Issue - 12

BIANUAL PUBLICATION OF THE BANGALORE DERMATOLOGY SOCIETY

IADVL: BDS BULLETIN

BANGALORE DERMATOLOGICAL SOCIETY SINCE 1998

2020

"DERMADRISHTI"

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Executive Editor - **Dr. Eswari .L**

BANGALORE DERMATOLOGICAL SOCIETY
BANGALORE,KARNATAKA
www.bdsmembers.com

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A note from the Presidential desk:

We are happy to continue with the BDS newsletter-“DERMADRISHTI”, which covers all aspects of the activities of our Bangalore Dermatological Society, a vibrant branch of the IADVL. This news letter is brought out biannual by the editorial team comprising of Dr. Umashankar Nagaraju and Dr. Eswari. I wish all the success to this academic activity and request all the members of BDS to contribute actively to the journal. This is an opportunity for young dermatologists and postgraduates to showcase their talent.

I wish Dr. Umashankar Nagaraju and Dr. Eswari all the very best to take this journal to greater heights.

Dr. Leelavathy B

President

Bangalore Dermatological Society

Presidential Message

It is with immense pleasure that I accepted the role of the Presidentship of Bangalore Dermatological Society for the year 2019- 2021.

First of all let me thank, all my senior colleagues and all the members of the BDS for having trust in me to hold this prestigious post and also to host the academically mind boggling CME's, workshops, conferences, etc. I also thank all my colleagues and executive committee members for their support and encouragement.

To continue the good work of all my predecessors, I would like to enhance our goals.

Integrity

1. To enhance professional cohesiveness & to encourage the spirit of fraternity and the collegial spirit

Academics

3. To provide a platform for the upgradation of professional information, standards and skills
4. To guard and enhance professional and ethical standards of the members and thereby enhance the honor of the profession.

Digital

5. To make BDS completely paperless. All communication will be through mails, web site, messages, whatsapp and through other digital media.

Rural camps

6. To improve quality of life of people living in the rural areas through camps and awareness .

Memberships

7. The strength of any organization lies in the membership. So to see to it that every practicing dermatologist in Bangalore becomes a BDS member

CME's

8. To make every CME unique and worth the change.

Postgraduates

9. To encourage every PG student to participate , present cases and papers, compete, research and discuss and win laurels .

Unique programmes

11. To continue the tradition of introducing something unique in every term , eg. Capsules, Hand outs, Research grants etc .

Non dermatology talks

12. To introduce non dermatology talks for the benefit of all members, eg. investment, spirituality, fitness, etc to lead a better life

All this can be done only with the active participation, encouragement and support of all my team members, senior colleagues and the BDS fraternity .

I would like to thank all members for their continued co-operation and to help me make BDS successful, an important arm of IADVL.

Thank you and regards ,

Dr. Leelavathy B

President

Bangalore Dermatological Society

Message from the Secretary

Dear friends and colleagues,

BDS newsleter wishes you a hearty welcome. With the mission to promote specific areas of dermatology, this newsletter is dedicated to showcase the recent developments in our field. As science and medical research are progressing in a rapid pace, it is necessary for the clinicians to review and refresh their knowledge and skills.

I congratulate Dr. Eswari. L on bringing out the BDS news letter highlighting the activities and achievements of BDS. This will definitely serve as an inspiration for those involved in the BDS activities and motivation for those who want to be part of the same in these testing times of COVID.

Long live BDS,

Dr. Mahesh Kumar C
Secretary
Bangalore Dermatological Society

Chief Editor's message

This year, Bangalore Dermatological Society brought the dermatologists of Bangalore closer, as a family, whilst sparing no effort in nurturing our knowledge of the subject.

The BDS members and post graduates have benefitted a lot from the monthly meets, especially the online meetings which have been organized during this COVID era.

I invite contributions to our biannual BDS Bulletin "DERMADRISHTI", from one and all, including innovative ideas, original research and, of course, the occasional, but essential write up that is the perfect amalgam of humour and subject matter. It is the perfect platform for the junior residents to gain visibility and hone their writing skills. I, especially, encourage them to contribute to this magazine.

I would like to sincerely thank the President, the Secretary and EC of BDS, for giving me the opportunity of being the editor of DERMADRISHTI.

Dr. Umashankar Nagaraju
Chief Editor
Bangalore Dermatological Society

Executive Editor's message

Bangalore Dermatological Society is a close knit group of vibrant dermatologists. The IADVL BDS bulletin "DERMADRISHTI" was a concept of Dr. Prabhakar M Sangolli. Eleven issues have been brought out so far.

This bulletin is a platform which unearths scientific and writing talents amongst dermatologists young and old.

I am happy to be of service as the executive editor to the best of my ability under the able guidance of our Chief Editor Dr. Umashankar Nagaraju and past Editor Dr. Leelavathy B

We are happy in bringing out the 11th issue of our bulletin and we hope that with continued patronage of all of you we would be able to index the journal in the near future.

I thank our president Dr. Leelavathy B, Secretary Dr. Mahesh C and all our executive committee members for helping me to serve our Bangalore Dermatological Society.

I wish that this bulletin will reach greater heights in future.

Dr. Eswari L, MD DVL, FRGUHS, FAADV
Executive Editor
Bangalore Dermatological Society

INDEX

CONTENTS:

Facial Moles and Blemishes

Dr. Eswari .L

Face is considered the index of mind and well being. Self regard and confidence is largely dependent on an attractive facial appearance. Any facial imperfection is considered a blemish and is a cause for concern. People solicit treatment and go to any extent to have spot less skin. Face is exposed to the vagaries and vicissitudes of climate and chemicals in the environment and hence is prone to various exposure related dermatoses.

In this chapter, moles and blemishes of the facial skin will be discussed. It can be broadly classified as follows

Table 1: Classification of Moles and Blemishes on face

Sun exposure induced lesions Freckles Actinic keratoses Solar comedones	**Seborrheic diathesis** Acne Acne scars Rhinophyma	**Pimentary disorders** Lentigenes Melasma Periorbital melanosis	**Benign epidermal lesions** Dermatoses papulosa nigra Seborrheic keratosis Kerato acanthoma Epidermoid cyst Dermoid cyst Milia	**Benign appendageal origin tumors** Syringoma Trichoepithelioma Steatocystoma
Benign neural crest origin tumors Melanocytic nevi Nevus of ota Neurofibroma	**Benign Mesenchymal origin tumor** Xanthelasma Soft fibroma/ acrochordon Lipoma Pyogenic granuloma Hemangioma	**Malignant tumors** Actinic keratoses Basal cell carcinoma Squamous cell carcinoma		

Sun Exposure Induced Lesions:

Freckles/Ephelis

Freckles are small (<0.5 cm) brown macules that occur profusely on the sun-exposed skin of the face, neck, shoulders and backs of the hands. They become prominent in summer and fade during winter.

Etiopathogenesis:

Hyperpigmentation in freckles is due to increased photo-induced melanogenesis and transfer of melanosomes from melanocytes to keratinocytes.

Clinical features: (Figure 1.a, 1.b)

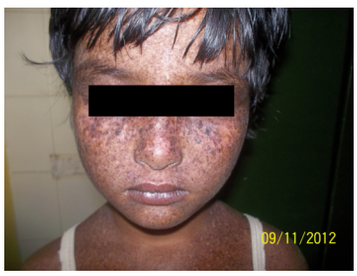

Figure 1.a: Multiple freckles seen on sun exposed areas in a girl with xeroderma pigmentosa

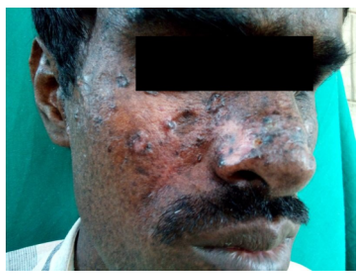

Figure 1.b: Multiple freckles and basal cell carcinoma lesions in a 40 year male with xeroderma pigmentosa

They are found on the sun exposed areas of the body like face, dorsal aspects of arm, upper chest and back. They are well demarcated, round, oval or irregular in shape measuring less than 3mm in diameter. They are benign lesions showing no malignant transformation. It has been found that presence of freckles increases the risk of melanoma.

Histopathology:
Increased melanin content is seen in the basal layer. The number of melanocytes are normal.

Differential diagnosis:
Lentigenes: Found on all areas of the body including mucosal surfaces
Café-au- lait macules: Larger size
Seborrheic keratosis
Junctional nevi

Management:
No treatment is needed as they are benign lesions. Sunscreens should be used to prevent the photoinduced damage. Hydroquinone and retinoids may be of some help. Pulsed dye lasers can be used to remove pigmentation.

Actinic keratoses
Also knows as solar keratosis, they are hyperkeratotic lesions occurring on chronically sun exposed lesions with a very low risk of progression to invasive squamous cell carcinoma (SCC).

Etiology:
Actinic keratoses occur on sun-exposed sites in fair-skinned people who have had excessive exposure to solar UV radiation[2]. Ionizing radiation or radiant heat is also implicated. It is also seen in workers exposed to pitch and other products of coal distillation.

Clinical features
They are found chiefly on the chronically sun exposed surfaces of the face, ears, balding scalp, dorsal hands, and forearms. They are usually multiple, discrete, flat or elevated, verrucous or keratotic, red, pigmented, or skin-colored. Usually, the surface is covered by an adherent scale, but sometimes it is smooth and shiny. The patient may complain of tenderness when the lesion is rubbed or shaved over with a razor. The lesions are usually relatively small, measuring 3 mm to 1 cm in diameter. The hypertrophic type, which may lead to cutaneous horn formation, is most frequently present on the dorsal forearms and hands.

Clinical pointers favoring the diagnosis of an early invasive SCC include: the presence of tenderness, induration or a raised shoulder that extends beyond the area of disorganized scaling[3].

Histopathology:
Actinic keratosis fulfills histopathologic criteria for an early squamous cell carcinoma[4] because:

(1) It is made up of epithelial cells that have crowded, large and pleomorphic nuclei, some of which are in mitosis,
(2) Its cells have an acidophilic cytoplasm in sections of tissue stained by hematoxylin and eosin
(3) Epithelial cells show signs of faulty cornification in the form of dyskeratotic cells and parakeratosis

Differential diagnosis
Discoid lupus erythematosus.
Lichen planus.
Seborrhoeic keratosis,
Bowen's disease
Management
Since it is considered an early stage of SCC, removal or destruction is the treatment of choice.
Curettage or shave excision, cryotherapy, ablative laser systems such as Carbon dioxide laser or Erbium:YAG laser. Topical treatment with 5-fluorouracil and combination of 5-fluorouracil with isotretinoin ordiclofenac have given good results. Imiquimod is an immune response modifier that activates pro-inflammatory cytokines. Administered three times a week overnight, it induces an inflammatory reaction that may cause complete resolution of actinic keratoses[5].Photodynamic therapy with methyl-(5-amino-4-oxopentanoate) aminolevulinate cream and red light is another treatment modality[6]

Solar comedones

Nodular elastoidosis with cysts and comedones or Favre-Racouchot Syndrome (FRS) is a peculiar complication of solar (se¬nile) degeneration of the skin found most commonly in the periorbital region of elderly individuals[7].
Etiology:
It is found pre¬dominantly in white men who have had ex¬tensive exposure to ultraviolet light and other physical climatic agents.
Clinical features (Figure 2)

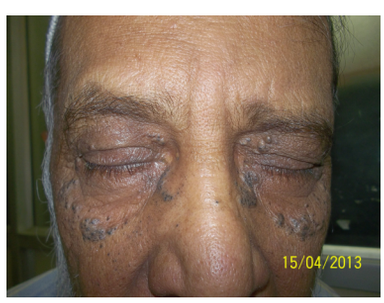

Figure 2: Senile comedones, follicular cysts, solar elastosis around the periorbital areas in a 68 year old male

The clinical presentation is that of multiple comedones, follicular cysts, furrows, and yellowish nodules superimposed on a sun damaged skin, usually symmetrically affecting the periorbital areas and the cheeks; occasionally the lesions are unilateral[8].
Histopathology[9]:
There is atrophy of the epidermis with mas¬sive basophilic (actinic) degeneration of the connective tissue in the upper dermis. Elastic tissue stains show nodular aggregates of elastotic material in areas

where degeneration is most pronounced. The infundibula of the pilosebaceous units are dilated to form keratin-filled comedones and follicular cysts.
Differential diagnosis:
Pseudoxanthoma elasticum
Basal cell carcinoma.
Management:
Acne peeling lotions, comedone extraction, dermabrasion, curettage can be tried though with limited benefits. Topical application of tretinoin gives better results.

Lesions on the face due to seborrheic diathesis

Acne Vulgaris and acne scars

Acne is a chronic inflammatory disease of the pilosebaceous unit.
Etiopathogenesis
Usually starts in adolescence with some genetic predisposition and hormonal influence. Four major factors are involved in the pathogenesis:
(i) Increased sebum production,
(ii) Hypercornification of the pilosebaceousduct,
(iii) Abnormality of the microbial flora especially colonization of the duct with P. acnes
(iv) Inflammation.
Clinical features(Figures 3.a, 3.b, 3.c, 4.a,4.b)

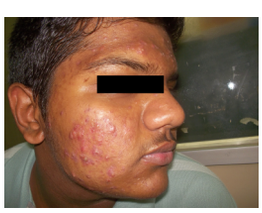

Figure 3.a: Acne vulgaris- comedones, papules, pustules and nodules in a male of 21 years

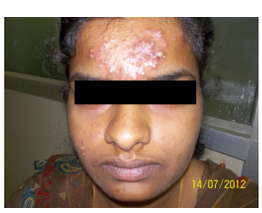

Figure 3.b: Steroid induced pustular acne on forehead in a 19 year old female

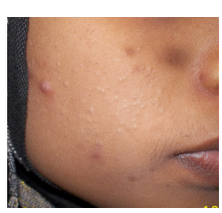

Figure 3.c: Multiple closed comedones in a 16 year old female

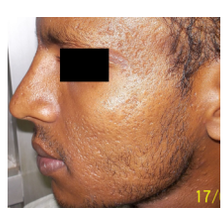

Figure 4.a: Acne scars- ice pick scars, rolling scars and boxcar scars in a 25 year old male

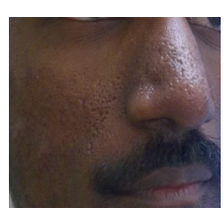

Figure 4.b: Acne scars- predominantly ice pick scars

Acne is a polymorphic, inflammatory disease of the skin which occurs most commonly on the face (in 99% of cases) and to a lesser extent on the back (60%) and chest (15%)[10].
Distribution of acne lesions in different age groups[11]: Table 2.

Table 2: Distribution of acne lesions in different age groups

Age groups	Sex	Type of lesions	Site of distribution
Neonates	Both	Papules and pustules, no comedones	Cheeks, chin, eyelids, forehead
Infants	Male	Comedones, papules, nodules, scars	Full face
Preadolescent	Both	Forehead, upper cheeks, nose	Forehead, upper cheeks, nose
Adolescent	Both	All types of lesions	Full face, seborrheic areas of torso
Adults	Females	Papules, excoriated papules	Chin, upper lip, jaws

Acne shows a variable and fluctuating mix of comedones, folliculocentric inflammatory lesions, scars, and pigmentary disturbances. The full spectrum includes many types of comedones, inflammatory lesions and acne scars[11]. Different morphology of lesions in acne is given in **Table 3.**

Table 3: Different morphology of lesions in acne

Comedones	Inflammatory lesions	Acne scars
Open comedones (blackheads) Closed comedones (whiteheads) Grouped comedones Missed comedones Macrocomedones Sandpaper comedones Submarine comedones Nevoid comedones Drug-induced comedones	Papules Pustules Nodules Cysts	Atrophic scars • Ice-pick scars • Boxcar scars • Rolling scars • Perifollicular elastolysis • Macular atrophic scars Hypertrophic scars • Keloids • Papular scars • Perifollicular fibrosis Mixed scars Unclassifiable scars

Grading of acne[11] is given in **table 4.**

Table 4: IAA guidelines of acne severity

Mild acne (Grade I)	Predominance of comedones	Comedones < 30 Papules < 10 No scarring
Moderate acne (Grade II)	Predominance of papules	Comedones any number Papules > 10 Nodules < 3 Scarring ±
Severe acne (Grade III)	Many nodules	Comedones any number Papules any number Nodules/ cysts > 3 Scarring +

Differential diagnosis
Rosacea
Perioral dermatitis
Lupus miliaris disseminatus facei (previously called acne agminata)
Papular sarcoidosis
Folliculitides (pityrosporum, candidal, demodex, staphylococcal, Gram negative, herpetic)
Adenoma sebaceum
Verruca plana
Molluscum contagiosum
Management
Topical therapy
Topical therapy is useful in mild and moderate acne, as monotherapy or as combination therapy.

Benzoyl peroxide: Available in different formulations (washes, lotions, creams, and gels) and concentrations (2.5-10%). Benzoyl peroxide is a broad spectrum bactericidal agent which is effective due to its oxidizing activity. The drug has an anti-inflammatory, keratolytic, and comedolytic activities, and is indicated in mild-to-moderate acne vulgaris.
The main adverse effects are cutaneous irritation or dryness and bleaching of clothes, hair, and bed linen.
Topical retinoids: Topical retinoid should be used as the first-line therapy, alone or in combination, and is also a preferred agent for maintenance therapy. It targets the abnormal follicular epithelial hyperproliferation, reduces follicular plugging and reduces microcomedones and both noninflammatory and inflammatory acne lesions. Tretinoin, adapalene, tazarotene, isotretinoin, are currently available topical retinoids. The main adverse effects are primary irritant dermatitis, which can present as erythema, scaling, burning sensation.

Topical antibiotics:
They inhibit the growth of P. acne and reduce inflammation. Topical antibiotics such as erythromycin and clindamycin are the most popular. Side effects though minor includes erythema, peeling, itching, dryness, and burning. To avoid development of bacterial resistance, antibiotics should not be used as monotherapy.

Other topical/new agents:
Combination therapy: Benzoyl peroxide combined with topical erythromycin or clindamycin, combination of topical retinoid and topical antimicrobial.
Azelaic acid: It is available as 10-20% topical cream which has been shown to be effective in inflammatory and comedonal acne and also reduces post inflammatory hyperpigmentation.
Systemic antibiotics
Oral antibiotics are indicated in moderate-to-severe inflammatory acne. Tetracyclines, macrolides, co-trimoxazole and trimethoprim are other alternatives for acne. Concomitant use of oral and topical therapy with chemically dissimilar antibiotics is to be avoided.

Oral isotretinoin
Oral retinoid is indicated in moderate-to-severe acne. It is the only drug that affects all four pathogenic factors implicated in the etiology of acne. The approved dose is 0.5-2 mg/kg/day, which is usually given for 20 weeks. Side effects include those of musculoskeletal, mucocutaneous, and ophthalmic systems, as

well as headache, and central nervous system effects. Oral isotretinoin is a potent teratogen.
Other physical modalities like, comedone extraction, photodynamic therapy, chemical peeling and lasers can be tried to treat active lesions as well as acne scars.

Rhinophyma

The phymas are localized swellings of facial soft tissues due to variable combinations of fibrosis, sebaceous hyperplasia and lymphoedema[12].

Etiology :

In many cases rhinophyma develops in patients with a long history of rosacea and it is often regarded as a complication or 'end stage' of the disease. Recurrent and fleeting vasodilatation of the face causes soft-tissue nasal hypertrophy, gradually giving rise to rhinophyma. Alcoholism, hormones like steroids and androgens, microorganisms like Demodex folliculorum, and sunlight play a role in progression of the condition.

Clinical features: (Figure 5)

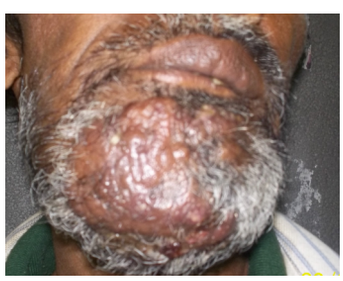

Figure 5:
Gnathostomia: nodular hypertrophy of the soft tissue of the jaw area

They develop predominantly in males. Other areas which may be affected include the forehead (metophyma), chin (gnathophyma), eyelids (blepharophyma) and ears(otophyma)[13]. The peculiar morphologic characteristics of rhinophyma are telangiectasia, hypervascularity, a thick nasal cutaneous layer and nodularity covered by atrophic skin with expanded pores.

Histopathology

Massive hyperplasia of sebaceous glands, dermal elastosis, moderate fibrosis in a myxoid edematous stroma, follicular cysts and perivascular lympho-histocytic infiltration are the characteristic features[14].

Management

Surgical paring of excess tissue, dermabrasion, electrocautery, Co2 laser, ionizing radiation are the different available options.

Pigmentary disorders

Lentigenes: `

It is derived from Latin word 'lentil', comparing the size of beans to the size of lesions.
Lentigo is a benign discrete hyperpigmented macule appearing at any age and on any part of the body, including the mucosa.

Etiopathogenesis :

The intensity of the color is not dependent on sun exposure. The solar lentigo appears at a later age, mostly in persons with long-term sun exposure. The backs of the hands and face(especially the forehead) are favored sites. They may also occur as part of syndromes.

Clinical features: (figures 6.a, 6.b, 6.c)

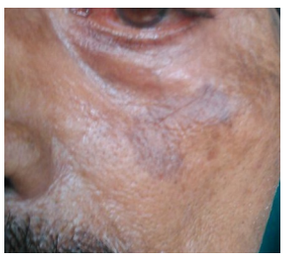

Figure 6.a: Solar lentigo: Hyperpigmented macule on the left side of cheek

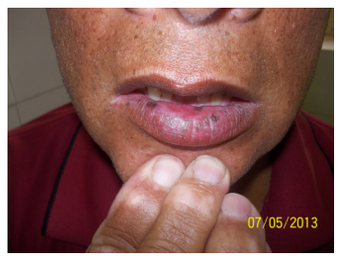

Figure 6.b: Multiple lentigenes including the buccal mucosa

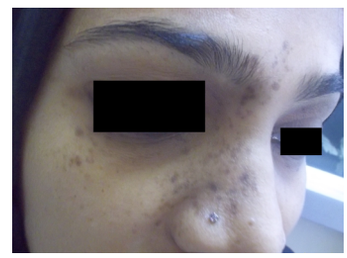

Figure 6.c: Segmental lentigenes

Lentigo simplex is the most common type which may be acquired or congenital. They are usually isolated well defined lesions measuring less than 5mm in size. Solar lentigo increases with age and sun exposure. The various associated syndromes include Peutz–Jeghers syndrome, the leopard syndrome (lentigines, electrocardiogram anomalies, ocular anomalies, pulmonary stenosis, abnormal genitalia, retardation of growth and deafness)[15], the Carney complex and the closely related NAME and LAMB syndromes. Difference between lentigenes and freckles is given in **Table 5.**

Table : 5: Difference between Lentigenes and Freckles

Lentigenes	**Freckles**
No genetic predisposition	Common in fair skinned individuals
Not related to sun exposure	Found on sun exposed regions with seasonal variation
Mucosal surface involved	Mucosa not involved
Becomes darker in pregnancy	Not related to pregnancy
Well defined regular borders	Well demarcated but irregular borders
Number of melanocyes are increased	Number of melanocytes are normal
Elongated club shaped reteridges	Epidermis is normal
Associated with syndromes: LEOPARD, LAMB, NAME	Not associated with syndromes

Histopathology:
Increased number of melanocytes at the dermoepidermal junction and elongated club shaped rete ridges.

Treatment:
When there are multiple lesions, reassurance is the main management. When the condition occurs at early age, syndrome associations must be looked for. Very rarely there is risk of melanoma. Strict avoidance of sun is a must. Cryotherapy, topical retinoids, hydroquinone and lasers are effective in the treatment of solar lentigines.

Melasma
It is also known as mask of pregnancy or chloasma. It is the most common acquired hypermelanosis particularly in females.
Etiopathogenesis

Sun exposure, pregnancy, estrogen containing hormone replacement therapy and genetic predisposition are all implicated in the causation of melasma.

Clinical features (Figure 7.a, 7.b)

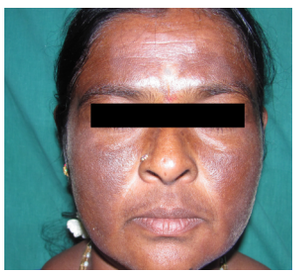

Figure 7.a: Melasma: Diffuse hyperpigmentation over forehead, malar area and chin

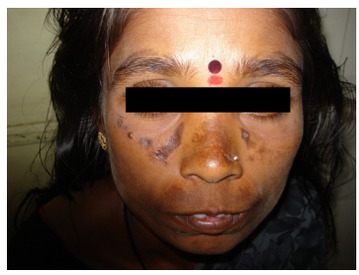

Figure 7.b: Melasma: well defined pigmented patches on the malar region, nose and upper lips

The hyperpigmented patches may range from single to multiple, usually symmetrical on the face and occasionally V-neck area. According to the distribution of lesions, three clinical patterns of melasma are recognized[16]. The centrofacial pattern is the most common and involves the forehead, cheeks, upper lip, nose, and chin. The malar pattern involves the cheeks and nose. The mandibular pattern involves the ramus of the mandible. Using the woods lamp the depth of pigmentation can be determined, and melasma is classified into epidermal, dermal and mixed type.

Histopathology

There is increased deposition of melanin in the epidermis, in the dermis within melanophages, or both. The number of melanocytes in the lesions is normal or increased. The melanosomes within the melanocytes and keratinocytes are increased in size[17].

Management

Sun avoidance and use of sunscreens is the main stay of management. The existing modalities which are used include hydroquinone, retinoic acid, kojic acid, azelaic acid, and peeling agents like glycolic, trichloroacetic acid, salicylic and lactic acid. Physical agents like lasers and dermabrasion have also been tried with limited success.

The most extensively used combination is the so-called 'triple combination', a formulation containing hydroquinone HQ, retinoic acid, and corticosteroids, proposed by Kligman and Willis, [55] the modified regimen includes HQ 4%, tretinoin 0.05%, and fluocinolone acetonide 0.01% and has been found to be highly effective. Many new topical agents like niacinamide, flavanoids and liquorice derivatives have been tried. Prolonged application of hydroquinone based combination cause exogenous ochronosis[18], a blue black pigmentation of the treated areas.

Periorbital melanosis

Periorbital melanosis (POM) (or dark circles) is of great cosmetic concern.

Etiology

Factors incriminated in etiology of POM include dermal melanin deposition, post inflammatory hyperpigmentation (atopic or contact allergic dermatitis), shadowing from lax skin, anemia, stress and infraorbital swelling. Familial periorbital hyper pigmentation is determined by an autosomal dominant gene[19] and in one study POM was found to be an extension of pigmentary demarcation lines over the face (PDL-F)[20]. Clinical features(Figure 8)

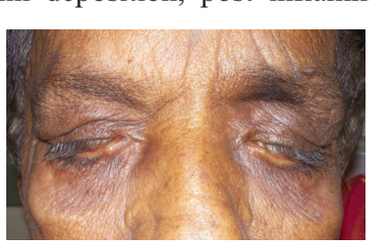

Figure 8: Periorbital melanosis

Periorbital hyperpigmentation is defined as bilateral, homogeneous hyperchromic macules and patches primarily involving the lower eyelids but also sometimes extending towards the upper eyelids, eyebrows, malar regions, temporal regions and lateral nasal root. The age of onset is usually after puberty or in early adulthood

Management

Correction of faulty habits and underlying causative factors go a long way in managing POM

Benign epidermal lesions

Dermatoses papulosa nigra

Dermatosis papulosa nigra (DPN) is a benign epithelial tumor that is common in dark-skinned people and females

Etiopathogenesis:

Lesions are genetically determined and more common in the blacks. They are due to naevoid developmental defect of pilosebaceous follicles. It is considered as a variant of seborrheic keratoses.

Clinical features[23]: (Figure 9)

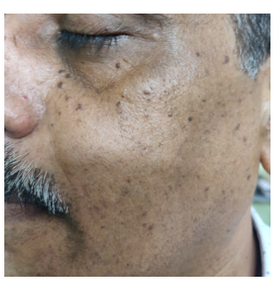

Figure 9:
Dermatosis Papulosa Nigra:
on the malar area

The individual lesions are black or dark brown, flattened or cupuliform papules 1–5 mm in diameter. They are most numerous in the malar regions and on the forehead.

Histopathology

The lesions show irregular acanthosis and hyperkeratosis, and somewhat resemble seborrhoeic keratoses.

Differential diagnosis

Seborrheic keratoses

Acrochordons

Melanocytic nevi

Lentigenes

Warts

Management

Snip excision with scissors, electrodessication and superficial cryotherapy are the common treatment modalities. It is generally treated for cosmetic concern.

Seborrheic keratoses

Seborrheic keratoses (SK) are the most common benign epidermal tumors composed of epidermal keratinocytes. The condition usually occurs in middle-aged individuals and can arise as early as adolescence

Etiopathogenesis:
Seborrheic keratoses are more common in areas of sun exposure. In about one-third or more of cases, solar lentigines and seborrheic keratoses both have gain-of-function mutations in FGFR3 and PI3K, the genes mutated in keratinocytic epidermal nevi. The rapid development of large numbers of SKs can occur in patients with an inflammatory dermatoses[24] or in association with underlying malignancies like stomach and colon cancer, where it is known as the sign of Leser–Trélat[25].
Clinical features (figure 10)

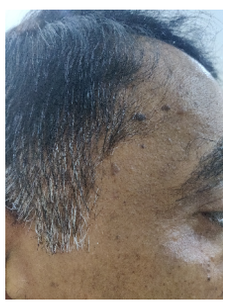

Figure 10: Seborrheic keratosis: Pigmented raised keratotic lesion with a stuck on appearance

Seborrheic keratoses are common and usually multiple. They present as oval, slightly raised, tan/light brown to black, sharply demarcated papules or plaques, rarely more than 3 cm in diameter. They appear "stuck on" the skin, as if they could be removed with the flick of a fingernail. They are located mostly on the chest and back, scalp, face, neck, and extremities. The surface of the warty lesions often becomes crumbly, like a crust that is loosely attached. When this is removed, a raw, moist base is revealed. Seborrheic keratoses may be associated with itching. Some patients have hundreds of these lesions on the trunk.

Histopathology:
Histologically, most seborrheic keratoses demonstrate acanthosis, varying degrees of papillomatosis, hyperkeratosis, and at times keratin accumulations within the acanthotic epidermis (pseudo-horn cysts). The epidermal cells lack cytologic atypia, except at times in the irritated variant where typical normal mitoses may occur. Six histologic types—hyperkeratotic,
acanthotic, adenoid or reticulated, clonal, irritated, and melanoacanthoma—are distinguished.
Differential diagnosis
Malignant lentigo
Actinic keratoses
Melanocytic nevus
Malignant melanoma
Management
Curettage, shave excision, cryosurgery, laser ablation are the various available treatment options.

Keratoacanthoma
It is also known as molluscum sebaceum. It is a rapidly developing benign tumor of the skin originating in pilosebaceous follicles.
Etiopathogenesis
Sun exposure, contact with mineral oil and coal tar, trauma and viral causes have been implicated.
Incidence increases with age and is more in males.
Clinical features (Figure 11)

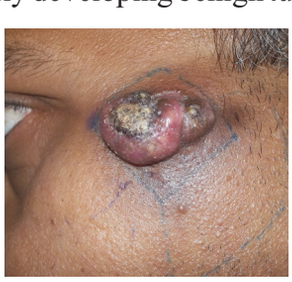

Figure 11: Keratoacanthoma : nodular lesion with central keratin filled crater in a 45 year old male

There are four types of keratoacanthoma: solitary, multiple, eruptive, and keratoacanthoma centrifugum marginatum. They begin as firm round skin or red colored papules and progress to dome shaped nodules with central crateriform ulcer or keratin plug that may project like a horn. They heal spontaneously over 4 to 6 months.

Histopathology[26]

The early lesion is composed of a mass of rapidly multiplying squamous cells. These are large and rather pale with vesicular nuclei, prominent nucleoli and frequent mitoses. Hyperchromatic cells, atypical mitotic figures, individual cell keratinization and other evidence of loss of polarity may be found. The marginal cells invade the surrounding dermis aggressively, while those more centrally placed keratinize to form a branched core of keratin that communicates with the surface. The stroma is vascular and infiltrated with round cells and histiocytes.

Differential diagnosis

Actinic keratoses

Cutaneous horn

Basal cell carcinoma

Verruca vulgaris

Management

Surgical excision is the main option. Systemic retinoids, bleomycin, intralesional methotrexate have been used. Laser therapy and cryotherapy are also successful.

Epidermoid cyst

It is commonly known as sebaceous cyst, which is a misnomer. It is a keratin cyst.

Etiopathogenesis

It occurs due to inflammation around a pilosebaceous follicle, may also result from deep implantation of a fragment of epidermis by a blunt penetrating injury.

Clinical features (Figure 12.a, 12.b)

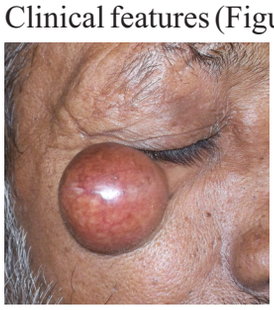

Figure 12.a: Epidermoid cyst: tense cyst with prominent visible vasculature

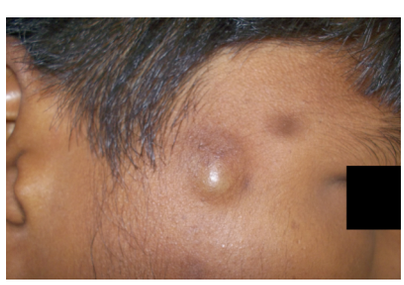

Figure 12.b: Sebaceous cyst with central punctum

It occurs as a firm nodular swelling, freely mobile over the underlying structures, and has a central keratin filled punctum. They enlarge slowly and may become inflamed and tender. Suppuration may occur.

Histopathology

The cysts are lined by an epidermis like epithelium including a granular cell layer. The cysts contain laminated keratin.

Differential diagnosis

Lipoma

Dermoid cyst

Neurofibroma

Pilar cyst

Management
Total excision of cyst is the treatment of choice.

Milia
It is a small subepidermal keratin cyst.

Etiopathogenesis
Milia are quite common at all ages. Usually arise in the proximal part of divided sweat ducts. The cause of the duct damage is usually avulsion accompanying an acute subepidermal
bulla, particularly in second-degree burns, epidermolysis bullosa, porphyria cutanea tarda and bullous lichen planus. They may also follow dermabrasion and occur in areas of chronic topical corticosteroid-induced atrophy.
Clinical features (Figure 13)

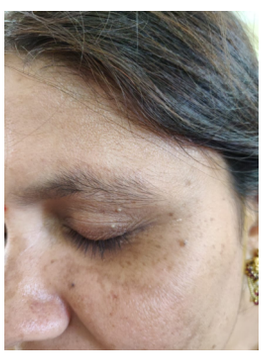

Figure 13: Milia: tiny yellow papule just beneath the epidermis, when slit open with a sharp needle, it releases a cheesy keratin material.

The lesions are firm, white or yellowish, rarely more than 1 or 2 mm in diameter and appear to be immediately beneath the epidermis. They are usually noticed only on the face, and occur in the areas of vellus hair follicles, on the cheeks and eyelids particularly.
Differential diagnosis
Syringoma
Trichoepithelioma
Management
They disappear spontaneously. Curettage with sharp needle or scalpel is usually effective.

Benign appendage origin tumors
Syringoma
These are benign sweat gland tumors which are usually multiple. Adult females are more affected
Clinical features (Figure 14.a, 14.b)

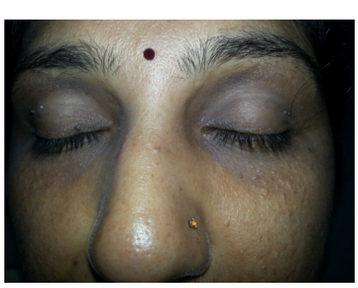

Figure 14.a: Syringoma: Multiple skin coloured translucent papules on both lower eyelids with periorbital melanosis and skin tags on upper eyelid in a 35 year old female

Figure 14.b: Syringoma: Multiple hyperpigmented papules with angulated borders on the lower eyelid

They are multiple skin colored papules occurring on face and neck. They vary in size from 1 to 5 mm and

contain clear fluid. They appear cystic and translucent with angular outline. Syringomas have also been reported during radiotherapy[27] for breast cancer

Histopathology
Syringoma consists of mature ductal structures lined by 2 layers of cuboidal cells. Epithelial elongation of the ducts into adjacent stroma gives a tadpole like appearance. The surrounding stroma is sclerotic.
Differential diagnosis
Plane warts
Milia
Xanthelasma
Management
Electrodessication, TCA application, cryotherapy are useful options. But recurrences are common.

Trichoepithelioma
It is also known as Brooke's tumor. It is hamartoma of the hair germ.

Etiology
Solitary lesion is due to deletions in the patched gene at 9q22. The gene for multiple trichoepitheliomas has been mapped to a locus on chromosome 9p21[28], and is inherited by autosomal dominant transmission. They are common in young adults.
Clinical features (Figure 15)

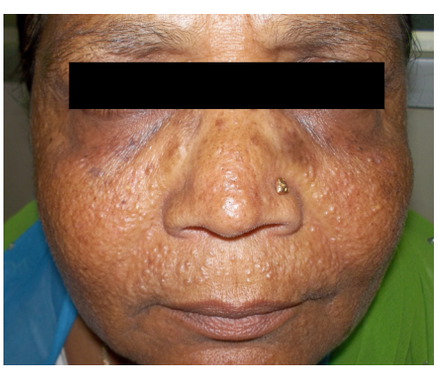

Figure 15: Trichoepithelioma: Multiple skin colored papules in central part of the face

Solitary lesion usually occurs on the face resembling a non ulcerated Basal cell carcinoma.
The presentation of a solitary lesion is that of a smooth nodule, usually on the face. Most affected patients are young adults. Multiple lesions are seen as small, pearly lesions, mainly on centrofacial skin.

Histopathology[29]
There are lobules of small, dark basaloid cells, often with a degree of peripheral palisading surrounding a central area of eosinophilic amorphous material. Occasionally, hair shaft-like structures can be seen in these central areas. A fibrous cellular stroma is seen around the cellular lobules.
Differential diagnosis
Basal cell carcinoma
Colloid millium
Steatocystoma multiplex
Management
Surgical excision, curettage, cryotherapy, dermabrasion and CO2 laser are effective treatment options

Steatocystoma

They occur as multiple cysts in the dermis having sebaceous gland lobules in their wall and containing sebum.

Etiopathogenesis

It has an autosomal dominant inheritance affecting adolescents and young adults.

Clinical features (Figure 16)

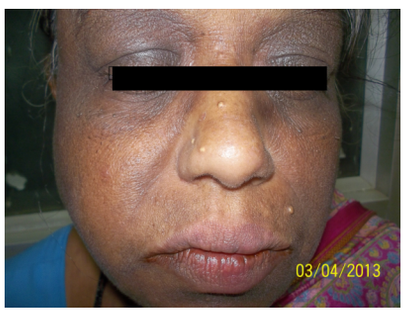

Figure 16: Steatocystoma: yellow colored cystic lesions on the nose and upper lip. Dermatosis papulosa nigra and melanosis is also seen in the periorbital regions

They occur as multiple smooth nodules varying in size, commonly occurring on the trunk and genitals. Sometimes they are seen on the forehead and malar areas of face. No punctum is seen. Oily fluid can be expressed out of the lesions.

Histopathology

The cysts are situated in mid dermis having sebaceous gland lobules in their walls and contain sebum, the wall has keratinized epithelium. They have oily content composed of unsplit esters of sebum.

Management

Done mainly for cosmetic reasons. Surgical excision for many lesions becomes impractical.

Benign neural crest origin tumors

Melanocytic nevi

They are commonly known as ‘moles’. Melanocytic nevi are benign proliferations of a type of melanocyte known as a "nevus cell." They have been broadly grouped into five types[30] **(table 6):**

Congenital melanocytic nevi	
Acquired melanocytic nevi	Junctional Compound Intradermal
Special variants	Spitz Nevus spilus
Dysplastic nevi	
Dermal melanocytic nevi.	

Etiopathogenesis

The emergence of nevi is under strong genetic control, whereas environmental exposures, like sun exposure, affect the mean number of nevi. Melanocytes have origin from neural crest.

Clinical features (Figure 17.a, 17.b, 17.c, 17.d)

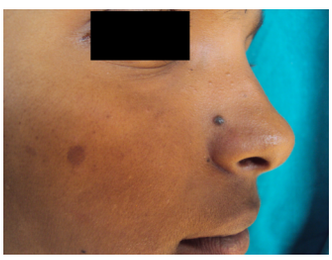

Figure 17.a: Acquired Melanocytic nevi: both junctional and compound nevi is seen in a 20 year old female

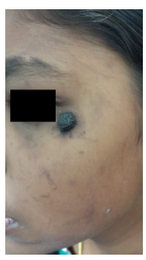

Figure 17.b: Compound melanocytic nevi

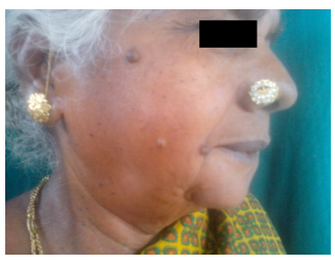

Figure 17.c: Dermal melanocytic nevi: multiple and skin colored in a 50 year old female.

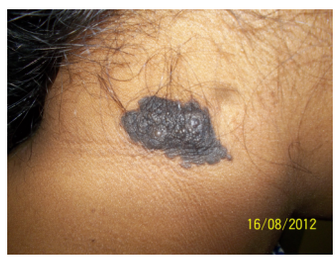

Figure 17.d: Dysplastic nevus

Congenital melanocytic nevi are present at birth as round to oval, fairly homogenous, brown, multishaded pigmented lesions with sharply demarcated border often with a mamillated surface and hypertrichosis. They vary in size from <1.5cm to >20 cm, commonly occurring on the trunk. Malignant transformation may occur rarely.

Acquired melanocytic nevi are more common and can be of junctional, compound or intradermal type. Junctional nevi appear as pigmented macules with well preserved surface markings. Compound nevi are raised circular plaques with a darker center and lighter periphery. Coarse hair may project from the surface. Intradermal nevi are dome shaped skin colored smooth papules.

Histopathology
Nevus cells are seen as single row (Indian File appearance), sheets or combinations in the collagen bundles of reticular dermis.
Differential diagnosis
Lentigenes
Freckles
Melanoma
Management
Treatment is dictated by cosmetic necessity. Surgical excision, shave excision or punch excision is often preferred, with variable rates of recurrence in each procedure. Laser ablation is also done. Careful follow up to identify malignant transformation is essential.

Nevus of Ota (NOO)
Also known as Oculodermal melanocytosis, it is more common in Japanese, mostly in women (9 times) with onset either in the perinatal period (50%) or around puberty (30%) [21].

Etiopathogenesis
Due to racial differences, genetic factors are thought to be important but familial cases are rare.
NOO represents aborted embryonic migration of melanocytes from neural crest to epidermis. Late pubertal onset is explained by pigmentation of the amelanotic nevoid cells present at birth by adolescent spurt of sex hormones.

Clinical features
NOO is characterized by speckled or mottled coalescing blue-grey pigmentation of the area supplied by ophthalmic and maxillary divisions of trigeminal nerve. It is usually unilateral (90%). In addition to skin,

pigmentation of NOO may involve oral mucosa and the eye (conjunctiva, sclera, retrobulbar fat, cornea, and retina). Based on the extent, NOO is classified into mild, moderate, severe and bilateral type[22]
Differential diagnosis
Hori nevus
Acquired bilateral nevus of Ota like macules (ABNOM)

Management
The lesions are usually asymptomatic but lifelong follow-up is required as a few cases of malignant melanoma have been reported in literature. Cosmetic camouflage, co2 snow with dermabrasion, argon laser, Q switched ruby laser, ND: YAG laser have given beneficial results.

Neurofibroma
They are tumors of neural origin, may occur as localized or diffuse lesion or as a part of neurofibromatosis syndrome.

Etiopathogenesis
Although it appears to be hamartomatous in nature, the demonstration of clonality suggests a neoplastic origin[31]. Neurofibromatosis is an autosomal dominant syndrome
Clinical features (Figure 18)

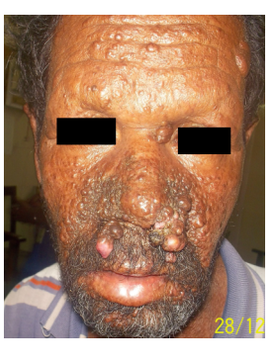

Figure 18:
Neurofibromatosis

They are soft painless tumors that can be pushed down into the subcutaneous fat, by light pressure with a finger ((button hole sign). They may occur as solitary lesion or multiple as in neurofibromatoses. Plexiform neurofibroma occurs as large nodules, giving a bag of worms feeling, with overlying hyperpigmented skin. Café-au-lait macules and axillary freckling are the other pathognomonic features seen in neurofibromatosis syndrome. Bilateral acoustic neuromas can also occur in type 2 neurofibromatosis.

Histopathology
Neurofibromas are well-circumscribed encapsulated, spindle cell proliferations with mucinous background and numerous mast cells.
Management
Planned surgical removal of disfiguring, plexiform lesions can be performed.

Benign Mesenchymal origin tumor
Xanthomas
The term xanthoma derives from the Greek ‘xanthos’ meaning yellow and is used to describe a variety of

subcutaneous lipid deposits. When they occur around eye they are known as Xanthelasmata.

Etiology

They are seen in familial hypercholesterolaemia, type III hyperlipoproteinaemia and in chronic cholestasis (especially primary biliary cirrhosis) [32]

Clinical features (Figure 19)

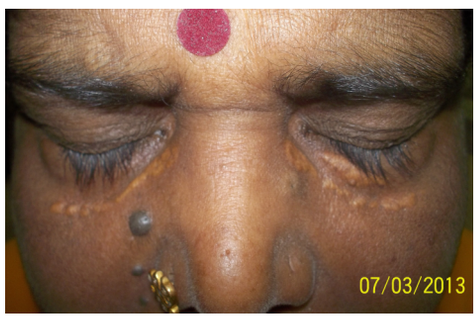

Figure 19:
Bilateral Xanthelasma palpebra

They usually occur around eyelids affecting upper eyelid and medial canthi initially. They are relatively soft to the touch and range from pale yellow to yellow-orange in colour.

Histopathology

Xanthomata contain macrophages loaded with cholesterol and cholesterol esters ('foam cells')

Management

They can be removed surgically, or by cauterization but often recur, particularly if LDL cholesterol levels are high. Cholesterol lowering agents like statins have to be given. Such treatment is often associated with regression of xanthelasmata in hypercholesterolaemic patients without the need for other interventions

Achrochordon

It is commonly known as skin tags or soft warts. They are most commonly seen in women at menopause or later.

Clinical features (Figure 20.a, 20.b)

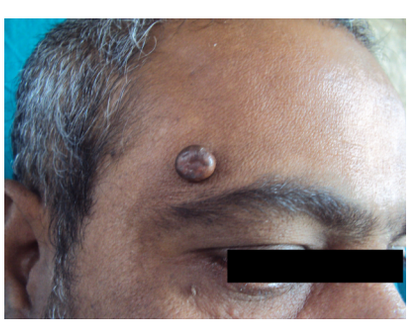

Figure 20.a:
Acrochordon: one large nodular lesion above the right eyebrow, and one small lesion on the lower eyelid.

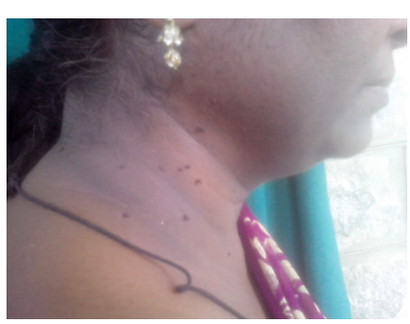

Figure 20.b:
Multiple Acrochordons on the neck

They are well circumscribed, soft, hyperpigmented lesions which may be sessile or pedunculated. They vary in size and are most commonly seen on sides of the neck and face. They are usually accompanied by seborrheic keratoses. Similar lesions may be found in and around the axillae and groins.

Histopathology
The epidermis is thin, and the basal cell layer is flat and often hyperpigmented. The bulk of the lesion is loose fibrous tissue, similar to that of the papillary dermis.
Differential diagnosis
Melanocytic nevi
Seborrheic keratoses
Warts
Management
Surgical removal, cautery, cryotherapy are the available effective options

Lipoma
Lipomas are benign tumors composed of mature fat cells. They are found in the subcutaneous tissue

Etiology
It has been demonstrated that loss of negative-feedback control regulatory enzymes (by citrate or phosphofructokinase) may be an early feature in the development of lipoma[33]

Clinical features
A lipoma is a subcutaneous nodule, often lobulated, with a characteristic soft, putty-like consistency. The overlying skin is normal and moves freely over the tumor. The most common sites are the neck, shoulders and upper arms, back and thighs. It may also occur on the face. There are rarely any subjective symptoms, but pain from pressure on the nerves is sometimes experienced.
Fat necrosis may cause enlargement, pain and tenderness.
They are an inconstant feature of Gardner's syndrome, in which they are associated with multiple sebaceouscysts, osteomas and polyposis of the colon. Multiple lipomas may also be a pointer to Proteus syndrome, Cowden's disease and Bannayan's syndrome.

Differential diagnosis
Sebaceous cyst

Management
If the patient desires, surgical excision is the treatment of choice.

Pyogenic granuloma
It is a lobular capillary hemangioma.

Etiology
It can occur in both sexes and is more common in the 2nd decade of life. Penetrative injury, burns, pregnancy have been implicated as causative factors. In pregnancy, mucosal lesions in oral cavity are common.
Clinical features (Figure 21)

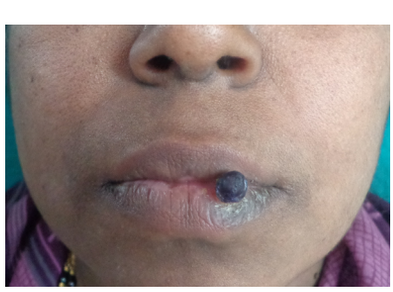

Figure 21:
Pyogenic granuloma on the lower lip with black crust due to repeated bleeding

It is a well circumscribed, partially compressible, bright red to bluish black in color and of varied sizes. The common sites are the hands, especially the fingers, feet, lips, head, upper trunk, and the mucosal surfaces of the mouth and perianal area.

Histopathology
Lobule of capillaries and venules separated by fibrous septa, epidermis forms a collaret at the base. Feeding vessels extend to adjacent dermis.
Differential diagnosis
Bacillary angioma
Inflamed seborrheic keratoses
Kaposis sarcoma
Glomus tumor
Management
Electrocautery, intralesional steroids or sclerosants, cryotherapy and lasers are used.

Hemangioma
Hemangiomas are benign hamartomatous proliferations of vascular endothelial cells
Incidence
Hemangiomas are more common in females. They may be self involuting or permanent.
Clinical features (Figure 22.a, 22.b)

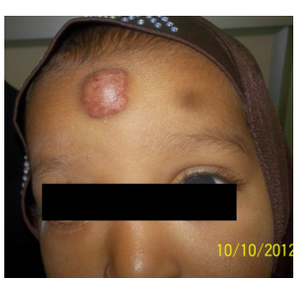

Figure 22.a:
Congenital Hemangioma

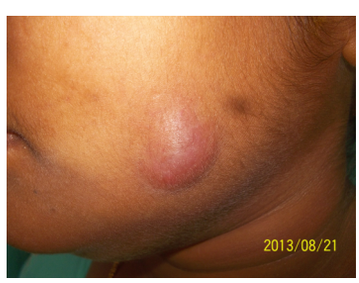

Figure 22.b:
Hemangioma: unresolved at the age of 30

They are present at birth, having a proliferating growth in infancy and spontaneous involution later in life. Most commonly seen on head and neck region, the lesions typically blanch with pressure. They are also called as strawberry hemangioma.

Histopathology
There is proliferation of single layer of endothelial cells and pericytes.
Differential diagnosis
Angiosarcoma
Pyogenic granuloma
Management
Topical or intralesional steroids, topical beta blockers, systemic propranolol and laser ablation are the therapeutic options.

Basal cell carcinoma
Basal cell cancer (BCC) is the most common type of skin cancer in Caucasians, Hispanics, and Asians. The frequency of BCC appears to be directly correlated with the degree of pigmentation in the skin.

Etiopathogenesis

UVR exposure is the most common etiologic factor for BCC in all racial groups, others include scars, ulcers, chronic infections, immunosuppression, previous radiation treatment, and both physical and thermal trauma. Genetic disorders such as albinism, xeroderma pigmentosum , and nevoid basal cell carcinoma syndrome are also risk factors for BCC.

Clinical features (Figure 23.a, 23.b, 23.c, 23.d)

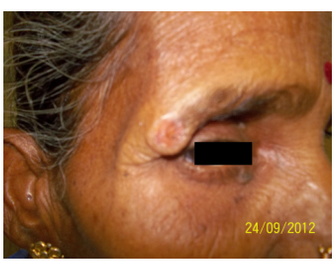

Figure 23.a: Basal cell carcinoma on the lateral end of right eyebrow in a 70 year old female

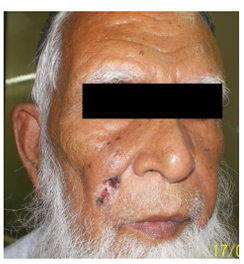

Figure 23.b: Basal cell carcinoma: ulcerative type

Figure 23.c: Basal cell carcinoma : cauliflower like lesion at the angle of mouth

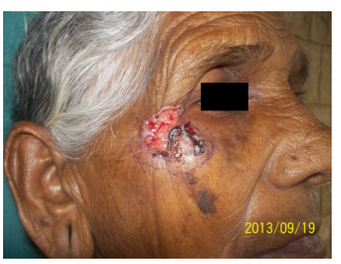

Figure 23.d: Basal cell carcinoma : ulcerative type. Dysplastic melanocytic nevi is also seen

Most patients with BCC are elderly and present with asymptomatic, translucent, solitary nodules with central ulceration. Telangiectasias and a pearly, rolled border in dark skin or in a pigmented tumor may be difficult to discern. Pigmentation is present in more than 50% of the tumors. Lesions can occur as nodules, plaques, papules, ulcers, or in more advanced cases, indurated or pedunculated masses. 89% of the lesions are present in the head and neck region.

Risk factors for metastasis include a tumor diameter greater than 2cm, location on the central part of the face or ears, longstanding duration and incomplete excision. The prognosis for metastatic disease is poor, with mean survival ranging from 8 months to 3.6 years[34].

Differential diagnosis

Seborrheic keratoses

Malignant melanoma

Nevus sebaceous

Management

Treatment for BCC includes Moh's micrographic surgery, cryosurgery, and electrodessication and curettage.

Squamous cell Carcinoma

Overall, Squamous cell carcinoma (SCC) accounts for ~20% of all skin cancers[35]

Etiopathogenesis

Predisposing factors for SCC in people of color include scars from thermal and chemical burns, chronic leg ulcers, and previous sites of radiation. Immunosuppressed patients, such as those with organ transplants and those infected with the human papillomavirus are also at increased risk for SCC. Patients with chronic inflammation, such as osteomyelitis, hidradenitis suppurativa, or lupus vulgaris, are also at increased risk for SCC. Chronic sun exposure is also a risk factor.

Clinical features (Figure 24.a, 24.b)

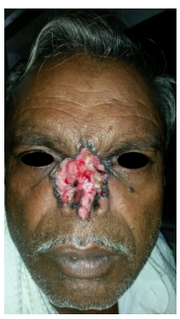

Figure 24.a:
Squamous cell carcinoma :
Ulcerative and mutilating type

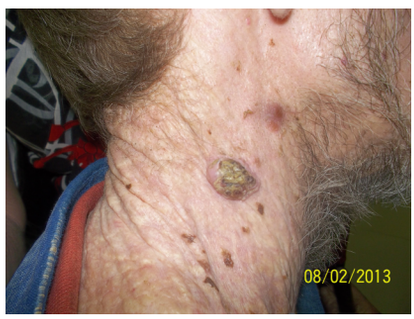

Figure 24.b:
Squamous cell carcinoma:
seen on the neck of an albino
at 55 years.

SCC's are often superficial, discrete, and hard lesions arising from an indurated, rounded, and elevated base. SCC arising from a chronic scarring process tends to be more aggressive and metastasis is a possible risk. If metastasis is present, 10-year survival rates are less than 20 percent for patients with regional lymph node involvement and less than 10 percent for patients with distant metastases[35].

Histopathology
The tumor may appear as single cells, small groups of nests of cells or a single mass. The aberrant cells have increased mitoses, mitotic figures, nuclear hyperchromasia and loss of intercellular bridges. Squamous differentiation is seen as foci of keratinisation in concentric rings of squamous cells called horn pearls.
Differential diagnosis
Wart
Seborrheic keratoses
Actinic keratoses
Keratoacanthoma
Management
Treatment for SCC includes Moh's micrographic surgery and electrodessication and curettage.

Melanoma
Melanoma is the third and most deadly form of skin cancer in all racial groups

Etiopathogenesis
Major risk factors for melanoma include intermittent high exposure to sunlight and chronic cumulative dosages of UV radiation. Host susceptibility factors include, dysplastic nevi, increased number of nevi, freckling, family history of melanoma, fair complexion, light eyes, and blonde or red hair

Clinical features
Clinically, melanomas typically present as dark, rapidly spreading patches. Clues to the diagnosis of subungual (under the nail) melanoma include a pigmented band on the nail with width greater than 3 mm (Hutchinson's sign), variable pigment, rapid increase in size, and the presence of solitary lesions[36]

Histopathology
Clark staging and Breslows staging are done depending on the level of invasion of tumor cells.
Management

Treatment for melanoma includes wide local excision or, sometimes, amputations for melanoma involving the limb, such as acral lentiginous melanoma. Metastatic melanoma is very difficult to treat, but includes isolated limb perfusion with chemotherapy, radiation, IL-2, and experimental cancer vaccines[37]

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Choosing the right Chemical Peel

Dr. Rasya Dixit

Introduction:

Chemical peels are a common tool used in the Dermatologist's office. The increasing awareness of these peels has made it a sought after treatment, not only to treat medical conditions, but also for general skin rejuvenation. These easy to perform treatments complement the medical therapies and help to accelerate skin recovery. However, as multiple new peels become available, it is important to learn to discern where a particular peel can be used appropriately.

Choosing the correct peel based on classification: Currently available peels are classified based on the depth of penetration as superficial, medium and deep peels. The depth of penetration is determined by the concentration, pH and the type of peeling agent used.[1] Application time, occlusion and contact time also determine the depth of the peels. Though the depth of the skin condition being treated is paramount to the peel being chosen, the following table shows the peels being chosen based on the depth.

	Depth of penetration	**Peeling agent**	**Indications**
Very superficial	Stratum corneum	Salicylic acid 20 to 30%, Glycolic acid 30 to 50% (applied for 1 to 2 minutes)	Acne, Post inflammatory hyperpigmentation, solar lentigines.
Superficial	Stratum corneum to papillary dermis	Glycolic Acid 50 to 70% (applied for 2 to 5 minutes), Lactic acid, salicylic acid, TCA<20%, Retinoic acid	acne vulgaris, mild acne scarring, pigmentary disorders (melasma, mild dyschromia, post-inflammatory hyperpigmentation) Periorbital melanosis
Medium	Papillary dermis to upper reticular dermis	70% Glycolic, 35% TCA, Jessner's peel	actinic keratoses, fine lines, rhytides, solar lentigines, pigmentary disorders, (melasma, mild to moderate dyschromia) seborrhoeic keratosis, superficial atrophic scars, keratosis pilaris
Deep	Upper reticular dermis to mid reticular dermis	TCA >50%	Severe photoageing (advanced rhytides), pigmentary disorders, premalignant skin tumours, scars

Choosing the correct peel based on clinical condition:

Acne: Chemical peels are used as an adjunct to medical therapy in acne.The primary effect may be on comedones with a concomitant reduction in inflammatory lesions. [2]Peels may allow topical acne agents to penetrate more efficiently into the skin and may improve PIH. Salicylic acid (SA) peels are used to treat seborrhoea and comedonal acne.[3] More recently, the Salicylic Mandelic (SM peel) combination peels are used to reduce both oiliness and pigmentation. Inflammatory acne is also well treated with this combination peel. Though glycolic acid (GA) can be used to treat acne as well, a split face study which compared glycolic acid to salicylic acid showed sustained efficacy and better tolerability of SA over GA peels. [3]

Acne scars: Macular pigmented scars or acne PIH can be treated with a combination of Glycolic and Retinol peels. Modified Jessner's peel can also be used as a leave on peel for these scars. Superficial atrophic scars respond well to 20 to 35% TCA peels. Ice pick scars are well treated with the TCA CROSS method. [4]

Melasma: Melasma has always been very challenging to treat for multiple reasons including the presence of melanin at varying depths in the epidermis and dermis. Because chemical peels remove melanin and improve skin tone and texture, they are commonly used in treating this condition. More superficial and more limited involvement melasma is often more responsive to treatment. Data from small studies suggest that melasma improvement occurs more rapidly when peels are combined with medical therapy. Several peels have been studied (SA, GA, TCA, tretinoin and resorcinol, retinoic acid and Jessner's), although GA is currently most popular. [5]

Facial rejuvenation: The goal of facial rejuvenation is to correct irregular pigmentation, wrinkling, loss of elasticity, and coarseness and restore the natural glow of the skin. Histologically, peels alter the epidermis creating a more normal pattern with columnar cells showing return of polarity, more regular distribution of melanocytes, and melanin granules. A wide range of chemical peels including AHA, SA, TCA, and phenol are used. The efficacy of treating photoaging with tretinoin is well established.[6]
Periorbital melanosis: The problem of periorbital melanosis is common especially in the Indian subcontinent. The skin here poses several challenges as it is significantly thinner than anywhere else on the body. The skin is also prone to rugosities due to the action of the orbicularis oculi, making the peel run off into the creases, leading to undesired side effects. Lactic acid, Arginine and TCA peels are studied and most popular in the treatment of this area.[7]

Acanthosis nigricans: The skin in Acanthosis nigricans appears hyperpigmented and the reduction of thickness of the stratum corneum with TCA peels or modified Jessner's peels help in the cosmetic improvement of the problem. The improvement of the metabolic profile helps in the long term resolution of the Acanthosis. [8]

Choosing the correct peel based on the correct patient:

To select the appropriate patient for chemical peel is as important as to select the peel itself. An ideal patient for the peel is someone who has a specific indication for the peel, understands the need for pre and post treatment care and maintenance, and is willing for multiple sessions of treatment. A patient with

unrealistic expectations is a bad choice for chemical peels. Similarly, any patient taking oral photosensitising medicines, any pregnant lady and anyone with active skin infection or eczema over the area to be treated are to be approached with caution. [9]

It is also important to assess the social calendar of the patient, especially if the peel is expected to have any downtime. A patient who desires cosmetic improvement may be happier with multiple milder peels than a stronger peel sessions because of their social commitments. Similarly, younger and inexperienced dermatologists should prefer gel based and buffered peels over the lotion based peels initially to avoid complications.

Choosing the correct peel based on the peel composition:

Newer chemical peels have more options for the dermatologist to choose from. Gel based preparations of peels have now increased the safety of the procedure as they offer a slow release of peel and lower the irritation to the patient and increase safety. However, there may be a slight compromise of efficacy and more number of sessions may be needed to achieve desired results. Similarly, buffered peels offer the reduction of irritation and increase peel tolerability.

Combination peels are peels which are premixed in the bottle. Here, two or more peels with synergistic action are combined together. They add the advantage of lower cost (when compared to the use of two different peels) and better side effect profile. Common combination peels are SM peel (Salicylic and Mandelic) and Glycolic Lactic peels. [10]

Knowing the various combinations helps the dermatologist to select peel for the appropriate patients.

Potential side effects

As the chemical peel become affordable and common, it is important to know how to prevent the complications of treatment. Superficial chemical peels are generally safe and tolerated with mild discomfort, such as transient burning, irritation, and erythema. Scarring is rare in superficial peels, as are PIH and infection. Care should be taken to feather peel solution at junctions with nonpeeled skin to avoid this effect. Side effects of peels can also include pigmentary changes (e.g., PIH for dark-skinned individuals), infections, allergic reactions, improper healing, hypersensivity, disease exacerbation, and those due to improper application.[11]

Care must also be taken to prophylactically treat patients with a history of herpes simplex infections. Herpetic episodes, usually on the lip or above the vermilion border, may be prevented with prophylactic oral acyclovir, valacyclovir hydrochloride, or famciclovir. Antiviral agents are especially useful in patients who indicate a strong history of multiple herpetic lesions each year.

The best way to prevent complications is to identify patients at risk and maintain an appropriate peel depth that balances efficacy with known adverse events. Patients at risk include those with PIH, keloid formation, heavy occupational sun exposure, a history of intolerability to sunscreens, and uncooperative patients.

Tolerability of peels may be influenced by many factors, such as peel agents, concentration, depth, skin

type, and concomitant use of skin care products. PIH can be exacerbated by sun exposure, so it is important to educate patients and closely monitor their recovery phase. Sunscreens should be used continuously to limit PIH development. Epidermal PIH responds well to various treatments, while dermal PIH remains problematic. Pretreatment with bleaching agents before beginning therapy with peels decreases the appearance of PIH. Treatment options include hydroquinone or kojic acid or other tyrosinase inhibitors.[12]

Considerations before and after peels

Medical history: As in any dermatological procedure, taking a complete history prior to peeling is critical. It is important to check on wound healing and response to previous cosmetic procedures. A list of current medications to look for photosensitising medications, and a history of herpes labialis which needs oral prophylactic antivirals is vital.

Pretreatment: Pretreatment can help to enhance outcomes and is often started 2 to 4 weeks prior to the peel and discontinued 3 to 5 days before the procedure. Topical retinoids, glycolic acid creams and non hydroquinone skin lightening agents are of choice to help to prep the skin to achieve an even penetration of peel and avoid post peel post inflammatory hyperpigmentation. Discussing peel after-effects with patients before the peel is also important to aid comprehension of the peeling process.

Photography and Written Informed Consent:

Baseline photography is highly recommended. Photographs may be used to guide peel selection and for before-and-after comparisons. A detailed consent discussion should be undertaken by the treating clinician and appropriately documented in the medical record. This process may be facilitated by photographs and videos of the peeling procedure, the provision of written information and the use of a dedicated consent form.

Postpeel: Patients should use a broad-spectrum, preferably physical sunscreen on a daily basis and implement a gentle cleansing regimen should be prescribed. Moisturizers may also be recommended to help repair the impaired barrier.

Maintenance: After a chemical peel, erythema, and desquamation may occur for 1 to 3 days for superficial peels and 5 to 10 days for medium peels. Patients should be instructed to avoid peeling or scratching the affected skin and to use only simple moisturizers.

Patients can restart the pre peel prep creams after the dryness and exfoliation is complete. These are essential to continue the improvement achieved by the peel.

Patients should also be advised to review after 4 weeks for the next peel session. Long term results of peels are noted with repeated sessions, and this must be explained to the patient before the first session.

Conclusion:

Chemical peels remain popular for the management of various dermatological conditions, commonest being acne, acne scarring, melasma and photoageing. Need for picking the correct peel is as important as selecting the appropriate patient. Repeated sessions and good maintenance of skin care in between

treatments is imperative for treatment success. Newer molecules that provide unique characteristics need to be studied further.

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BOTOX IN FACIAL AESTHETICS

Dr. Vidya T S, Dr. Aneesa

INTRODUCTION

Botulinum neurotoxin (BT) is is best known to clinicians as a deadly poison/ miracle poison. Its an exotoxin produced by clostridium botulinum. It exerts its effect by paralyzing striated muscles or the autonomic-innervated muscles. The term botulinum is derived from the latin word for sausage, "botulus". When ingested in large quantity, this induces a bilaterally symmetrical descending neuroparalysis called botulism. Botox A is used for several disorders in the field of medicine, particularly in dermatology, for aesthetic purposes.

TYPES OF BOTOX

BT is a powerful neurotoxin produced by bacterium Clostridium botulinum. There are 8 types of Botulinum toxin (A, B, C1, C2, D, E, F, and G). Type A is the most potent toxin followed by B and F.[1]

VARIOUS PREPARATIONS AVAILABLE

BOTULINUM TOXIN A

BOTOX - was the first commercially available type in the US.
DYSPORT - The potency of 4 unit of Dysport is approximately equal to 1 unit of BOTOX.
XEOMIN- One unit of Xeomin is equal to 1 unit of Botox and even at high doses it has least chances of developing neutralizing antibody.
NEURONOX
PURTOX
PROSIGNE
LINURASE.[1,2,3]

BOTULINUM TOXIN B

NEUROBLOC (Myobloc) – not yet approved for cosmetic use.[1]

TOPICAL BOTULINUM TOXIN

Rt001

MECHANISM OF ACTION

- BT is a polypeptide consists of a protein molecule with a heavy and light chain held together by a heat-labile disulfide bond. Disruption of disulfide bond inactivates the neurotoxin, so BT storage at the correct temperature is necessary. The BT heavy chain binds to receptors on the nerve terminal membrane which allows internalization of BT. Then it undergoes conformational changes, disulphide bond is reduced. Light chain gets released into the cytosol. The light chain acts as endopeptidase, that cleaves the SNARE protein (SNAP-25) specific to the particular neurotoxin. Thereby inhibiting the release of acetyl choline and neuromuscular signal transmission. Although the binding of toxin and protein complex is permanent, paralytic effect lasts only for 2–6 months. The reason for this reversal is the reestablishment of neurotransmitter pathway due to new axonal sprouts formation.[4,5]

PREPARATION, STORAGE, AND TECHNIQUE

BT comes in an insulated glass vial as 50/100 units of freeze-dried powder, stored before reconstitution either frozen at −5° or in a refrigerator at 2°–8°; once opened and reconstituted the product should be stored at 2°–8° and can be used upto 6 weeks.[3,6]

Usually 100 unit vial is reconstituted with 2.5ml sterile preservative free normal saline to get a concentration of 4U/0.1ml. The product is vacuum sealed, while reconstitution the plunger of syringe should be held in controller manner, so that air bubble is not formed. Then gently rotate the vial. Do not shake it.

One must have basic knowledge about the anatomy of muscles before injecting the toxin. Its better to mark the area with marking pencil before the injection. Then the procedure is carried out with aseptic precaution. Men require higher dose of botox as they have higher muscle mass than women.[5]

The clinical effect are seen approximately 12-72 hrs after injection, followed by 1–3 weeks of maximum effect, which will resolve after 3–4 months.[3,5]

INDICATIONS IN FACIAL AESTHETICS

- Forehead lines
- Crow's feet
- Vertical and horizontal frown lines
- Bunny lines
- Nasal tip elevation
- Nasal flare
- Gummy smile
- Nasolabial fold
- Upper lip rhytids
- Marionette lines
- Pebbly chin
- Horizontal and vertical neck bands,
- Scar management
- Brow lift
- Masseter hypertrophy
- Nefertiti lift
- Infraorbital wrinkles.[3,4,7]

PROCEDURE for some of the commonly performed indications.[3,7]

Indication	Targeted Muscle	Procedure
FOREHEAD LINES	Frontalis	Mark the area in Jacob ladder pattern, 1-2 cm apart and atleast 2 cm above the eyebrow margin. About 1 unit of toxin is injected into each point to raise a wheal. Donot cross the mid papillary line.
CROWS FEET	Lateral fibres of Orbicularis occuli	Points are marked 1 cm lateral to orbital rim at the level of lateral canthus, and 1 cm above and below this. Two to three units injected at each point. Make sure that it doesn't cross below the zygomatic arch
FROWN LINES	Transverse rhytids-procerus Vertical rhytids-procerus and corrugator supercilii	Mark a point just above the meeting point of 2 imaginary lines running form medial end of the eye brow to contralateral inner canthus. Four to ten units injected here in the centre of procerus muscle. Four units in the medial belly of the corrugators above the supraorbital ridge in line with the medial canthus and 2 units each given to tail of corrugator lateral to previous injection, but not beyond the mid pupillary line. Always place your thumb on the supra orbital ridge and inject above it
BUNNY LINES	transverse nasalis muscle	Ask the patient to sniff, to laugh or to squint. Injections points are marked on either side of nasal bridge. Inject 2-4 into the superior portion of the muscle across the lateral nasal wall
GUMMY SMILE	levator labii superioris alaeque nasi	Inject 2 units on each side of the nose within the triangle between ala, nose and cheek
MASSETER HYPERTHROPHY	Masseter	Draw line form the lowest part of external auditory meatus to angle of mouth. Ask the patient to grind the teeth. palpate for the anterior and posterior border of masseter, with ramus of mandible being the lower border. Locate the maximum muscle bulge while the patient is biting and mark this point. Mark 2 more points lateral to the 1st point to form a triangle. Around 25-30 units injected to each side.[8]
PERIORAL LINES	Orbicularis oris	One unit per site

MESOBOTOX (microbotox)

- Injection of multiple microdroplets of diluted botulinum toxin into the dermis or the interface between the dermis and the superficial layer of facial muscles.
- Indication : Decreases sebum production, pore size and wrinkles and gives the skin a smooth lustrous appearance.
- Acts by decreasing sweat and sebaceous gland activity, target the superficial layer of muscles that are attached to the under surface of the dermis and at the same time, not to allow diffusion of the toxin into the entire muscle to paralyze it completely.
- Recnstituted botox is further diluted 3-5 times with normal saline. To full face upto 20 units can be given.[9]

CONTRAINDICATIONS

Hypersensitivity to any component of BT
Dysport is contraindicated in patients with cow's milk protein allergy Pregnancy & lactation,
Neuromuscular junction disorders (myasthenia gravis), amyotrophic lateralizing sclerosis, myopathies
Concomitant use aminoglycosides, cholinesterase inhbitir, calcium channel blockers].[2,3,7]

COMPLICATIONS

botulinum toxin injections are generally well tolerated and side effects are very few.
Mild pain, edema, erythema
Transient numbness
Headache, malaise or mild nausea.
Temporary upper lid or brow ptosis
Lip ptosis
Neck weaknes
Repeated injections – bone loss on repeated injections because of disuse atrophy.[1,10]

Conclusion

Botulinum toxin is a good, reversible, minimally invasive and safe method for facial rejuvenation. Examine the patient carefully and look for any asymmetry. Proper knowledge of anatomy, technique, and proper selection of patient much essential for successful treatment.

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Microblading

Dr. Shilpa K, Dr. Lakshmi D V

INTRODUCTION

Body art techniques have always fascinated the world of medicine from its roots of origin as a cultural identity; thence transforming into the modern world medical tattooing. The secrets of these techniques has been discovered and reinvented with improvised electric or battery driven tattoo machines, better quality pigments and asepsis to widen its applications in medico-cosmetic arena...Under its roof,an emerging body art technique is 'Microblading'an art of creating realistic and semi permanent hair strokes that resemble natural hair fibres.

How microblading is different from Tattooing?

In Traditional tattoos, the desired pigments are introduced into the deep reticular dermiseither manually or with tattoo machines which are then engulfed by macrophages and remain under the skin permanently. As the pigment laden cells dip into the deeper dermis, the tattoo acquires a greenish hue rather than the black colour. The colour achieved is diffuse in traditional tattoos and multiple colours are used.

In microblading, black pigments are deposited more superficially at the junction of papillary and reticular dermis with the help of microfine needle blades. These blades are used to create a linear strokes along which the pigments get deposited mimicking a natural hair . As pigments are deposited in superficial layers of skin these pigments are extruded out of the skin as the keratinocytes are shed. Thus making the procedure semipermanent and require periodic partial redoing sessions to replicate the primary appearance at6 to 9 months interval.

Indications

The most common indication is cosmetic eyebrow enhancement and the others include madarosis and loss of eyebrow due to varied aetiology, camouflaging traumatic or iatrogenic scars, asymmetric, drooping or short eyebrow.

Pre procedural counselling

It is essential to disclose the nature of the procedure, anaesthesia, complications, post procedure care to the patient Semi permanent nature of the procedure and need for periodic partial redoingsessions ever 6 to 9 months should be made aware. It is mandatory in every situation to consider the patient expectations of the procedure, if found to be overzealous and/or unrealistic, a medical fitness certification by a psychiatrist is worth the time. Written informed consent should be taken before the procedure.

Required instruments

Microbladingpen (Figure 1) and microfine blades (Figure 2)
Pigment(Figure 3) -
For Indian skin, Brown black colour is preferable. Apart from colour density, pigment size also to be considered.

Figure 1

Figure 2

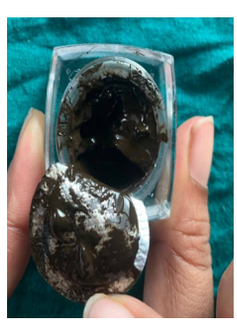


Figure 3

Anaesthesia
Procedure is done under topical anaesthesia. Emla cream is applied one hour before procedure.

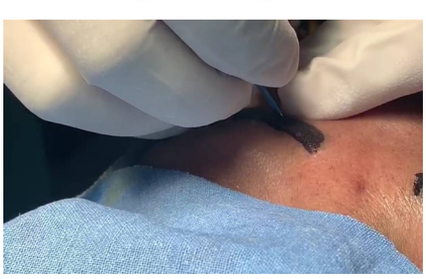
Designing of eyebrow:
Designing of eyebrow is the most time consuming part of the procedure. Design must suit age, sex and also shape of the face to give a natural look.

A proper anatomical knowledge with respect to eyebrow shape size and direction in both male and female is very important especially if you are doing microblading in completely absent eyebrows.Generally male eyebrows are bushier as compared to females.

Anatomy of eyebrow:
Eyebrows are arched eminences measuring 4.5-5.5cm comprising of head medially, middle body and tail laterally. Head end measures 0.5-1cm with a vertical or rounded medial border and lies about 1-1.5cm lateral and cephalic to central glabella. The body forms the widest part and area of maximum density measuring 2.5cm and gives the prominence to eyebrow structure. It has a flat lower border medially which the arches upwards with a gradual narrowing laterally (pronounced in females). In few males, narrowing may be absent and it may appear wider instead.The lateral end is the narrowest part with least hair density; extends laterally and descends downwards from the arch peak and measures 1cm
Preformed stensils are available which can be used.However,these do not take into consideration the shape of the face.

Standard principles of infection control like hand hygiene, hand wash, personnel protection equipments, cleaning and disinfection of parts,surrounding environment and waste disposal are mandatory.

Procedure proper
After designing the eyebrow,take patient approval (Design and approval signature to be a part of the written consent form). Skin is stretched with left index and thumb finger. The direction of stretching should be perpendicular in medial part and parallel to the eyebrow as we move laterally.With the microfine blade attached to the handle and is dipped in pigment paste and linear strokes are put parallel to the existing hairs(Figure 4). The depth should be such that serum oozes out of the slits. In general, the medial most part of the eyebrow the hairs remain vertical.In this area strokes are given sparsely in a fanning fashion to reproduce natural look and as we move laterally they tend to become parallel to the long axis of the eyebrow. In the middle part, strokes should be given in a converging pattern so that the strokes in the upper part are directed downwards and laterally and lower part of strokes directed upwards and laterally so together they form a crisscross pattern. In the middle part of the eyebrow more closer strokes to be given to give prominent eyebrow. In the lateral most part progressively sparse strokes should be given. Lower lateral part is feathered to give natural soft appearance.



After completing strokes from medial to lateral eyebrow extra pigment paste is covered on the entire eyebrow and left for four to five minutesfor pigment to settle in the linear cuts.A petrolatum jelly is applied and the extra pigment is wiped off using a guaze. Linear pigmented streaks mimicking hairlines become visible. Touch up strokes can be put again to give uniform density. Always make sure that the

symmetry is maintained between the two eyebrows(Figure5).

Post procedure

Immediately after the procedure the eyebrow looks darker. Erythema and swelling can be there for a day or two. Next 1-3 days there will be serum oozing which forms the crust. Crust will fall off in 7-10 days. Patient experiences lightening or loss of pigment as the crust falls off. Pigment regain appears 25 to 30 days following the procedure. Touch up sessions to be done after 45 to 60 days.

Patient should be advised not to touch the part and wet the part for minimum three days. Scabs should not be picked as it will result in pigment loss. Avoid swimming, strenuous exercise, make ups, cosmetics for the next one week.

Complications

Infection, scarring, non-uniform pigmentation asymmetric eyebrow design, Pigment loss, colour alteration over time due to deep placement are some of the complications which can be avoided by adopting proper technique.

Conclusion

Microblading is a minimally invasive semi-permanent technique for cosmetic enhancement which gives hair lines that mimic natural hair fibres. A proper designing, colour, depth, direction, density and post procedure determines the optimum outcome of the procedure.The fast gaining popularity of the procedure can be attributed to its affordability, compliance and minimal risks due to evanescent nature makes it a promising technique in developing world.

Legends

1.Microblading pen with one end holding blade
2. Microfine blades-Microfine needle arrranged in linear fashion in between spaces holding pigment
3.Brown black pigment paste
4. Strokes being given parallel to existing hairs
4.Pre and post microblading

THINKING INSIDE THE BOX

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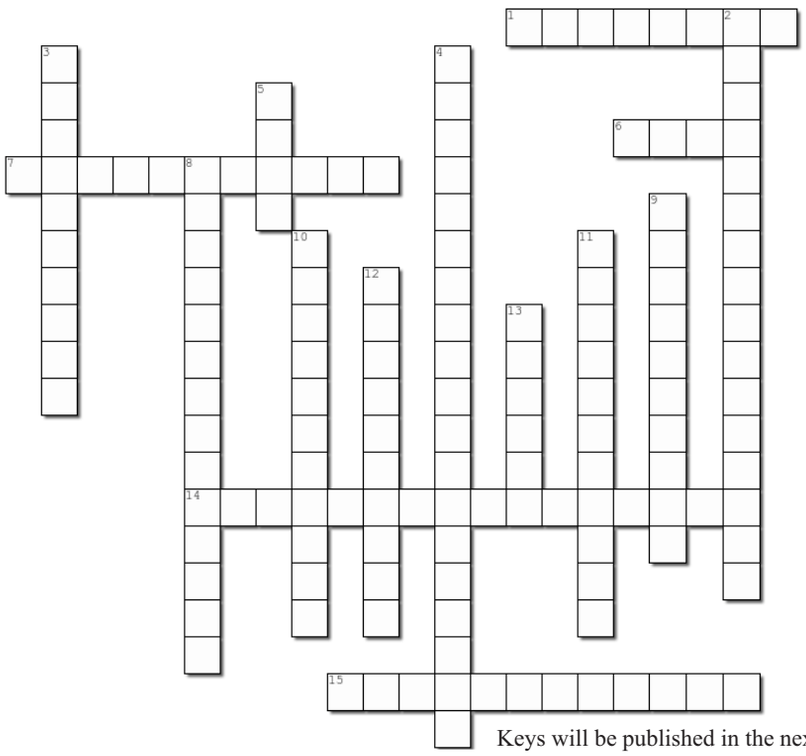

Keys will be published in the next issue

Across

1. Type of elastic fibre that does not contain elastin
6. Trichoscopic feature characteristic of trichotillomania is _____ sign
7. Mupirocin is derived from which pseudomonas species
14. Enzyme defect in ichthyosis inherited in this manner (image-2)
15. Half and half cells are seen in which disease

Down

2. Parchment pulps appearance of fingers seen in
3. Hair shaft lines equivalent of Beau's lines seen after periods of illness or chemotherapy
4. Tiger tail hair on polarizing microscopy seen in (image-1)
5. Mitosoid cells in stratum granulosum is seen in which disease
8. French dermatologist who proved an early description of skin lesions associated with sarcoidosis, introducing the name 'lupus
9. US FDA approved monoclonal antibody for treatment of HIV 1
10. Stain than can stain both collagen and elastin simultaneously
11. Carvajal syndrome is due to defect in
12. HIV belongs to which genus
13. CK 20 is expressed in which cells in skin

Image-1

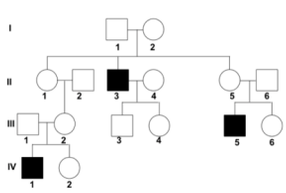

Image-2

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