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Oral Pigmented Lesions: Classification and a Literature Review

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ABSTRACT

Introduction: Pigmentations are commonly found in the mouth. They distinguish in several clinical patterns that can range from physiologic pigmentation to oral manifestations of systemic diseases and malignancies. A variety of pigmentations can be in the form of blue/purple vascular lesions, brown melanotic lesions, brown heme associated lesions, gray/black pigmentations. We must assess several parameters associated with pigmented lesions such as location, shape, color, size and conduct a thorough medical history with relevant physical examination to identify possible adrenal, gastrointestinal or genetic disorders that are usually associated with these types of lesions. If a patient with systemic disorder is suspected then it is promptly referred to the appropriate health-care provider for further evaluation and management.

Materials and Methodology: Scientific databases were searched for the literature and relevant articles were selected or the review.

Conclusion: Multidisciplinary care is often essential to effectively manage patients with these conditions. Since the dentist come across a number of pigmented lesions in the oral cavity, it is important to have a widespread knowledge about pathogenesis, clinical manifestations, possibility of malignant transformations and their management. In this review, various pigmentations of the oral cavity are discussed.

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Introduction

Oral pigmentation is a frequent finding in the mouth. Pigmentation can be normal or abnormal discoloration of oral mucous membrane [1]. It typically occurs due to increased melanin production, increased number of melanocytes or accidental deposition of exogenous materials. They are both physiologic and pathologic. The manifestation of mucosal pigment is variable and can range from focal to diffuse macular coloration or from a small nodular growth to a large mass [2]. Blue, brown and black pigmented discolorations of the oral mucosa reflect the underlying health status and such colour changes can be attributed to the deposition of either endogenous or exogenous pigments. Exogenous pigmentations are because of foreign-body implantation in the oral mucosa. [Table1] Endogenous pigments comprise melanin, hemoglobin, hemosiderin and carotene [3]. [Table 2] Pigmentations in pathological conditions range variety from localized anomalies to potentially life-threatening conditions. Etiology of these lesions may be a local phenomenon and associated with an underlying systemic disorder [4]. The identification of pigmented lesions within the oral cavity may present a diagnostic dilemma for the clinician. Therefore, classification has been made for proper management depending on the lesion. [Table 3] Diagnostic and therapeutic modalities must be cautiously considered as these lesions encompass the spectrum of clinical pathology, ranging from benign to malignant [1]. Evaluation of a patient presenting with a pigmented lesion should comprise a full medical and dental history, extraoral and intraoral examinations and in some cases, biopsy and laboratory investigations. In this review, we have discussed various pigmentations of the oral cavity.

Table 1: Exogenous Pigmentation

Source	Color	Disease Process
Silver amalgam	Gray, black	Tattoo, iatrogenic trauma
Graphite	Gray, black	Tattoo, trauma
Lead, mercury, bismuth	Gray	Ingestion of paint or medicinals
Chromogenic bacteria	Black, brown, green	Superficial colonization

Table 2: Endogenous Pigmentation

Pigment	Color	Disease Process
Hemoglobin	Blue, red, purple	Varix, hemangioma, Kaposi's sarcoma, angiosarcoma, hereditary hemorrhagic telangiectasia
Hemosiderin	Brown	Ecchymosis, petechia, thrombosed varix, hemorrhagic mucocele, hemochromatosis
Melanin	Brown, black or gray	Melanotic macule, nevus, melanoma, basilar melanosis with incontinence
Chromogenic bacteria	Black, brown, green	Superficial colonization

Classification of Oral Pigmented Lesions

Based on Clinical Presentation of Lesions

Based on Location

- Focal: Oral Melanotic Macule, Nevus, Amalgam Tattoo, Melanoma, Graphite Tattoo, Hemangioma
- Multifocal: Kaposi sarcoma, Hereditary hemorrhagic telangiectasia, Physiologic pigmentation, Hemochromatosis, Neurofibromatosis, Lichen planus, Addison's disease, Drug-induced pigmentation, Petechiae, Peutz-Jeghers syndrome, Heavy-metal ingestion pigmentation.

Based on Color

- Blue/purple Lesions: Hemangioma, Varix, Angiosarcoma, Kaposi's Sarcoma, Hereditary Hemorrhagic Telangiectasia.
- Brown Melanotic Lesions: Ephelids, Oral melanotic macule, Nevocellular nevus, Melanoma, Drug-induced melanosis, Physiologic pigmentation, Café au Lait pigmentation, Smoker's melanosis, Pigmented Lichen Planus, Endocrinopathic pigmentation, HIV Oral Melanosis, Peutz-Jeghers syndrome.
- Brown Heme-Associated Lesions: Ecchymosis, Petechiae, Hemochromatosis.
- Gray/Black Pigmentations: Amalgam Tattoo, Graphite Tattoo, Hairy Tongue, Pigmentation Related to heavy-metal ingestion.

Based on Syndromes Associated with Oral Pigmented Lesions
Addison's Disease, Peutz-Jeghers Syndrome, Albright Syndrome, Neurofibromatosis, Laugier-Hunziker Syndrome, Nelsons Syndrome.

Based on Lesions Caused by Exogenous Pigmentation
Sliver amalgam tattoo, Lead, Mercury, Bismuth, Chromogenic bacteria, Graphite tattoo, Drug-induced Pigmentation, Medicinal Metal-induced Pigmentation, Hairy Tongue, Accidental impregnation, Iatrogenic impregnation.

Based on Lesions Caused by Endogenous Pigmentation
Normal racial variation, Melanin, Focal Melanocytic Pigmentation, Freckle/ Ephelids, Oral Melanotic Macule, Oral Melanoacanthoma, Melanocytic Nevus, Malignant Melanoma, Blood and bile pigments, Carotene, Lipofuscin, Varix, Addison's disease, Peutz-Jeghers Syndrome, Albright's Syndrome, Hyperfunction of the pituitary gland, Pregnancy and female sex hormones, Bilirubin, Haemochromatosis, Smoker's melanosis, Lentigo, HIV/AIDS.

Table 3: Clinical Classification of Pigmented Lesions

	Solitary		Multifocal
Color	**Focal**	**Diffuse**	
Blue/Purple	▪ Varix ▪ Hemangioma	▪ Hemangioma	▪Kaposi's sarcoma ▪ Hereditary hemorrhagic telangiectasia
Brown	▪ Melanotic macule ▪ Nevus ▪ Melanoma	▪ Ecchymosis ▪ Melanoma ▪ Drug-induced pigmentation ▪ Hairy tongue	▪ Hemochromatosis ▪ Physiologic pigment Neurofibromatosis ▪ Lichen planus ▪ Addison's disease ▪ Drug-induced pigmentation ▪ Petechia ▪ Peutz-Jeghers syndrome
Gray/Black	▪ Amalgam tattoo ▪ Graphite tattoo ▪ Melanoma ▪ Nevus	▪ Amalgam tattoo ▪ Hairy tongue ▪ Melanoma	▪ Heavy-metal ingestion pigmentation

Oral Pigmented Lesions

Physiologic (Racial) Pigmentation

Physiologic pigmentation is the most common cause of oral pigmentation. It is widespread in African, Asian and Mediterranean populations caused due to greater melanocyte activity rather than a greater number of melanocytes. It occurs as diffuse discoloration of oral mucosa in dark-skinned individuals [5]. Discolouration is frequently seen on the gingiva, labial mucosa, buccal mucosa, hard palate, lips and the tip of the fungiform papillae of the tongue. The colour can range from light brown to black. Most common site of occurrence is attached gingiva where it appears as bilateral, dark brown, well demarcated band that usually spares the marginal gingiva [6]. Diagnosis is made by a typical clinical appearance. No treatment is necessary, as it is asymptomatic. There are no chances of malignant transformation since these lesions are physiological.

Smoker's Melanosis

It is a common benign diffuse pigmentation of the oral mucosa from cigarette or pipe smoking. This process is due to chemicals in tobacco smoke such as nicotine and heat stimulating melanin production resulting in pigmentation. It is proved that distinction of pigmentation related to duration and amount of smoking. It occurs in up to 21.5% -31% of tobacco product consumers [7]. Widespread macular melanosis of the anterior labial gingiva, lateral tongue, palate, buccal mucosa and floor of the mouth is seen. The lesions are brown, flat and irregular some are still geographic or maplike in configuration. It can be used as a clinical finding to identify the smoking history. Females are most commonly affected which suggest that strong influence between female sex harmone and smoking. Microscopically, basilar melanosis with melanin incontinence is observed, and the lesions have no premalignant potential [7]. It usually disappears without any treatment after cession of smoking habit. If a lesion is elevated or occurs in an unusual site, a biopsy should be performed. There is no evidence of the malignant transformation of smoker's melanosis.

Drug-Induced Pigmentation

A variety of medications cause oral pigmentation such as antimalarial agents (chloroquine, hydroxychloroquine and quinidine), tranquilizers (chlorpromazine), oral contraceptives, clofazimine, ketoconazole, amiodarone, busulfan, doxorubicin, bleomycin, cyclophosphamide, 5-fluorouracil, chemotherapeutic agents, minocycline, estrogen or medications to treat AIDS [8]. The etiology varies depending on the causative drug. It develops possibly due to increased melanin production usually temporary but it may be permanent also [9]. This type of pigmentations are

flat, diffuse, well demarcated, bluish grey discoloration of the hard palate, maxillary anterior alveolar mucosa or tongue and gingiva without any evidence of nodularity or swelling. These are focal reactions seen in oral mucosa and no malignancy reported [9].

Peutz Jeghers Syndrome

It is a rare genetic disorder associated with germline mutations in the STK11/LKB1 tumor suppressor gene. The syndrome consisted of mucocutaneous macules, intestinal polyposis and increased risk of carcinomas of the small intestine, colon, stomach, pancreas, breast and genital tract [10,11]. The macular pigmentation deposits often involve lips, buccal mucosa and fingers. Lesions may also develop on the gingiva, palate and tongue but this is not common. The spots are normally found to fade or disappear in older age. Histologically, the oral lesions demonstrate an increase in melanin in the basal layer, without an obviously increased melanocyte count. The melanotic spots do not need treatment and are not associated with increased risk of melanoma [12-14]. However, the patient should be observed for the development of internal malignancies. Such oral lesions facilitate in early diagnosis and should alert the clinician to prompt the patient to screen for cancers in organs implicated in this syndrome.

Varix and Thrombus

A varix is a pathologic dilated vein. It is seen mostly in patients older than 60 years of age as this is progressively prominent with age [9]. The site of intraoral involvement is the ventral surface of the tongue, where varices appear as multiple bluish purple, irregular, soft elevations that blanch on pressure which is subjected to increased hydrostatic pressure but inadequately supported by surrounding tissue [8,9]. Also represent a degenerative change in the adventitia of the venous wall and are of no clinical consequence. They are asymptomatic and are not subjected to rupture and hemorrhage. If the varix comprises a thrombus, it presents as a firm bluish-purple nodule that does not blanch on pressure. Thrombi are more common on the lower lip and buccal mucosa. The lesion can be excised or removed by electrosurgery or cryosurgery [15].

Malignant Melanoma

Malignant Melanoma is a neoplasm of epidermal melanocytes. It is rare and accounts for less than 1% of all oral malignancies. The most common site is palate followed by gingiva & other oral mucosal sites may be affected. It is generally encountered in males than female between 4th and 7th decade of life [15]. Clinically, it may present as an asymptomatic, slow growing, brown or black patch with irregular borders with ulceration, bleeding, pain and destruction. Oral mucosal melanoma tends to be more aggressive than the cutaneous counterpart. Superficial spreading melanoma, nodular melanoma, lentigo maligna melanoma, acral lentiginous melanoma, mucosal lentiginous melanoma is clinicopathological types of melanomas. Of these acral lentiginous melanoma and mucosal lentiginous melanoma occur in oral cavity. Histopathologically, characterized by proliferation of malignant melanocytes along the junction between the epithelial and connective tissue as well as within the connective tissue [16,17]. Treatment includes radical surgical excision with clear margins. The depth of invasion (Breslow depth) decides the prognosis. Immunotherapy and chemotherapy can act as adjunct. The prognosis for patients is worse, only 15% of patients survived for 5 years so the early detection is of paramount importance.

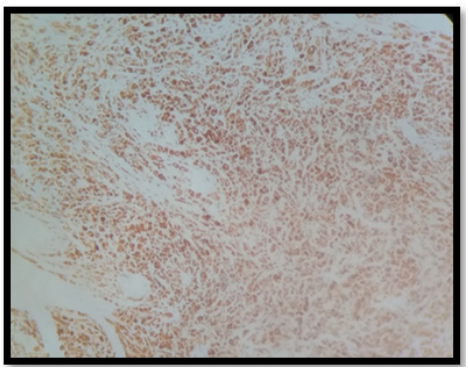

Figure 1: Histopathology of Malignant Melanoma

Kaposi's Sarcoma

It is a multicentric proliferation of vascular and spindle cells components. This malignancy is predominantly associated with AIDS progression. Human herpes virus (HHV-8) is implicated in the etiology of Kaposi's sarcoma. Immunosuppression is related with Kaposi's sarcoma [8,12,17]. Transplant recipients are another group that gets Kaposi's sarcoma. It is much more common in men than in women and it is rarely seen in children. Most common oral lesions occur on hard palate, gingiva and tongue. Initially lesion appears as a flat or slightly elevated brown to purple that often bilateral, advanced lesion appears dark red to purple plaques or nodules that may exhibit ulceration, bleeding and necrosis [18]. Definitive diagnosis requires biopsy which shows the proliferation of spindle shaped cells, surrounded by poorly formed vascular spaces or slits with numerous extravasated red blood cells. Anti-retroviral therapy is useful for AIDS associated Kaposi's sarcoma. Early detection of HIV will turn down further risk.

Melanocytic Nevi

Pigmented nevi are rare cause of focal oral pigmentation. It is a benign hamartomatous proliferation of the nevus cells either in the epithelium or in the connective tissue. Most common sites are hard palate, buccal mucosa and gingiva along with female predilection. It is classified as congenital and acquired, based on their size the congenital melanocytic nevi are further classified as giant melanocytic congenital nevi and small melanocytic congenital nevi [15,17,18]. The giant congenital melanocytic nevi are also called as garment nevus, bathing trunk nevus or giant hairy nevus. The acquired melanocytic nevus is commonly called as mole. 80% of nevi are smaller than 1cm. Intramucosal nevus is the most common variant seen on the buccal mucosa followed by blue, compound and junctional nevi. Histopathologically, based on location they are classified as junctional, intradermal, intramucosal and compound nevi. Superficial nevi like junctional nevi are dark brown as compared to deeper intramucosal and compound nevi which are light brown. In blue nevi, blue colour can be accounted when dermal melanocytes proliferates within deeper part of tissue. It is believed that nevi may represent precursor lesion to oral mucosal melanoma because of their most common site is palate [19,20].

Surgical excision of all intraoral pigmented nevi is the treatment of choice to rule out early melanoma or malignant transformation [20].

Hemangioma

It is a benign proliferation of the endothelial cells that lines vascular channels. It is a developmental abnormality & may be congenital or traumatic in origin. The cause is unknown but suggested its role of estrogen signaling in hemangioma proliferation [21]. The common site is tongue and lip mucosa. The lesion may be flat or slightly raised and varies in colour from red to bluish purple (port wine stain) to a nodular blue tumefaction. Vascular malformation is a structural anomaly of blood vessels without endothelial proliferation and persist throughout life [9]. The most characteristic histopathological feature of hemangioma is presence of numerous endothelia lined vascular channels containing RBCs. Lesions also show numerous proliferating plump endothelial cells. Based on size of the blood vessels hemangioma can be Capillary hemangioma- it is characterized by multiple, small proliferating capillaries lined by single layer of endothelial cells supported in the connective tissue stroma of varying density. Cavernous hemangioma- It is characterized by large irregular blood vessels lined by single layer of endothelial cells in the connective tissue stroma. Treatment includes surgery, flash lamp pulsed laser, intralesional injection of fibrosing agent and electrocoagulation [9].

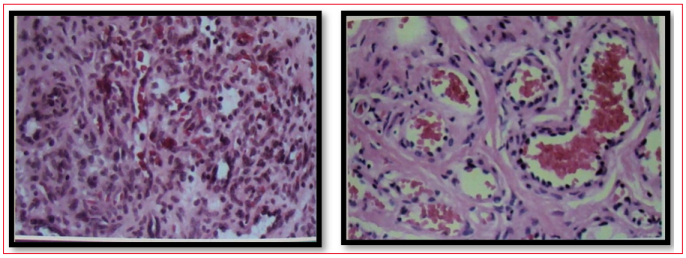

Figure 2: Photomicrograph Hematoxylin and eosin-stained sections showing
A. multiple, small proliferating capillaries lined by single layer of endothelial cells
B. large irregular blood vessels lined by single layer of endothelial cells in the connective tissue stroma.

Ephelids

Ephelids are a common small, brown, hyperpigmented macule of the skin that represents an increased melanin production. It effects on sun-exposed areas of perioral skin and lips. They are small (less than 1 cm) red or light to dark brown macules, multiple and regular in outline. Use of sun screen lotions prevents the appearance of new freckles and helps prevent the darkening of existing lesions. No treatment is necessary for ephelids. There is no chance of malignant transformation [22,23].

Amalgam Tatoo (Focal Argyrosis) and Other Foreign-Body Pigmentation

Amalgam tattoo is the single most common type of focal intraoral pigmentation by an exogenous agent such as dental amalgam and twice as common as melanotic macules and 10 times as common as oral nevi. It is localized, flat, solitary or multiple, discoloration which can be gray, blue or black. The borders may be irregular, well defined or diffuse; majority are 6 mm or less in dimension [24]. The most common location is gingiva and alveolar mucosa occasionally it is also seen on buccal mucosa, hard palate and floor of the mouth. Amalgam particles are implanted into the oral mucosa during restoration or removal of an amalgam filling, or during the extraction of an amalgam-filled tooth. A biopsy should be performed to exclude the possibility of mucosal melanoma. Microscopically, it shows several a fine brown granular stippling of reticulum fibers, particularly around vessel walls and in many instances, large chunks of black metallic particles can be seen. Diagnosis is made on the basis of the clinical information regarding previous prosthetic dental work history and radiographically fine radiopaque granules of metallic particles finding can safely rule out mucosal melanoma. No treatment is necessary for amalgam tattoo. Extensive lesion in cosmetic zone may be successfully treated with surgical excision and gingival grafting. Graphite can be surrounded into the oral mucosa through accidental injury with a graphite pencil. The lesion occurs most commonly in the anterior palate of young children, appearing as an irregular gray to black macule [25].

Oral Melanoacanthoma

It is an acquired, benign, uncommon pigmented lesion of oral mucosa characterized by proliferation of dendritic melanocytes scattered throughout the thickness of acanthotic and hyperkeratotic surface epithelium [18,23]. The cause is unknown, traumatic or reactive etiology has been proposed. Lesion appears as a flat or raised color ranges from dark brown to black. Buccal mucosa is most common site and affects young black females. It has tendency to enlarge rapidly which raises the possibility of malignancy. It appears to be a reactive lesion with no malignant potential [23].

Post-Inflammatory Pigmentation

Long-standing mucosal diseases can cause mucosal pigmentation. It is frequently seen in dark-skinned individuals [1]. The pathogenesis of post-inflammatory pigmentation is unclear. It is mostly seen in reticular or erosive type of lichen planus. Clinically, multiple brown- black pigmented areas are noted adjacent to lesion. Microscopically, increased production of melanin by melanocytes and accumulation of melanin laden macrophages in the connective tissue. No evidence of transformation into malignancy [1,8].

Addison Disease (Hypoadrenocorticism)

Addison's disease is the result of inadequate production of cortisol and aldosterone due to progressive bilateral destruction of the adrenal cortex caused by autoimmune disease, infection (tuberculosis) and malignancy. It has no gender predilection and affects all age groups. In response to insufficient corticosteroid levels in the blood, the anterior pituitary gland synthesizes and secrets increased amount of ACTH [24,25]. As ACTH level rises, there is a corresponding increase in alpha melanocyte stimulating hormone and this will cause melanogenesis. Clinically, oral involvement presents as diffuse brown patches on the gingiva, buccal mucosa, palate, and tongue. It can be fatal if left be untreated. It moreover occurs secondary to overproduction of the pro-opio melanocortin by product- lipotropin. Diagnosis involves exogenous ACTH stimulation testing with subsequent measurement of serum cortisol and plasma ACTH level. Management involves treatment of underlying cause and corticosteroid replacement therapy [25].

Cafe-Au-Lait Macule

These are brown patches of melanin pigment on the skin with irregular margins. Neurofibromatosis, Albright's syndrome, Noonan syndrome, Watson syndrome, bloom syndrome and ring chromosome syndrome are associated with Cafe-au-Lait macule [8].

Microscopically, excess amount of melanin is seen in basal keratinocytes and macrophages. No treatment is required, but proper clinical evaluation should be done to rule out the presence of any syndrome [8].

Hematoma and other Hemorrhagic Lesions

Hematomas, ecchymoses, petechiae and purpurae are produced

by extravasation of blood into the soft tissues. They appear as nonbranching flat or elevated pigmented lesions [1]. The colour, produced by the degradation of hemoglobin to bilirubin and biliverdin, varies among red, purple, blue and bluish black depending on the length of time the blood has been present in the extravascular spaces [1]. The colour gradually returns to normal but this can take up to 2 weeks. These Hemorrhagic lesions may occur spontaneously in certain systemic conditions such as idiopathic thrombocytopenic purpura, or they may result from trauma. If these lesions occur in the absence of recent trauma, the patient should be investigated for platelet disorders and coagulopathies [9,23].

Diagnosis for Oral Pigmented Lesions

History and proper clinical examination have enormous diagnostic value for drug induced pigmentations, racial pigmentation, smoker's melanosis, amalgam tattoo and heavy metal pigmentation. Histopathology is of paramount importance for the diagnosis of melanocytic nevus, oral melanoacanthoma and oral malignant melanoma. Immunohistochemical analysis also has a diagnostic appraisal to differentiate oral melanotic macules (HMB 45 negative) and oral malignant melanoma (HMB 45 positive). Although the provisional diagnosis is made based on the clinical findings, histopathological evaluation is essential for a decisive diagnosis to delineate the true biological nature. Laboratory findings (high level of urinary VMA) along with the age of occurrence have a pragmatic value for the analysis of pigmented neuroectodermal tumor of infancy. Thus, history, clinical findings, laboratory investigations and immunohistochemical analysis attached with histopathological evaluation must for the diagnosis of oral pigmented lesions [26-29].

Conclusion

Exact treatment modalities depend on the nature of the pigmented lesion. Pigmentation may be physiological or pathological pigmentation. Causative agents can be exogenous or endogenous. Appropriate diagnosis of oral pigmented lesions is important because these pigmented lesions may be traumatic, reactive, neoplastic, or associated with systemic pathogenesis and syndromes. The dentist should be attentive of the various lesions to aid in the proper treatment plan.

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Conflicts of Interest

There are no conflicts of interest.

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