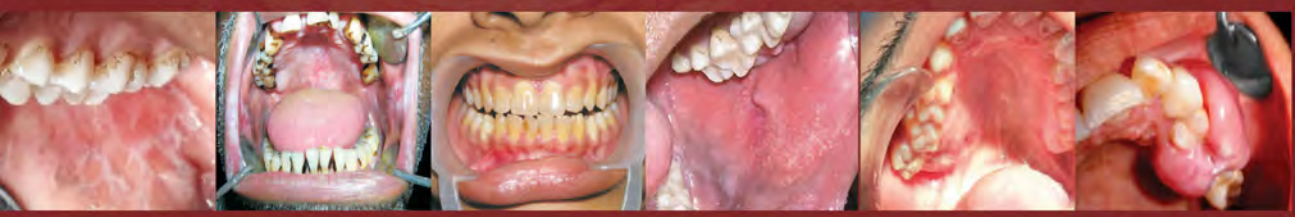
Swapan Kumar Purkait

Essentials of
ORAL PATHOLOGY



4th Edition

Forewords
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CONTENTS

CHAPTER

2

Benign and Malignant Neoplasms of the Oral Cavity

NEOPLASM (TUMOR)

DEFINITION

A neoplasm is an abnormal mass of tissue, the growth of which exceeds and is incoordinated with that of the surrounding normal tissues and persists in the same excessive manner after cessation of the stimuli that evoked the change (Rupert Willis 1950).

Important characteristics of a neoplasm:

- Abnormal growth—due to multiplication of cells that cannot be controlled
- Ceaseless—growth of a neoplasm never ends
- Purposeless—it is an unnecessary mass of cells
- Uncoordinated—growth pattern of tissue/cells is far in excess than that of the surrounding normal tissue
- Cessation of stimuli—a neoplasm continues to grow even after the stimulus or the initiating factor is removed.

Neoplasms can occur from virtually any tissue anywhere in the body and the oral cavity is an important location where a large variety of neoplasms often develop with diverse pathogenicity.

The modern classification of oral neoplasms is based primarily on the structural basis or in other words, several neoplastic conditions are put into different categories on the basis of their tissue of origin.

Depending on the pathologic state, the oral neoplasms can be divided into two broad categories or groups, namely:

1. Benign neoplasm
2. Malignant neoplasm

In the following section, we will see how a benign neoplasm may differ clinicopathologically from its malignant counterpart (Table 2.1).

Generally, the benign tumor is designated by attaching the suffix "oma" to the cell type from which it arises. For example, a benign tumor arising from the fibrous tissue is called a "fibroma" while a benign cartilaginous tumor is called a "chondroma". A benign epithelial tumor arising from the gland is known as "adenoma".

A malignant tumor arising from the epithelial tissue is called "carcinoma" and a malignant tumor arising from the connective tissue is known as "sarcoma". Recent literatures have documented about another malignancy, which is called "carcinosarcoma" and it is characterized by simultaneous malignant transformation of both the epithelial and mesenchymal components of the tissue.

LOCAL INVASION

When a tumor penetrates into the adjoining tissues due to its increased rate of growth, it is known as invasion. Most of the malignant tumors as well as few benign tumors show this behavior. Invasion is an important pathological

Table 2.1: Difference between benign and malignant neoplasms.

	Features	*Benign*	*Malignant*
On the basis of clinical features	Size of the tumor	Usually small	Usually large
	Rate of growth	Slow	Very fast
	Pain	Absent	Mostly painful
	Hemorrhage	Not usual	Very common
	Ulceration	Absent	Present
	Paresthesia	Does not occur	Commonly occurs
	Induration	Absent	It is often present
	Symptoms	Asymptomatic	Always symptomatic
	Superadded infection	Usually absent	Commonly present
	Necrotic areas	Usually absent	Commonly present
	Metastasis	Usually Absent	Very common
On the basis of histopathologic features	Cell multiplication rate	Slow	Very fast
	Cell maturation	Good	Cells are often immature
	Cell uniformity	Uniform	Irregular size and shape
	Cell morphology	Not changed	Normal cell morphology is lost
	Cell function	Restored	Mostly lost
	Stroma	Almost normal	Exhibits invasion
	Tissue architecture	Intact	Mostly lost or altered
	Capsule	(Resembles normal tissue) Usually present	Absent
Prognosis		Good	Mostly poor

change in any malignant neoplasm, which determines the future course of the neoplasm as well as the prognosis.

METASTASIS

Metastasis can be defined as the distant spread of tumor cells anywhere in the body away from its primary location. This is an important characteristic of the malignant tumor. The tumor which occurs initially is called the primary tumor; while the newly formed tumor developing as a result of metastasis at a distant site is called the metastatic or secondary tumor.

During metastasis, the tumor cells spread either via the lymphatic channels or the blood vessels, besides this, in some cases, the metastatic cells can spread via the nerve sheath or even through other natural tissue spaces. With some exceptions, the carcinomas generally metastasize via lymphatic channels while the sarcomas metastasize via blood vessels.

CLASSIFICATION OF ORAL NEOPLASMS (TUMORS)

In the oral cavity, several types of neoplasms often develop and these entire varieties of neoplastic lesions are broadly divided into two categories:

1. Odontogenic neoplasm and
2. Nonodontogenic neoplasms.

Odontogenic neoplasms: These are a group of neoplastic conditions either benign or malignant, which develop from the dental formative tissues or their remnants.

Nonodontogenic neoplasms: These are the neoplastic lesions, which arise from virtually any tissue in the oral cavity excepting from those arising from the dental formative organs.

The nonodontogenic neoplasms can develop from several tissues like skin or mucous membrane, fibrous connective

Table 2.2: Neoplasms of epithelial tissue origin.

Benign neoplasms	*Malignant neoplasms*
Papilloma	Basal cell carcinoma
Keratoacanthoma	Squamous cell carcinoma
Pigmented cellular nevus	Verrucous carcinoma
Papillary hyperplasia	• Adenoid squamous cell carcinoma • Adenosquamous cell carcinoma • Malignant melanoma • Spindle cell carcinoma • Primary intra-alveolar carcinoma • Multicentric oral carcinoma

tissue, blood vessels, muscles, bone, cartilage, neural tissue and lymphoid tissue, etc. It is important to remember that unlike the odontogenic neoplasms which can arise only in the oral cavity or its surrounding areas, the nonodontogenic neoplasms are not always confined to the oral region, rather they can develop in other parts of the body as well.

Classification of Oral Nonodontogenic Neoplasms

The neoplasms of epithelial tissue origin and neoplasms of mesenchymal tissue origin are given in Tables 2.2 and 2.3, respectively.

Table 2.3: Neoplasms of mesenchymal tissue origin.

Benign neoplasms	*Malignant neoplasms*
Neoplasms of fibrous connective tissue	*Neoplasms of fibrous connective tissue*
Fibroma	Fibrosarcoma
Fibromatosis	Malignant fibrous
Desmoplastic fibroma	Histiocytoma
Pyogenic granuloma	
Fibroepithelial polyp	
Giant cell fibroma	
Peripheral ossifying fibroma	
Central ossifying fibroma	
Peripheral giant cell granuloma	
Central giant cell granuloma	
Benign fibrous histiocytoma	
Nodular fasciitis	
Myxoma	
Neoplasms of adipose tissue	*Neoplasms of adipose tissue*
Lipoma	Liposarcoma
Angiolipoma	
Neoplasms of vascular tissue	*Neoplasms of vascular tissue*
Hemangioma	Hemangiopericytoma
Lymphangioma	Hemangioendothelioma
Juvenile angiofibroma	Angiosarcoma
Hereditary hemorrhagic telangiectasia	Kimura's disease
Glomus tumor	

Contd...

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Benign neoplasms	***Malignant neoplasms***
Neoplasms of osseous tissue	*Neoplasms of osseous tissue*
Osteoma	Osteosarcoma
Osteomatosis	Parosteal osteosarcoma
Osteoid osteoma	Ewing's sarcoma
Osteoblastoma	
Osteoclastoma	
Torus palatinus	
Torus mandibularis	
Neoplasms of cartilaginous tissue	*Neoplasms of cartilaginous tissue*
Chondroma	Chondrosarcoma
Chondroblastoma	Mesenchymal
Chondromyxoid fibroma	Chondrosarcoma
Neoplasms of neural tissue	*Neoplasms of neural tissue*
Neurolemmoma	Neurosarcoma
Neurofibroma	Olfactory neuroblastoma
Neurofibromatosis	
Multiple endocrine neoplasia syndrome	
Melanotic neuroectodermal tumor of infancy	
Neuroblastoma	
Ganglioneuroma	
Traumatic neuroma	
Plexiform neuroma	
Neoplasms of smooth muscle tissue	*Neoplasms of smooth muscle tissue*
Leiomyoma	Leiomyosarcoma
Angiomyoma	Angiomyosarcoma
Neoplasms of striated muscle tissue	*Neoplasms of striated muscle tissue*
Rhabdomyoma	Rhabdomyosarcoma
Granular cell myoblastoma	
Congenital epulis of newborn	
Neoplasms of lymphoid tissue	*Neoplasms of lymphoid tissue*
No benign neoplasm	Hodgkin's lymphoma
	Non-Hodgkin's lymphoma
	Burkitt's lymphoma
	Mycosis fungoides
	Leukemias
	Multiple myeloma
	Plasmacytoma
Neoplasms of mixed tissue	*Neoplasms of mixed tissue*
Teratoma	
Neoplasms of salivary gland tissue	*Neoplasms of salivary gland tissue*
See the chapter of *Salivary Gland Neoplasm* (Chapter 4)	

BENIGN NEOPLASMS OF THE EPITHELIAL TISSUE ORIGIN

Papilloma

Definition

Papilloma is a common benign neoplasm of the oral cavity, arising from the epithelial tissue. It is characterized by an exophytic papillary growth with a typical "cauliflower like" appearance.

This lesion constitutes about 2% of all oral neoplasms and it is believed by many investigators that they are caused by human papilloma virus (HPV).

HPV virus subtypes 6 and 11 frequently detected from neoplastic tissues of papilloma.

Clinical Features

- *Age*: Any age but mostly third, fourth and fifth decade
- *Sex*: Both sexes are equally affected
- *Site*: Tongue, lips, buccal mucosa, gingiva, hard and soft plate, etc.

Clinical Presentation (Figs. 2.1 and 2.2)

- Clinically, papilloma appears as a slow growing, exophytic, soft, usually pedunculated, painless, nodular growth often with a typical "cauliflower-like" appearance.
- Papillomas often characteristically have numerous finger-like projections on their surface, which can be either blunt or pointed.

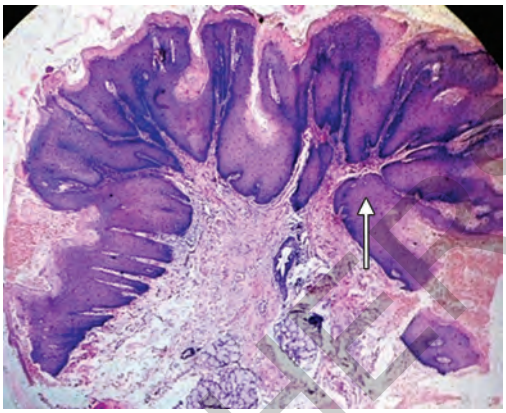
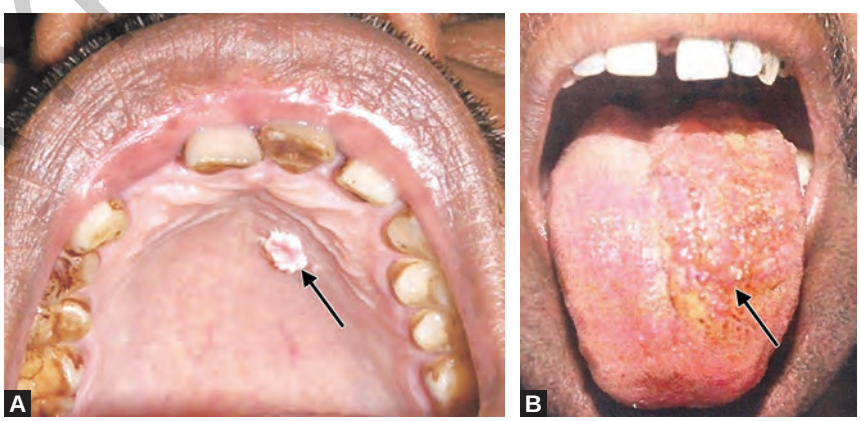


Fig. 2.2: Histopathology of papilloma (low power).

- Because of these projections, the papilloma often appears as an ovoid swelling with a rough, corrugated surface.
- The size of the lesion is usually small and that varies from few millimeters to about one centimeter in diameter.
- The base of the lesion can be either pedunculated or sessile (broad based) but papilloma is mostly a well-circumscribed growth.
- The lesion is mostly white in color and is firm in consistency as the surface is highly keratinized.
- On rare occasions, papillomas may grow in an inwardly direction (inverted type) instead of growing in the usual exophytic manner. Such lesions are mostly



Figs. 2.1A (A) Papilloma on the palate; (B) Papillomatosis of tongue.

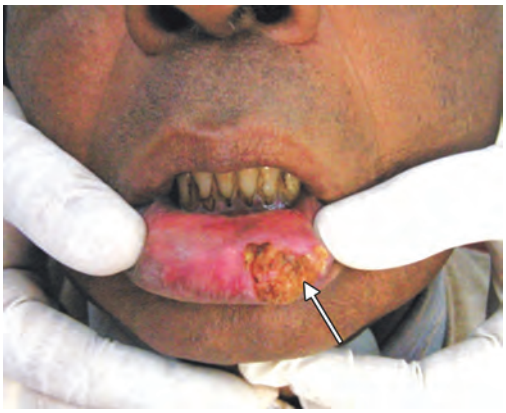


Fig. 2.3: Keratoacanthoma of lower lip.

seen in the lateral nasal wall, paranasal sinuses and in the maxillary antrum, etc. Moreover, they have great tendency for local destruction and malignant transformation.

- Multiple papillomas may sometimes coalesce together and form a large lesion in the oral cavity and the condition is commonly known as "papillomatosis" (Figs. 2.3 and 2.4).
- Papillomatosis of oral mucosa may sometimes occur in association with skin disorders, e.g. focal dermal hypoplasia syndrome, nevus unius lateris, Cowden syndrome and acanthosis nigricans, etc.

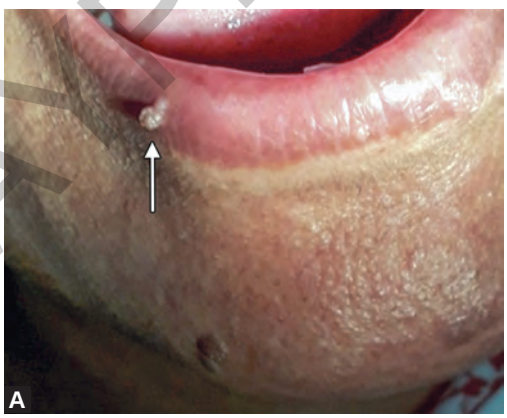
Histopathological Features

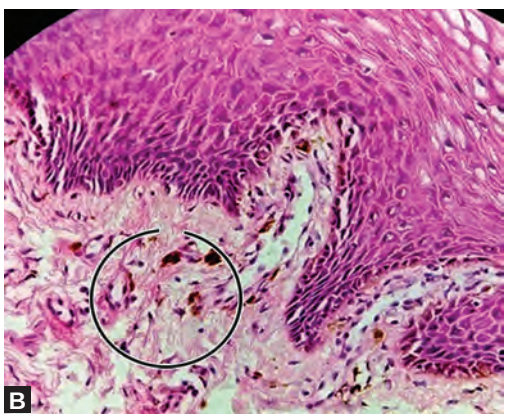
Microscopically, papillomas present the following features:

- Proliferating keratinized stratified squamous epithelium in the form of multiple fingers-like projections.
- Every single finger-like projection has a fibrovascular connective tissue core in the center, which contains few inflammatory cells.
- The covering squamous epithelium shows hyperkeratosis and acanthosis. Thickening of the keratin is seen in lesions which are clinically whiter.
- In the spinous cell layer "koilocytes" are sometimes seen, these are virus-altered epithelial clear cells with small dark (pyknotic) nuclei surrounded by clear halos.
- There can be little cellular atypia in some papillomas, however, the dysplastic changes in the epithelium is rarely found.
- Papilloma is not a premalignant lesion and malignant transformation in preexisting oral papillomas has not been documented.

Differential Diagnosis

- Verruca vulgaris
- Focal dermal hyperplasia
- Verruciform xanthoma
- Verrucous carcinoma
- Condyloma acuminatum.





Figs. 2.4A and B: (A) Nevus on the lip; (B) Nevus cells.

Treatment

Conservative surgical excision of the lesion including the base is the common treatment. Recurrence is rare.

Keratoacanthoma (Self-healing Cancer)

Definition

Keratoacanthoma is a benign endophytic epithelial tissue neoplasm with profound clinical and histological resemblance to well-differentiated squamous cell carcinoma (SCC). It commonly occurs in the sun-exposed skin of the face and it usually appears as a circumscribed keratin filled crater.

Origin

Keratoacanthoma of the skin surfaces probably develops from the hair follicles above the sebaceous glands.

On the mucosal surfaces these lesions are extremely rare but if they occur at all, they probably develop from the superficial epithelium of the sebaceous ducts.

Causes

- Chronic sun exposure
- HPV infection (especially types—9, 11, 13, 16, 18, 25, 33, 37 and d5)
- Immunosuppression
- *Heredity*: Chromosomal aberrations such as gains on 8q, 1p, and 9q with deletions on 3p, 9p, 19p, and 19q.
- Trauma.

Clinical Features

- *Age*: Middle-aged adults are frequently affected between the age group of 50 years and 70 years
- *Sex*: Male to female ratio in this tumor is about 2:1
- *Site*: Keratoacanthoma chiefly develops over the sun-exposed skin surface of the lips (both upper and lower lips) near the outer edge of vermilion border. Besides this, the lesion can also occur on the cheeks, nose, eyelids and ear. Intraoral lesions of keratoacanthoma are rare, although few have been reported in the palate and gingiva.

Presentation (Fig. 2.3)

- Keratoacanthoma initially begins as a small, red macule that soon turns into a well-circumscribed, elevated and umbilicated, crater-like lesion with a central depression.
- The lesion is firm, painless and sessile in nature; and can be single or multiple in number.
- The fully developed lesion of keratoacanthoma clinically presents a well-circumscribed, elevated nodule, which has a sharply delineated, rolled margin and a central keratotic core.
- Clinically, the outer surface of the lesion shows normal skin color or slight erythema, while the central keratin plug appears yellow, brown or black with an irregular crusted appearance.
- The disease is often painful and sometimes it may have an associated lymphadenopathy.

Stages of Development of Keratoacanthoma

- *Growth phase*: The lesion initiates as a small lump or a bud like growth on the sun-exposed skin surface of the face, it grows rapidly and achieves the maximum size (1–2 cm in diameter) over a period of about 4–8 weeks.
- *Stationary phase*: After the initial growth, the disease remains static for an indefinite period of up to 4–8 weeks and then it starts to regress spontaneously.
- *Involution phase*: Within the next 6–12 months time, the lesion regresses completely leaving only a small depressed scar.

Muir–Torre syndrome: Gastrointestinal carcinoma, keratoacanthoma, and sebaceous neoplasm.

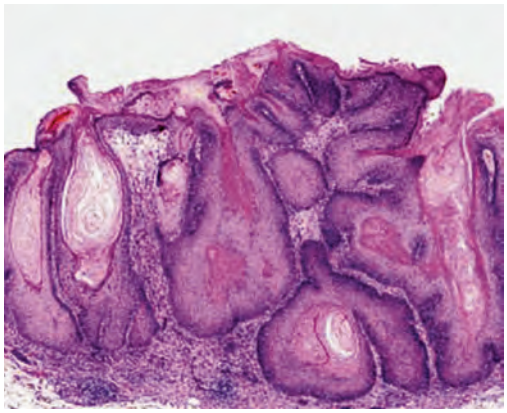


Fig. 2.5: Photomicrograph of keratoacanthoma.

Histopathology (Fig. 2.5)

- Keratoacanthoma clinically and histologically appears very similar to well-differentiated SCC and because of this, it is often known as "self-healing" cancer (Box 2.1).
- The cells appear mature and often there is individual cell keratinization and even keratin pearl formation in the tumor.
- The lesion consists of a thick hyperkeratinized covering epithelium with a central zone of keratin or parakeratin plugging.
- Pseudoepitheliomatous hyperplasia may be observed in some cases.
- Pathognomonic nonmalignant feature of this neoplasm can be identified at the margin, where the lesion shows a crater-like area, plugged with keratin and is surrounded by hyperplastic normal epithelium.
- This abrupt transition of the normal surrounding epithelium at the margin of the crater-like area is an important diagnostic clue for keratoacanthoma.
- Although, keratoacanthomas are benign and self-limiting conditions, serial sectioning is always required of the available tissue sample for confirmation of the diagnosis.
- Moreover, careful long-term follow-up evaluations are necessary since the neoplasm is often confused with SCC.

Box 2.1 The distinction between keratoacanthoma and squamous cell carcinoma.

- The epithelium in this neoplasm exhibits a pseudocarcinomatous rather than a true carcinomatous growth pattern
- Dyskeratosis is always absent in keratoacanthoma
- The epithelium is composed of well-differentiated spinous cells with abundant cytoplasm, minimal nuclear pleomorphism, infrequent mitotic figures and no abnormal mitosis

Differential Diagnosis

- Basal cell carcinoma
- Squamous cell carcinoma.

Treatment

Surgical excision is the treatment of choice for keratoacanthoma, usually before the lesion reaches its maximum size of 2–2.5 cm diameter. Waiting for spontaneous regression of the lesion is not advisable for the following reasons:

- Confusion with SCC
- The scar developing after spontaneous regression is depressed and cosmetically unacceptable
- Surgical treatment always provides good tissue specimen for confirmation of the diagnosis.

Pigmented Cellular Nevus

Definition of Nevus

The term "nevus" has got several meanings; in *Latin* nevus means birth marks, however, the common lay term used for nevus is "mole". A nevus can be defined as a congenital, developmental, tumor-like malformation of the skin or mucous membrane; which are often present at birth or they can be seen any time after birth (Box 2.2).

Oral nevi are much rarer than their cutaneous counterparts with a prevalence of 0.1% among the general population; Women are affected twice as common as men and most cases are seen in the third and fourth decades. In the mouth these are commonly seen in the hard palate, buccal and labial mucosa.

Box 2.2 Different types of nevi.

- Intradermal (intramucosal) nevus
- Junctional nevus
- Compound nevus
- Blue nevus
- Halo nevus (halo mole)

Nevus is composed of "nevus cells" which are neuroectodermal in origin, these cells, except for their tendency to form cell nests and their less prominent dendritic processes, are nothing but melanocytes or their precursors. After their formation, nevus cells migrate through the peripheral nerves and finally reach to the basal layer of the skin or mucous membrane (*See* Figs. 2.4A and B).

The function of nevus cells is to produce melanin, this pigment, after being synthesized within the nevus cells is passed on to the adjacent keratinocytes of the oral mucous membrane.

Intradermal (Intramucosal) Nevus

The term intradermal nevus and intramucosal nevus are synonymous, the former occurs on the skin surfaces while the later occurs over the mucous membrane.

Clinical Features

- Intradermal nevus is a very common lesion of the skin (comprise more than 50% of all types of nevi) and it usually occurs in children
- This lesion is often referred to as common "mole"
- Intradermal nevus clinically appears as a raised or flat area on the skin surface, with a typical tan or dark brown color
- The mucosal lesions clinically appear as asymptomatic, slightly elevated papules or flat macules with a pigmented surface
- The color of these nevi varies from brown to black
- The intraoral lesions are often slow growing and their size is usually less than 1 cm in diameter.

Histopathology

- Microscopically intramucosal nevus reveals clusters or nests of nevus cells which are confined within the connective tissue.
- The cells may appear as epithelioid cells or lymphocyte like cells; however few cells may be even spindle-shaped.
- Multiple multinucleated giant cells may be found in some cases.
- Intramucosal nevus often characteristically presents a narrow zone of connective tissue devoid of nevus cells, which separates the zone of nevus cells from the overlying epithelium.
- The amount of melanin produced by these nevus cells varies; some cells are heavily pigmented whereas other cells are almost nonpigmented.

Treatment

Intramucosal nevi usually do not require any treatment. However, when these lesions are subjected to persistent trauma during mastication, surgical excision should be preferred. Once excised, they usually do not recur.

Junctional Nevus

Clinical Features

- Junctional nevus is usually a less common variety as compared to the intradermal nevus.
- It appears as an asymptomatic, brown or black macule, affecting both skin as well as the oral mucosal surfaces.

Histopathology

- Histologically, junctional nevus reveals focal areas of proliferating nevus cells or in some cases clusters of cells at the basement membrane zone of the epithelium.
- The cluster of nevus cells is often specifically present at the apex of the epithelial rete-pegs.

Treatment

Junctional nevus should be excised surgically. Postsurgical recurrence is uncommon.

Compound Nevus

The compound nevus characteristically presents the combined features of intradermal nevus and the junctional nevus.

Clinical Features

- This lesion occurs far more commonly in skin as compared to the oral mucosa
- Intraorally they appear as pigmented papules or macules over the hard palate or the gingiva.

Histopathology

Microscopically, compound nevus reveals the presence of nevus cells, which are distributed both in the basal layer of the epithelium as well as in the adjacent superficial connective tissue.

Treatment

Surgical excision is the treatment of choice for compound nevus.

Blue Nevus

Blue nevus is a relatively common pigmented lesion of the oral cavity.

- Two major types are recognized—(1) the common blue nevus and (2) the cellular blue nevus.
- The common blue nevus is the second most frequent melanocytic nevus encountered in the mouth.

Clinical Features

- Clinically, it often appears either as a dome-shaped, dark blue papule or as a flat pigmented macule over the skin or the oral mucous membrane.
- Intraoral blue nevi are commonly seen on the mucosal surfaces of the hard palate.

Histopathology

- Instead of being round or epithelioid in shape, the cells of blue nevus are usually elongated, bipolar and spindle-shaped.
- Sometimes, fusiform dendritic cells can also be present in these lesions.
- The spindle-shaped cells are mostly oriented parallel to the overlying epithelium and are not arranged in clusters.
- Few pigmented macrophages may be present among these dendritic nevus cells and they are known as "melanophages".

Treatment

Blue nevus often clinically resembles a melanoma and therefore surgical excision of the lesion with subsequent histopathological evaluation blue nevi do not have much tendency to undergo malignant transformation.

Halo Mole (Halo Nevus)

It is a peculiar nevus on the skin, which characteristically has a white ring around it; (also known as Sutton nevus or leukoderma aquisitum centrifugum) (Fig. 2.6).

It develops like any other nevus of the skin with the typical nevus cell being present in it, but later on, an autoimmune reaction develops against the nevus to destroy it, and there is activation of cytotoxic T lymphocytes, which start destroying the nevus cells. The white ring is

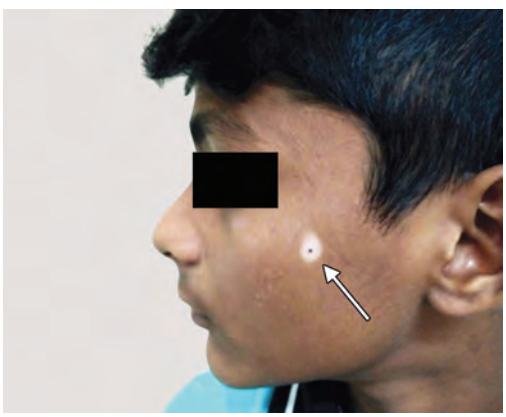


Fig. 2.6: Halo nevus on the facial skin.

Box 2.3 Symptoms of malignant transformation in a preexisting nevus.

- Sudden faster rate of growth
- Size becoming much more than 5 mm in diameter
- Asymmetric growth pattern
- Irregular border of the lesion
- Sudden change of color (becoming darker in color).
- Development of surface ulceration and pain (sometimes)

(*Histopathological confirmation is always required*)

created due to the elimination of the pigmented cells at the periphery of the nevus (Box 2.3).

MALIGNANT NEOPLASMS OF THE EPITHELIAL TISSUE ORIGIN

Squamous Cell Carcinoma

Definition

Squamous cell carcinoma is the most common malignant epithelial tissue neoplasm of the oral cavity, which is derived from the stratified squamous epithelium. Since oral SCCs constitute bulk of the oral malignancies (above 90%) it is thus commonly referred to as oral cancer (although there are several other malignancies of the oral cavity besides SCC).

Epidemiology

- SCC is also termed as epidermoid carcinoma and it is by far the most common malignant neoplasm of the oral cavity.
- The incidence of oral SCC varies in different countries and also in different population groups. It represents the eleventh most common cancer in males and the sixteenth most common in females.
- On an average, oral SCCs represent about 3% of all cancers in males and about 2% of all cancers in females.
- This disease is responsible for 2% of all annual deaths in males and 1% of all annual deaths in females.
- The incidence of oral cancer varies in different countries depending upon the frequency of tobacco usage and other related habits throughout the world.
- The general trend indicates that the incidence of oral SCC increases alarmingly in the societies, where extensive tobacco use begins in the early life and is continued for a longer period.
- The incidence of oral SCC increases with age and most of the cases occur usually after the age of 40 years.
- Although, oral SCCs can arise from virtually any intraoral site, but they develop more frequently from the lower lip, lateral borders of the tongue, buccal mucosa and floor of the mouth, etc.
- According to this ICD classification, oral cancers are numerically categorized in the following manner:
  - Lip cancer–ICD No. 140
  - Tongue cancer–ICD No. 141
  - Cancer of the gingiva and alveolar mucosa–ICD No. 143
  - Cancer of the floor of the mouth–ICD No. 144
  - Cancer of other parts of the mouth–ICD No. 145.
- The annual age-adjusted incidence rates of oral cancer per 100,000 (one lakh) population varies from continents to continents, from countries to countries and also from places to places within the same country.
- In Europe, it varies from 2.0, in UK to 9.4, in France, in USA it varies from 4.4, in Columbia to 13.4 in Canada.
- In Asia, the annual incidence rates of oral cancer per 100,000 population vary from 1.6 in Japan to as high as 13.5 in India.
- In Sri Lanka, the oral cancers constitute about 40% of all malignancies.
- In the Manipuri districts in India, the annual incidence rate of oral cancer is about 21.4 per 100,000 population (Wahi 1968).
- Survey among textile mill workers in Ahmedabad (Gujarat), India, indicates that

the average incidence rate of oral cancer among individuals above 35 years of age is 25 per 100,000 population (Malaowalla et al.).

- Recent trends besides few exceptions indicate that the incidence and mortality rates of oral cancer are declining and it can be due to the reduced exposure to various etiological agents.

Etiology of Oral Cancer

A large number of etiological factors have been implicated in the development of oral cancer, specifically the oral SCCs, these include the following:

- Tobacco smoking
  - Cigarettes
  - Beedies
  - Pipes
  - Cigars
  - Reverse smoking
- Use of smokeless tobacco
  - Snuff dipping
  - Tobacco sachets (*Gutkha*)
  - Tobacco chewing
  - Tobacco as a toothpaste
- Consumption of alcohol
  - Drinking spirits
  - Drinking wines
  - Drinking beers
  - Tobacco and alcohol synergism (smoking and chewing tobacco with drinking of alcohol)
- Diet and nutrition
  - Vitamin A, B-complex and C deficiency
  - Nutritional deficiency with alcoholism
- Dental factors
  - Chronic irritation from broken teeth
  - Ill-fitting or broken prosthesis
- Radiations
  - Actinic radiation, X-ray radiation
- Viral infections
  - Herpes simplex virus (HSV)
  - Human papilloma virus (HPV)
  - Human immunodeficiency virus (HIV)
  - Epstein-Barr virus (EBV)
- Immunosuppression
  - AIDS
  - Organ transplants
- Chronic infections
  - Candidiasis
  - Syphilis
- Occupational hazards
  - Woolen textile workers
- Genetic factors
  - Oncogenes
  - Tumor-suppressor genes
- Preexisting oral diseases
  - Lichen planus
  - Plummer–Vinson syndrome
  - Oral submucous fibrosis
  - Leukoplakia
  - Discoid lupus erythematosus.

Role of Tobacco in Oral Cancer

- One person dies in every 10 seconds in the world due to the use of tobacco.
- Epidemiological and experimental studies categorically indicate that tobacco plays an important role in the development oral cancer.
- Tobacco is used in various smoking forms such as cigarettes, cigars, pipes and *beedies*, etc.
- It can also be used in smokeless forms like paan-beetel quid, snuff, tobacco sachets (*khaini*), *zarda* and other popular forms.

Factors Influencing the Carcinogenic Effect of Tobacco

- Frequency of smoking—number of cigarette or Pipe used per day (smoking 40 or more cigarettes per day increases 10–20 times more cancer risk).
- Duration of smoking—habit continued for how many years.
- Age of beginning of the habit—smoking from a younger age has more risk.
- Composition of tobacco—variation in tar, nicotine and nitrosamine content, e.g. "beedi" (country made cigarette) is made up of crude form of tobacco containing more harmful chemicals and it has no filter.

Essentials of
Oral Pathology

Salient Features

- All chapters are adequately covered, revised and updated within a concise volume
- Inclusion of large numbers of clinical and radiographic photographs and several photomicrographs of very important cases is the hallmark feature of this edition
- Simple and lucid language for easy understanding
- Concise and comprehensive presentation of each topic will be helpful for both undergraduate and postgraduate students
- Latest classifications of oral diseases, recent grading and staging system of tumors have been added
- Several diagrams, tables and flowcharts have been provided to help students understand the subject better.

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