is probably not caused by melanin and might be a medical issue. The most common pigmentations of gingiva are lesions of the melanocytic origin or gingival melanosis that happen due to the extreme deposition of melanin in the basal and suprabasal epithelium layers [8]. A dark color changing of the gingiva is a result of melanin accumulation. It is probably caused by a genetic condition. Dummett believes the degree of pigmentation is relative and could be dependent on chemical, mechanical, and physical stimuli [9].

Gingival melanosis is more common among dark-skinned people, the French, Filipinos, Arabs, and Chinese people. It is more been observed in the anterior area of the mandible; however, it has been found in the palatal mucosa and the tongue, the attached gingiva, the papilla gingiva, the gingival margin, and the alveolar mucosa [10,11]. The melanocytic lesions appear in brown or black color. They could be developed as an oral melanotic macule, melanocytic nevus, or melanoma. Dummett in his research has stated gingival pigmentation in newborns could be appeared as early as 3 hours after birth. He indicated that gingival pigmentation is seen from

early infancy which is consistent with the majority of the published data [9]. In another research among Japanese children, Kuroda and colleagues presented that gingival pigmentation increases up to 6 years of age [12].

Smoking

Smoking can cause discoloration of the gums. This is known as smoker's melanosis. The nicotine in tobacco can produce melanocytes to produce more melanin [13,14]. Discoloration may appear in form of patches or affect the entire mouth. Hedin observed smokers' melanosis on the mandible which is most common in the attached gingiva on the labial side of the canines and incisors [15]. The color may also change inside the cheeks and lower lip. Smoker's melanosis must not be misdiagnosed with gingival melanosis (a natural pigmentation in the mouth) and it is often observed in ethnic groups with darker skin. It is hardly seen in European and white populations [16,17].

Smoker's melanosis is a condition that has been remarkably recognized among smokers' people or tobacco chewers whether they are from a fair-skinned or dark-skinned ethnic group [18]. In other studies, the researchers showed that tobacco smoke from parents in the home environment could cause smokers' melanosis in their children [19,20]. Wallstrom and his colleague revealed that nicotine tablets could also stimulate melanin pigmentation of the oral mucosa [21]. Research has been shown a link between quitting smoking and reducing gingival pigmentation [22]. This suggests that dark patches on the gums that are caused by smoking may be reversible. In another study, the researchers found a correlation between exposure to environmental tobacco smoke and gingival pigmentation in children, change in flora of the mouth, periodontal disease, primary and permanent tooth decay, tooth loss, and delayed eruption of teeth [23].

Drugs

Taking some medications may lead to oral mucosal pigmentation. It can include the accumulation of melanin, deposition of the drug, and production of pigments under the influence of the drug [24]. The most-reported drugs that cause oral mucosa melanin pigmentation are the following: *Chloroquine, Quinine, Minocycline, Zidovudine, Chlorpromazine, Ketoconazole, Bleomycin, and Cyclophosphamide*. Minocycline is another one to causes gingival and lips pigmentation [25,26]. Consumption of drugs such as Phenothiazines and Phenytoin can cause blue, blue-grey, or brown mucosal pigmentation [27,28]. Amiodarone may have adverse effect of grey orofacial and oral mucosal pigmentation [29]. In HIV patients, drug-induced melanotic pigmentation may appear after the therapy with Clofazimine, Zidovudine, and Ketoconazole [30,31].

Amalgam tattoo

Amalgam tattoos can appear anywhere on the mouth, but they usually appear next to a filling. It could be caused by the traumatic implantation of metal particles on soft tissue when the restoration is removed, during endodontic treatment, or when penetrating the alveoli during tooth extraction. The patch appears black, gray, or blue inside the mouth. If the particles of this substance are destroyed, they can appear under the skin of the gums. An amalgam tattoo is common, painless, and benign, but it can be mistaken for melanoma [32]. The diagnosis can be confirmed histologically by excisional biopsy that rules out nevi and melanomas. Graphite tattoos may appear in the oral mucosa through accidental injury with a graphite pencil. It has predominantly been seen in women and youngsters from age 5 to 21 years [33]. The color appearance of this type of tattoo is blue-gray. The most frequently commonly involved site is the anterior palate of young children [33]. The diagnosis can be determined by the history of the injury area.

Necrotizing ulcerative gingivitis (NUG)

This periodontal disease is a gum infection distinguished by gingival necrosis. It can be presented by the distinctive features of rapid onset of gingival pain, interdental gingival necrosis, and gingival bleeding [34]. NUG is frequently associated with HIV infection that seems to play a major role in the pathogenesis of NUG. However, the disease is more common among young adults, but children are no exception and complications are more frequent in children aged 2–7 years [35,36]. In malnourished, immunocompromised patients, particularly in children, NUG may lead to necrotizing ulcerative periodontitis and spread as Necrotizing Stomatitis or Noma if the disease is left untreated [37,38]. This condition has also been known as Vincent disease, trench mouth, and acute necrotizing ulcerative gingivitis that cause black or grey gums. Factors such as tobacco smoking, preexisting gingivitis, and trauma have been reported as predisposing factors of NUG [34,39].

Addison's disease

Addison's disease affects the adrenal glands, which produce a variety of hormones. Disruption of these glands stops them from producing enough hormones. It is also known as autoimmune adrenalitis and is now the most common cause of Addison's disease in both children and adults [40]. Most cases of Addison's disease are due to a problem with the immune system. This causes the body to attack and damage the adrenal glands. The other causes of Addison disease in children could involve trauma and adrenocortical hemorrhage, which is most commonly associated with meningococcemia [41]. Addison's disease can cause severe complications if left untreated. If hormone levels drop, it can lead to an adrenal crisis. As Addison's disease progresses, the person may have dark gums and lips. The medical term for this is hyperpigmentation. The oral hyperpigmentation of Addison's disease can appear on the areas such as the tongue, gingiva, buccal mucosa, and hard palate [42]. Addison's disease can also cause darker patches of skin on other parts of the body.

Peutz-Jeghers syndrome, McCune-Albright syndrome, Laugier hunziker syndrome

Peutz-Jeghers syndrome is a genetic disorder featured by mucocutaneous pigmentation and intestinal hamartomas. It usually appears in childhood and disappears with age. The pigmentations appear like macules on the hands, perioral skin, and the gingiva, buccal, and labial mucosa. Pigmented spots are particularly found on the lower lip and buccal mucosa; however, are rarely seen on the upper lip, tongue, palate, and gingiva [43]. The oral pigmentations in McCune-Albright Syndrome are developing in mid-childhood to early adulthood and may appear as developed new hyperpigmented macules on the gingiva, tongue, lips, and labial and buccal mucosa [44]. Laugier-Hunziker syndrome occurs predominantly among middle-aged adults and occurrence is usually seen after puberty. LHS is a rare, acquired skin disorder featured by diffuse hyperpigmentation of the oral mucosa and lips, and melanonychia of nails. The buccal mucosa and the lips are the most commonly involved sites, but the gingiva, tongue, soft palate, and hard palate can also be involved [45]. The pigmentation is in the form of a brown-black macule.

Oral melanocytic nevus, Melanotic macules

Pigmented nevi of the oral cavity are uncommon. It can be observed in any age group and seen commonly on the palate and gingiva. The clinical features include brownish-black to blue papules with a well-defined border. Nevi can be classified as congenital and acquired. Nevus represents a benign proliferation of melanocytes [46].

- a) **Oral nevi and melanomas:** Could also have oral pigmentation similar to Addisonian pigmentation [47]. Oral melanocytic nevus (OMN) is quite uncommon [48]. In the case of developing intraorally, they appear most commonly on the gingiva, hard palate, and buccal mucosa [48, 49].

- b) **Oral melanotic macule:** Known as focal melanosis, is a single benign, well distinguished, dark brown color lesion that includes 3% of the normal population. The commonly involved areas are the gingiva, lip, palate, buccal mucosa, and alveolar ridge [50].

In contrast to nevi and melanomas, melanotic macules have no malignancy potential [51]; hence, the treatment is not essential.

Mucosal melanomas

Melanoma is a malignant neoplasm of melanocytes that is more common in the skin and only rarely involves the oral mucosa. It accounts for less than 1% of all oral malignancies. The most common involved oral area is the palate (40%), and the gingiva (30%) cases respectively which count for one-third of all cases. Other oral mucosal sites may also be affected [52]. Generally, oral melanoma has the characteristic of one isolated brown or black patch that can be determined by asymmetric and irregular borders. Malignant melanoma may appear with different clinical features, with early lesions as a macule or plaque with different colors (brown, black, blue, red, or white) or occasionally as ulceration that does not heal [53]. The presence of multiple lesions, their clinical aspects, and their signs and symptoms of systemic involvement could rule out melanoma from others without performing a biopsy [54].

The prevalence is higher in Japanese people with extremely rare occurrences. It mostly appears on the anterior labial gingiva and the anterior aspect of the hard palate. The early stages become visible as brown or black plaques and then change to more diffuse, nodular [55,56]. If a Patient with oral melanoma had a previous pigmentation in the same area before the melanoma diagnosis, it might recall the emergency need to perform a biopsy for any oral pigmented lesion that is not readily diagnosed. Early biopsy of focal pigmentations of undetermined etiology is extremely important to detect oral melanomas at an early stage [57]. A biopsy is strongly advisable for any new oral pigmentation because an early melanoma may be mistaken as melanocytic nevi [58].

HIV oral melanosis

In these patients' hyperpigmentation of the skin, nails, and mucous membrane had been observed; however, the etiology of such hyperpigmentation could be associated with medication or adrenocortical involvement by opportunistic parasites [30,59]. Ficarra et al. found that 6.4% of HIV patients had developed oral pigmentation. Even though the majority of these patients had multiple macules on the oral mucosa, labial, palatal and gingival pigmentation, it had been seen in others too [30]. Due to infection by *Mycobacterium Avium* in HIV patients the adrenal cortex involves in hormonal changes that cause an increase in the level of adrenocorticotropin hormone (ACTH) and MSH. Consequently, this dysfunction leads to hyperpigmentation in these patients.

Furthermore, the medications like ketoconazole, and zidovudine that are used for the treatment of these patients would also cause oral pigmentation [60,61]. *Kaposi sarcoma* is a very important

pediatric cancer in the HIV epidemic. Kaposi sarcoma in pediatrics is rare, but the prevalence greatly has changed worldwide, especially in developed countries. In a study of some African countries, KS was the most common and the second most common childhood cancer in many areas of Southern and Eastern Africa [62]. The oral site of involvement in children is commonly the oropalatal area [63]. The most affected areas inside the mouth in Kaposi sarcoma are the palate and gums. Intraoral lesions are easily injured which can result in pain, bleeding, ulceration, or secondary infection [64,65]. Early lesions have the characteristic of flat or slightly elevated brown to purple lesions that are often bilateral. Advanced lesions have dark red to purple plaques or nodules appearance that may present ulceration, bleeding, and necrosis [66].

Other endocrine diseases

Research show that few endocrine disturbances could cause oral pigmentations. Some conditions such as Cushing's syndrome, Acromegaly, and hyperthyroidism, also show pigmentation similar to Addison's disease [60,67]. In Nelson Syndrome hyperpigmentation usually appears with a linea nigra and pigmentation of the extensor surfaces, scars, and gingiva [68].

Conclusion

The development of gingival pigmentation in children can be due to physiological or pathological factors. These factors may include systemic diseases, genetics, use of medication, hormonal changes, smoking, acute gingivitis, and amalgam tattoo. The onset of oral pigmentation could be present from birth in certain individuals. Nonetheless, a sudden occurrence of pigmentation has suggested that some genetic and systemic conditions such as Peutz-Jeghers syndrome and Addison's disease may be involved. Occasionally, these lesions may result from an increase in the number of cells, which can range from benign nevi to fatal oral melanoma. Patients should be evaluated to rule out any systemic diseases or neoplasm associated with gingival pigmentation.

References

1. Lanza (2009) Oral Pigmentation as a Sign of Addison's disease: A Brief Reappraisal. The Open Dermatology Journal 3: 3-6.
2. Dummett CO, Barrens G (1967) Pigmentation of oral tissues: a review of the literature. J Periodontol 38(5): 369-378.
3. Anil Kumar N, Divya P (2015) Adverse drug effects in the mouth. International Journal of Medical and Applied Sciences 4: 82-91.
4. Meleti M, Vescovi P, Mooi WJ (2008) Pigmented lesions of the oral mucosa and perioral tissues: a flow-chart for the diagnosis and some recommendations for the management. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 105(5): 606-616.
5. Hedin CA, Axell T (1991) Oral melanin pigmentation in 467 Thai and Malaysian people with special emphasis on smoker's melanosis. J Oral Pathol Med 20(1): 8-12.
6. Tamizi M, Taheri M (1996) Treatment of severe physiologic gingival pigmentation with free gingival autograft. Quintessence Int 27: 555-558.
7. Peeran SW, Ramalingam K, Peeran SA, Altaher OB, Alsaad FM, et al (2014) Gingival pigmentation index proposal of a new index with a brief review of current indices. European Journal of Dentistry 8(2): 287.

8. Pavlic V, Brkic Z, Marin S, Cicmil S, Gojkov-Vukelic M, et all (2018) Gingival melanin depigmentation by Er: YAG laser: A literature review. *J Cosmet Laser Ther* 20(2): 85-90.
9. Dummett CO (1945) Clinical observation on pigment variations in healthy oral tissues in the Negro. *J Dent Res* 24(1): 7-13.
10. Tal H, Oegieser D, Tal M (2003) Gingival Depigmentation by Erbium: YAG Laser: Clinical Observations and Patient Responses. *J Periodontol* 74(11): 1660-1667.
11. Roshna T, Nandakumar K (2005) Anterior Esthetic Gingival Depigmentation and Crown Lengthening: Report of a Case. *J Contemp Dent Pract* (6)3: 139-147.
12. Kuroda K, Karazumi I, Fujino M, Tani Z, Nagai A, et al. (1961) Biochemical studies on gingival pigmentation in children. *J Biochem* 49: 693-699.
13. Boissy RE (2003) Melanosome transfer to and translocation in the keratinocyte Experimental Dermatology 12(Suppl 2): 5-12.
14. Hearing VJ (1999) Biochemical control of melanogenesis and melanosomal organization. *Journal of Investigative Dermatology Symposium Proceedings* 4: 24-28.
15. Hedin CA (1977) Smoker's melanosis, Occurrence, and localization in the attached gingiva, *Arch Dermatol* 113(11): 1533-1538.
16. Becker SW Jr (1969) Pigmentary lesions common to the skin and oral cavity. *Oral Surg Oral Med Oral Pathol* 28: 526-533.
17. McCarthy PL, Shklar G (1964) Disturbances in pigmentation, Diseases of the Oral Mucosa: Diagnosis, Management, Therapy. McGraw-Hill Book Co, New York, USA pp. 228-238.
18. Axell T, Hedin CA (1982) Epidemiologic study of excessive oral melanin pigmentation with special reference to the influence of tobacco habits. *Scand J Dent Res* 90: 434-442.
19. Sridharan S, Ganiger K, Satyanarayana A, Rahul A, Shetty S (2011) Effect of environmental tobacco smoke from smoker parents on gingival pigmentation in children and young adults: a cross-sectional study. *J Periodontol* 82: 956-962.
20. Hanioka T, Tanaka K, Ojima M, Yuuki K (2005) Association of melanin pigmentation in the gingiva of children with parents who smoke. *Pediatrics* 116: 186-190.
21. Wallstrom M, Sand L, Nilsson F, Hirsch JM (1999) The long-term effect of nicotine on the oral mucosa. *Addiction* 94: 417-423.
22. Kato T, Takiuchi H, Sugiyama S, Makino M, Noguchi S, et al. (2016) Measurement of reduced gingival melanosis after smoking cessation: A novel analysis of gingival pigmentation using clinical oral photographs. *Int J Environ Res Public Health* 13(6): 598.
23. Moravej Salehi E, Moravej Salehi E, Hajifattah F (2015) Passive Smoking: Oral and Dental Effects. *Iran J Public Health* 44(4): 600- 601.
24. Dereure O (2001) Drug-induced skin pigmentation. *American journal of clinical dermatology* 2(4): 253-262.
25. Bortuluzzi EA, Araújo GS, Guerreiro Tanomaru JM, Tanomaru-Filho M (2007) Marginal gingival discoloration by gray MTA: a case report. *J Endod* 33: 325-327.
26. De Melo Filho MR, da Silva CA, da Rocha Dourado M, de Oliveira Pires MB, Pêgo SP, et al. (2012) Palate hyperpigmentation caused by prolonged use of the anti-malarial chloroquine, *Head and neck pathology* 6(1): 48-50.
27. Manor A, Sperling I, Buchner A (1981). Gingival pigmentation is associated with antimalarial drugs. *Refuat Hapeh Vehashinayim* 28(4): 13-16.
28. McAllan LH, Adkins KF (1986) Drug-induced palatal pigmentation. *Aust Dent J* 31: 1-4.
29. Bucknall CA, Keeton BR, Curry PV, Tynan MJ, Sutherland GR, et al. (1986) Intravenous and oral amiodarone for arrhythmias in children. *Br Heart J* 56: 278-284.
30. Ficarra G, Shillitoe EJ, Adler-Storthz K, Gaglioti D, Di Pietro M, et al. (1990) Oral melanotic macules in patients infected with human immunodeficiency virus. *Oral Surg Oral Med Oral Pathol* 70: 748-755.
31. Porter SR, Glover S, Scully C (1990) Oral hyperpigmentation and adrenocortical hypofunction in a patient with acquired immunodeficiency syndrome. *Oral Surg Oral Med Oral Pathol* 70: 59-60.
32. Ajit Auluck (2008) Primary Malignant Melanoma of Maxillary Gingiva — A Case Report and Review of the Literature. *JCDR* 7(4).
33. Shahna N (2019) Gingival pigmentation: A review of literature. *International Journal of Applied Dental Sciences* 5(2): 83-91.
34. Rowland RW (1999) Necrotizing ulcerative gingivitis. *Ann Periodontol* 4: 65-73.
35. Taiwo JO (1996) Effect of social class on the prevalence and severity of necrotizing ulcerative gingivitis in Nigerian children. *Afr J Med Sci* 25: 357-360.
36. Reade PC (1963) Infantile acute ulcerative gingivitis: a case report. *J Periodontol* 34: 387-390.
37. Enwonwu CO (2006) Noma (cancrum oris) (Review). *Lancet* 368: 147-156.
38. Enwonwu CO (1999) Pathogenesis of cancrum oris (noma): confounding interactions of malnutrition with infection. *Am J Trop Med Hyg* 60: 223-232.
39. Niklaus Lang, Soskolne WA, Gary Greenstein, David Cochran, Esmonde Corbet, et al. (1999) Consensus Report: Necrotizing Periodontal Diseases. *Annals of Periodontology* 4: 78.
40. Aaron Michels, Nicole Michels (2014) Addison Disease: Early Detection and Treatment Principles. *Am Fam Physician* 89(7): 563-568.
41. Baker JR (1997) Autoimmune endocrine disease. *JAMA* 278: 1931-1937.
42. Samir S Shah, Catherine H, Susan E Coffin, Albert C Yan (2005) Addisonian Pigmentation of the Oral Mucosa. *Pediatric Dermatology* 76: 97-99.
43. Kopacova M, Tachecl, Rejchrt S, Bures J (2009) Peutz-Jeghers syndrome: diagnostic and therapeutic approach. *World Journal of Gastroenterology: WJG* 15(43): 5397.
44. Richard, Dominique C (2014) Oral pigmentation in McCune-Albright syndrome. *JAMA dermatology* 150(7): 760-763.
45. Sachdeva S, Sachdeva S, Kapoor P (2011) Laugier Hunziker syndrome: a rare cause of oral and acral pigmentation. *Journal of cutaneous and aesthetic surgery* 4(1): 58.
46. Sreeja C, Ramakrishnan K, Vijayalakshmi D, Devi M, Aesha I, et al. (2015) Oral pigmentation: A review. *Journal of pharmacy & bioallied sciences* 7(2): 403.
47. Alessandro Lanza, Inam Heulfe, Letizia Perillo, Antonio Dell'Ermo, Nicola Cirillo (2009) Oral Pigmentation as a Sign of Addison's Disease: A Brief Reappraisal. *The Open Dermatology Journal* 3: 3-6.
48. Ferreira L, Jham B, Assi R, Readinger A, Kessler HP (2015) Oral melanocytic nevi: a clinicopathologic study of 100 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol* 120(3): 358-367.
49. Meleti M, Mooi WJ, Casparie MK, van der Waal I (2007) Melanocytic nevi of the oral mucosa - no evidence of increased risk for oral malignant melanoma: an analysis of 119 cases. *Oral Oncol* 43(10): 976-981.
50. Kaugars GE, Heise AP, Riley WT, Abbey LM, Svirsky JA (1993) Oral melanotic macules, a review of 353 cases. *Oral Surg Oral Med Oral Pathol* 76(1): 59-61.
51. Kauzman A, Pavone M, Blanas N, Bradley G (2004) Pigmented lesions of the oral cavity: review, differential diagnosis, and case presentations. *J Can Dent Assoc* 70(10): 682-683.
52. Symvoulakis EK, Kyrmizakis DE, Drivas EI, Koutsopoulos AV, Malandrakis SG, et al. (2006) Oral mucosal melanoma: a malignant trap. *Head & face medicine* 2(1): 7.

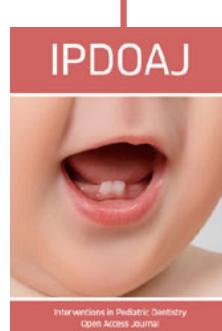
53. Lim GF, Cusack CA, Kist JM (2014) Perioral lesions and dermatoses. *Dent Clin N Am* 58(2): 401-435.
54. Eisen D, Voorhees JJ (1991) Oral melanoma and other pigmented lesions of the oral cavity. *J Am Acad Dermatol* 24: 527-537.
55. Pour MSH (2008) Malignant melanoma of the oral cavity: a review of the literature. *Ind J Dent Res* 19(1): 47e51.
56. Glasgow BJ, Steinsapir KD, Anders K, Layfield LJ (1985) Adrenal pathology in the acquired immune deficiency syndrome. *J Am Clin Pathol* 84: 594e7.
57. Garzino Demo P, Fasolis M, Maggiore GM, Pagano M, Berrone S (2004) Oral mucosal melanoma: a series of case reports. *J Craniomaxillofac Surg* 32(4): 251-257.
58. Müller S (2010) Melanin-associated pigmented lesions of the oral mucosa: presentation, differential diagnosis, and treatment. *Dermatologic Therapy* 23: 220-229.
59. Esposito R (1987) Hyperpigmentation of skin in patients with AIDS (letter). *Br Med J* 294: 840.
60. Regezi JA, Sciubba JJ, Jordan RC (2009) editors, *Oral Pathology, Clinical Pathologic Correlations*. 5th Ed. WB Saunders, Philadelphia.
61. Smith KJ, Skelton HG, Yeager J, Ledsky R, McCarthy W, et al. (1994) Cutaneous findings in HIV-1-positive patients: A 42-month prospective study. *Military Medical Consortium for the Advancement of Retroviral Research (MMCARR) J Am Acad Dermatol* 31: 746-754.
62. Stefan DC (2015) Patterns of distribution of childhood cancer in Africa. *J Trop Pediatr* 61: 165-173.
63. Mohanna S, Bravo F, Ferrufino JC, Sanchez J, Gotuzzo E (2007) Classic Kaposi's sarcoma presenting in the oral cavity of two HIV-negative Quechua patients. *Medicina Oral, Patología Oral y Cirugía Bucal* 12(5): 365-368.
64. Nichols CM, Flaitz CM, Hicks MJ (1993) Treating Kaposi's Lesions in HIV-Infected Patient. *The Journal of the American Dental Association* 124(11): 78-84.
65. Lydia L Cairncross, Alan Davidson, Alastair JW Millar, Komala Pillay (2009) Kaposi sarcoma in children with HIV: a clinical series from Red Cross Children's Hospital. *Journal of Pediatric Surgery* 44(2): 373-376.
66. Lager I, Altini M, Coleman H, Ali H (2003) Oral Kaposi's sarcoma: a clinicopathologic study from South Africa. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* 96(6): 701-710.
67. Neville BW, Damm DD, Allen CM, Bouquot JE (2009) *Oral and Maxillofacial Pathology*. 3rd ed. St. Louis: Saunders Elsevier Publications, Elsevier Publications pp. 308-313.
68. Banasiak MJ, Malek AR (2007) Nelson syndrome: a comprehensive review of pathophysiology, diagnosis, and management. *Neurosurgical Focus FOC* 23(3): 1-10.



This work is licensed under Creative Commons Attribution 4.0 License

To Submit Your Article Click Here: [Submit Article](#)

DOI: [10.32474/IPDOAJ.2022.08.000277](https://doi.org/10.32474/IPDOAJ.2022.08.000277)



Interventions in Pediatric Dentistry : Open Access Journal

Assets of Publishing with us

- Global archiving of articles
- Immediate, unrestricted online access
- Rigorous Peer Review Process
- Authors Retain Copyrights
- Unique DOI for all articles