K.M. SHAH DENTAL COLLEGE & HOSPITAL
Estd. 1999
Vadodara
SUMANDEEP VIDYAPEETH
Deemed to be University

BDS Year 4 Regular batch
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Subject: Oral Medicine and Radiology
Topic: Pigmented Lesions of Oral Cavity

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Lecture objectives

- Knowledge of physiologic or abnormal lesion
- Differentiation of various pigmented lesions in oral cavity
- For further evaluation and treatment plan

LECTURE CONTENT

- **Introduction**
- **Healthy oral mucosa**
- **Classification**

- **Endogenous pigmentations**
  - Focal pigmentation
  - Multifocal/diffuse pigmentation
  - Melanosis associated with systemic or genetic disease

INTRODUCTION

- Pigment" is derived from the Latin word meaning "Colour".
- Pigmentation- process of deposition of pigments in tissues

HEALTHY ORAL MUCOSA

- Normally various shades of red.
- Attributed to
- Different structural colours, Degree of keratinization, Numbers and melanogenic activity of melanocytes, Vascularization, Type of submucosal tissue (muscle, bone, cartilage)

<u>The physiologic colour of the oral mucosa ranges from</u>

- White to red-purple in light-skinned people
- Black to brown colour of dark-skinned people

Endogenous

Focal Melanocytic Pigmentation

- Freckle/ Ephelis
- Oral/Labial Melanotic Macule
- Oral Melanoacanthoma
- Melanocytic Nevus
- Malignant Melanoma

Multifocal/Diffuse Pigmentation

- Physiologic Pigmentation
- Drug-induced Melanosis
- Smoker's Melanosis
- Post-inflammatory Hyperpigmentation
- Melasma(Chloasma)

Melanosis Associated With Systemic Or Genetic Disease

- Hypoadrenocorticism (Adrenal Insufficiency
- Cushing's Syndrome
- Hyperthyroidism (Graves' Disease)
- Primary Biliary Cirrhosis
- Vitamin B12 Deficiency
- Peutz-Jeghers Syndrome
- Café au Lait Pigmentation
- HIV/AIDS-Associated Melanosis

Most common endogenous sources of mucosal colour change

1.Haemoglobin-

- Red, blue, or brown appearance
- Produced by lysis of red blood cells

2.Hemosiderin

3.**Melanin** (Derivative of tyrosine)

- Melas-black
- Synthesized by melanocytes in the basal cell layer of the epithelium
- Appear brown, blue, or black (dependent on the amount of melanin and its location within the tissue)
- Actions:
- Protect from damaging effects of actinic irradiation
- Act as scavengers (against various cytotoxic intermediate)
- Composed of eumelanin (brown-black pigment)and pheomelanin (red-yellow pigment)

Melanocytes in basal layer → **Stimulation (tobacco, heat, smoke, hormonal drug, idiopathic)**

↓

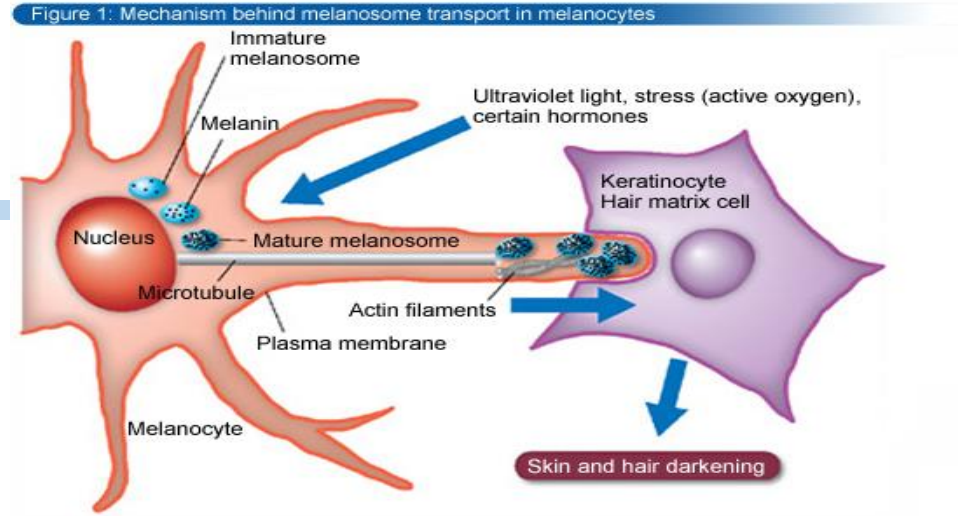
Activation of tyrosinase enzyme → **Formation of melanin from DOPA (dihydroxyphenyl alanine)**

↓

Melanin incorporated in melanosomes → **Melanosomes release melanocyte within keratinocyte cells**

↓

Brownish black appearance



When melanocytes are activated by ultraviolet light etc., melanin (schematically shown as granules) is formed around the nucleus and stored in small organelles known as melanosomes. Melanosomes are transported along microtubules and actin filaments to the cell periphery. Subsequently, melanosomes bind to the plasma membrane and transfers melanosomes to keratinocytes and hair matrix cells, resulting in skin and hair blackening.

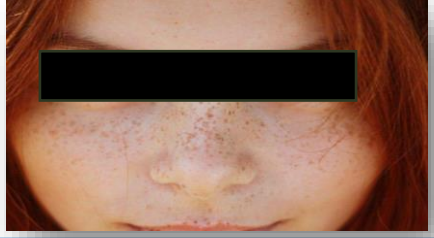
Increase in melanin pigmentation

Overproduction
(basilar melanosis)
Increased sun exposure
Drugs
Pituitary adrenocorticotropic hormone (ACTH)
Genetic factors (in association with certain syndromes)

Overpopulation
Benign nevi
Malignant melanomas

FOCAL PIGMENTATION

Clinical presentation



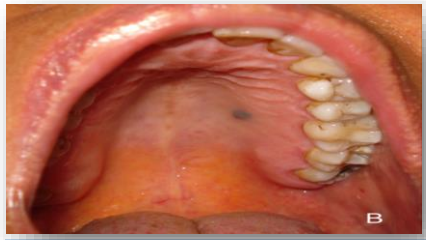
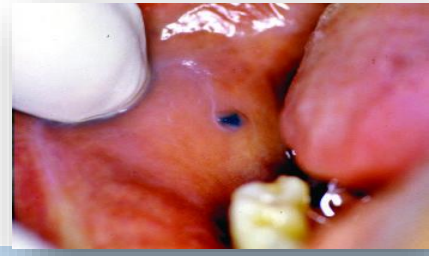
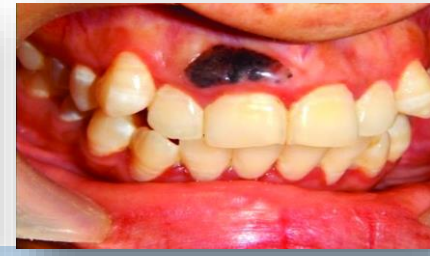
Pigmentations	Freckles	Melanotic macule	Melanoacanthoma
Size Shape Lesion	Small **(1–3 mm)** Well-circumscribed **Macule**	Small **(<1cm)** Well circumscribed, oval or irregular	**Several centimetres**, ill-defined, irregular **Macular or plaque-like**
Etiology	Sunlight	Trauma	Trauma/history of chronic irritation
Site	Sun exposed (facial and perioral)	Lower lip and gingiva	Buccal mucosa(50%)
Other	• Light-skinned individuals • Darker: Spring, summer • Lighter: Autumn, winter	• Sun exposure is not a precipitating factor • Does not generally recur following surgical removal	• Benign • Mixed lesion(Keratinocyte, dendritic melanocytes) • Tendency to enlarge rapidly
Biopsy	Not required	Rule out melanoma	Lead to spontaneous regression of the lesion)
DD		Melanocytic nevus, Malignant melanoma, Amalgam tattoo, Focal ecchymosis	Pigmented nevus, melanotic macule, melanoma

Melanocytic Nevus

- Benign
- Solitary, asymptomatic, <1 cm, brown or blue, well circumscribed nodule or macule
- Etiology: Genetic and environmental
- Consequence of melanocytic growth and proliferation
- Common in Females
- Over the age of 30
- Hard palate >buccal and labial mucosae > gingiva

CONGENITAL	ACQUIRED
• Small nevi • >1cm but <5cm • Large nevi • > 10cm	**Intradermal/intra mucosal (most common 50%)** • Junctional • Compound • Spindle cell or epithelioid • **Blue nevus (2nd most common 25-30%)**

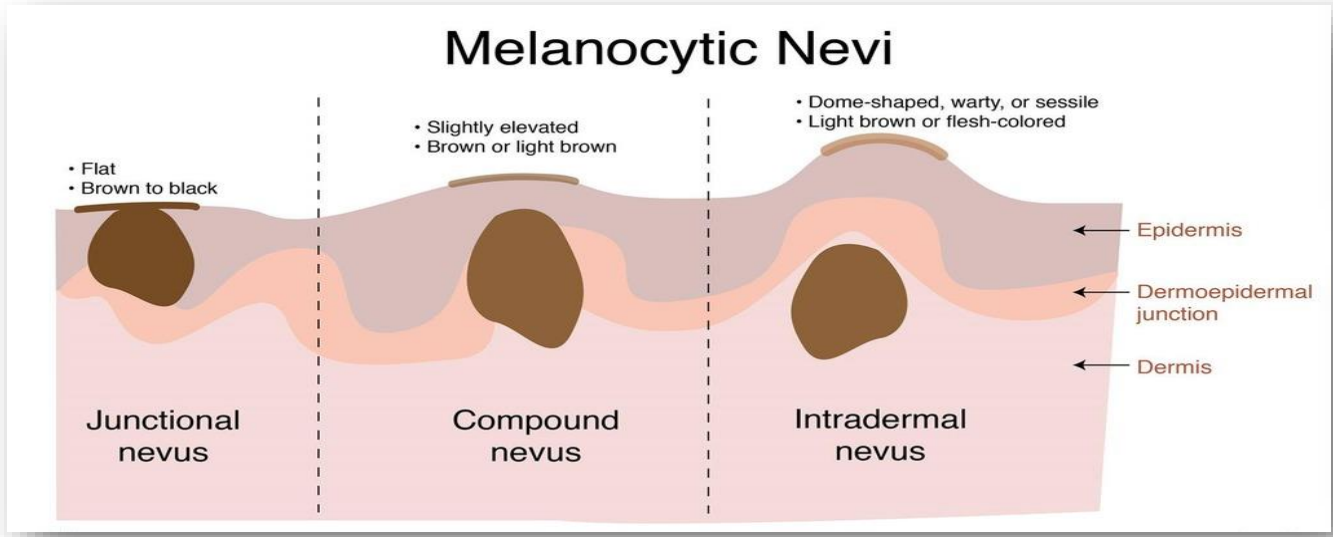
- Intra-mucosal nevus > common blue nevus > compound nevi > junctional nevus

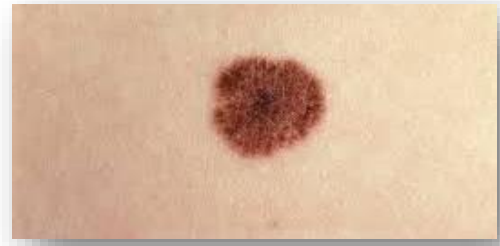
- solitary black pigmented papule at hard palate, retromolar mucosa, gingiva.

Histologically, nevi are classified into subtypes

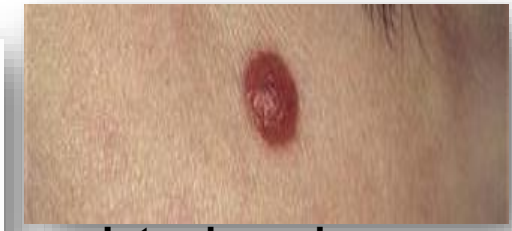
- Junctional nevi : In the epithelium at the connective tissue junction
- Intradermal nevi or intramucosal nevi : In the lamina propria and do not contact the basement membrane
- Compound nevi : In a combination of zones (both the epithelium and the connective tissue)



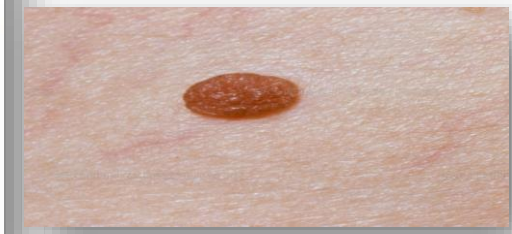
Clinical Presentation



Junctional nevus



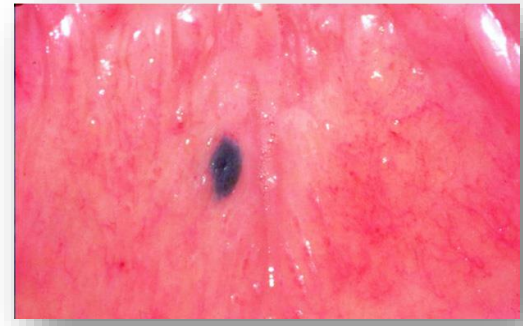
Intradermal nevus



Compound nevus

Blue Nevi/ Jadassohn-tieche nevi

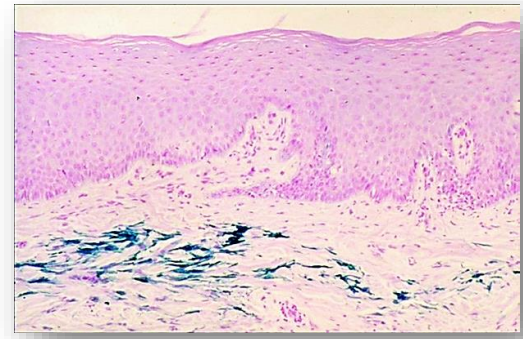
- Derived from neural crest cells migrating ventrally along the developing nerves during embryogenesis
- Bluish colour: preferential scatter of the short wavelength component of visible light (blue) by melanin particles, so-called **Tyndall effect**
- Acquired(solitary lesion) but may also be congenital (multiple sites)



Blue nevus of the palate.

- AGE – any age , >30 years
- SEX – M > F Cuteneous nevi
  - M < F oral melanotic nevi

Site – Hard palate commonly ,buccal & labial mucosa , gingiva



Histopathologic features of a blue nevus. Spindle shaped nevomelanocytes

Two widely known histological subtypes:

Common blue nevus -Variants of common nevi include epithelioid, sclerosing, amelanocytic, and combined

Cellular blue nevus

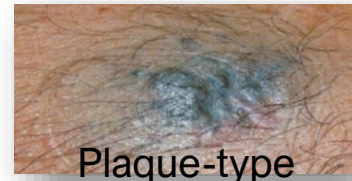
Potential precursor of malignant melanoma(complete removal needed)

Variants of blue nevus:

1 Congenital nevus
2 Spitz nevus
3 Balloon cell nevus
4 Cellular
5 Epithelioid
6 Plaque-type


Spitz nevus


Balloon cell nevus


Plaque-type

Nevus Susceptibility In Various Inherited Diseases

- Familial atypical multiple mole melanoma syndrome- Atypical nevi
- Carney complex- Epithelioid blue nevus
- Turner's syndrome, Noonan's syndrome- Common nevi
- Neurocutaneous melanosis- Congenital nevi

- Biopsy is necessary

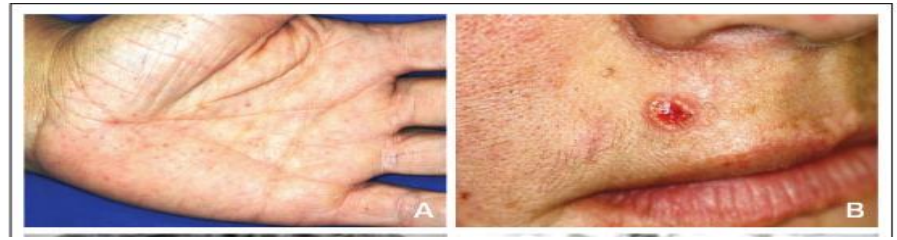
- DD: Various vascular phenomena
- Treatment :
- Complete but conservative surgical excision
- Recurrence has only rarely been reported

Nevus of Ota

- Benign melanosis that primarily involves Trigeminal nerve, namely the ophthalmic v1 and the maxillary v2 are most commonly involved.
- Gray-blue hyperpigmentation of the conjunctiva and sclera along with ipsilateral facial skin.
- Palatal involvement may also occur.

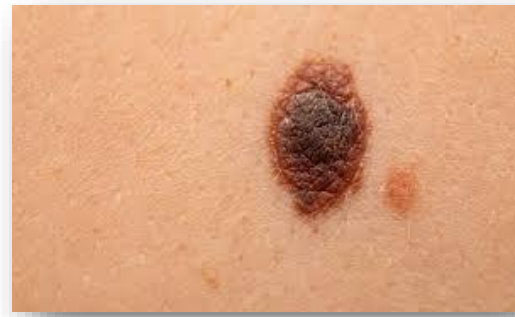
Gorlin-goltz syndrome

- Nevoid basal cell carcinoma syndrome (BCCS) or basal cell nevus syndrome (BCNS)
- Multiple basal cell nevi carcinomas
- Keratocystic odontogenic tumors
- Bifid ribs



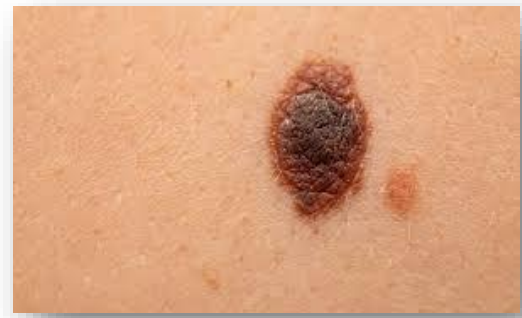
Malignant Melanoma

- Least common, deadly of all primary skin cancers, arising from malignant melanocytes
- Known risk factors play a role in the pathogenesis of melanoma are
- History of multiple episodes of acute sun exposure
- Young age
- Immunosuppression
- Multiple cutaneous nevi
- Family history of melanoma



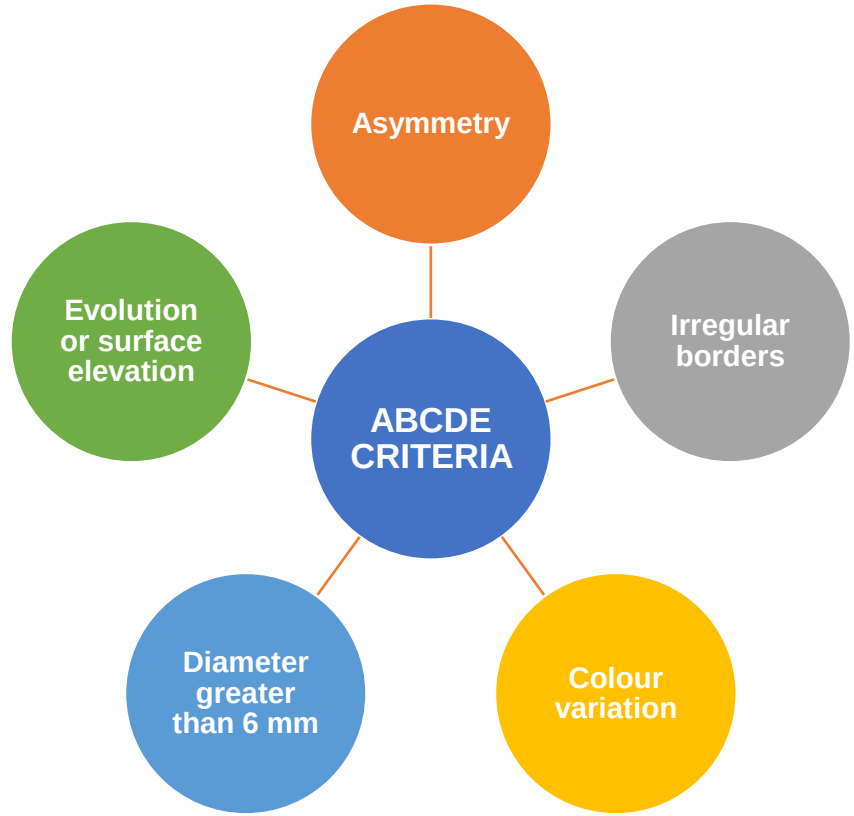
Malignant Melanoma

- Gene related to this are
- CDKN2A (cyclin-dependent kinase inhibitor 2A) (p16), CDK4 (Cyclin-dependent kinase 4), RB1(Retinoblastoma gene)
- Exhibit mutations in BRAF, HRAS, NRAS (proto-oncogenes)
- MC1R (Melanocortin 1 receptor) polymorphisms
- PTEN (phosphatase and tensin homologue) alterations or loss of function



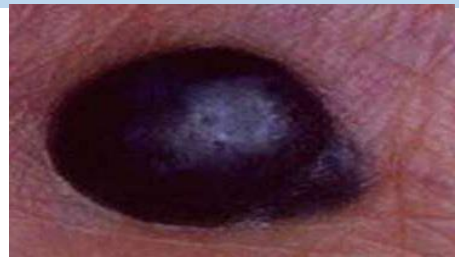
Clinical Features

- White populations that live in the sunbelt regions of the world
- Mortality rates are higher in blacks and Hispanics
- Most commonly occurring cancers in women of child-bearing age
- Facial skin, the malar region is a common site

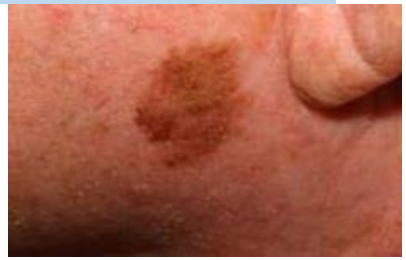


ABCDE criteria

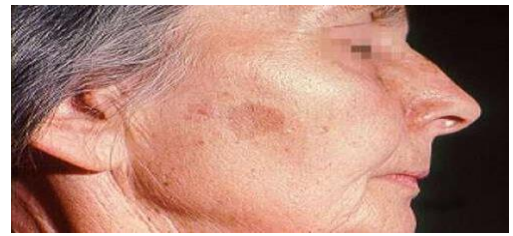
- **Types of melanoma**
- Superficial spreading
- Nodular melanoma
- Lentigo maligna melanoma
- Acral lentiginous melanoma
- Mucosal lentiginous melanoma



Nodular melanoma



Lentigo maligna melanoma

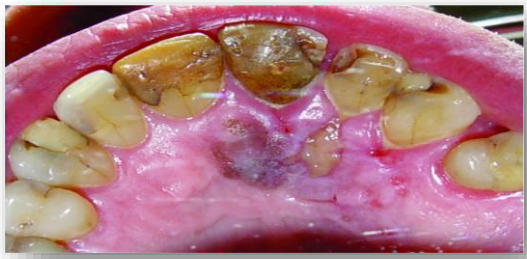


Acral lentiginous melanoma

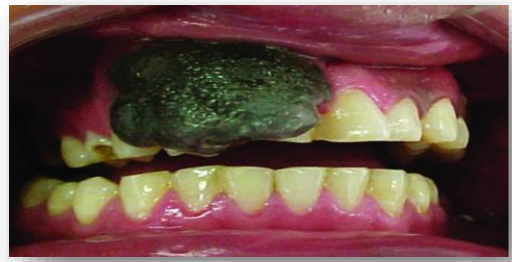
- In the first three subtypes, initial growth: radial growth phase-melanocytic tumor cells spread laterally and therefore superficially
- These lesions have a good prognosis if they are detected early and treated
- Nodular lesions- vertical growth phase(invasion into the deeper connective tissue)

Oral Melanomas

- Develop in the head and neck, most in the sinonasal tract and oral cavity
- Prevalence: among black-skinned and Japanese people
- Frequently in males
- BRAF mutations are rarely observed
- Most present over the age of 50
- Palate : most common site of involvement
- Maxillary gingiva/alveolar crest : second most frequent site



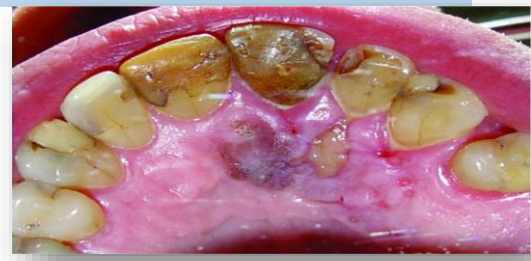
Malignant melanoma exhibiting macular involvement of the anterior hard palate.



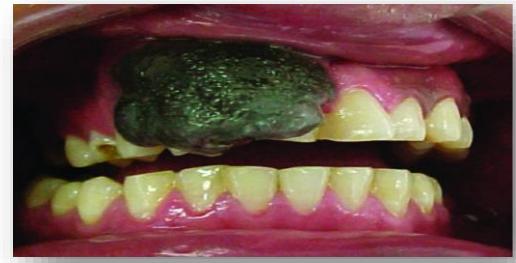
Malignant melanoma presenting as a mass on the maxillary gingiva.

Oral Melanomas

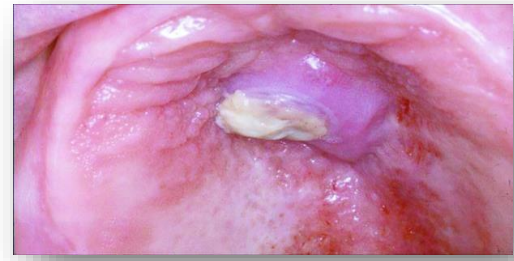
- Macular, plaque like or mass-forming
- Well circumscribed or irregular
- Focal or diffuse areas of brown, blue, or black pigmentation
- Up to one-third of oral melanomas may exhibit little or no clinical evidence of pigmentation (amelanosis)
- May exhibit both melanotic and amelanotic areas
- **Signs and symptoms**
- Ulceration, Pain, Tooth mobility or spontaneous exfoliation
- Root resorption, Bone loss, Paraesthesia/anaesthesia
- Tumours may be completely asymptomatic



Malignant melanoma exhibiting macular involvement of the anterior hard palate.



Malignant melanoma presenting as a mass on the maxillary gingiva.



Amelanotic melanoma of the palate

Oral Melanomas

- **Clinical Differential Diagnosis**
- Melanocytic nevus
- Oral melanotic macule
- Amalgam tattoo
- Various vascular lesions
- Other soft tissue neoplasms

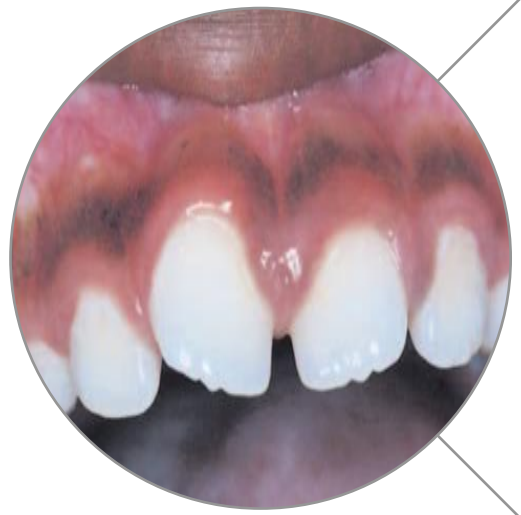
- Biopsy is always warranted
- Oral mucosal malignant melanoma -a very poor prognosis (five-year survival rates of 15%–40%)
- Palate shows the worst prognosis
- Regional lymphatic metastases are frequently identified

Management

- Primary oral melanomas:
- Ablative surgery with wide margins, adjuvant radiation therapy may also be necessary
- CT and MRI: Explore metastases to the regional lymph nodes
- Chemotherapeutic and immunotherapeutic strategies
- Adjuvant interferon-α-2b therapy

- Discovery of KIT and BRAF mutations and the development of novel immune-therapeutic agents that specifically target and inhibit these oncogenic pathways have provided new alternative treatments

MULTIFOCAL/DIFFUSE PIGMENTATION



Physiologic Pigmentation

Dark- complexioned individuals

Site: **attached gingiva(most common),** buccal mucosa, hard palate, lips and tongue

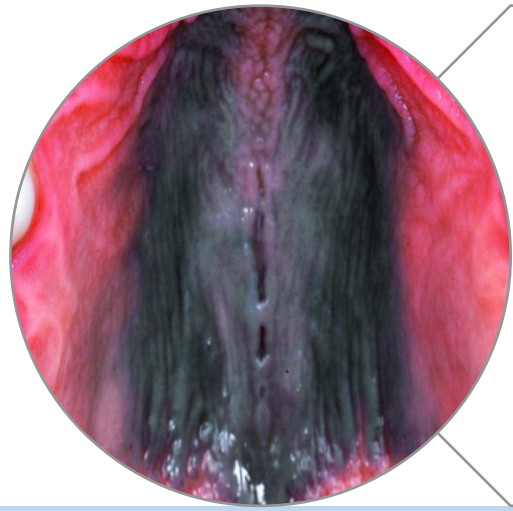
Bilateral, well-demarcated, ribbon-like, dark brown band that usually Spares the marginal gingiva

basilar melanosis

Treatment:

Gingivectomy, laser therapy, cryosurgery

May eventually recur.



Drug-Induced Melanosis

Site: hard palate

Flat and without any evidence of nodularity or swelling

Etiology Drugs or drug metabolites stimulate melanogenesis

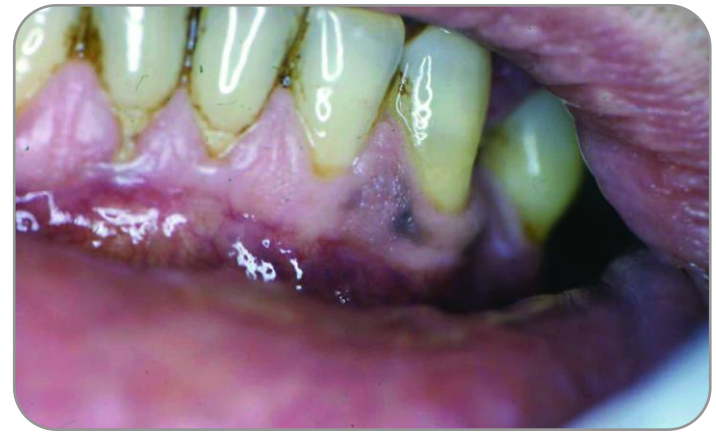
Basilar hyperpigmentation

Treatment:

Tends to fade within a few months after the drug is discontinued

Smoker's Melanosis

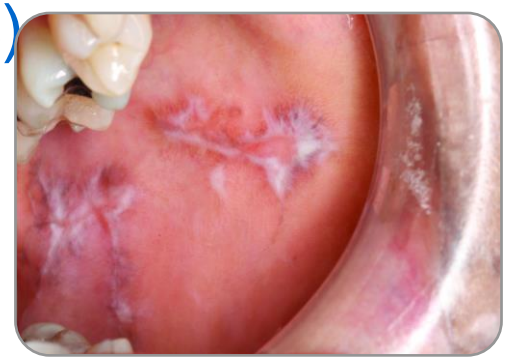
- Seen among cigarette smokers
- Site: gingivae, buccal mucosa, lateral tongue, palate, and floor of the mouth involved
- Brown, flat, and irregular; some are even geographic or map-like,
- Stimulated by tobacco smoke products, Heat of the smoke: stimulate the pigmentation



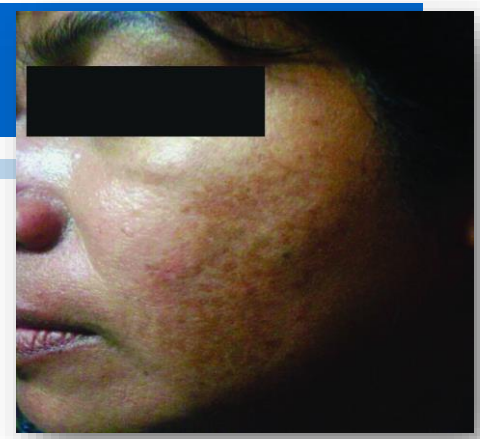
- Not a preneoplastic condition
- Basilar melanosis with melanin
- incontinence
- Treatment:
- Reduction in smoking may lead to fading of the pigmentation

Post Inflammatory Hyperpigmentation

- Dark-complexioned individuals
- In areas that were subjected to previous injury or inflammation
- Acne prone face is a relatively common site
- Oral pigmentation has also been described in patients with lichen planus (lichen planus pigmentosus)
- Basilar melanosis with melanin
- incontinence
- Treatment:
- Resolution of the lichenoid lesion, pigmentation eventually does subside



Melasma (Chloasma)



- Acquired, symmetric melanosis
- Female predilection, mask of pregnancy
- Darker-skinned individuals
- Most commonly affected areas of face : Forehead, cheeks, upper lips, and chin
- Associated with sun exposure, hormonal factors including pregnancy and contraceptive hormones
- Combination of estrogen and progesterone that induces the pigment
- Idiopathic cases
- Elevated levels of luteinizing hormone(LH) in both sexes
- Decreases in serum estradiol (in women) and testosterone (in males)

Melasma (Chloasma)

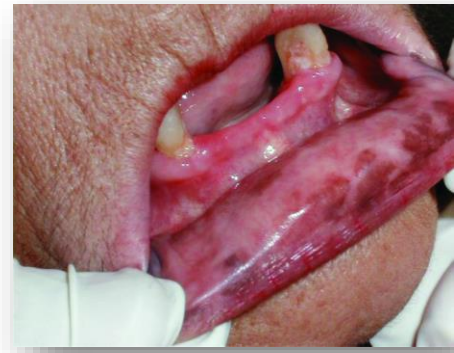
- Treatment: Topical administration of a triple Combination product
- 4% hydroquinone, 0.05% tretinoin, 0.01% fluocinolone acetonide along with photo protection (SPF 30 sunscreen)

Title	Melasma Treatment: An Evidence-Based Review
Authors	Jacqueline McKesey, Andrea Tovar-Garza, Amit G. Pandya n Evidence-Based Review. *Am J Clin Dermatol* **21**, 173–225 (2020).
Objective	to conduct an evidence-based review of all available interventions for melasma
Material and method	A systematic literature search of the PubMed electronic database was performed using the keywords ‘melasma’ and/or ‘chloasma’ in the title, through October 2018.
Conclusions	Hydroquinone monotherapy and triple combination cream are the most effective and well-studied treatments for melasma, whereas chemical peels and laser- and light-based therapies are equal or inferior to topicals, but ofer a higher risk of adverse effects. Oral tranexamic acid may be a safe, systemic adjunctive treatment for melasma, but more studies are needed to determine its long-term safety and efficacy. Limitations of the current evidence are heterogeneity of study design, small sample size, and lack of long-term follow-up, highlighting the need for larger, more rigorous studies in the treatment of this recalcitrant disorder.

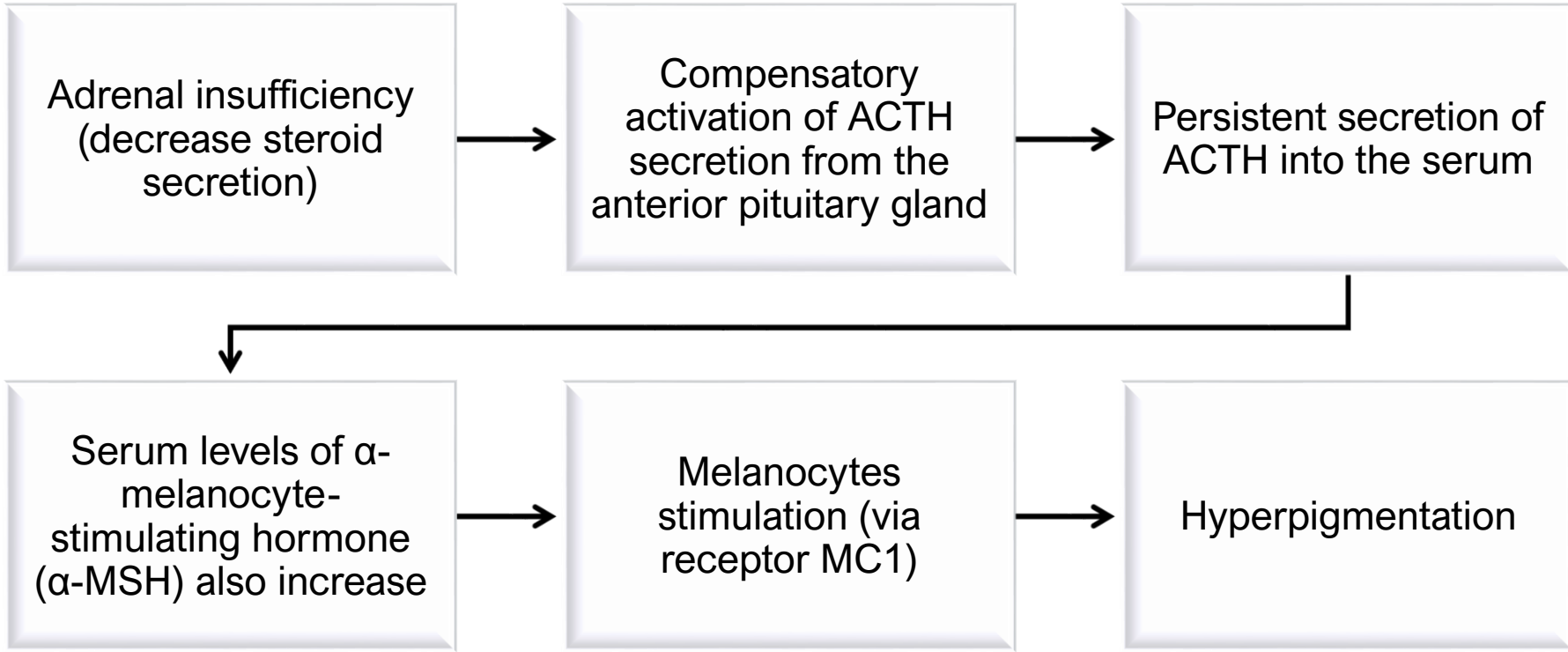
MELANOSIS ASSOCIATED WITH SYSTEMIC OR GENETIC DISEASE

Hypoadrenocorticism (Adrenal Insufficiency or Addison's Disease)

- **Etiology**
- Decrease in endogenous corticosteroid levels
- Infectious agents, neoplasia, trauma, certain medications, and iatrogenic causes may lead to adrenal destruction or an impairment of endogenous steroid production



- **Clinical Features**
- Weakness, poorly defined fatigue, depression
- First sign of disease- Mucocutaneous hyperpigmentation
- Macules diffusely on the tongue, gingiva, buccal mucosa, and hard palate



Mechanism Underlying Hyperpigmentation In Addison's Disease

Diagnosis

- Oral biopsy
- Laboratory tests
- Serum cortisol levels of less than 100 nmol/L at 9:00 a.m. is a diagnostic of deficiency
- Hyponatremia, hyperkalaemia are frequently associated with adrenal insufficiency

Differential Diagnosis

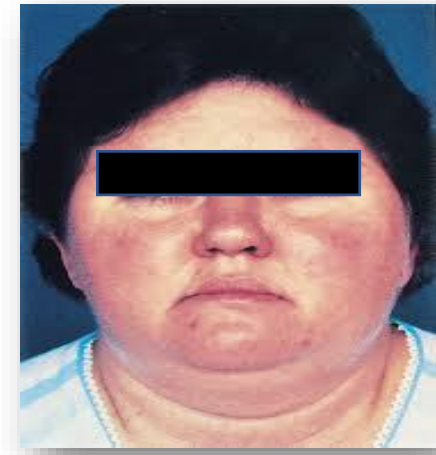
- Physiologic pigmentation ,Drug-induced pigmentation

Treatment

- Exogenous steroid replacement therapy with glucocorticoids and mineralocorticoids
- Dehydroepiandrosterone to improve the quality of life of patients with Addison's disease
- With appropriate therapy, the pigmentation will eventually resolve

Cushing's Syndrome/Cushing's Disease

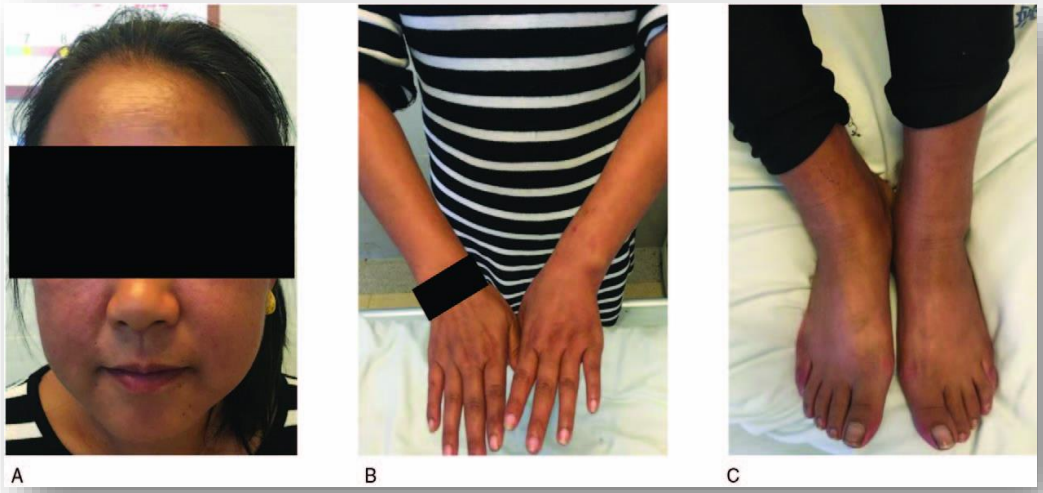
- Excessive ACTH production from a pituitary tumor → Excessive MSH in → conjunction hyperpigmentation
- **Clinical Features**
- More prevalent in female, Prepubertal - in boys
- Weight gain and the characteristic "moon facies"
- Diffuse Mucocutaneous pigmentation
- Primary pituitary neoplasm
- **Three main tests**
- Low-dose dexamethasone suppression test, Midnight plasma cortisol, 24-hour urinary free cortisol
- **Treatment**
- Surgical radiation, or drug therapy for the specific source of the endocrinopathy
- Pasireotide (a somatostatin analogue) has been approved for the treatment of Cushing's syndrome



Moon facies

Hyperthyroidism (Graves' Disease)

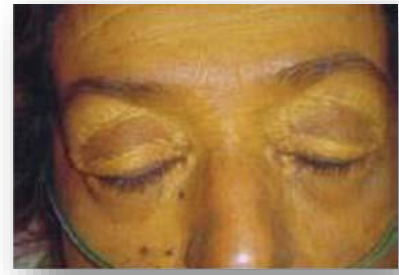
- Melanosis, dark-skinned individuals
- Excessive thyroid activity stimulates melanin synthesis remains unclear
- Brown diffuse pigmentation or may be localized to the face, neck and palmar creases
- No mucosal or genital hyperpigmentation in contrast to addison's disease
- Pigmentation tends to resolve following treatment of the thyroid abnormality



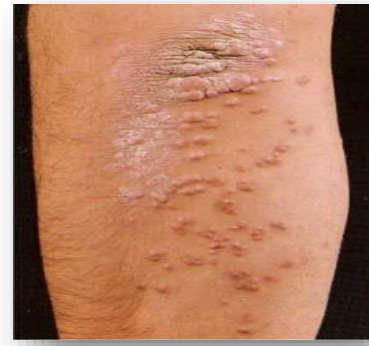
Hyperpigmentation especially on the face, upper limbs as well as lower extremities

Primary Biliary Cirrhosis

- Middle-aged women
- Results from damage to small intrahepatic bile ducts
- Jaundice usually an end-stage complication
- Excessive levels of serum bilirubin (a breakdown product of haemoglobin)
- Induces a yellowish discoloration of the skin, eyes, and mucous membranes
- Treatment of the underlying disease will lead to resolution of jaundice
- **Differential Diagnosis**
- Carotenemia (excessive β –carotene levels) , Lycopenemia (excessive lycopene)
- However, the oral mucosal tissues are not affected in either of these latter conditions



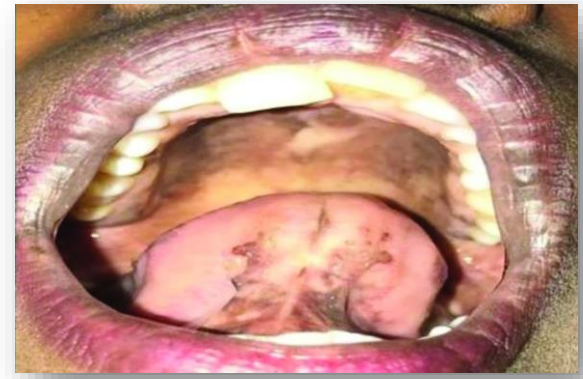
Bilateral xanthelasmata of upper eyelids



Papular eruptive xanthomas–multiple, discrete, papules becoming confluent At the level of elbow

Vitamin B12 (Cobalamin) Deficiency

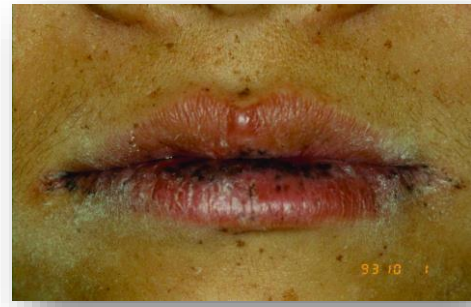
- Megaloblastic anemia
- Various neurologic signs and symptoms(vision problems, paraesthesia, memory loss)
- Various cutaneous and oral manifestations
- Generalized burning sensation
- Erythema
- Atrophy of the mucosal tissue
- Diffuse Mucocutaneous hyperpigmentation
- Mechanisms by which melanosis develops are unknown
- Pigmentation resolves following restoration of vitamin B12 levels



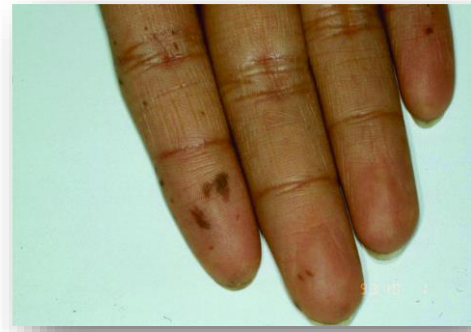
Peutz–Jeghers Syndrome

- Autosomal dominant disease
- Mutations in the STK11/LKB1 tumor suppressor gene
- Clinical manifestations include
- **<0.5 cm in diameter, multiple, pigmented macules of the lips, perioral skin, hands, feet**
- **Intestinal polyposis**
- **Cancer susceptibility**

- Other genetic diseases associated with triad of Gastrointestinal disease, Cancer susceptibility, Mucocutaneous pigmented macules include:
- **Cowden syndrome**, Cronkhite–Canada syndrome



Multiple small macules and patches with a perioral distribution in an 11-year-old male with Peutz–Jeghers syndrome.



Multiple pigmented macules on the fingertips in in an 11-year-old male with Peutz–Jeghers syndrome.

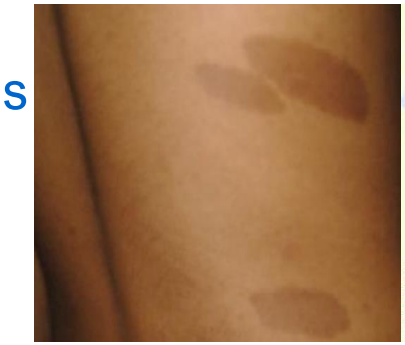
Café au Lait Pigmentation

- Solitary- Idiopathic café au lait **("coffee with milk") spots**
- Multiple -Genetic disorder
- Tan- or brown-coloured, **irregularly shaped macules of variable size**
- May occur anywhere on the skin

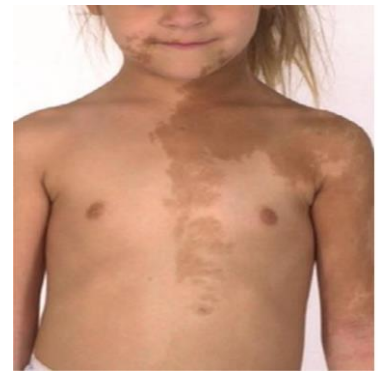
TABLE 6-6 Diseases Commonly Associated With Café au Lait Pigmentation

Ataxia-telangiectasia
Familial café au lait spots
Familial cavernous malformation
Fanconi's anemia
Hereditary nonpolyposis colorectal cancer
Idiopathic epilepsy
Johanson–Blizzard syndrome
McCune–Albright syndrome
Microcephalic osteodysplastic primordial dwarfism
Neurofibromatosis type 1
Neurofibromatosis type 1/Noonan's syndrome
Neurofibromatosis type 2
Nijmegen breakage syndrome
Noonan's syndrome
Ring chromosome 7 syndrome
Ring chromosome 11 syndrome
Ring chromosome 12 syndrome
Ring chromosome 15 syndrome
Ring chromosome 17 syndrome
Russell–Silver syndrome
Tuberous sclerosis
Turcot's syndrome

- Two main types
- **Coast of California**
- Regular and clearly demarcated margins which is more common
- Size from a few millimetres to several centimetres (>20cm)
- Solitary or multiple spots
- Seen neurofibromatosis 1 (NF1)

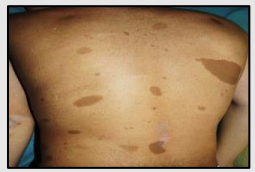


- **Coast of Maine**
- Second type of CALM
- Irregular margin, which is less common
- Larger and solitary
- Seen in a segmental pigmentary disorder

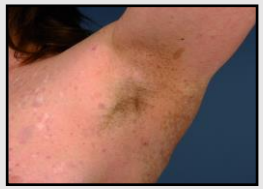


NF 1:

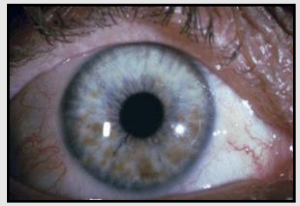
- Multiple neurofibromas
- **Cutaneous café au lait spots**



- **Axillary and/ or inguinal freckling (Crowe's sign)**

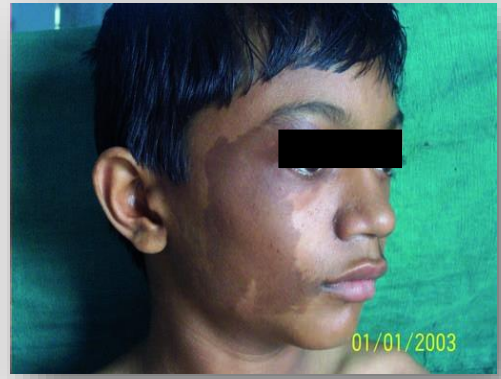


- **Pigmented lesions of the iris (Lisch nodules)**



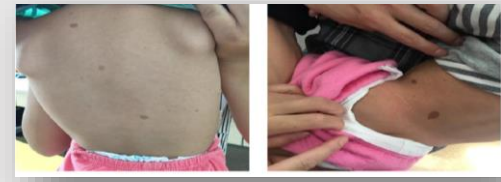
Mccune–albright syndrome

- Polyostotic fibrous dysplasia
- Various endocrinopathies
- Soft tissue myxomas (mazabraud disease)
- **Borders of the pigmented macules are irregularly** outlined, whereas in nf1, the borders are typically smooth

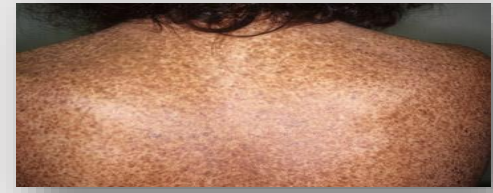


Noonan's syndrome & LEOPARD syndrome

- Café au lait spots are more characteristically seen in patients with the Noonan's phenotype

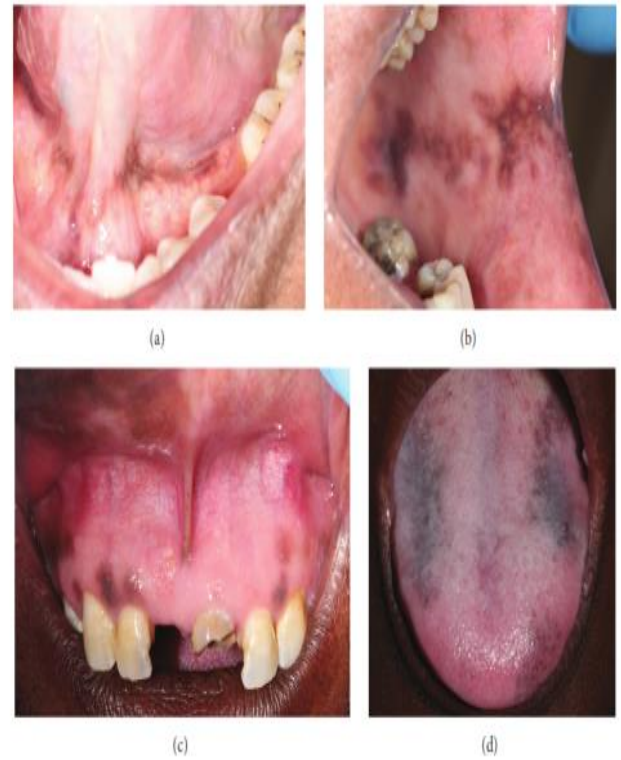


- LEOPARD phenotype: numerous, small, freckle-like macules often involving the facial skin



HIV/AIDS-Associated Melanosis

- Late-stage manifestation- diffuse or multifocal Mucocutaneous pigmentation
- Antifungal and antiretroviral drugs as a result of adrenocortical destruction by virulent infectious organisms
- Mucocutaneous pigment and CD4 counts cells/µlf200
- **Increased secretion of α-MSH from the anterior pituitary gland, which may also stimulate increased melanin synthesis**
- Skin, nails, and mucous membranes involvement
- Buccal mucosa > gingiva, palate, and tongue



Melanin hyperpigmentation of the floor of the mouth, buccal mucosa , gingiva, tongue in HIV-seropositive patient

CCES MCQS

Acquired, symmetric hyper pigmentation of the sun exposed skin of the face & neck which is strongly associated with pregnancy & use of oral contraceptives is called as_____?

A. Cafe-au-lait-spots
B. Melanoma
C. Freckle
D. Melasma

Cafe-au-lait spots on the skin are characteristic of_____?

A. Peutz-Jeghers syndorme
B. Addision's disease
C. Von recklinghausen disease
D. Hyper pituitarism

Disease which increase oral melanin pigmentation_____?

A. Addison's disease
B. Nephritis
C. Hyperthyroidsim
D. All of the above

CCES MCQS

The most common intraoral location for a pigmented nevi is the_____?

A. Hard palate
B. Buccal mucosa
C. Soft palate
D. Floor of mouth

Melanin pigmentation in pregnancy is known as_____?

A. Melasma
B. Epulis
C. Melanoma
D. Melanosis

References

- BURKET'S ORAL MEDICINE 13TH EDITION
- PEEYUSH SHIVHARE, TEXTBOOK OF ORAL MEDICINE AND ORAL RADIOLOGY
- MARCO MELETI ET AL , PIGMENTED LESIONS OF THE ORAL MUCOSA AND PERIORAL TISSUES, OOOOE, MAY 2008
- CRAIG L. HATCH, PIGMENTED LESIONS OF THE ORAL CAVITY, 2005 DOI:10.1016/J.CDEN.2004.07.013
- SREEJA C, RAMAKRISHNAN K, VIJAYALAKSHMI D, DEVI M, AESHA I, VIJAYABANU B. ORAL PIGMENTATION: A REVIEW. J PHARM BIOALL SCI 2015;7:S403-8.
- DR. RESHNA ROY, CLASSIFICATION OF ORAL PIGMENTED LESIONS: A REVIEW, INTERNATIONAL JOURNAL OF APPLIED DENTAL SCIENCES 2019
- FAIZAN ALAWI, ET AL, PIGMENTED LESIONS OF THE ORAL CAVITY: AN UPDATE, DOI:10.1016/J.CDEN.2013.07.006.

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THANK YOU