"A CLINICAL STUDY ON PIGMENTED LESIONS OF THE ORAL CAVITY AND LIPS"

BY

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ABSTRACT

BACKGROUND

Pigmentation of the oral mucosa is caused by the accumulation of one or more pigments leading to cellular hyperplasia and featuring a change in color of tissues. Pigmentation of the oral mucosa reflects the underlying health status either local or systemic. Oral pigmentation could be physiological or pathological that can range from benign nevi to fatal oral melanoma. A large number of lesions must be considered in the differential diagnosis, and the treatment may vary from observation to radical surgery. The presence of oral pigmentation may be an important clue to underlying systemic disease, and the dermatologist needs to be vigilant and informed in this regard and be prepared to refer the patient for appropriate work-up by other specialists. By this, treatment modalities can be employed at an early stage avoiding undue complications.

There have been very few studies regarding pigmented lesions of oral cavity and lips. Hence this study is undertaken to characterize the frequency of pigmentary lesions of oral cavity and lips in Kolar district region. This study will facilitate in deriving a region specific list of disorders which in turn, will help in taking preventive steps towards various complications.

OBJECTIVES

To determine the frequency of pigmented lesions of oral cavity and lips in patients attending Dermatology OPD, R.L. Jalappa Hospital, Tamaka, Kolar.

METHODS

This study was conducted out between January 2011 to September 2012. All patients reporting to Department of Dermatology were evaluated and those who had oral pigmentation were enrolled into the study. A written consent was taken. A detailed medical and dental history including demographic data, chief complaints related to skin, presence of oral pigmentary lesions and associated medical disorders were recorded. A detailed physical examination was made to see any associated lesions elsewhere in the body.

RESULTS

Total out patients aged 18 years and above attending department of dermatology were 18000. Among these 200 patients (1.11%) had oral pigmentary lesions. Most patients belonged to the age group of 35-50 years (130 patients, 65%), followed by the age group 50-65 years (70 patients, 35%). Most patients were asymptomatic (120 in number, 60% cases) followed by discoloration of oral cavity (55 in number, 27.5% cases). Other complaints were swelling and burning. 40 patients (20%) had more than one complaint. Lesions noted exclusively on oral mucosa were seen in 89 cases (44.5%) of 200 patients. Other unassociated skin diseases were also diagnosed in 111 cases (55.5%) during screening. In both male and female patients, anterior labial gingiva (70 patients, 35%) was the most common site of involvement, followed by buccal mucosa (65 patients, 32.5%) and hard palate (50 patients, 25%). A total of 13 different types of oral pigmentary lesions were noted in our study. The most common was melanoplakia (60 cases, 30%) followed by 53 cases (26.5%) of smoker's melanosis, 36 (18%) cases of nicotinic chelitis and 17(8.5%) cases of drug induced melanosis.

INTERPRETATION & CONCLUSION

Our study brings to light various oral pigmenatry lesions in general population. This study highlights the importance of diagnosing oral pigmentary lesions and removes the general misconception of considering most oral lesions to be precancerous. Thus clinicians need to differentiate the various causes of oral pigmentary lesions. Most of our patients are from rural or semi urban areas with poor oral hygiene and a list of incriminating factors for oral pigmentation like chronic smoking, betel chewing, spicy foods and paan masala. Therefore, it is of immense importance for all general physicians to be aware of all the conditions enlisted above to facilitate easy diagnosis and relieve the patient from unnecessary psychological trauma.

Key words: Oral pigmentation, Melanoplakia, Smoker's melanosis.

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INTRODUCTION

Pigmentation of the oral mucosa is caused by the accumulation of one or more pigments leading to cellular hyperplasia and featuring a change in colour of tissues. Healthy oral mucous membranes are normally various shades of red. When either the patient or the clinician notices areas of pigmentation, there is often an element of increased concern. Changes in colour of the oral mucosal surfaces can be of paramount significance because they frequently represent diagnostic evidence of either local or systemic disease. The range of diagnosis extends from variations of physiological to benign and malignant neoplasms.

Careful inspection of all the oral cavity surfaces is often necessary to detect subtle alterations in colour, and associating the abnormality with an undiagnosed systemic disorder may require a complete physical examination and laboratory testing. This is especially the case when oral pigmentation is diffuse and involves multiple surfaces of the oral cavity. Biopsy of oral pigmented lesions that are focal and of undetermined etiology is required to detect oral melanomas at an early stage. A large number of lesions must be considered in the differential diagnosis, and the treatment may vary from observation to radical surgery.

The presence of oral pigmentation may be an important clue to underlying systemic disease, and the dermatologist needs to be vigilant and informed in this regard and prepared to refer the patient for appropriate work-up by other specialists. By this, treatment modalities can be employed at an early stage avoiding undue complications.

AIMS AND OBJECTIVE OF THE STUDY

To determine the frequency of pigmented lesions of oral cavity and lips in patients attending Department of Dermatology, R. L. Jalappa Hospital and Research centre, Tamaka, Kolar**.**

REVIEW OF LITERATURE

ORAL MUCOSA

Development, Anatomy and Functions

Mucosa

Mucosa is a lining of a body cavity that opens to outside of body[1].

Oral mucosa is a moist lining of the oral cavity.[1] It consists of:

I. A covering epithelium

II. An underlying connective tissue - the lamina propria

The oral mucosa is considered as an organ, as it performs a similar function like skin and other mucous membranes lining the body cavities.[2]

Organization of oral mucosa

The oral cavity consists of two parts.[3, 4, 5, 6, 7, 8, 9]

I. An outer vestibule, which is bounded by lips and cheeks.

II. The oral cavity proper, separated by an alveolus bearing gingiva and the teeth.[10]

The superior zone of oral cavity proper is formed by hard and soft palates, and the inferior zone is formed by the floor of the mouth and base of the tongue. Posteriorly, it is bounded by the faucial pillars and the tonsils.[2]

Clinical Features

Generally, oral mucosa appears pale pink and its mucosal surface is moist and its glandular component is represented by minor salivary glands primarily.[2, 3, 4, 5, 6, 8, 9]

The surface of the oral mucosa tends to be smoother and have fewer folds. Papillae are seen on the dorsal surface of the tongue as rugae and transverse ridges are seen on the palatal surface.[2]

Depending on location, oral mucosa varies considerably in its firmness and texture. The lining mucosa of the lips and cheeks is soft and pliable whereas the gingiva and the hard palate are covered by a firm immobile layer.[2]

Development of Oral Mucosa

The oral cavity is lined by epithelium derived from both the ectoderm and endoderm. The regions of the oral cavity lined with the epithelium of ectodermal origin include the gingiva, the mucosa lining the cheeks, hard and soft palates. The structures derived from the endoderm are the tongue, the floor of the mouth, the pharynx and the epiglottis.[4, 5, 6, 7, 8, 9, 11]

HISTOLOGY

Oral mucosa is composed of:

a. Epithelial tissue which is a stratified squamous epithelium analogous to epidermis of the skin.[4, 5, 6, 7, 8, 9, 11]

b. The underlying loose connective tissue component called lamina propria, analogous to dermis of skin. A basal lamina - basement membrane

complex separates the epithelium from the lamina propria.[3, 4, 5, 6, 7, 8, 9]

c. Sub mucosa.[3, 6]

A. EPITHELIAL PORTION

The epithelium of the oral mucosa is stratified squamous in character. It may be keratinized, non- keratinized and para-keratinized depending on the location.[3, 4, 5, 6, 7, 8, 9]

In humans, the epithelial tissues of gingiva and hard palate are keratinized, although in many individuals the gingival epithelium is para keratinized. The cheek, faucial and the sub lingual tissues are non- keratinized.[3, 4, 5, 6, 7, 8, 9]

Based on keratinization, the epithelium of oral mucosa is classified into two types.[3, 4, 5, 6, 7, 8]

i. Keratinized epithelium

The keratinized epithelium is composed of four layers.[3, 4, 5, 6, 7, 8, 9]

a) Basal cell layer (Stratum Basale)

b) Prickle cell layer (Stratum Spinosum)

c) Granular cell layer (Stratum Granulosum)

d) Cornified layer (Stratum Corneum)

ii. Non keratinized epithelium

Like keratinized epithelium, it also has four layers, but in this type of epithelium,

granular and cornified layers are replaced by intermediate and superficial cell layers.

a) Basal cell layer (Stratum Basale)

b) Prickle cell layer (Stratum Spinosum)

c) Intermediate cell layer (Stratum Intermedium)

d) Superficial cell layer (Stratum Superficialis)

Cells of oral mucosa

The epithelial lining the mucous membrane of the oral cavity is composed of two types of cells.[3, 4, 5, 6, 7, 8, 9] They are:

1. Keratinocytes

2. Non keratinocytes

i. Langerhan cells

ii. Merkel cells

iii. Melanocytes

iv. Inflammatory cells

1. Keratinocytes

Keratinocytes are the epithelial cells containing tonofilaments. They are of cuboidal or low columnar type. These cells are attached to the basement membrane by means of hemidesmosomes. The mitotic activity of these cells is greatest.[3, 4, 6, 7]

2. Non keratinocytes

In contrast to keratinocytes, non-keratinocytes do not contain tonofilaments, do not generally participate in any series of maturation like keratinocytes.[3, 4, 6, 7, 9]

Four types of non keratinocytes are found in oral epithelium.[3, 4, 6, 7, 9]

i. Melanocytes

Melanocytes are melanin pigment producing cells located in the basal layer. These cells arise embryologically from the neural crest ectoderm and they enter the epithelium at about 11 weeks of gestation. In basal cell layer, the melanocytes are present in a ratio of 1:4 to basal epithelial cells.

ii. Langerhan cells

Langerhan cells are dendritic cells of unknown origin. These cells are usually found in stratum spinosum and occasionally in the stratum basali. These cells are characterized by the presence of rod shaped granules called as **Birbeck's granules.** Langerhan cells migrate from epithelium to the regional lymph nodes.[7]

iii. Merkel cells

Merkel cells are clear cells that are present exclusively in the basal cell layer and even in the underlying lamina propria. Merkel's cells are sensory cells responding to touch. These cells may also arise from division of epithelial cells.[3, 4, 7, 9]

iv. Inflammatory cells

Inflammatory cells are often seen in the nuclear cell layers, when the section of a clinically normal mucosa is examined microscopically. These cells are transient and do not represent themselves in the epithelium as other non-keratinocytes do. The cells most frequently seen are the lymphocytes.

B. LAMINA PROPRIA

It forms the connective tissue of oral mucosa and is composed of cells, fibres and amorphous ground substance. It also contains blood vessels and nerves.[5] The cells of lamina propria include fibroblasts, macrophages, mast cells, lymphocytes and plasma cells.

Lamina propria is divided into:

a. Papillary portion, which is a superficial layer of papillae and is composed of finger like projections of connective tissue extending into the deep layer of epithelium called as epithelial ridges.[3, 4, 6, 7]

b. Reticular portion, which is a deep layer lying between the papillary layer and the underlying structures. It is composed of reticular fibers.[3, 4, 5]

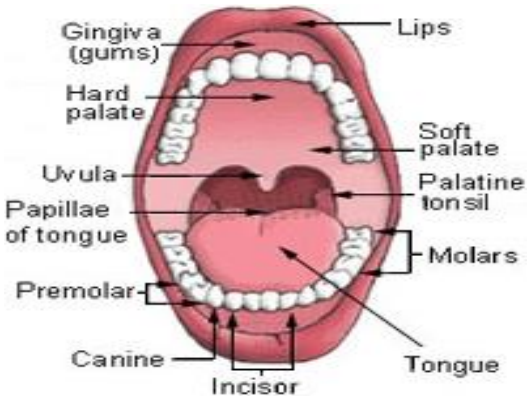
C. SUB MUCOSA

Sub mucosa lies beneath the lamina propria. It consists of connective tissue of varying thickness. It also contains glands, blood vessels, nerves and adipose tissue. It is the site of minor salivary glands in the oral cavity. It contains lymphoid nodules found at the base of tongue - lingual tonsil and palatine tonsils between the

glossopalatine and pharyngopalatine arches. In masticatory mucosa, the submucosa is totally absent and lamina propria itself is attached directly to bone to form a muco periosteum. Sub mucosa also plays a role in nutrition and defense mechanism.[3, 4, 6]

TYPES OF ORAL MUCOSA

Functionally, the oral mucosa is divided into three types.[3, 4, 6, 7, 8, 9]



A. Masticatory Mucosa

i. Gingiva

ii. Hard palate

B. Lining Mucosa

i. Lips and cheek (labial and buccal mucosa)

ii. Vermilion border of lips

iii. Floor of mouth

iv. Ventral surface of tongue

v. Soft palate

vi. Alveolar mucosa

C. Specialized Mucosa

i. Dorsum of the tongue

A. Masticatory Mucosa

Masticatory mucosa covers areas of the oral cavity such as gingiva and hard palate that are exposed to compressive and shear forces and abrasion during mastication. In an edentulous mouth, the masticatory mucosa covers the chewing surfaces of the dental arches.[4, 6, 7]

i. Gingiva

The gingiva is that part of oral mucosa that covers the alveolar process and surrounds the necks of the teeth like a collar.[6]

Parts of gingiva:

It is divided into:

- Marginal gingiva
- Attached gingiva
- Interdental gingiva
- Gingival sulcus
- Muco gingival junction

The gingiva is normally pink in colour but sometimes it may have a grayish tint. The surface may be translucent or transparent, permitting the colour of the underlying tissues to be seen. The presence of melanin pigment in the epithelium may

give it a brown to black coloration. Pigmentation is most abundant at the base of interdental papilla.[7]

ii. Hard palate

The mucous membrane of the hard palate is tightly fixed to the underlying priosteum. The epithelium is uniform in nature with a well keratinized surface.

Based on the nature of submucosa, hard palate is divided into various regions. They are:

i. Gingival region

ii. Palatine region

iii. Antero-lateral region

iv. Postero-lateral region or glandular zone

B. Lining Mucosa

Lining mucosa covers the ventral surface of the tongue, inside of the lips and cheeks, floor of the mouth, alveolar processes and soft palate.[4, 6, 7]

The epithelium of the lining mucosa is thicker than that of masticatory mucosa which is non-keratinized in nature. The surface is thus flexible and able to withstand stretching.[4, 6, 7]

It consists of:

i. Lips and Cheek

The epithelium of the mucosa lining the lips and cheek is stratified squamous non-keratinized type. The lamina propria of the lips and cheek contains long papillae with anastomosing capillary loops giving a rich vascular supply.[3, 4, 6, 7, 8, 9]

The cheek and commissure may contain heterotropic collections of sebaceous glands called Fordyces spots.[4, 6, 7]

ii. Vermilion border of the lip

The term Vermilion is because of its reddish appearance in the Caucasian race.[4] It is the junction between the skin and the mucous membrane of the lip, where the epithelium changes from the keratinized stratified squamous type to moist non keratinized stratified squamous type of epithelium of the oral cavity.[4, 6]

iii. Floor of the mouth

The mucous membrane on the floor of the mouth is thin and loosely attached to the underlying structures to allow for free movements of the tongue and is of non-keratinized stratified squamous type.[4, 6, 7]

iv. Ventral surface of the tongue

The mucous membrane covering the inferior surface of the tongue is smooth and relatively thin. The epithelium is non - keratinized.[6, 7]

v. Soft palate

The mucous membrane of the soft palate is relatively thin, non keratinized stratified squamous epithelium type. The mucous membrane is highly vascularized and reddish in colour differing from pale colour of the hard palate.[4, 6, 7]

vi. Alveolar mucosa

It is relatively thin and is non-keratinized, stratified squamous epithelium type.[6] The connective tissue contains short papillae and some elastic fibres. It contains capillary loops close to the surface running superficially to the periosteum. The sub mucosa is loose and contains minor salivary glands. It contains elastic fibres attaching it to the periosteum of alveolar process.[6, 7]

C. Specialized Mucosa

Specialized mucosa covers the dorsal surface of the tongue which is rough and irregular.[6, 7]

i. Dorsal surface of the tongue

A V-shaped line called as Sulcus terminalis divides the dorsal surface of the tongue into an anterior part or body, and a posterior part or base. The anterior portion forms about two thirds of whole length and posterior portion forms about one-third of the length of organ.[6, 7]

The majority of papillae present in anterior keratinized stratified region are filiform papillae which are pointed, cone shaped giving a velvety appearance and do not contain taste buds.[4, 6]

Interspersed between filiform papillae are mushroom shaped fungiform papillae that are round and reddish and contain a few taste buds found only on their dorsal surface.[6, 7]

In front of Sulcus terminalis at the posterior limit of body of tongue, a row of rounded papillae called circumvallate papillae are present. These papillae do not protrude above the surface of tongue but are bounded by a deep circular furrow. On the lateral surface of circumvallate papillae, the taste buds are present. The ducts of small serous glands called Von Ebner's glands open into the trough.[6, 7]

Functions of Oral Mucosa

1. Mastication
2. Respiration
3. Ingestion, Digestion and Absorption
4. Sensory function
5. Secretion and Excretion
6. Protection

PHYSIOLOGY OF PIGMENTATION

The physiologic pigmentation of oral mucosa is due to melanin pigment containing cells called as melanocytes.

Melanocytes are non-keratinocytes that originate from the neural crest. These cells are present in the basal cell layer. Melanocytes are also found in external hair root sheaths, and in the bulbs of hair follicles.

Based on their secretory action, the melanocytes are divided into two types.

They are:

A. Secretory Melanocytes

These melanocytes produce melanosomes which are pigmented granules. These melanosomes are transferred to surrounding basal cells. The transfer of melanosomes involves the active participation of keratinocytes. The tip of dendrite of melanocyte becomes embedded to the cytoplasm of keratinocyte and the end becomes pinched off and melanasomes are transferred into keratinocytes that act as phagocytes.[8, 9, 10]

B. Non Secretory Melanocytes

These melanocytes are known as melanophores which are dermal histiocytes that carry small quantities of pigment shed by epidermal melanocytes.[11, 12]

Embryology of Melanocytes

Melanocytes arise from the neural crest. The appearance of melanocytes in the epidermis and oral mucosa takes place in a cranio caudal direction, in accordance with the development of the neural crest, from which the melanocytes are derived.[13, 14]

Ultra structural studies on early human embryos have shown the presence of melanocyte in the epidermis by the eighth week of gestation and by tenth week these cells contain melanosomes showing early melanization.[13] Melanocytes appear in the epidermis and oral mucosa by 14th week of intrauterine life.[15] Melanocytes with

recognizable melanosomes may be seen in the fetal epidermis at a gestational age of 8 to 10 weeks.

PIGMENT OF IMAGINATION [16] - Melanin

Melanin is a complex brown - black pigment synthesized from an amino acid Di hydroxy phenyl alanine (L - DOPA).[13, 14, 17, 18, 19]

Melanin pigment may exist in any of these three forms.

They are:

a. Eumelanin

Eumelanins are black or brown nitrogenous pigments, insoluble in all solvents, which arise by oxdiative polymerisation of 5, 6 dihydroxy indoles derived biogenetically from tyrosine via dopa.[13, 17]

b. Pheomelanin

Pheomelanins are alkali soluble pigments ranging from yellow to reddish brown in colour. Most of them contain sulphur and nitrogen. They form by oxidative polymerisation of cysteine-s-yl-dopa via 1, 4-Benzothiazine intermediates.[13, 17]

c. Trichosomes

Trichosomes are sulphur contaning pheolmelanic pigments with a well defined structure, characterized by a 1, 4-Benzothiazine chromophore.

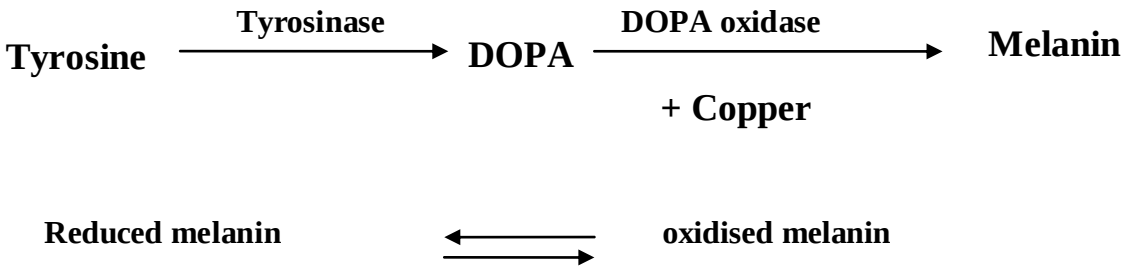
MELANOGENESIS

The formation of melanin pigment in humans is a dual process, which involves.

I. The production of melanosomes within the melanocyte, termed as melanogenesis.[13, 15]

II. The distribution and transfer of these pigment granules to surrounding basal cells.[13, 15]

Melanin is synthesized by hydroxylation of tyrosine to hydroxy phenylalanine (DOPA) and oxidation of DOPA to DOPA quinone. Before tyrosinase acts on tyrosine, two cupric atoms present in the tyrosinase are reduced to cuprous atoms. It is believed that DOPA, apart from its role as a substrate, it activates the reduction of cuprous atoms, there by acting as a co - factor in the reaction.[14, 17, 18, 19, 20]



REGULATION OF MELANOGENESIS

A. Genetic Factors

Interaction of various genes can influence the size, shape of melanosomes, biosynthesis of tyrosine and the organization of melanosomes.[17, 19]

B. Ultra - Violet Radiation

Ultraviolet B radiation is the most effective and efficient spectrum since it increases the number and size of melanosomes.[17]

C. Pharmacologic Agents

i. Melanotropins

The best known melanotropins such as α and β melanocyte stimulating hormones are derived from a protein called pro opio melanocortin (POMC). Clinically, POMC is synthesized exclusively in the anterior and intermediary lobes of pituitary gland. It is cleaved into smaller peptides such as α- Melanocyte stimulating hormone, (α MSH) or adrenocorticotropic hormone (ACTH), β - lipotropin or β Melanocyte stimulating hormone (β MSH) and endorphins by proteases.[17]

ii. Prostaglandin and Leukotrines

Prostaglandin E (PGE) is the most potent stimulant of melanogenesis and melanocyte growth. Prostaglandin D (PGD) also enhances melanogesis but to a lesser extent. Prostaglandin A (PGA) inhibits the process of melanogenesis by suppressing tyrosinase activity in melanoma cells in culture.[17]

Leukotrine C_4 (LTC_4) functions as a mitogen for the growth of human melanocytes in culture.[17]

iii. 1, 25 - Dihydroxy vitamin D_3

In culture, Vitamin D_3 seems to have a inhibitory action on tyrosinase. As human melanocytes posses a receptor for 50% of vitamin D_3 indicating its role in

pigment system, humans deficient to vitamin D_3 do not readily form melanin in response to such as exposure to ultraviolet B (UVB) radiation.[17]

iv. Interleukins, Lymphokines and Interferon

IL - 1 inhibits the melanogenic effects of α - MSH on melanocytes. Interleukin - 6 (IL-6) suppress both tyrosinase activity and the growth of normal human melanocytes in culture.[17]

Tumor necrosis factor α (TNF α) which is similar to IL-1 in many of its functions and to some degree in its structure has similar effects on melanocytes as IL-1.[17]

The peptide α -MSH is a potent stimulator of melanogensis. Interferon λ (1FN -λ) by itself has no effect on melanogenesis, but when combined with α-MSH, IFN λ exhibits synergistic stimulatory effect of melanogenesis. It probably acts by altering expressions of surface receptors for MSH.[17]

D. Growth Factors

- ***Basic Fibroblast Growth Factor (b-FGF)***

Basic Fibroblast Growth Factor which is synthesized by keratinocytes act as an essential growth factor for maintenance of normal melanocytes in cultures.[17, 18]

- ***Nerve Growth Factor***

In culture, receptors for nerve growth factor on melanocytes have been identified. Nerve growth factor plays a role in migration of melanocytes.[17, 18]

E. Therapeutic Agents

- ***Topical Steroids***

Moderately potent steroids decrease the tryosinase activity and suppress melanin formation. Even topical steroids when applied frequently will cause decreased melanization.[17, 18]

- ***Hydroquinone***

Hydroquinone in a test tube directly blocks the activity of enzyme tyrosinase and its ability to form melanin.[17, 18]

- ***Mono Benzene***

Monobenzene is a derivative of hydroquinone. The addition of aliphatic or aromatic side chains to hydroquinone alters its pharmacologic properties and this addition makes the molecule toxic to the melanocytes.[17]

In contrast to hydroquinone, mono benzone also tends to kill melanocytes.

- ***Ascorbic Acid***

Ascorbic acid tends to reduce the process of melanin formation.[14]

PATHOGENESIS OF DISORDERS OF MELANIN PIGMENTATION

Disorders of melanin pigment can be divided into two types on morphological grounds. [13] They are:

a. **Hyper-melanosis** - wherein there is an increased amount of melanin.

b. **Hypo-melanosis** - wherein there is a lack of melanin pigment

c. **Amelanosis** - wherein in there is a total lack of melanin pigment.

Changes in pigmentation can arise in number of ways.[13] These include:

a. By the formation of melanosomes in melanocytes.

b. By melanization of melanosomes.

c. By secretion of melanosomes into keratinocytes.

CLASSIFICATION OF PIGMENTED LESIONS OF THE ORAL CAVITY

I. McCarthy in 1980 classified pigmented lesions of oral cavity as follows.[21]

A. Endogenous

a. Due to Melanin

i. Normal racial pigmentation

ii. Addison's disease

iii. Peutz - Jeghers Syndrome

iv. Albright's Syndrome

v. Neurofibromatosis

vi. Pigmented nevi

vii. Malignant melanoma

b. Due to Bilirubin

i. Jaundice

c. Due to Iron

i. Haemochromatosis

ii. Ecchymosis

B. Exogenous

a. Pigments introduced into body systemically

i. Bismuth

ii. Lead

iii. Silver

iv. Mercury

v. Gold

vi. Arsenic

vii. Anti malarials

viii. Chlorpromazine

b. Pigments introduced into the oral mucosa locally

i. Amalgam tattoo

ii. Industrial accidents involving various materials such as lead/ copper

iii. Graphite (pencil tips)

C. Miscellaneous

i. Black hairy tongue

ii. Stains from tobacco and lozenges

II. SURENDER GOPAL in 1991 proposed the following classification.[22]

A. Endogenous pigmentation

a. Normal variations

b. Pathological variations

i. Addison's Disease

ii. Peutz Jegher's Syndrome

iii. Albright's Syndrome

iv. Hyperfunction of pituitary glands

v. Pregnancy and female sex hormones

vi. Von – Reckling Hausen disease

vii. Haemochromatosis

viii. Fixed drug reactions E.g. quinacrine and related anti-malarials, quinidine, sulfa group of drugs, diuretics, chlorpromazine, nasal drop containing silver salts.

ix. Carotenemia

x. Jaundice

B. Exogenous pigmentation

a. Pigments introduced into the body either systemically, therapeutically or accidentally:

i. Bismuth

ii. Lead

iii. Silver

iv. Mercury

v. Gold

vi. Arsenic

b. Substances introduced into oral tissues locally

i. Amalgam Tattoo

ii. Industrial accidents and various materials

c. Local environmental changes

i. Black Hairy tongue

ii. Stain caused by Lozenges, Tobacco and medicaments

d. Miscellaneous

i. Oral Sub Mucous Fibrosis

ii. Basal Cell Carcinoma

iii. Malignant melanoma

iv. Cutaneous leishmanians

v. Juveline acanthosis nigricans

vi. Pigmented lichen planus

vii. Colonization

III) CLINICAL CLASSIFICATION OF ORAL PIGMENTATION [23]

Based on distribution, they are classified as:

A. Solitary

a. Focal

b. Diffuse

B. Multi Focal

A. Solitary Lesions

a. Focal lesions

i. Blue / Purple lesions:

Eg: Varix, Haemangioma

ii. Brown lesions

Eg: Melanotic macule, Nevus, Melanoma

iii. Grey/Black lesions

E g.: Amalgam tattoo, Graphite tattoo, Nevus, Melanoma.

b. Diffuse lesions

i. Blue / Purple lesions

Eg: Haemangioma

ii. Brown lesions

E.g.: Ecchymosis, melanoma, drug induced pigmentation

iii. Grey / Black lesions

E.g.: amalgam tattoo, melanoma, Hairy tongue

B. Multi Focal Lesions

i. Blue / Purple lesions

E.g.: Kaposis sarcoma, Hereditary Haemorrhagic Telangiectasia

ii. Brown lesions

E.g.: Physiologic pigmentation, Neurofibromatosis, Haemochromatosis, Lichen planus, Addison's disease, drug induced pigmentation, petechiae and ecchymosis, Peutz-Jegher's syndrome.

iii. Grey / Black lesions

E.g.: Heavy metal ingestion

A modified version of the clinical classification is followed below for the description of individual conditions:

I. TATTOO

Tattoo is tissue stained by a pigmented foreign material.[24]

The most common foreign material to cause an oral tattoo is dental amalgam although graphite from pencils and other foreign materials are occasionally implicated.[24]

a. Amalgam tattoo

Amalgam tattoos are common clinical lesions.

Clinical Features

Amalgam tattoos are typically asymptomatic, benign, solitary or multiple lesions. They are bluish gray to bluish black or brown in colour. They present as flat macules and occasionally as a slightly raised lesion. The margins of these lesions may be well defined or irregular or diffuse in nature. The size of these lesions varies approximately from few millimeters to 1 to 2 cm in diameter. These lesions are found adjacent to teeth with large amalgam restorations. The most frequently affected sites are gingiva, buccal mucosa, palate, tongue and alveolar mucosa.[22, 23, 25, 26, 27, 28, 29]

Occasionally these lesions are symptomatic exhibiting mild to moderate inflammatory reaction.[29, 30]

Histological Features

Microscopically, amalgam particles are typically aligned along collagen fibers and around blood vessels. Few lymphocytes, macrophages and multi-nucleated giant cells may be seen.[22, 30, 31]

Diagnosis

Diagnosis can be established by:

History, radiographs and biopsy.

Differential Diagnosis

It Includes:

Graphite tattoo, superficial haemangioma, melanoma, melanotic macule, blue nevus and varix.[32, 33]

Treatment

Treatment is considered when the location of the lesion is esthetically displeasing by reconstructive surgery.[33]

b. Graphite tattoo

Aetiology

It occurs due to traumatic implantation from lead pencil into oral soft tissues.[22, 23]

Clinical Features

The lesions are usually macular, focal and gray or black in colour.[22] They commonly occur on palate.[22]

II. ALBRIGHT'S SYNDROME

Albright's Syndrome is a rare disease of unknown etiology.[31, 34, 35, 36, 37, 38] It is characterized by:

a) Polyosteotic fibrous dysplasia

b) Pigmentation of skin and rarely oral mucous membrane

c) Endocrine dysfunction

Clinical Features

The disease manifests itself early in life.[34, 38]

Cafe au lait spots which occur on the skin surface may also appear on oral mucous membrane. When oral mucosa is affected, lips are the most commonly affected site although any site of oral mucosa may be involved.[22, 23, 29, 31, 36, 39]

Diagnosis

Cafe au lait spots present in this condition should not be considered as pathognomonic. To arrive at a definitive diagnosis, other manifestations of the disease process should be considered.[38]

Differential Diagnosis

It includes:

Neurofibromatosis, Addison's disease and Peutz-Jegher's Syndrome

III. DRUG INDUCED MELANOSIS

Melanosis of oral mucosa caused by certain medications is a known phenomenon. The exact mechanism by which these drugs cause pigmentation is unknown. The lesions are usually homogenous, macular lesions with sharp delineated borders. The lesions may progress for certain period of time and may disappear with discontinuity of drug usage.[39, 41, 42]

The colour produced by these lesions varies depending on drug used. For example, lesions caused by anti-malarials exhibit yellow and mepacrine bluish black. Busulphan, oral contraceptives, phenothiazines and anti convulsants exhibit brownish colour.[22, 23, 27, 29, 41, 42]

These pigmented lesions may be localized usually to hard palate or are multifocal occurring in different sites of oral cavity.[37, 39, 42]

Aetiology

a) Drugs containing heavy metals such has lead, bismuth, gold, mercury, arsenic and Bromides.[22, 27, 36]

b) Anti- malarial drugs such as chloroquinine, hydroxychloroquine, Amadioquine.[22, 27, 41, 43, 44]

c) Quinidine, an alkaloid which is used in the treatment of cardiac arrhythmias.[43]

d) Minocycline, a synthetic tetracycline which is used in treatment of Acne vulgaris. It has an ability to pigment many tissues such as thyroid, skin, bone, teeth, oral mucosa and other sites.[44, 45, 46]

e) Oral contraceptives

f) Zidovudine, an anti viral drug which is used to treat AIDS causes nail and oral mucosal pigmentation.[36]

g) Hormones such as ACTH, progesterone.[47]

h) Anticonvulsants such as phenytoin and their related compounds.[48, 49]

i) Phenothiazines such as chlorpromazine and related compounds.[48, 49]

j) Others - cisplastin, busulphan, nitrogen mustard, cyclophosphamide, heroin, doxurubicin, melphalan and clofazimine also cause oral pigmentation.[48, 49]

Differential Diagnosis

When drug induced pigmentation presents as brownish macules, Addison's disease is considered. Brown macules present in both conditions are indistinguishable. To arrive at a definite diagnosis proper history and serum laboratory studies may be required.[23, 31, 37]

Diagnosis

Diagnosis is made on the basis of drug history.[22, 23, 27, 29, 41, 42]

Treatment

These lesions do not require any treatment. For elimination of pigmented lesions the patient is advised to discontinue the drug responsible.[22, 23, 27, 29, 41, 42]

IV. ECCHYMOSIS AND PETECHIAE

Aetiology

1. Trauma

The injury may be related to cheek bite, coughing, fellatio, trauma from prosthetic appliances, injudicious oral hygiene procedures and iatrogenic dental injuries.[22, 23, 27, 29]

2. Various Blood Dyscrasias

Leukemia, Platelet and Bleeding Defects, Hemophilia

3. Other Conditions - Infectious Mononucleosis and it may be due to thrombocytopenia.[22, 23, 26, 27]

Clinical Features

The colour of these lesions appears red initially and then turns into brown in a few days when extravasated blood cells are lysed and degraded into hemosiderin pigment. These lesions disappear after a short period of time. These lesions usually occur in trauma accessible areas such as buccal mucosa, lateral borders of tongue, lips and junction of the hard and soft palate.[22, 23, 26, 27, 31, 36, 41]

Differential Diagnosis

A. Amalgam Tattoo, oral melanotic macule, junctional nevus, melanoma

B. Petechiae and ecchymosis occurring as solitary lesions at or near junction of hard and soft palates should be differentiated from the following conditions.[23. 36, 38]

i. Trauma from Fellatio

ii. Trauma from severe vomiting and coughing

C. Infectious Mononucleosis

V. HEAVY METAL PIGMENTATION

Heavy metal pigmentation is caused by ingestion or inhalation of materials containing lead, bismuth and mercury. The other metals such as copper, zinc, phosphorous, copper, manganese may rarely cause pigmentation of oral tissues.[29, 31, 36, 41]

Aetiology

There are two important mechanisms by which these heavy metals get deposited and cause pigmentation.

- It may be an occupational hazard.[22, 23, 27, 29, 31, 36, 41]
- By ingestion of medication containing heavy metals as their components for example, historically arsenic and bismuth compounds were used to treat syphilis, lichen planus and other dermatoses.[29, 31, 36, 37, 41]
- Cis - platinum, the salt of the heavy metal has anti- neoplastic activity and is used to treat some malignancies.[29]

Clinical Features

In oral cavity, the pigmentation is usually found along the free gingival margin which outlines the gingival cuff like a cylinder. The characteristic color of this

pigmentation is gray to black. The most likely pigmentation in free gingiva may be due to inflammation as there is an increased capillary permeability which permits peri-vascular infiltration of metal into tissues.[22, 23, 26, 27, 36]

Apart from gingiva, the pigmentation can also occur in other sites such as buccal mucosa, lips, and the ventral surface of tongue or in any localized areas of inflammation such as partially erupted third molars or around a periphery of an ulcer.[22, 36]

Heavy metal pigmentation may occasionally be associated with systemic symptoms such as burning sensation, metallic taste, increased salivation & tremors. Symptoms of toxicity such as behavioral changes, neurologic disorders and intestinal pain may occur which are rare.[22, 50, 51]

VI. PHYSIOLOGIC PIGMENTATION (MELANOPLAKIA)

It is naturally occurring pigmentation. Physiologic pigmentation of oral mucosa is characterized by multifocal or diffuse melanin pigmentation with variable prevalence in different ethnic groups. The pigmentation depends on amount of melanin in the normal oral mucosa which varies in intensity and distribution with racial differences varying for the greatest.[22, 25, 29, 31, 41]

Clinical Features

Clinically it presents as a diffuse or multifocal melanin pigmentation which is symmetric, persistent and does not alter the normal architecture of the tissues affected. The pigmentation may be seen at any age and the intensity of pigmentation diminishes with increasing age.[22, 23, 31, 36, 41]

The clinical appearance of melanin varies from the light brown to blue black in colour, depending on the amount and its depth in tissues. The deeper and the heavier the tissues, the darker it appears.[22, 23]

Intra orally the most commonly affected site is gingiva. But it also occurs in other sites such as buccal mucosa, alveolar mucosa, tongue and to a lesser extent in soft palates.[22, 27, 29, 31, 41] The pigmentation of gingiva produces black discoloration of gingival which is commonly known as Black Gum.[52]

Histological Features

Physiologic pigmentation is characterized by increased melanin pigmentation with normal number of melanocytes. The melanin pigment is found in surrounding basal keratinocytes and subjacent macrophages.[22, 31, 36]

Differential Diagnosis

Smoker's melanosis, Addison's disease, melanoma, post–inflammatory pigmentation.

VII. NAEVI

The term Nevus refers to tumor like malformation of skin and oral mucosa. They are thought to be congenital or developmental in nature. They may arise from surface epithelium or from underlying connective tissue or both.[31, 36, 53]

Naevi are collection of nevus cells that are round or polygonal in shape and are typically seen in a nested pattern.[31, 36, 53]

The origin of nevus cell is thought to be from neural crest cell that migrate from neural crest to the epithelium and sub mucosa or from altered resident melanocytes.[31]

Naevi are classified into:

1. Congenital Melanocytic Nevus [29, 31, 36, 53]

2. Acquired melanocytes nevus (Naevo cellular nevus) [29, 31, 36, 53]

1. Congenital Melanocytic Nevus-

Congenital melanocytic nevus affects approximately 1 to 2.5 percent of neonates. Approximately 15% of them occur on the skin of head and neck. Intraoral occurrence is extremely rare.[29, 31, 36, 53]

2. Acquired melanocytic naevi

Acquired melanocytic naevi are extremely common. These naevi begin to develop on the skin during childhood and they reach peak incidence in late third decade of life. They occur in both men and women with women having more predilections. Naevi are more commonly present in Whites than in Blacks. Most of these lesions are distributed above the waist, head and neck region being the common site of involvement.[23, 24, 29, 31] Interestingly, these naevi begin to decrease in number as one ages, so that elderly person will have fewer naevi than young adults.[53]

Blue naevus (Dermal melanocytoma, Jadassohn -Tieche naevus)

Blue naevus is a benign proliferation of dermal melanocytes usually presents deep to the sub epithelial connective tissue.[31, 36] The blue colour is explained by

Tyndall effect, which relates to interaction of light with particles in a colloidal suspension.

Types

Two types of blue naevus are recognized:

i. The Common Blue Naevus

ii. The Cellular Blue Naevus

INTRA ORAL NAEVI

Intra oral naevi occur in all decades of life with highest incidence being in third and fourth decades of life. In the oral cavity, intra mucosal naevi are the most common variety and blue naevi are as the second most common. It occurs more commonly in hard palate and buccal mucosa followed by lips and gingiva.[40, 53, 54, 57, 58] Occasionally, few naevi occur in soft palate and tongue.[58]

Oral naevi occur more commonly in females than in males with a ratio of 1.7:1.[29, 36, 57, 58, 59] The size of these lesions vary from 0.1 to 3.0 cm in diameter with an average size of 0.47 cms.[31, 58]

Most naevi present as raised, pigmented lesions with smooth borders and these are palpable.[57, 59] The color of the naevi may be gray, pale, brown, dark brown, blue, black or combination of these colors. The color of the junctional, compound and intra mucosal naevi ranged from light to dark brown in color where as blue naevi are blue - gray in colour.[31, 36, 57, 59, 60] Eighty five percent of oral naevi are pigmented and 15% of them are non pigmented lesions.[61]

Histologic Features

Melanocytic naevus is characterized by benign, uncapsulated proliferation of small ovoid cell; naevus cells. A characteristic microscopic feature is that these superficial naevus cells are organized into small, round aggregates.[31, 36, 58, 59]

Histologically, melanocytic naevi are classified according to their stage of development which is based on the relationship of naevus cells to the surface epithelium and connective tissue.[22, 29, 31, 36, 57, 59]

1. Junctional Naevus

2. Compound Naevus

3. Intramucosal Naevus

4. Spindle cell or Epithelioid Naevus

5. Common Blue Nevo-Cellular Blue Naevus

Differential Diagnosis

It includes

Hematoma, varix, haemangioma, amalgam tattoo, foci of haemorrhage, melanoma and oral melanotic macule.[31, 38, 40, 59]

Treatment and Prognosis

No treatment is indicated for oral melanocytic naevi unless it is cosmetically unacceptable. If surgery is indicated, conservative surgical excision is the treatment of choice.[31, 61]

VIII. ORAL MELANOTIC MACULE

Oral melanotic macule or focal melanosis is a focal pigmented lesion which may represent in three distinct entities.

i. Post inflammatory or post traumatic pigmentation.[31, 36, 62]

ii. An intra oral freckle / ephelide.[31, 36, 62]

iii. A unique lesion for which there is no cutaneous counterpart.[31, 36, 62]

The term oral melanotic macule was suggested by Page and his Co-workers which is reserved for those lesions without an identifiable etiologic factor.[63]

Aetiology

These lesions are due to increase in melanin synthesis by melanocytes without an increase in number of melanocytes.[31, 36, 62, 63]

a. Post traumatic or inflammatory reaction is a possible cause.[63]

b. Local factors such as chronic inflammation (periodontal disease), heat (smoking), traumatic irritation (cusp appliance etc.). Chemicals such as tobacco chewing which leads to excessive formation of melanin within the tissues.[63]

Clinical Features

Oral melanotic macule may occur at any age but is usually seen in individuals over 40 years of age. It occurs with equal frequency in males and females. The lesions are oval or round in shape with irregular outlines or may be demarcated and measures

less than 6 to 7 cm in diameter. The colour of the lesions may be blue, black or brown. The lesions are usually focal in nature but multiple lesions also occur. They commonly occur in the lower lip and gingiva. They also occur in hard palate, buccal mucosa, and occasionally in muco buccal fold and tongue.[36, 49, 61, 62, 63]

Oral melanotic macules may occur as solitary lesions or they may be associated with other conditions.[31, 36, 63]

Histologic Features

Microscopically, these lesions are characterized by melanin accumulation in basal keratinocytes with normal number of melanocytes. Melanin incontinence into sub mucosa is also commonly seen. Rarely melanin containing dendritic cells are seen extending into a thickened spinous layer. Chronic inflammatory cells are also seen.[36, 63, 64]

Differential Diagnosis

It includes:

Melanoplakia, amalgam tattoo, ecchymosis, superficial spreading melanoma and naevi.[61, 63]

Treatment and Prognosis

No treatment is indicated. Biopsy may be required to establish the definitive diagnosis of the lesion.[31, 36, 61, 63] It has little or no propensity for recurrence.[31, 63]

IX. ORAL MELANOACANTHOMA

Oral melanoacanthoma is a benign and rare acquired pigmentation of oral mucosa characterized by dendritic melanocytes dispersed throughout the epithelium and keratinocytes. It is considered to be a benign mixed tumour of keratinocytes admixed with pigmentation laden dendritic melanoctyes. It appears to be reactive in origin, often arising as a result of local trauma.[64, 65]

Clinical Features

It occurs almost exclusively in Blacks, but also reported to occur in Whites. It shows predilection to occur in females and occurs commonly in the third and fourth decades of life.[65, 66]

Most oral melanoacanthomas occur on stress bearing areas such as hard palate and alveolar ridge or in common sites of trauma such as buccal mucosa. It is also occurs on gingiva, lip, soft palate and oropharynx.[65, 66, 67]

Clinically it present as smooth, flat or slightly raised lesion with varying degree of pigmentation from dark brown to black in colour. The lesions often demonstrate a rapid increase in size and occasionally reach a diameter of several centimeters within a period of few weeks.[65, 66]

Differential Diagnosis

It includes:

Melanotic macule and Melanoma.[65, 66]

Treatment

Because of tendency of lesion to rapidly increase in size, incisional biopsy is usually indicated to rule out the possibility of melanoma.[67, 68]

It is reparative lesion with no malignant potential. In instances, the lesion undergoes spontaneous resolution after biopsy.[23, 68]

X. PIGMENTATION ASSOCIATED WITH LICHEN PLANUS

Lichen planus is a relatively common chronic mucocutaneous disease of unknown aetiology affecting skin and oral mucosa.[61] Lichen planus was first described as a separate disease entity by a British physician Erasmus Wilson in 1869.[69, 70, 71]

The oral lesions of lichen planus were further noted and described by **Unna P.G and Crochber H.R.**[71]

Thibierge first described the oral lesions systematically.[71]

In approximately 40 to 50% of cases of lichen planus, oral lesions appear simultaneously with dermal lesions. In 25% of cases, the oral lesion may appear without the occurrence of skin lesions. When it involves oral cavity, it commonly occurs on buccal mucosa followed by tongue lips, palate and gingiva.[71, 72]

Types

Andreasen divided oral lesions into six forms.[71, 72]

a) Reticular form

b) Papular form

c) Plaque form

d) Atrophic form

e) Erosive form

f) Bullous form

Aetiopathogenesis

The pigmentation may be due to increased melanogenesis stimulated by infiltration of T lymphocytes into basal cell layers.[72]

Clinical Features

Pigmentation following oral lesions is rare, present in only 11% of cases. Oral lesions are occasionally associated with pigmentation, while the appearance of pigmentation in resolving skin lesions is common. Among oral lesions, erosive and reticular types are associated with pigmentation.[2] The pigmentation may begin with lesions or it may appear subsequently.[73]

Histologic Features

The thickness of spinous layer varies. The rete ridges show a pointed or **saw-toothed** appearance. Basal cell layer of epithelium shows hydropic degeneration which is accompanied by an intense band of T lymphocytic infiltrate immediately subjacent to epithelium. Degeneration of keratinocytes, termed as **colloid, cytoid, hyaline** or **civatte bodies** are seen at the junction of connective tissue and the epithelium. Basal cell layer in pigmented conditions shows melanosis and melanin incontinence.[71, 72, 73]

XI. PEUTZ – JEGHER'S SYNDROME

Peutz – Jegher's Syndrome was described by Jeghers and is associated with generalized intestinal polyposis and pigmented macules on the face, oral cavity and occasionally on hands and feet.[74, 75]

Clinical Features

Peutz – Jegher's Syndrome is characterized by multiple, focal, melanotic brown macules concentrated around the perioral region. Lesions on the perioral areas are essentially pathognomonic. Intra orally, the most common site of occurrence is buccal mucosa followed by gingiva, hard palate, lips and tongue. The macule appears as a freckle or an ephelide measuring less than 0.5cm in diameter.[75, 76]

The intestinal polyps are usually present throughout the intestine but clinically manifest in small intestine as gastrointestinal bleeding, abdominal pain, recurrent bouts of intestinal obstruction, small bowel intussusception, anaemia and reccurent diarrhoea. These intestinal polyps have a slight potential to undergo malignant transformation.[75, 76, 78]

Differential Diagnosis

Albright's syndrome, Addison's disease and Neurofibromatosis.[75, 76, 78]

XII. SMOKER'S MELANOSIS

Smoker's melanosis is a benign focal pigmentation of oral mucosa caused by exposure to tobacco smoke.[79, 80]

Aetiopathogenesis

In smokers, increased melanosis of oral mucosa is due to polycyclic amines such as benzopyrine, nicotine present in tobacco as well as free radicals produced by use of tobacco stimulates melanocytes to produce melanin. With melanin present in tissues, these agents may be bound to melanin, and the deleterious effect on these cells is prevented. The harmless melanin-toxic complex is transported within the advancing cells up through epithelial layers and finally expelled with aging cells at the surface layer.[79, 80, 81]

Clinical Features

It is commonly seen in male smokers and in female smokers with elevated estrogen and progesterone concentrations resulting from high dose birth control pills.[81] It is commonly seen in adults and occurs after third decade of life.[79, 80, 81]

Clinically, the lesions are brown, flat and irregular in shape and some of them present as geographic or map like configuration. It predominantly occurs in anterior labial gingiva but also occur in other sites such as lips, palate, buccal mucosa, lateral borders of tongue and floor of the mouth.[77, 81, 82]

Histologic Features

Melanocytes show increased melanin production. There is an accumulation of melanin in basal keratinocytes and melanocytes, with normal number of melanocytes. Melanophagocytosis is also seen.[81, 82, 83]

Differential Diagnosis

Physiologic pigmentation (Melanoplakia).[81, 82, 83]

Treatment

These lesions are asymptomatic. Patient is advised to discontinue the smoking habit and with this improvement will be present over course of months or years.[80, 81, 83, 84]

XIII. HIV Oral Melanosis

Aetiology

In most of the cases reported, the cause for hyperpigmentation is unknown.[85]

It may be related to:

a. Primary adrenocortical insufficiency.[85, 86]

b. Due to drugs such as clofazimine, zidovudine and ketoconazole.[50, 85, 86]

Clinical Features

In this condition, buccal mucosa is the most frequently affected site. The other sites such as gingiva, palate, tongue, upper lip may also be involved. Lesions appear as diffuse, multifocal macules which are brown in colour.[86, 87]

Histologic Features

It is characterized by increased melanin accumulation in the epithelial basal layer and in the epithelial phagocytes.[86, 87]

Differential Diagnosis

Addison's disease, Albright's Syndrome and Neurofibromatosis.[85, 86, 87]

XIV. HAIRY TONGUE

It is a relatively common condition of unknown etiology.[88, 89]

Predisposing Factors

Although hairy tongue is generally idiopathic, there are various initiating or contributory factors. They are:

- use of broad-spectrum antibiotics such as penicillin and systemic corticosteroids.[88, 89]
- use of oxygenating mouth rinses containing hydrogen peroxide, sodium perborate and carbonate peroxide have also been cited.[88, 89]
- Intense smoking.[89]
- In individuals who have undergone radiotherapy to head and neck region for malignant disease.[89, 90]
- By various food stuffs particularly coffee and tea.[89, 90]
- Candida albicans infection.[89, 90]
- Poor oral hygiene.[89,90]

Clinical features

Clinically it manifests by hyperplasia of filiform papillae, with concomitant retardation of normal rate of desquamation.[89, 90]

The hyperplasia of papillae results in thick matted surface that serves to trap bacteria, fungi, cellular debris and foreign material. The pigmentation due to colonization of chromogenic bacteria can impart a variety of colour ranging from green to brown to black.[89, 90] Symptoms are generally minimal. Occasionally there may be gagging or tingling sensation.

Histological Features

Microscopically, it is characterized by elongated and hyperplastic papillae. Keratinization may extend into the mid portions of stratum spinosum. External colonization of the papillae by basophilic microbial colonies is a prominent feature. The underlying lamina propria is generally inflamed.[90, 91]

Differential Diagnosis

Candidiasis and Hairy leukoplakia.[90, 91]

Treatment

Identification and elimination of possible etiologic factors will result in improvement of the condition within a few weeks. Brushing the tongue and maintenance of meticulous oral hygiene should be done.[90, 91, 92] Application of 1% solution of podophyllium resin has also been described as a useful treatment.[92]

This process is entirely benign, self limiting and returns to normal after institution of physical debridement and proper oral hygiene.[91, 92]

XV. HAEMANGIOMA

Haemangioma refers to benign proliferation of blood vessels. It is a tumor of childhood and infancy, but it may also develop in adults. It is usually congenital or it may occur due to vascular malformations. Congenital haemangiomas seldom invade the surrounding tissues. About 85% of them regress spontaneously after puberty.[93, 94]

Classification

I. Classification by Shafer [95]

Based on study of about 1308 tumors, Watson S and Mc Carthy classified as:

a. Capillary haemangioma
b. Cavernous haemangioma
c. Angioblastic haemangioma
d. Racemose haemangioma
e. Systemic haemangioma
f. Metastasizing haemangioma
g. Nevus vinosus or port wine stains
h. Hereditary haemorrhagic telangiectasia

Clinical Features

Commonly, haemangiomas are present by birth or may arise at an early age. They are found in 1.1 to 2.6% of newborns. Haemangimas more frequently occurs in head and neck region than any other part of the body. In head and neck region, it accounts for about 56%.[94, 95]

Oral Features

Oral haemangiomas may present as superficial or as extensive lesions involving large areas of underlying connective tissue and musculature resulting in gross deformity. Haemangiomas often develop as multicentric lesions, in which there may be grouping of several haemangiomas. As these lesions develop, they blend into large lesions with a pebbled surface.[94, 95]

Generally the oral lesions are present at birth or may appear in childhood. Usually oral lesions are not associated with pain. Clinically, the oral lesions vary considerably in size and shape. They occur as solitary or multiple lesions.[94, 95]

In the oral cavity, the most common sites of occurrence are lips, buccal mucosa, palate and tongue. These lesions show a tendency to fluctuate which indicates the variation in amount of fluid engorgement of vascular spaces. Other features such as pulsation or presence of fluid thrill may be noticed. These lesions usually blanch and show emptying on pressure.[94, 95]

In the oral cavity when jawbones are affected, mandible is more commonly involved than maxilla. In the affected region, there may be loosening of teeth, bleeding of the gingivae and the anesthesia of mucous membrane. Radiographically, these lesions present a honey combed pattern, with radiating spicules at the periphery giving a sun-burst appearance.[94, 95]

Differential Diagnosis

Mucocele, ranula, superficial cysts, arterio-venous fistula or aneurysm and varicosity.[94, 95]

Treatment

Many congenital haemangiomas have been found to undergo spontaneous regression at early stages. Surgical excision, radiotherapy, sclerosing agents, cryosurgery and embolization have been tried.[94, 95]

XVI. HAEMATOMA

Haematoma is a pool of effused blood confined within the tissues.[96, 97]

Aetiology

- Traumatic incidents, e.g. accident, surgery, administration of local anesthetic
- Thrombocytopenia
- Disseminated intravascular coagulation
- In viral infections such as Infectious Mononucleosis and Measles

Clinical Features

It appears as a non- blanching, flat or elevated lesion varying from red or purple blue or black in colour. Haematomas are commonly seen in the more trauma prone areas such as labial or buccal mucosa.[96, 97]

Diagnosis

Haematoma is often confirmed by:

- Its recent onset and any history of trauma.
- The lesion is tender, fluctuant and cannot be evacuated by digital pressure.[63]

- On palpation, it generally induces a stinging sensation as the pressure on the contained pool of blood causes further suppression of tissues.
- No fluid thrill or crepitus seen.
- On aspiration, blue venous fluid is obtained.[97]

Differential Diagnosis

It should be differentiated from other bluish lesions that occur in oral cavity such as:

Haemangioma, mucocele, ranula and superficial cysts and varicosity.[96, 97]

XVII. HEREDITARY HAEMORRHAGIC TELANGIECTASIA

Hereditary Haemorrhagic Telangiectesia is a rare genetic condition characterized by multiple macular or papular, vascular lesions of skin, mucosal surfaces and occasionally viscera.[98, 99] The term **Hereditary Haemorrhagic Telangiectesia** was first used by Hanes in 1909. It is also known as **Osler's disease or Rendu – Osler's -Weber disease.[99]**

Clinical Features

It affects both males and females with equal frequency and is transmitted by both sexes in an autosomal dominant pattern.[99] Occasionally telangiectatic lesions are present at or shortly after birth, but in majority of cases they become conspicuous after puberty.

The lesions are round or oval in shape and measures 2 to 6 mm in diameter with sharply delineated margins at periphery.[99] A common feature of this disease is

recurrent and often profuse epistaxis in 90% of cases when nasal mucosa is involved. It progressively worsens over the time.

In this condition, as multiple organ systems are involved, there will be haemoptysis, gastrointestinal bleeding, haematuria and cerebral haemorrhage which are recurrent in nature.[99]

Oral features

In 60% of cases, the oral mucosa is affected with this disease. Tongue is common site of occurrence in oral mucosa. The other sites such as lips, gingiva, buccal mucosa palate and floor of mouth are also affected. Clinically, these lesions appear as bright red to purple in color and will blanch on pressure, a characteristic feature that distinguishes Hereditary haemorrhagic telangiectatic lesions from petechiae which do not blanch on pressure. The size of the lesions range from 1 to 3 mm in diameter, although larger lesions may be seen.[98, 99]

Differential Diagnosis

It includes:

Petechiae, angiokeratoma and others like CREST syndrome, scleroderma, sarcoidosis, Ataxia-Telangiectasia and lupus erythematosus.[97, 98, 99]

Treatment

The treatment of the disease depends upon the severity of the condition.[98, 99] The angiomatous or telangectatic areas are treated with electrocoagulation, cryotherapy, cauterization, laser therapy.[98, 99]

XVIII. LINGUAL VARICES

Varices are pathologic dilation of veins or venules.

Aetiology

It may result from partial blockade of the vein proximal to the distension either by structure causing external pressure or from a plaque that has formed on the lumen side of wall as a result of injury.[100]

Clinical Features

In oral cavity, the common site of occurrence is on the ventral surface of tongue. It also occurs in other sites such as lips and buccal mucosa. Clinically, they appear as torturous bluish red or purple elevations that course along the ventral surface of tongue which extends anteriorly. Varices are painless and they blanch on pressure. Rarely these lesions do not blanch due to formation of intravascular thrombi. When many of sub lingual veins are involved, this condition is called as **Caviar Tongue** or **phblectesia linguae.**[100]

Differential Diagnosis

Haemangioma, aneurysm, mucocele, ranula and non-keratotic cyst.[89, 90, 100]

Treatment

No treatment is required unless it is frequently traumatized or it is cosmetically objectionable. Intralesional injection of sclerosing agents such as 1% sodium tetra decyl sulphate, surgical excision, arteriole embolization, electrosurgery, cryosurgery, laser therapy have been tried.[100]

XIX. NEUROFIBROMATOSIS (VON RECKLINGSHAUSEN DISEASE, FIBROMA MOLLUSCUM)

Neurofibromatosis is an autosomally dominant inherited disease which is characterized by pigmentation, by formation of pedunculated neurofibromas of skin, oral mucous membrane and nerves.[101]

Types

a. Type 1 (Peripheral type)

It is associated with NF1 gene.[101]

It is classical autosomal dominant neurocutaneous disorder associated with multiple cutaneous neurofibromas, soft papillomas, cafe-au-lait spots, axillary frecklings, iris fibroma, plexiform neurofibroma, spinal neurofibromas, aqueductal stenosis, scoliosis, endocrine tumors. Oral lesions are typically associated with this type.[101]

b. Type *2* (Central form)

It is associated with NF2 gene. It is also an autosomal dominant disorder associated with few cutaneous lesions, bilateral acoustic neuromas, cerebral and optic nerve gangliomas, meningiomas and spinal neurofibromas.[101]

Aetiology

The origin of cell of neurofibromas is not clearly known. Many of them believe it is the Schwann cell and others, it is perineural fibroblasts.[101]

Clinical Features

Oral features are typically associated with Type 1 which is characterized by multiple neurofibromas, cafe-au-lait spots, bone abnormalities, central nervous changes, and other stigmata.[101]

The term **Cafe au lait** means coffee colored macules, which may appear in childhood and overlie the neurofibromatous swellings. Discrete, non-ulcerated nodules with overlying normal mucosa are seen which occur on tongue, buccal mucosa, vestibule, palate, alveolar ridge, pharyngomaxillary space and tonsillar area. Macroglossia may be present when there is a diffuse involvement of tongue surface by neurofibromas.[101] Occasionally neurofibromas occur within jawbone. When involved, mandible is more commonly affected in which lesions arise from mandibular nerve resulting in pain or paresthesia.[101]

Differential Diagnosis

It includes:

Addison's disease, Albright's Syndrome and Peutz - Jegher's Syndrome.[98, 99, 101]

XX. ANGIOSARCOMA

Angiosarcoma is a rare malignant tumor of endothelial cells of blood vessels or of lymphatics. More than 50% of them occur in head and neck region. It accounts for 1-2% of soft tissue malignant tumors.[102, 103]

Oral Features

Occurrence of primary or metastatic angiosarcoma in oral cavity is an extremely rare condition. They present as superficial red, blue or purple lesion. They rapidly proliferate and present as elevated nodular masses which tend to ulcerate. The mandible is most common site of occurrence.[102, 103]

Treatment and Prognosis

These tumours are treated by radical excision as they exhibit aggressive clinical course and a poor prognosis.[102, 103]

XXI. KAPOSI'S SARCOMA

Kaposi's sarcoma is a multifocal, multi organ neoplasm consisting of aberrant vascular structures lined by abnormal endothelial cells, extravasated erythrocytes and a mononuclear cell infiltrate consisting predominantly of plasma cells.

It affects skin, oral mucosa, lymph nodes, gastro-intestinal, visceral organs such as liver, lung, heart, pancreas, spleen, adrenal gland, testis and bone. Kaposi's sarcoma is the most common tumor associated with AIDS. Half of the patients with Kaposi's sarcoma develop oral lesions.[104, 105]

Aetiology

HHV-8 or KSHV plays a significant role in induction and maintenance of Kaposi's sarcoma through perturbation of focally released cytokines and growth factors. Various other viruses such as HTLV – III, lymphoadenopathy virus, HIV (Human immunodeficiency virus), Cytomegalovirus (CMV), have been implicated in pathogenesis of Kaposi's sarcoma. HLA antigen may also play a role in the

development of Kaposi's sarcoma.[104, 105]

Types

Four clinical variants have been described:

1. Classic Type

2. African Type (Endemic Kaposi's sarcoma)

a. Cutaneous variant

b. Lympho adenopathic variant

3. Iatrogenic / Immunosuppressive Type

4. AIDS related Kaposi's sarcoma

Clinical Features

In the oral cavity, hard palate followed by gingiva are common sites involved.[180, 183, 185] Very rarely, it also occurs in other sites such as tongue, buccal mucosa, oropharynx, lips, masseter muscle, and the parotid gland.[104, 105]

In the early stages, the oral lesions appear as bluish, blackish or reddish macules and in the later stages they become darker, elevated, often lobulated and ulcerated. Rarely they may not be discoloured.[105]

In case of gingival lesions, there will be an aggravation of the lesions due to presence of plaque and calculus on the teeth surrounded by lesions, it is important for the clinician to recognize the co existing periodontitis. The nodular and papular forms are often troublesome may cause obstruction often interfere with speech, mastication

and result in minor or major episodes of hemorrhage. Although oral lesions appear as focal lesion, typically these lesions are multifocal with numerous isolated and coalescing plaques.[104, 105]

Differential Diagnosis

Haemangioma, melanoma, erythroplakia, pyogenic granuloma and bacillary angiomatosis.[104, 105]

Treatment

Majority do not require any treatment.

Various treatment modalities have been implicated in the treatment of Kaposi's sarcoma. These include- Chemotherapy, radiotherapy, immunotherapy, lasers and sclerosing agents.[104, 105]

XXII. MALIGNANT MELANOMA

Malignant Melanoma is a neoplasm of epidermal melanocytes, the dendritic cells located in basal layers of skin and oral mucosa.[106, 107]

It is postulated that this neoplasm occurs in two phases which are:

a. Radical Growth Phase which is the initial phase in which the neoplastic process is confined to the epidermis[106]

b. Vertical Growth Phase in which the neoplastic cells populate the underling dermis.[106]

Clinical Features

Cutaneous melanomas are classified into four types [106, 107]

I. Superficial Spreading Melanoma

It accounts for approximately 65% and it presents as a tan, brown black lesion on sun exposed skin especially on the back.

II. Nodular Melanoma

It accounts for approximately 13% to 15%. It occurs on back, head and neck regions. It presents as sharply delineated nodular lesion.[106]

III. Lentigo Maligna / Melanotlc Freckle of Hutchinson

It accounts for approximately 10%. The lesions occur characteristically as macular lesions on malar surface of skin in middle aged and elderly individuals.[106, 107]

IV. Acral Lentiguous Maligna

It is most common form of Melanoma in blacks. It presents as darkly pigmented macule with irregular margins and later exhibits papular forms.[106, 107]

Oral Malignant Melanoma

Primary oral malignant melanoma is an extremely rare condition which carriers a poor prognosis. For a lesion to be considered a primary malignant melanoma of oral cavity it should have following criteria.[107]

a. Demonstration of malignant melanoma both clinically and histologically.

b. The presence of intra epidermal (Junctional) activity.

c. Absence of any other primary site.

Clinical Features

It occurs most commonly in individuals between the age of 40 and 70 years with a mean of 55 years. It also occurs as early as 7 years and as late as 95 years. The more commonly affected sites are hard palate and gingiva, especially maxillary gingiva.[106, 107,108]

Clinical presentation of the lesion varies from a macular spot to a raised nodular mass. In the early stages, oral melanomas present as macular brown or black lesions with irregular outlines. In later stages, they become more diffuse, nodular and tumefactive with foci of hyper- or hypo-pigmentation. These lesions exhibit separate discolored areas adjacent to primary lesions. These lesions are called as satellite lesions and are indicative of malignancy.[27] Foci of ulceration may also be present. Occasionally, the lesion may be hemorrhagic which tends to progressively increase in size.[107] This malignancy has a tendency for both regional and distant metastasis through lymphatic and haematogenous routes to sites such as lung, liver, brain and the bones.[106, 107, 108]

Differential Diagnosis

Oral melanotic macule, amalgam tattoo, naevi, ecchymosis, melanoplakia and melanoacanthoma.[98, 99, 100, 106]

Treatment

Various modalities like Surgery, chemotherapy, radiotherapy, and immunotherapy have been tried.[108]

XXIII. ADDISION'S DISEASE

Addison's disease, Adreno-cortical insufficiency is divided into two categories.[109]

A. Conditions associated with primary inability of the adrenal gland to release sufficient quantities of hormone. These include:

a. Anatomic destruction of the gland

i. Idiopathic atrophy (autoimmune)

ii. Surgical removal

iii. Infections-tuberculosis, fungal infection, AIDS

iv. Haemorrhage

v. Metastatic tumor

b. Metabolic failure in hormone production

i. Congenital adrenal hyperplasia

ii. Enzyme inhibition by drugs such as ketoconazole, aminogluthimide

iii. Cytotoxic agents

c. ACTH-blocking antibodies

B. Condition associated with a secondary failure due to primary failure in the elaboration of ACTH hormone. These include:

a) Hypo-pituitarism due to hypothalamic pituitary disease.

b) Agents suppressing hypothalamic pituitary axis such as exogenous steroids and endogenous steroids from tumor.

Patho-physiology of Pigmentation

The hyperpigmentation in this disease is due to over secretion of ACTH with melanocyte stimulating properties.

Clinical Features

General Features

Constitutional symptoms such as fatigue, malaise weakness, anorexia, weight loss and arterial hypotension are seen. Gastro intestinal symptoms are often prominent which includes mild anorexia, nausea, vomiting, diarrhoea and ill defined abdominal pain. Hyperpigmentation occurs on both exposed and unexposed areas of skin such as elbows, knees and palmar creases. Bluish black lesions are seen over the mucous membranes.[108, 109]

Oral Features

The hyperpigmentation of oral mucosa is usually an early prominent sign of Addison's disease. The pigmentation varies from bluish –black to dark brown in colour which occurs in various configurations such as streaks, blotches or spots and it commonly occurs on buccal mucosa, gingival mucosa and tongue.[108, 109]

Diagnosis

Diagnosis cannot be solely made based on the presence of hyperpigmentation. The other following features are taken into consideration to arrive at definite diagnosis.[34, 109]

- Low blood pressure.

- Increased blood potassium with decreased sodium levels.
- High levels of ACTH.
- Decreased cortisol levels.
- Impaired ACTH stimulation (synacthen test).

Differential Diagnosis

It includes:

Haemochromatosis and Albright's syndrome.

XXIV. CUSHING'S SYNDROME

Cushing's syndrome is characterized by truncal obesity, hypertension, weakness, amenorrhoea, hirsutism, purplish abnormal striae, edema, glycosuria, osteoporosis and a basophilic tumour of the pituitary gland.[110]

Types

It is of two types.[110]

A. **ACTH dependant**

It may be due to:

a) Iatrogenic therapy (ACTH therapy)

b) Pituitary dependant bilateral adrenal hyperplasia (Cushing disease)

c) Ectopic ACTH Syndrome

B. Non ACTH dependant

a. Iatrogenic therapy (E.g. Prednisolone)

b. Adrenal carcinoma

Patho-physiology of pigmentation

The pigmentation in Cushing's syndrome may be due to deposition of melanin granules as a consequence of hormone dependant melanogenesis.[110]

Clinical Features

General Features

The affected persons exhibit round appearance of the face- commonly known as **Moon's face.** There is a characteristic obesity with deposition of fat over the neck, shoulders, abdomen and hips- commonly known as Buffalo Hump. Hirsutism and acne may develop. Skin shows painless striae which present as reddish streaks over the thighs, gluteal region, abdomen and axillae. Bruising, purpura and pigmentation may be seen. Some patients show secondary polycythemia.[110]

Psychic symptoms like depression may be present. Involved bones show osteoporosis which is prominent in axial skeleton and they may exhibit tendency to fracture.[110]

Oral Features

Pigmentation commonly occurs on buccal mucosa, palate and gingiva. The pigmented areas present as blotchy brown or blackish areas.[110]

XXV. ARGYRIA

Argyria results from chronic exposure to silver compounds.[111]

Aetiology

It may occur as an occupational hazard or as a result of therapeutic silver compounds such as silver arsphenamine or silver nitrate.[111]

Clinical Features

General Features

Diffuse grayish blue pigmentation of skin is present on exposed areas associated with neurological and hearing damage.[111]

Oral Features

When oral cavity is affected, it presents as diffuse bluish green discoloration of oral mucosa.[111]

Differential Diagnosis

Addison's disease, haemochromatosis and cyanosis.[86, 89, 90, 111]

XXVI. HAEMOCHROMATOSIS (BRONZE DIABETES)

Haemochromatosis is a storage disease in which excess amounts of iron are deposited in body tissues.[90, 112]

Aetiology

It may due to:

a. Patient's inability to control iron absorption – idiopathic haemochromatosis.

b. As a manifestation of disorder of erythropoiesis – erythropoietic haemochromatosis.

c. In alcoholics with liver disease.

d. Due to excessive intake of iron.

Clinical Features

General Features

It includes hepatomegaly, bronze pigmentation of skin and rarely of oral mucous membrane, diabetes, arthropathy, cardiac disease and hypogonadism.[90, 112]

Oral Features

Oral manifestations include brownish to gray pigmentation of oral mucous membrane especially on palate and gingival and is seen in 15-20% of the patients. Pigmentation of oral mucous membrane is as a result of iron deposition in sub mucosa.[90, 112]

XXVII. JAUNDICE

Jaundice or icterus is a metabolic condition which is characterized by yellowish discoloration of skin, oral mucous membrane and the sclera.[100, 113]

Types

i. Haemolytic jaundice

ii. Hepato-cellular jaundice

iii. Cholestatic jaundice

Aetiology

The discolouration may be due to an increase in the concentration of bilirubin and deposition of bile pigment in the tissues. Jaundice appears when serum concentration of bilirubin exceeds 2-3mg /dl.[100, 113]

Clinical Features

Clinical manifestations of jaundice includes yellowish discolouration of skin, sclera and oral mucous membrane. Discolouration of oral mucosa is most apparent at the junction of hard and soft palates. This may be an accentuation of the yellow colour by the fat in this area. Depending on the cause of jaundice, it exhibits various clinical symptoms such as pruritus, pain and enlarged liver. The faeces will be very light in colour if there is any biliary obstruction and urine will be darker.[113]

Differential Diagnosis

Carotenemia.[113]

MATERIAL AND METHODS

The study was carried out between January 2011 to September 2012. All patients attending Department of Dermatology in R.L. Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar were evaluated and those who had oral pigmentation were enrolled into the study. A written consent was also taken.

A detailed medical and dental history including demographic data, chief complaints related to skin, presence of oral pigmentary lesions and associated medical disorders was elicited and recorded. The oral cavity and lips were examined and findings noted. The clinical evaluation of a lesion included observation of the lesion by first drying the area with gauze, applying digital palpation to the area to assess changes in texture and to determine whether the lesion is fixed to the tissue or movable, and then applying pressure to blanch the area and observe for changes in vascularity. A detailed physical examination was made to see any associated lesions elsewhere in the body. Biopsy and histopathological examination were done as and when required to establish the diagnosis.

Inclusion criteria:

All patients of both sexes aged 18 years and above with pigmented lesions in the oral cavity were included into the study.

Exclusion criteria:

1. Patients aged below 18 years of age.
2. Patients with post traumatic oral pigmentation.

OBSERVATIONS

The following observations were made in the study:

1. Total number of cases studied – 200
2. Total number of male patients- 129
3. Total number of female patients- 71
4. Most common age group affected- 35 to 50years (130 cases)
5. Most common site of involvement- Anterior labial gingiva (70 cases)
6. Most common oral pigmentary lesion noted- Melanoplakia (60 cases)
7. Total types of different oral pigmentary lesions observed in this study -13
8. Number of patients with exclusive oral lesions – 89
9. Number of patients with other unassociated skin diseases - 111
10. Number of patients who complained of discoloration of oral cavity – 55
11. Number of patients who complained of burning sensation – 40
12. Number of patients with swelling- 5

RESULTS

The present study includes totally 200 clinically diagnosed cases of oral pigmentary lesions. These patients belonged to both sexes and the age of these patients ranged from 22 years and above.

INCIDENCE

The study was carried out between January 2011 to September 2012. Total out patients aged 18 years and above attending Department of Dermatology were 18000. Among these 200 patients (1.11%) had oral pigmentation.

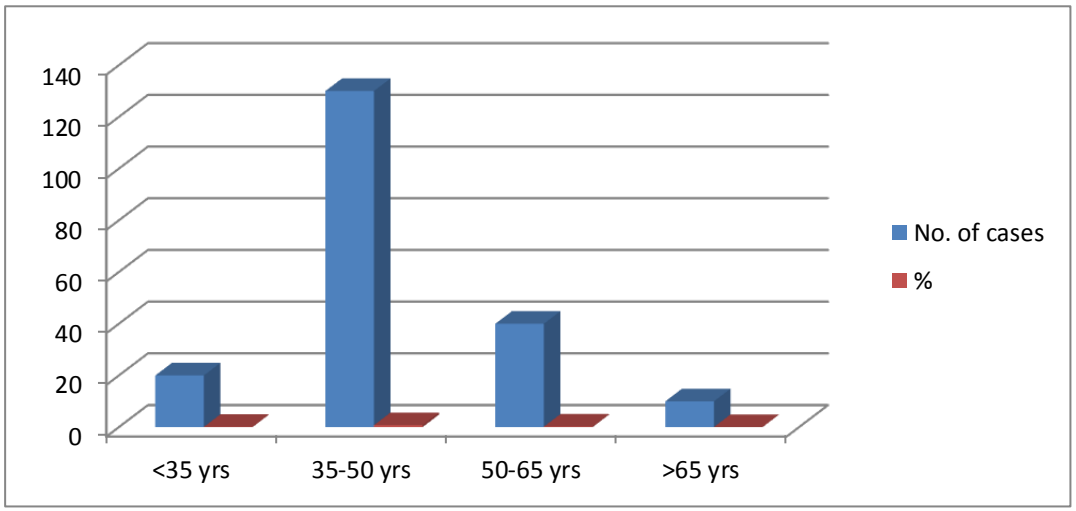
AGE INCIDENCE

In the present study the age group ranged from 18 years and above. Majority of the patients, 130 were in the age group of 35-50 years, constituting of 65% cases. The next common group affected was 50-65 years, 40 cases (20%). The youngest patient was 22 years and the oldest was 70 years of age. The mean age of the patients in this study was 45.8 years.

Table: 1- Age incidence

Age group	No. of cases	%
<35yrs	20	10%
35-50 yrs	130	65%
50-65yrs	40	20%
>65 yrs	10	5%

Diagram 1: Age wise distribution of oral pigmentary lesions.



SEX INCIDENCE

Of the total incidence of 1.11%, males constituted 0.71% and females 0.39%. Out of 200 patients with oral pigmentation, 129 males (64.5%) were affected and 71 female cases (35.5%) were affected, giving a male: female ratio of 1:1.8.

Table 2: Sex incidence

Sex	No. of cases	%
Total males	129	64.5%
Total Females	71	35.5%
TOTAL	**200**	**100%**

Diagram 2: Gender wise distribution of oral pigmentary lesions

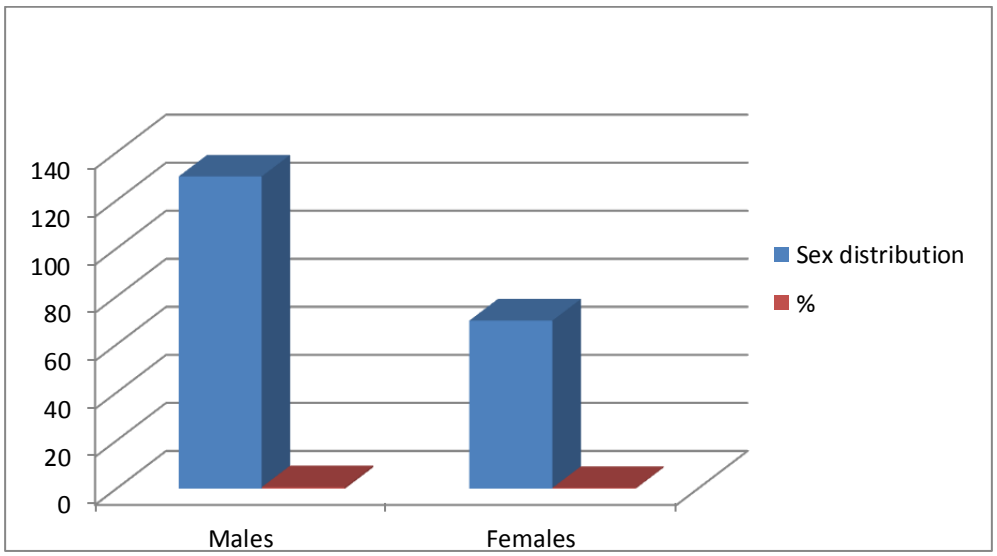


Table 3: Number of oral pigmentary lesions observed in total number of males and females

Oral lesions	No. of males	No. of females	Total	% of total
Melanoplakia	40	20	60	30%
Smoker's melanosis	41	12	53	26.5%
Nicotinic chelitis	20	16	36	18%
Oral lichen planus	4	7	11	5.5%
Drug induced melanosis	10	7	17	8.5%
Jaundice	3	2	5	2.5%
Pregnancy related	0	5	5	2.5%
Amalgam tattoo	3	1	4	2%
Melanotic macule	3	1	4	2%
Nutritional	2	0	2	1%
Hairy tongue	1	0	1	0.5%
Haemangioma of lip	1	0	1	0.5%
Intra oral nevi	1	0	1	0.5%
TOTAL	**129**	**71**	**200**	**100%**

SITE OF INVOVLEMENT

In the present study, the commonest site affected was the anterior labial gingiva involved in 70 patients (35%), next being buccal mucosa involved in 65 patients (32.5%).

Table 4: Site of involvement

Sites involved	Number of cases (n=200)	%
Gingiva	70	35%
Buccal mucosa	65	32.5%
Hard palate	50	25%
Labial mucosa	30	15%
>2 sites	120	60%

Diagram 3: Site of involvement of different oral pigmentary lesions.

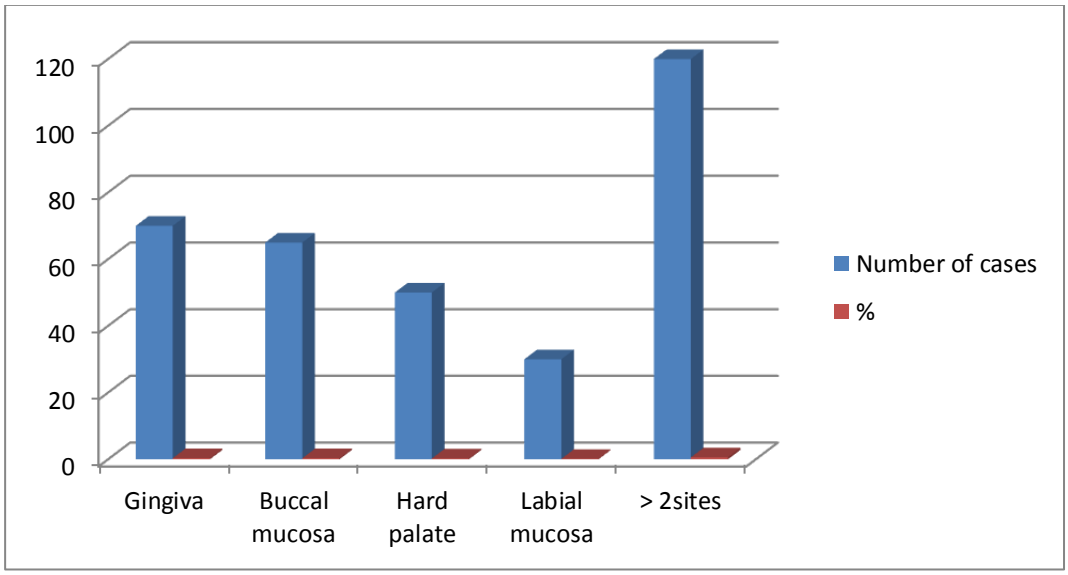


Table 5: Commonest site involved and duration of disease

Oral lesions	Most common site involved	Duration of disease in most patients
Melanoplakia	Gingiva	>6weeks
Smoker's melanosis	Hard palate	>6weeks
Nicotinic chelitis	Anterior gingiva	>6weeks
Oral lichen planus	Buccal mucosa	<6weeks
Drug induced melanosis	Lips and gingiva	>6weeks
Jaundice	Hard palate	<6weeks
Pregnancy related	Hard palate	<6weeks
Amalgam tattoo	Gums	>6weeks
Melanotic macule	Lips	<6weeks
Nutritional	Tongue	>6weeks
Hairy tongue	Tongue	>6weeks
Hemangioma of lip	Lips	>6weeks
Intra oral nevi	Tongue	>6weeks

DURATION OF DISEASE

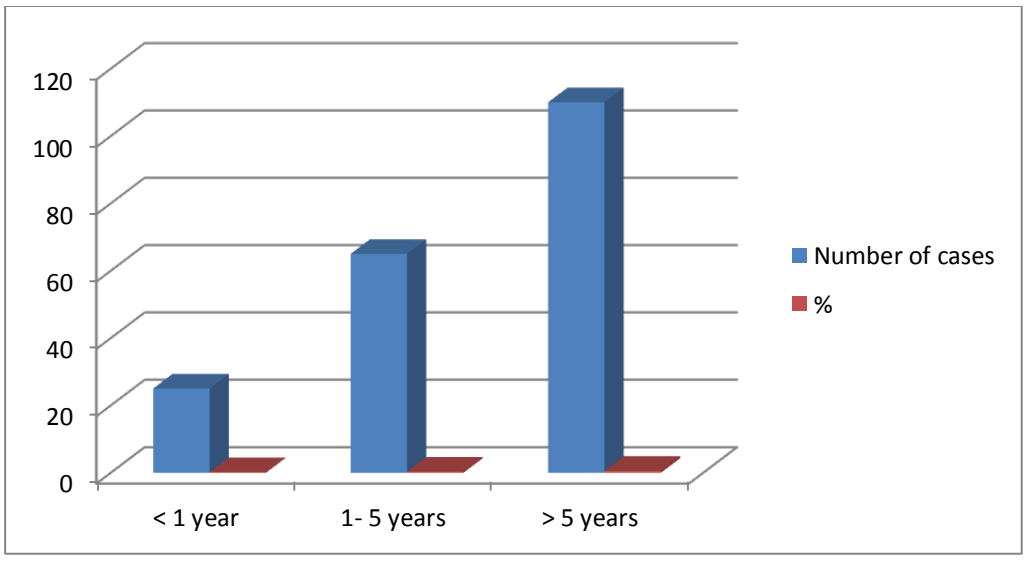
A total of 110 patients (55%) in the present study had oral pigmentation of 5-10 years duration before consultation. The mean duration of disease before consultation was 4 years. The longest duration was 15 years and the shortest 2 days. The study population in our study series were mainly of a rural background and hence the delay in presentation of a relatively asymptomatic condition.

In the present study we observed that majority of the patients 144 in number (72%) had lesions of chronic (>6 weeks) duration.

Table 6: Duration of disease

Duration of Disease (Years)	Number of Patients	%
<1.0	25	12.5%
1.0-5.0	65	32.5%
>5.0	110	55%

Diagram 4: Duration of different oral pigmentary lesions observed



SYMPTOMS

A majority of patients, 120 (60%) were asymptomatic. Discoloration of varying degree was complained by 55 patients (27.5%). Other less common complaints were pain, redness, burning and bleeding. 40 (20%) patients had more than one complaint.

TABLE 7: Presenting symptoms

Symptoms	No. of cases (n=200)	%
Asymptomatic	120	60%
Discoloration	55	27.5%
Burning	20	10%
Swelling	05	2.5%
> 1 complaint	40	20%

Diagram 5: Different presenting symptoms noted

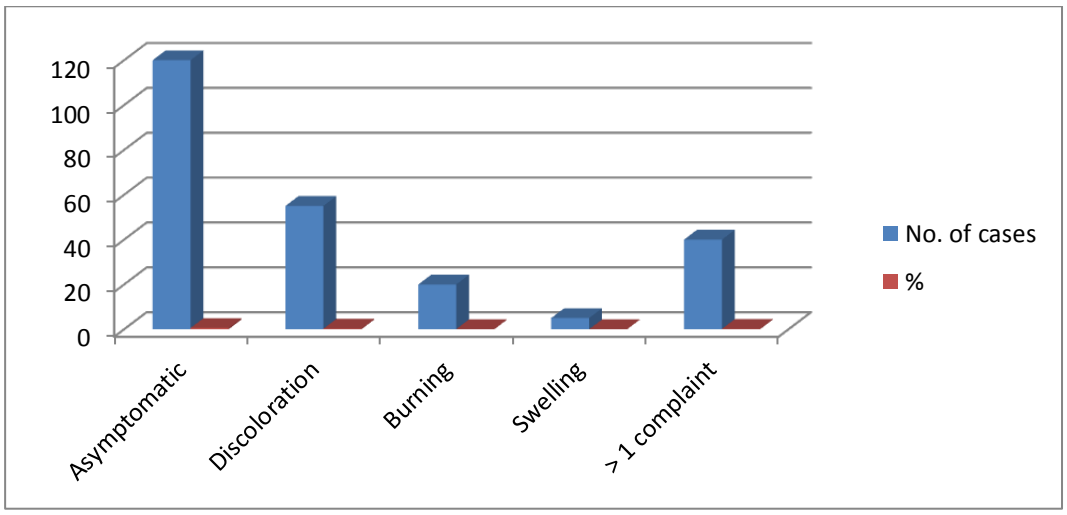


Table 8: Commonest symptom with aggravating factors

Oral lesions	Most common symptom with %	Aggravating factors
Melanoplakia	Discoloration (100%)	Nil
Smoker's melanosis	Discoloration (45.5%)	Tobacco smoke, spicy food
Nicotinic chelitis	Burning (20.8%)	Betel quids, paan masala, spicy foods
Oral lichen planus	Burning (80%)	Dental fillings, betel quids, spicy foods
Drug induced melanosis	Itching (90%)	Nil
Jaundice	Discoloration (100%)	Drugs, fatty foods
Pregnancy related	Discoloration (100%)	-
Amalgam tattoo	Itching (40.7%)	Dental fillings
Melanotic macule	Discoloration (100%)	-
Nutritional	Burning (67.9%)	Spicy foods, smoking, betel quid
Hairy tongue	Burning (100%)	Poor oral hygiene
Hemangioma of lip	Bleeding (100%)	Trauma
Intra oral nevi	Discoloration (100%)	-

SITES OF DISTRIBUTION

In the present study, lesions present only on oral mucosa were seen in 89 patients (44.5%) of 200 patients. 111 cases (55.5%) with other unassociated skin diseases were also diagnosed during screening.

Table 9: Sites of distribution

Sites	No. of cases	%
Exclusive oral lesions	89	44.5%
Associated with other skin lesions also	111	55.5%

Diagram 6: Number of patients with exclusive oral involvement and with other associated skin lesions

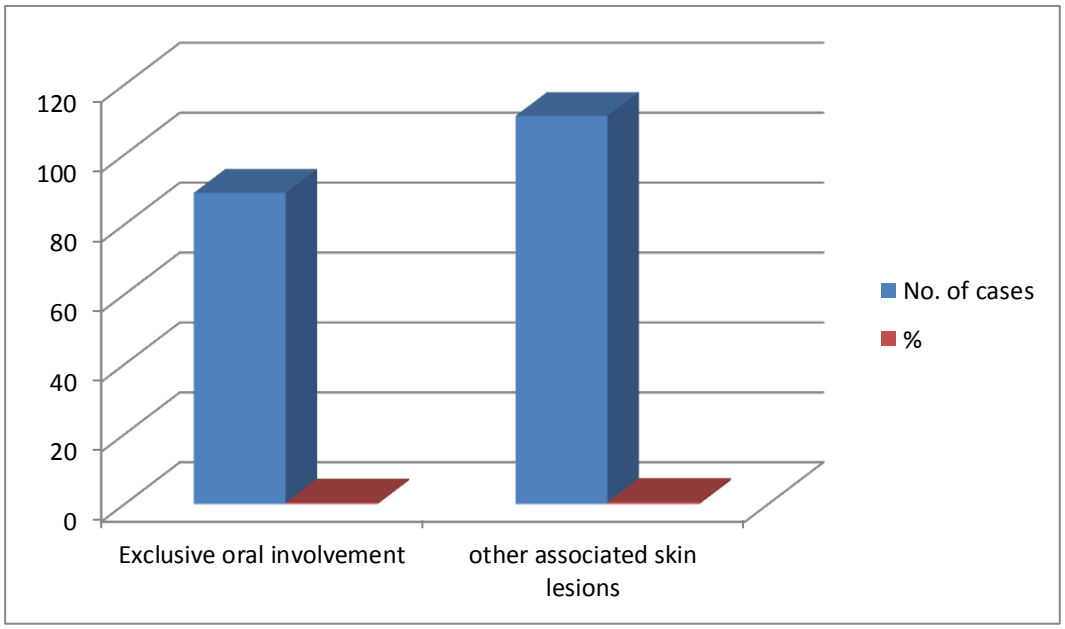


Table 10: Number of patients with exclusive oral involvement and with other unassociated skin lesions.

Disease	Number of patients	Pt. with exclusive oral pigmentatary lesions (no.) / % with group	Pt. with other unassociated skin lesions (no.) / % with group
Melanoplakia	60	25 (12.5%)	35 (17.5%)
Smoker's melanosis	53	23(11.5%)	30 (15%)
Nicotinic chelitis	36	16 (8%)	20 (10%)
Oral lichen planus	11	7 (3.5%)	4 (2%)
Drug induced melanosis	17	10 (5%)	7(3.5%)
Jaundice	5	1 (0.5%)	4 (2%)
Pregnancy related	5	-	5 (2.5%)
Amalgam tattoo	4	4 (2%)	-
Melanotic macule	4	4 (2%)	-
Nutritional	2	-	2 (1%)
Hairy tongue	1	1 (0.5%)	-
Hemangioma of lip	1	1 (0.5%)	-
Intraoral nevi	1	1 (0.5%)	-

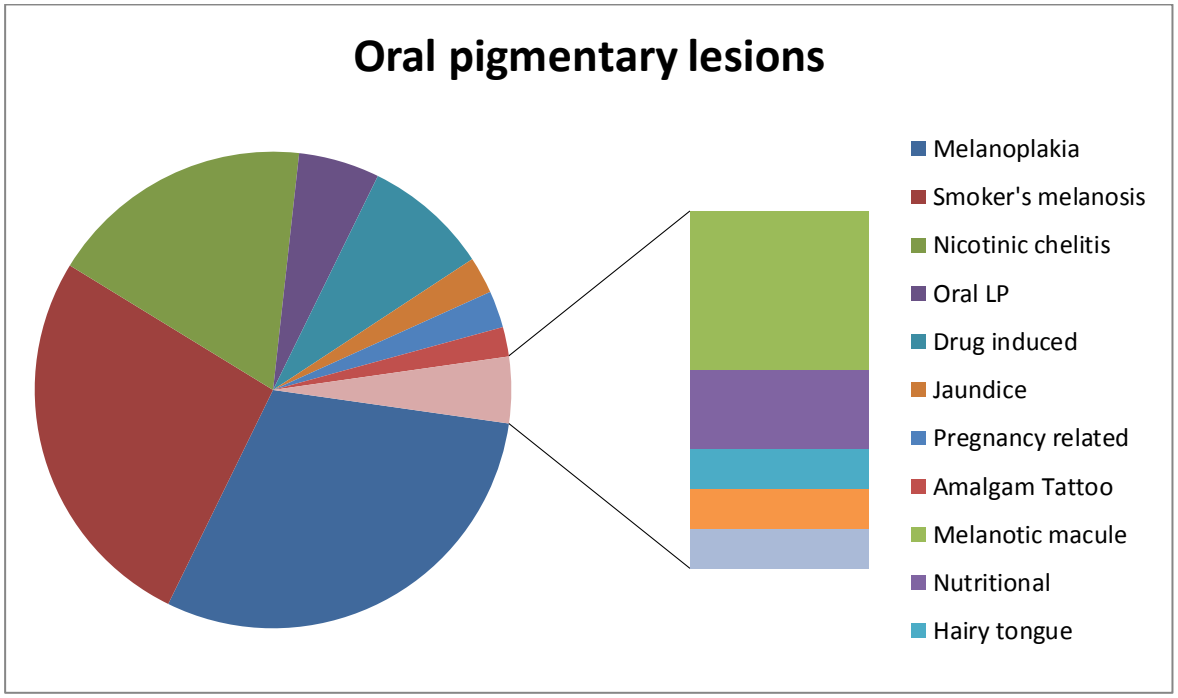
TOTAL NUMBER OF ORAL PIGMENTARY LESIONS NOTED

A total of 13 different types of oral pigmentary lesions were noted in our study. The most common was melanoplakia which constituted 60 (30%) cases followed by 53 (26.5%) cases of smoker's melanosis, 36 (18%) cases of nicotinic chelitis and 17 (8.5%) cases of drug induced melanosis.

Table 11: Total number of oral pigmentary lesions noted

Oral lesions	Total	% of total
Melanoplakia	60	30%
Smoker's melanosis	53	26.5%
Nicotinic chelitis	36	18%
Oral lichen planus	11	5.5%
Drug induced melanosis	17	8.5%
Jaundice	5	2.5%
Pregnancy related	5	2.5%
Amalgam tattoo	4	2%
Melanotic macule	4	2%
Nutritional	2	1%
Hairy tongue	1	0.5%
Hemangioma of lip	1	0.5%
Intra oral nevi	1	0.5%

Diagram 7: Total number of oral pigmentary lesions noted in our study.



CLINICAL PHOTOGRAPHS

- **Melanoplakia (Figures 1 & 2)**

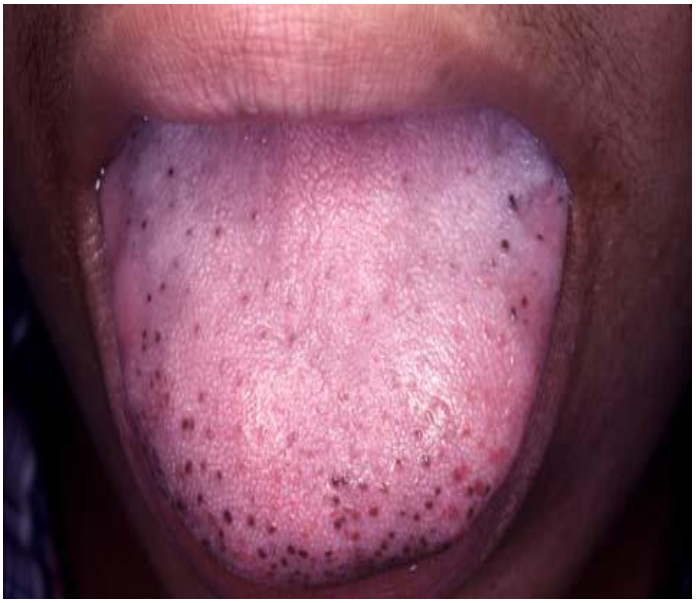


Figure1: **Multiple pigmented macules present on dorsum of tongue.**

- **Melanoplakia (Fig.2)**

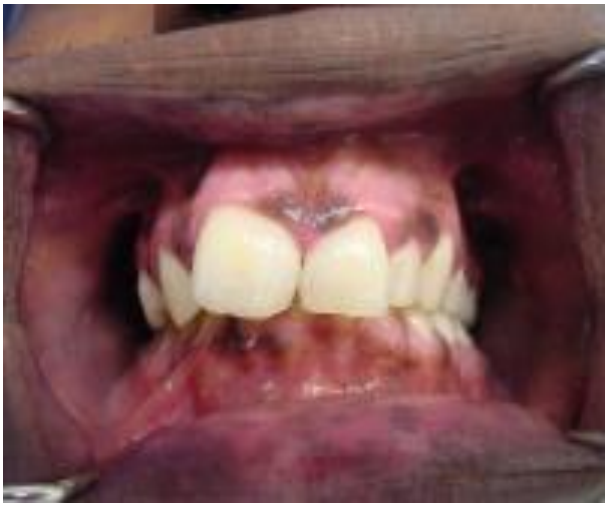


Figure-2: **Pigmented patches seen on the anterior labial gingiva.**

- **Smoker's melanosis (Fig.-3)**

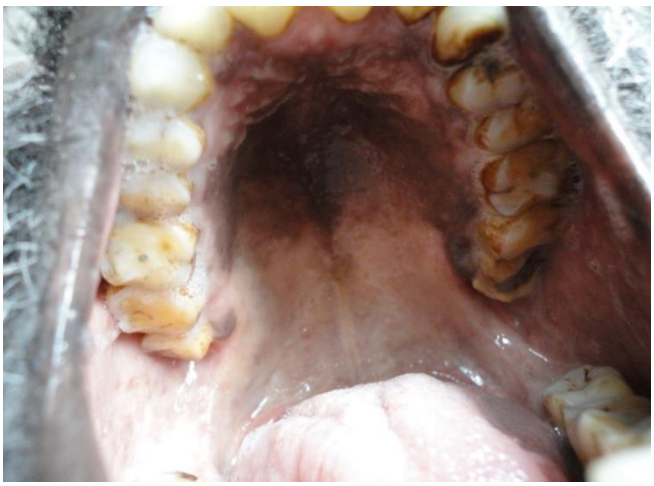


Figure 3: Hyperpigmented patch seen over the hard palate.

- **Nicotinic Chelitis (Fig. 4)**

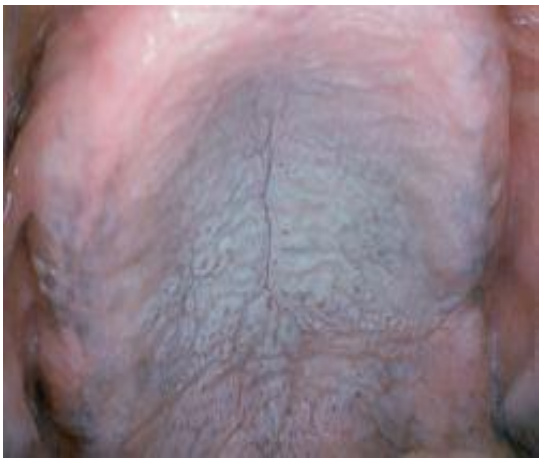


Fig. 4: Pigmented patch studded with multiple papules seen on the hard palate.

- **Oral Lichen Planus (Fig.5)**

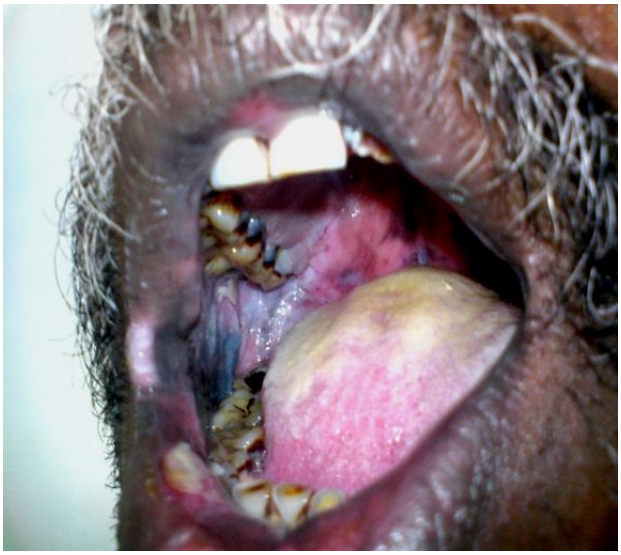


Figure 5: Pigmented lesions of lichen planus involving the buccal mucosa.

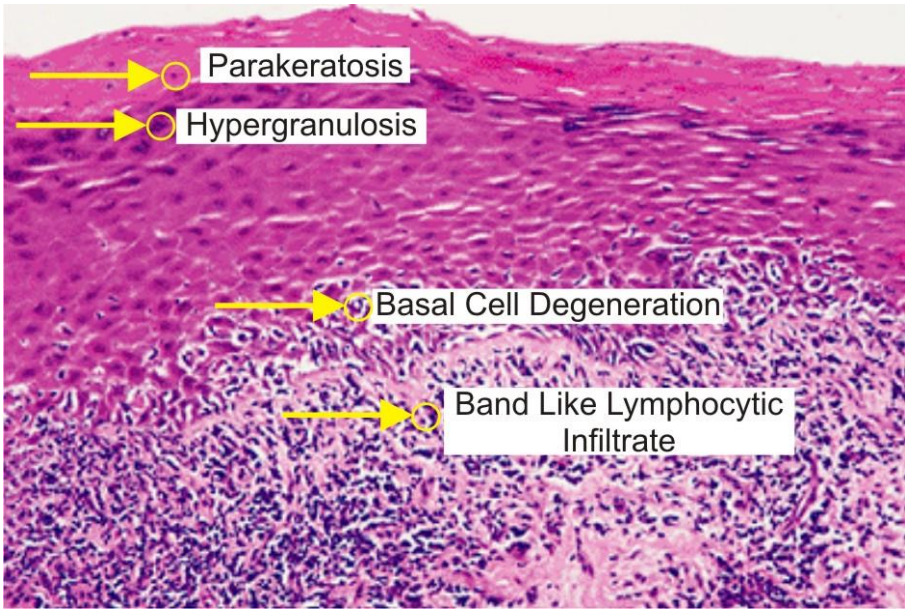


Fig.6: Oral lichen planus histopathology (H& E).

Showing parakeratosis, hypergranulosis, degeneration of basal epidermal cells and band like lymphocytic infiltrate in the upper dermis.

- **Melanotic Macule (Fig.7)**

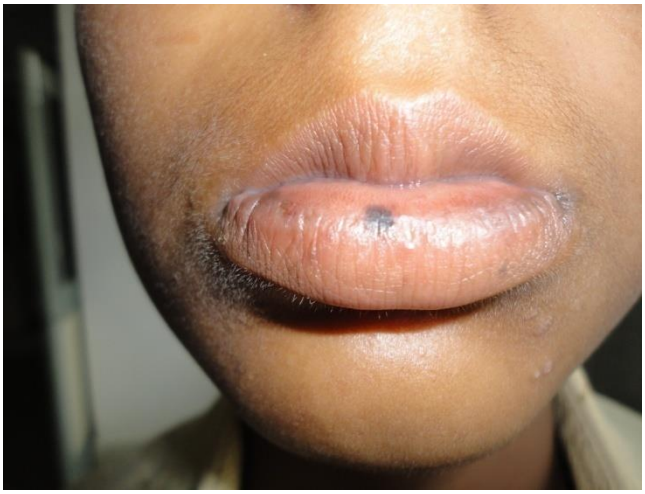


Fig. 7: Pigmented macule seen over the lower lip

- **Amalgam tattoo (Fig.8)**

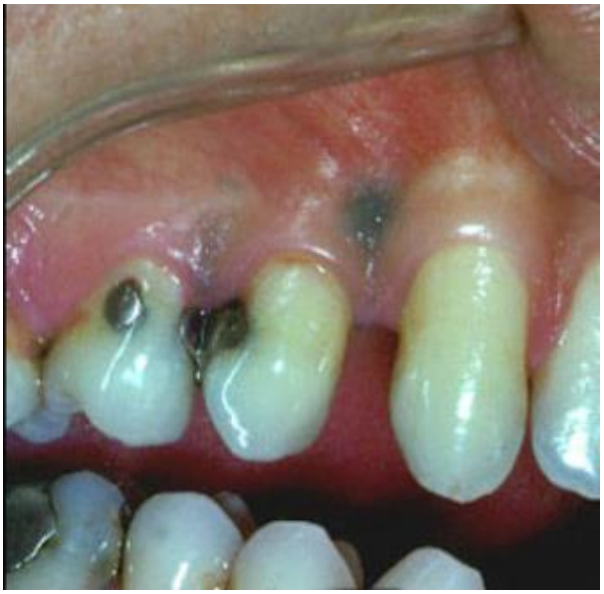


Fig.8: Pigmented plaque seen around dental fillings involving the gingiva

- **Intra Oral Naevi (Fig. 9)**

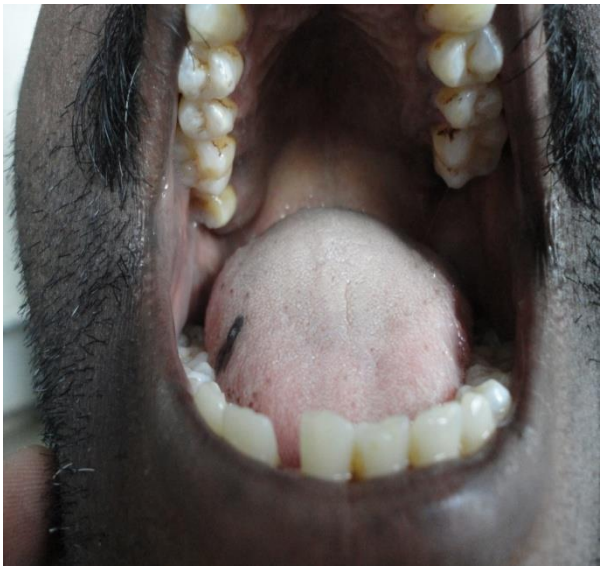


Fig. 9: **Hyperpigmented patch seen on the dorsum of the tongue**

- **Haemangioma of tongue (Fig.10)**

Figure 10: Pigmented nodule seen on the lateral margin of tongue

DISCUSSION

Pigmentation of oral cavity and lips include a spectrum of diseases with varied etiology. It may be associated with severe psychological trauma and fear in the mind of patients. Therefore, it is of immense importance to diagnose these lesions in order to relieve the patient from the stigma of carcinomatous changes. There is a dearth of comprehensive studies on the pattern of oral pigmentary lesions from a developing country like ours.

Melanoplakia:

Melanoplakia is a benign variation of melanin pigment and is not associated with aggressive change. The deeper and heavier the deposits, the darker the lesions appear.

It was the most common oral pigmentary lesion in our study seen in 60 out of 200 patients, constituting 30 % of cases. This condition was most commonly seen in the age group of 35-50 years (25 cases, 12.5%), with a mean age of 42.8 years. It was least seen in age group 35 years and below (7.5%). The most common site affected was anterior labial gingiva (40 cases, 20%) with a male: female ratio of 2:1. These findings are comparable to other studies. In a study done on melanoplakia, the incidence was 27.9%.[114] In a study on oral pigmented lesions, the gingiva was the most common site involved as is the case in our study.[115]

Smoker's melanosis :

Tobacco consumption increases oral melanin production and results in dark pigmentation

It was the second most common oral pigmentary lesion in our study (53 patients, 26.5%). The most common age group affected was 35-50 years (25 cases, 12.5%) and the most common site affected was buccal mucosa (30 cases, 15%). In this study a large number of female patients (12 cases, 22.44% of smoker's melanosis) were noted.[116] This high number can be attributed to the fact that many of them were working in beedi making industry and were also heavy beedi smokers and tobacco chewers. These findings are comparable to other studies. In a study on tobacco and oral diseases, prevalence of smoker's melanosis was 23.5%. In the same study, anterior attached gingiva was the most common site affected whereas buccal mucosa was the most common site affected in our study.[116] This finding can be attributed to the fact that most of our patients are primarily tobacco chewers rather than heavy smokers. In a study on excessive oral melanin pigmentation with special reference to the influence of tobacco habits, smoker's melanosis is reported in 21.5% of smokers and the intensity of the pigmentation is related to the number of cigarettes consumed.[117]

Nicotinic chelitis:

It is characterised by diffuse gray to white thickened appearance on the palate with numerous red papules in the centre. It was seen in 36 patients, constituting 18 % of cases. The most common age group affected was 35-50 years (28 cases, 14%). Total number of males affected were 20 (10%) and females 16 (8%). Commonest site of invovlement was buccal mucosa (58 % of cases). In a study done on nicotinic chelitis in smokers, the incidence of nicotinic chelitis was 5% of all the smokers screened. In the same study, total males affected were 72 and females 28 in number with a male: female ratio of 2.57.[118] In the above mentioned study, only smokers

were screened for nicotinic chelitis alone. This could explain the discrepency in results.

Oral lichen planus:

It was seen in 11 patients (5.5%). Females 7 (3.5%) outnumber males 4 (2%). All patients were in the age group of 35-50 years (100%) and the most common site involved was buccal mucosa (6 cases, 3%). Most of the patients complained of burning sensation (7 cases, 3.5**%).** Out of 11 patients, 7 patients (3.5%) had oral lichen planus exclusively while 4 (2%) of them had involvement of other body areas. The worldwide prevalence of oral lichen planus in the general population has been estimated to range from 0.1% to 2.2%.[119] In Swedish and Japanese populations, the incidences of oral lichen planus were 1.9% and 1.8%, respectively.[120, 121] Since our study is institutional based and not a population based study, this incidence cannot be commented upon.

Females were more affected in our study which is in concordance with all the other studies done earlier.[123]

In our study all female patients were in the age group of 35-50 years whereas in earlier study the most common age group in females was 50-59 years.[124] The most common site involved was buccal mucosa (6 cases, 3%) which is in agreement with other studies also wherein buccal mucosa was most commonly involved.[125]

Drug induced melanosis:

Multifocal mucosal pigmentations may be a side effect of drug therapy. Drug-induced oral mucosal pigmentation generally resolves within weeks to months when the offending drug is withdrawn, although sometimes it is permanent. Fixed drug

eruptions present as well-demarcated areas of hyperpigmentation commonly affecting the oral mucosa and lips.

Drug induced melanosis was seen in 17 patients (8.5%) and the most common site affected was hard palate. 8 patients were diagnosed with fixed drug eruptions caused by antibiotics and remaining 9 patients were HIV positive patients on zidovudine treatment. In a study done on HIV patients on anti-retroviral treatment, 2 patients had pigmentation of the hard palate accounting for an incidence of 12%.[126] This study was done on a small sample size of only HIV patients on treatment and hence cannot be compared with our study.

Amalgam tattoo:

The etiology of amalgam tattoos can be iatrogenic or traumatic. Metal particles may over time leach in the soft oral tissues, resulting in the discoloration. It was seen in 4 patients, constituting 2% of cases and the most common site involved was gingiva seen in 3 patients (1.5% of cases). In one patient it was associated with oral lichen planus. Most patients, 3 (1.5%) were asymptomatic. The results were comparable to other studies. In one study on amalgam tattoo, the incidence was 1.3%.[127] In another study, the most common site involved was gingiva as was found in our study.[128]

Melanotic macule:

Melanotic macules are caused by an increased production and deposition of melanin within the basal cell layer, the lamina propria, or both. It was seen in 4 patients (2%) of which 3 (1.5%) patients were males and 1 (0.5%) was a female. All were asymptomatic and present on the lower lips (100%). In a study, melanotic macules were seen in 1.7% of the patients studied which is comparable to our

study.[129] In another study, lower lip was the commonest site involved which is in agreement with our study.[130] In other large study involving 773 cases of solitary melanocytic lesions, the most common site of involvement was the lips and the vermillion border as is the case in our study.[131] Gingiva was the most commonly affected subsite.[131]

In earlier studies females were affected more than the males whereas in our study males were more affected.[122] Another review reported a prevalence of 0.4% among 86,202 biopsies from the oral cavity.[132]

Haemangioma:

It was seen in 1 patient (0.5%) in a 30 year old male with a history of repeated bleeding episodes due to trauma. In one study oral haemangioma of tongue represented 2% of the lesions of the oral mucosa and this difference is due to the fact that the study mentioned was done exclusively on haemangioma and not with other conditions. In the same study, the most common age group affected ranged from 65 to 74 years.[133]

Hairy tongue:

Black hairy tongue is a benign and self limiting disorder characterised by elongation and hypertrophy of the filliform papillae on the dorsal tongue.

In our study it was seen in 1 male patient with a long duration of 7 years and a history of heavy smoking since 30 years. This is not in accordance with other study, which showed a prevalence of 23.7%.[134] This difference is due to the fact that the study mentioned was done exclusively on hairy tongue and not with other conditions.

Jaundice:

In our study it was seen in 5 out of 200 patients, constituting 2.5 % of cases. All patients were seen in the age group of 35-50 years (5 cases, 2.5%) and in all the patients, the affected site was hard palate with a male: female ratio of 1:1.5. These data are not mentioned in other studies on oral pigmentary lesions with jaundice.

Pregnancy induced pigmentation:

In our study it was seen in 5 out of 200 patients, constituting 2.5 % of cases. All cases were seen in the age group of 25-35 years (5 cases, 2.5%) and in all of them anterior labial gingiva was involved. These data are not mentioned in other studies involving oral pigmentary lesions.

CONCLUSION

Our study brings to light various oral pigmenatry lesions in general population. This study highlights the importance of diagnosing oral pigmentary lesions and removes the general misconception of considering most oral lesions to be precancerous. Thus clinicians need to differentiate the various causes of oral pigmentary lesions.

Though a largely rural population attends our hospital belonging to the lower socio-economic strata with a relatively poor oral hygiene, the frequency of oral pigmentary lesions such as melanoplakia, smoker's melanosis, nutritional disorders and drug induced melanosis in our study is comparable to it's occurrence in other populations worldwide.

Most of our patients are from rural or semi urban areas with poor oral hygiene and have a list of incriminating factors for oral pigmentation like chronic smoking, betel chewing, spicy foods and paan masala. Therefore, it is of immense importance for all physicians to be aware of the various conditions enlisted above to facilitate easy diagnosis and relieve the patient from unnecessary psychological trauma.

SUMMARY

This study was carried out between January 2011 to September 2012. Total out patients aged 18 years and above attending department of dermatology were 18000. Among these 200 patients (1.11%) had oral pigmentary lesions.

Most patients belonged to the age group of 35-50 years (130 patients, 65%), followed by the age group 50-65 years (70 patients, 35%).

Most patients were asymptomatic (120 in number, 60% cases) followed by discoloration of oral cavity (55 in number, 27.5% cases). Other complaints were swelling and burning. 40 patients (20%) had more than one complaint.

Lesions noted exclusively on oral mucosa were seen in 89 cases (44.5%) of 200 patients. Other unassociated skin diseases were also diagnosed in 111 cases (55.5%) during screening.

In both male and female patients, anterior labial gingiva (70 patients, 35%) was the most common site of involvement, followed by buccal mucosa (65 patients, 32.5%) and hard palate (50 patients, 25%).

A total of 13 different types of oral pigmentary lesions were noted in our study. The most common was melanoplakia which constituted 60 cases (30%) followed by 53 (26.5%) cases of smoker's melanosis, 36 (18%) cases of nicotinic chelitis and 17(8.5%) cases of drug induced melanosis.

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ANNEXURES:

CLINICAL PROFORMA:

DEPARTMENT OF
DERMATOLOGY, VENEREOLOGY & LEPROSY.

R.L. JALAPPA HOSPITAL AND RESEARCH CENTRE, KOLAR.

PIGMENTED LESIONS OF ORAL MUCOSA AND LIPS

PATIENT PARTICULARS: **CASE NUMBER:**

NAME : OP/ IP No :

AGE & SEX : DATE :

OCCUPATION : RELIGION :

MARITAL STATUS:

ADDRESS & PHONE NUMBER:

CHIEF COMPLAINTS:

1. Oral lesions- flat/ elevated

2. Discomfort/ burning sensation/ pain/ swelling/ discolouration

HISTORY OF PRESENTING ILLNESS:

-Onset: acute/ insidious

-Duration: since childhood /<6 weeks/ >6weeks

-Site: cheek/ tongue/ lips/ gingiva/ hard palate/ soft palate

-Progression: slow/ rapid

-Risk factors:

- Smoking/ alcohol/ betel and areca chewing/ other forms of tobacco chewing/ spicy foods/ dental fillings

- Stress-

- Dentures-

- Medications taken - Antibiotics/ steroids/ immunosuppressive drugs/ other drugs

- Associated medical illness- Diabetes/ TB/ HIV/ Other skin lesions

PAST HISTORY

-any medications

-any other systemic illness:

PERSONAL HISTORY

-Food habits- vegetarian/ non-vegetarian/ spicy diet

-Sleep- sound/ disturbed

-Bowel and bladder regular/ altered

FAMILY HISTORY

-similar complaints:

-other skin problems:

-consanguinity:

EXAMINATION:

General physical examination:

-Built and Nourishment:

-Pallor/ Icterus/ Clubbing/ Cyanosis/ Lymphadenopathy/ Edema

-Pulse:

-Blood pressure:

-RR:

Examination of Oral cavity

1. SITES:

- Lips
- Labial mucosa

-Buccal mucosa

-Floor of mouth and ventrum of tongue

-Dorsum of tongue

-Palate and fauces

-Labial mucosa

-Gingivae

2. Morphology of lesion: Macule/ Patch/ Papule/ Plaque/ Nodule.

3. Surrounding area: pale/ erythematous/ discoloured/ indurated

4. Secondary changes: erosion/ atrophy/ ulceration.

5. Teeth: dentulous/ edentulous/ amalgam fillings/ dentures

6. Other mucosa: ocular/ anogenital

Cutaneous examination:

-Morphology of lesions

-Distribution of lesions

Hair and nail examination:

Systemic examination:

- CVS
- RS
- PER ABDOMEN
- CNS

Provisional diagnosis:

Investigations:

-Complete haemogram

-Biopsy: Histopathological findings

-Others if any:

FINAL DIAGNOSIS:

TREATMENT

CONSENT FORM

I Mr. / Mrs. / Ms. _____

Age _____ years,

R/O_____

Hereby give consent to Dr. Nikhil Kumar Singh, for performing the procedures related to the study as previously explained to me and any other procedures necessary or advisable to complete the study include the use of local anaesthesia.

I have completely understood the purpose of the procedure. I also agree to co-operative with him.

I have carefully understood the procedure and possible complications and agree to do it by my own free will and in complete consciousness without any influence.

I shall in no way hold the doctor responsible for any of the procedures or their consequences whatsoever.

Signature of Doctor Signature of Patient

Date:

KEY TO MASTER CHART

1. Serial number:

2. OP/ IP number:

3. Age:

4. Sex: Male-**M**, Female-**F**

5. Religion: Hindu- **H**, Muslim- **M**, Christian- **C**

6. Duration: **1**- <6 wks, **2**- >6wks, **3**- since birth

7. Site: **1**-Gingiva **2**- Buccal mucosa

3- Labial mucosa **4**- Lips

5- Tongue **6**- Hard palate

7- Floor of mouth

8. Symptoms: **1**- Asymptomatic **2**- Pruritus

3- Burning **4**-Pain

5- Redness **6**- Swelling

9. Previous treatment: **1**- Present **2**- Absent

10. Morphology: **1**-Macule **2**-Patch

3-Papule **4**-Plaque

5- Nodule **6**- Erosion

11. Risk factors: **1**- Smoking **2**- Poor oral hygiene

3- Tobacco quid **4**- Paan masala

5- Alcohol **6**- Spicy food

7- Dentures **8**- Dental fillings

10. Extra oral lesions: **1**- Present **2**- Absent

11. Oral pigmentary lesions: **1-** Melanoplakia **2-** Smoker's melanosis

3- Tobacco stains **4-** Oral lichen planus

5- Drug induced melanosis **6-** Jaundice

7- Pregnancy related **8-** Amalgam tattoo

9- Melanotic macule **10-** Nutritional related

11- Hairy tongue **12-** Haemangioma

13- Intra oral naevi

S.No.	OPD No.	Age/Sex	Reg	Dur(wks)	Locatn	Sympt	Prev trt	Morph	Risk fac	Othr skin condtn	Diag
1	696244	30/M	H	2	1	1	2	1	1,2,3	1,3	1
2	546570	45/M	H	2	1	1	2	1	1,2,3	1,3	1
3	429919	38/F	H	2	1	1	2	1	1,2,3	1	3
4	518234	55/M	H	2	1	1	2	1	1,2,3	1,3	3
5	542994	76/F	H	2	1	1	2	1	1,2,3,5	3	3
6	578645	32/M	M	2	1	1	2	1	1,2,3	3	1
7	493173	54/M	H	2	1	1	2	1	2,3	1	1
8	547990	66/M	M	2	1	1	2	1	1,5	1,3	2
9	546732	25/F	M	2	1	1	2	1	1,2,3	3	3
10	598233	30/M	H	2	1	1	2	1	1,2,3	1,3	1
11	599629	24/M	H	2	1	1	2	1	1,3,4	Nil	2
12	519963	79/F	H	2	1	1	2	1	1,2,3	1,3	3
13	595427	53/M	H	2	1	1	2	5	1,2,3	Nil	1
14	546796	48/M	M	2	1	1	2	1	1,2,3	1,3	1
15	575129	38/F	M	2	6	1	2	1	1,2,3	3	3
16	641048	23/M	M	2	6	1	2	1	1,2,3	Nil	1
17	641990	65/M	C	2	1	1	2	1	1,2,3	1	1
18	634567	35/M	M	2	1	1	2	5	2,4,5	Nil	2
19	645290	51/F	M	2	1	1	2	2	1,2,3	Nil	4
20	488824	90/M	C	2	1	1	2	2	1,6	1	3
21	509184	43/M	H	2	1	1	2	4	1,3,5	Nil	2
22	509448	39/F	H	2	1	1	2	1	1,2,3,4,5	1,3	3
23	514726	31/M	H	2	1	1	2	1	1,3	3	2
24	530329	23/M	H	2	1	1	2	1	2	Nil	1
25	504009	47/F	M	2	1	1	2	1	1,3	1,3	3
26	698595	30/M	M	2	1	1	2	1	1,2,3,4,5	Nil	1
27	563344	21/M	M	2	1	1	2	1	1,4,5	1,3	2
28	708514	39/F	M	2	1	1	2	1	1,2,3	3	3
29	687163	67/M	H	1	1	1	2	4	2,4,5	3	1
30	637268	61/F	M	2	1	1	2	2	2,4	3	3
31	563344	39/M	H	2	1	1	2	2	1,3,6	1,3	1
32	708514	67/F	H	2	1	1	2	2	1,2,3	1	3
33	708023	55/M	H	2	1	2	2	2	1,3,6	Nil	2
34	790064	41/M	H	2	1	1	2	2	1,2,3,4	1	1
35	659015	39/F	H	2	1	1	2	2	1,3,6	1	5
36	709688	29/M	H	1	6	1	2	2	2,4,5	3	2
37	710354	61/M	H	2	6	1	2	2	1,2,3,4	Nil	1
38	698073	51/F	H	2	6	1	2	2	1,3,4	Nil	4
39	710141	32/M	H	2	6	1	2	2	1,2,3	Nil	1
40	744984	30/F	H	2	6	1	2	2	1,2,3,4	3	3
41	609781	30/M	H	2	6	1	2	2	2	Nil	1
42	515040	76/F	H	1	6		2	2	3,4	3	3
43	691717	68/M	H	2	6	2	2	2	2,4,6	3	1
44	570475	54/M	H	2	6	1	2	2	2	Nil	2
45	851632	31/F	H	2	2	1	2	2	4,6	1,3	3
46	545121	27/M	H	2	2	1	2	2	2,3,6	Nil	2
47	539656	51/F	H	2	2	1	2	4	1,2,3	Nil	4
48	517053	49/M	H	2	1	1	2	1	3,6	1	1
49	533178	57/M	M	2	1	1	2	1	3,4,6	1	3
50	562831	51/F	M	2	1	1	2	1	1,2,3	1	3
51	553304	49/M	M	2	1	1	2	2	1,2,3,4	3	2
52	513024	34/F	H	2	1	1	2	2	1,2,3,4	1,3	3
53	549835	31/M	H	2	1	1	2	1	2,3,5	Nil	1
54	571721	29/M	H	2	1	1	2	1	1,2,3	Nil	1

55	496301	56/F	H	2	1	1	1	1	2,4,5,6	1	5
56	579971	60/M	C	2	1	1	2	1	2,4,6	Nil	4
57	517004	32/M	H	2	1	1	2	2	1,2,3	3	1
58	560856	37/M	H	2	1	1	2	2	1,2,3	1,3	2
59	537199	48/F	H	2	1	1	2	2	2,3,6	Nil	5
60	555684	57/M	H	2	1	1	2	2	2,3,4	3	2
61	531199	31/F	H	2	1	1	2	2	1,2,3	1	4
62	520487	35/M	H	1	1	2	2	5	1,2,3	Nil	1
63	537189	25/M	H	1	1	2	1	1	1,2	Nil	1
64	520487	65/F	H	2	1	3	1	1	1,5,6	Nil	5
65	555892	53/M	H	2		1	2	1	1,3	1	1
66	518477	59/F	H	2	6	1	2	1	1,3,6	3	4
67	518846	34/M	H	2	6	1	2	2	1,2,3,4	Nil	1
68	518824	55/F	M	2	6	1	2	2	1,3,5	Nil	4
69	580933	57/M	M	2		1	2	2	1,2,3	3	2
70	521401	58/F	H	2	6	1	2	2	2,4,5	1	5
71	581124	39/M	H	2	6	4	2	2	2	1,3	3
72	581071	34/M	H	1	6	1	2	5	2	Nil	1
73	552217	59/M	M	2	6	1	2	4	1,2	3	1
74	581221	52/F	H	2	6	1	2	1	1,2,3,4	Nil	2
75	494863	32/F	H	2	6	1	2	1	1,2,3	3	2
76	581371	31/F	H	2	6	1	2	1	1,5,6	3	2
77	528101	30/M	M	2	2	2	2	4	1,3,6	Nil	1
78	523150	23/M	H	1	2	1	2	2	1,2,3,4	Nil	2
79	530508	59/F	H	2		1	2	2	1,2,3,4	2	5
80	570928	67/M	H	2	2	1	2	2	1,2,4	1,3	3
81	573031	54/F	M	2	2	3	2	2	2,4,5	Nil	1
82	521049	31/F	H	2	3	3	1	4	2	3	2
83	580497	29/M	M	2	3	3	2	1	2,4,5	1	1
84	581832	55/M	H	2	3	3	2	1	1,2,3	Nil	2
85	581966	59/F	H	2	2	3	2	1	1,2,3	3	2
86	581931	50/M	H	2	3	4	1	5	1,2,6	Nil	1
87	511036	50/F	H	2	3	4	2	1	1,3,5	3	1
88	510932	54/M	M	2	1	1	2	1	1,4	Nil	1
89	520838	37/F	H	2	3	4	2	4	1,2,3,4	Nil	2
90	520862	38/M	C	1	1	4	2	2	1,2	3	2
91	510481	35/M	H	2	1	2	2	2	1,2,3	Nil	1
92	468417	23/F	H	2	1	2	2	2	1,3,5	3	1
93	582232	31/M	H	2	1	2		2	2,3	Nil	1
94	532249	30/F	H	2	6	2	2	2	2	3	4
95	531983	45/M	H	2	6	1	2	1	3,4,5	1,3	2
96	532343	30/M	H	2	6	1	2	2	4,5	3	1
97	582313	47/F	H	2	6	1	2	2	1,2,3,4	Nil	2
98	581857	69/M	H	2	3	1	2	5	1,2,3	Nil	3
99	582444	52/F	H	2	2	1	2	1	1,2,3	Nil	1
100	582172	30/M	M	2	4	1	1	1	3,4,5	1	1
101	520411	57/F	M	2	4	1	2	1	1,2,3,4	1,3	4
102	530881	69/M	H	2	2	1	2	1	1,2,3,4	3	3
103	540701	34/M	H	2	1	1	2	1	1,2,3	Nil	1
104	493573	32/F	H	2	2	1	2	1	2,3	Nil	1
105	544857	29/M	H	2	2	2	2	2	1,2,4	3	3
106	582763	55/M	H	2	2	2	2	2	1,2,3	Nil	1
107	552133	54/F	H	2	2	2	2	2	2,4,6	Nil	1
108	492512	47/F	H	2	3	4	2	1	1,2,3	3	2
109	538698	34/M	H	2	3	4	2	2	2,4,5	Nil	1
110	502574	49/F	H	2	3	5	2	1	1,2,3	3	1

111	513665	50/M	H	2	1	1	2	2	2	1	3
112	530541	32/F	H	1	3	1	2	2	4	Nil	2
113	510481	31/M	H	2	3	1	2	2	2,5,6	1,3	3
114	524597	45/F	H	2	3	1	2	2	3,4	Nil	1
115	526671	49/M	C	2	3	1	1	2	3,4,5	3	4
116	526368	60/M	M	2	3	1	2	2	1,2,3	Nil	5
117	546546	56/F	H	1	3	1	2	2	4,5	3	2
118	581987	49/M	H	2	5	1	2	2	1,2,3	2	5
119	496304	43/F	H	2	3	2	2	4	3,4,5	3	3
120	537983	39/M	H	2	6	1	2	1	1,2	3	3
121	583694	45/M	H	2	6	4	2	1	2,3,4	Nil	2
122	583745	49/M	H	2	6	1	2	1	2,3	3	3
123	526427	54/F	H	2	6	1	2	1	1,2,3	1	1
124	536281	31/M	H	2	1	1	2	2	1,2,3	3	2
125	510481	43/F	H	2	1	1	2	2	2,3	Nil	2
126	583989	41/M	C	1	1	1	2	2	1,3,4	3	2
127	583953	67/M	H	2	1	1	2	1	1,2,3	1	3
128	546169	49/F	H	2	2	3	2	1	2,4,5	Nil	1
129	528823	50/M	H	2	2	3	2	2	1,2	3	2
130	408011	54/F	H	2	2	3	2	2	1,2,7	Nil	8
131	539160	29/M	H	2	2	3	2	2	1,2,3,4	Nil	2
132	511890	32/F	H	2	2	4	2	2	1,2,3	3	1
133	667121	39/M	M	2	2	4	2	1	3,5	Nil	5
134	676180	34/M	H	2	2	4	1	1	1,2,3,4	Nil	2
135	696244	38/F	H	2	4	1	2	1	1,2	3	3
136	719297	57/M	H	2	4	1	2	2	2	1,3	2
137	724016	46/M	H	2	4	1	2	4	1,3,6	3	3
138	729864	44/M	H	2	4	1	2	1	1,2,3	3	2
139	738922	30/M	M	1	4	1	2	5	2,4,5	2	5
140	741253	49/F	H	2	4	5	2	1	2	Nil	1
141	744102	58/F	H	2	6	1	2	1	2,5,6	1	1
142	753031	33/M	H	2	6	1	2	1	1,2,3,4	1,3	2
143	752817	44/M	H	2	6	1	2	1	2,5,6	1	3
144	663655	66/F	H	2	6	1	2	2	2,4,6	Nil	2
145	545988	25/M	H	2	6	2	2	1	1,2,3,4	Nil	2
146	495005	34/M	M	2	3	1	2	2	2,4,6	Nil	2
147	525761	39/M	H	2	3	1	2	1	1,2,3,4	3	5
148	537284	35/F	H	2	3	1	2	2	2,3,6	3	7
149	507523	32/M	H	2	1	1	2	2	1,3,5	1,3	2
150	758588	56/F	M	2	2	3	2	2	1,2,4	3	1
151	575331	64/M	M	1	2	5	2	2	1,6	Nil	5
152	611978	31/M	H	2	3	3	2	2	1,5	3	2
153	550368	39/F	H	2	3	1	2	2	1,4	3	2
154	610516	38/M	H	2	3	1	2	2	1,2,3	3	3
155	546852	28/M	H	1	2	1	2	2	1,3	3	2
156	517278	49/M	H	2	2	1	2	5	1,2,3,4	Nil	5
157	541352	34/F	H	2	2	1	2	1	1,2,3	1,3	6
158	537546	35/F	H	2	3	1	2	1	2,6	1,3	2
159	524932	30/F	H	2	5	1	2	1	2,4,6	Nil	1
160	508677	55/F	H	2	1	1	2	1	1,2	1	1
161	516956	59/M	H	1	1	1	2	1	2,3	3	3
162	500092	40/M	H	2	1	1	2	1	2	Nil	5
163	508413	35/M	H	2	3	1	2	2	4,5	1	3
164	541597	43/M	M	1	2	1	1	1	1,2,3	3	1
165	541506	59/M	M	2	1	1	2	2	1,2,3,4	Nil	5
166	639623	31/F	H	2	2	1	2	1	1,3,4	Nil	2

167	493634	31/F	H	2	5	1	2	2	2	Nil	9
168	528739	30/M	H	2	3	2	2	2	1,2,3,4	1,3	6
169	491804	47/F	H	2	6	2	2	2	1,3	Nil	2
170	528522	49/M	M	2	6	3	2	2	2,4,7	Nil	8
171	511905	54/M	M	2	6	3	2	1	2	Nil	9
172	550333	58/M	H	1	6	3	2	1	3,5	1,3	6
173	592915	35/F	H	2	1	3	2	1	2,5,6	3	2
174	437365	59/M	H	1	1	4	2	1	1,3,7	Nil	8
175	544865	59/F	H	2	1	4	2	1	2,3,6	Nil	1
176	533809	68/M	H	2	1	4	2	1	3,5	1	3
177	523986	24/F	H	2	2	4	2	1	2,3	3	7
178	553981	59/F	H	2	2	1	2	2	1,4	Nil	5
179	556313	69/M	H	1	2	1	2	2	3,5	Nil	9
180	531388	31/M	H	2	2	1	2	4	2,4,6	1,3	6
181	531455	68/M	H	2	2	1	2	1	1,2,3	Nil	5
182	545940	26/F	M	2	1	1	2	1	2,3,4	3	7
183	546042	55/F	H	2	3	1	2	5	2	Nil	1
184	515469	30/F	H	2	3	1	2	1	3,4,6	Nil	2
185	526394	34/M	M	2	3	1	2	1	1,3,5	Nil	12
186	529664	69/F	C	2	3	1	2	1	1,2,3,4	3	1
187	507756	54/M	H	2	4	1	1	1	2,5,6	3	10
188	685904	28/F	H	2	2	1	2	2	2	3	7
189	541611	49/F	H	2	4	1	2	2	1,2,3,4	1,3	2
190	494950	32/M	M	1	4	1	2	2	2,3	Nil	9
191	685903	43/M	H	2	4	2	2	2	2,5	Nil	12
192	522209	44/M	H	2	4	2	2	4	1,2,3,6	Nil	11
193	685905	32/F	H	2	5	2	2	2	1,3,4	3	1
194	516672	56/F	M	1	5	2	2	2	2,5,6	1,3	2
195	506622	43/M	H	2	5	1	2	2	1,2,3	3	10
196	552827	65/F	H	2	5	1	1	2	1,3,5	3	4
197	501722	32/F	H	2	6	1	2	2	2,3	3	7
198	532524	32/F	M	2	6	1	2	2	2,4,5	3	1
199	573064	50/M	H	2	6	1	2	2	1,3,4,7	Nil	8
200	508639	38/F	H	2	6	1	2	1	1,2,3,4	1,3	6