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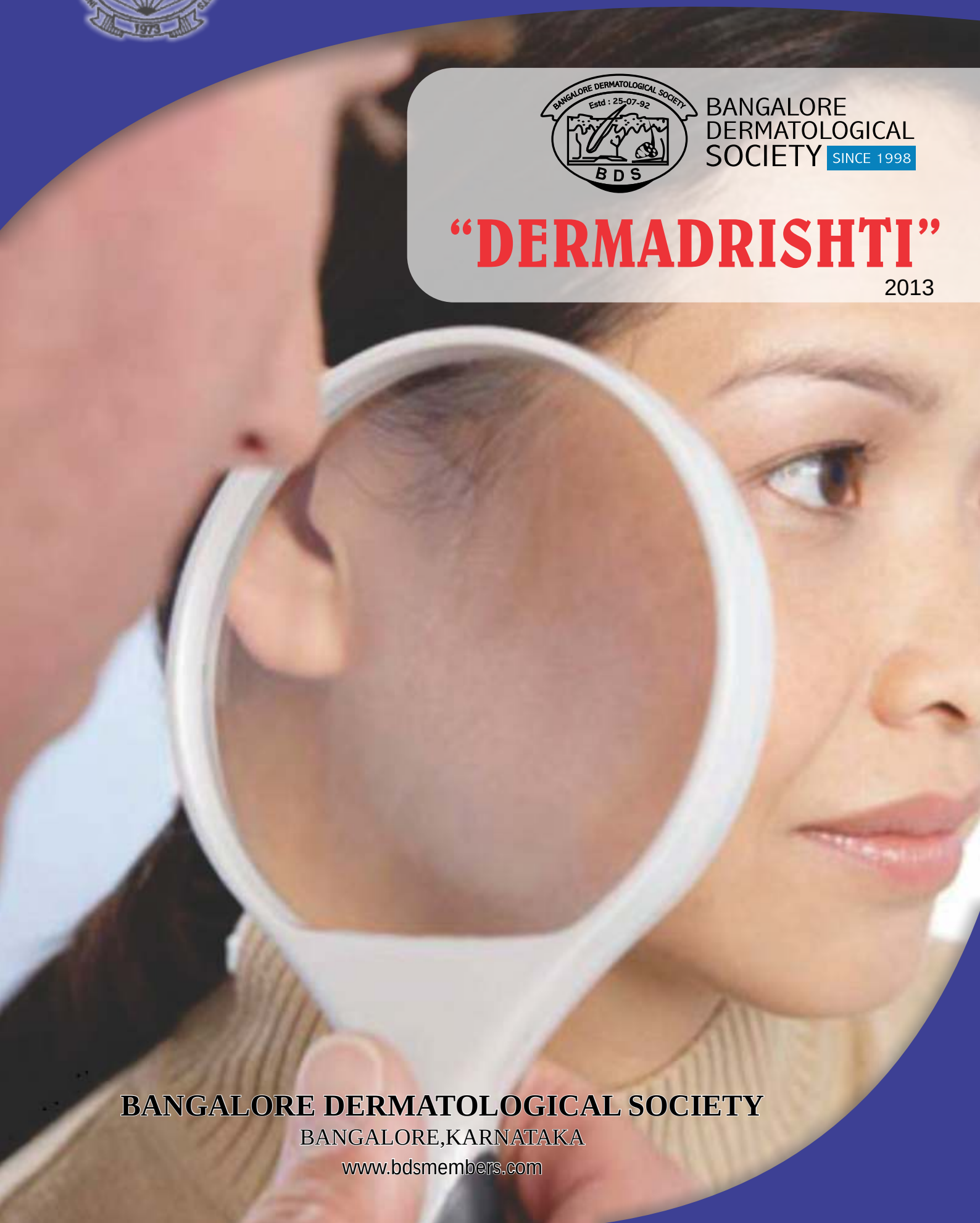

BANGALORE DERMATOLOGICAL SOCIETY

BANGALORE,KARNATAKA

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MESSAGE FROM THE PRESIDENT, BANGALORE DERMATOLOGICAL SOCIETY

I am happy to note that Dr. Prabhakar M. Sangolli, editor for Bangalore Dermatological Society News Letter along with editorial team consisting of Dr. Harikishan and Dr. Praveen, is bringing out first edition after I assumed the office of President of Bangalore Dermatological Society.

I wish him success on bringing out this letter every quarterly. News letter “DERMA DRISHTI” will feature excerpts of scientific content of academic meets, cartoon, quiz, conferences, news, and update on various Dermatological conditions like acne. I hope, soon this letter will be indexed also. Under Dr. Sangolli's guidance the news letter will reach out to all the members at its elegant best.

Thanking you,

Dr. H.V. Nataraja
President, BDS

MESSAGE FROM THE BDS SECRETARY, BANGALORE DERMATOLOGICAL SOCIETY

I wish to congratulate Dr.Prabhakar Sangolli, (our past president) Dr. Praveen and Dr. Hari Kishan on bringing out the BDS NEWS Letter, highlighting our activities and achievements.This should serve as inspiration for those involved with BDS activities and bring the best out of them.

Long live BDS

Dr.Nagaraju.T.S.
BDS Secretary

MESSAGE FROM THE EDITOR, BANGALORE DERMATOLOGICAL SOCIETY

Bangalore Dermatological Society is one the most vibrant branch of IADVL.BDS new letter “DERMA DRISHTI “ will show case the activities of BDS. It also will unearth hidden talent among BDS members by including cartoons, quiz, crosswords etc...We have young dynamic editorial team members namely Dr. Hari Kishan and Dr. Praveen. I urge all the members of BDS to actively contribute to the news letter, so that it can be indexed shortly.

Suggestions,feed back are welcome.

Dr. Prabhakar M Sangolli

Editor BDS Newsletter

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A. ACADEMIC CONTENT OF BDS MEETS (LAST 3 MEETS)

DETAILS OF MEET HELD ON 22-09-13

The fifth Bangalore Dermatological society CME was hosted by ST. John's medical college , under the chairmanship of Dr. Sujatha Harshad. Invited guest lecture was presented by DR. D.A Satish on 'Acne and PCOS': what a dermatologist should know. There were interesting case presentations by postgraduates. Dr. Vijay Aithal (associate professor) presented a talk on Expiry date of drugs: is it scientific or just a marketing strategy?. There was a lecture by Dr. Harikishan on 'Clinical and therapeutic approach to Facial melanosis, an update. IADVL quiz Zonal round was conducted by Dr.Shashikumar and the winning team was Dr. Amit and Dr. Nikitha from M.S. Ramaiah Medical college and teaching hospitals, Bangalore

DETAILS OF MEET HELD ON 20-10-13

The 6th Bangalore Dermatological Society CME was hosted by M.S.Ramaiah Medical college under the guidance of Dr. A.L. Shyam Prasad HOD, there were session of journal review and interesting case presentations by residents. An interactive sesion on desmoscopy and a photoquiz was presented by Dr. Praveen Kumar. Dr. Urmila Nischal presented an informative talk on management of Hirsutirm. Dr. Hammanthaya Kelogi delivered an interesting lecture on basics in HIV and its applied aspects. The First BDS bulletin was launched by Dr. Prabhakar M Sangolli with his team Dr. Harikishan and Dr. Praveen. Dr. S.D.N. Gupta presided over the launch of the bulletin.

DETAILS OF MEET HELD ON 17-11-13

The 7th Bangalore Dermatological Society CME was held on 17-11-13 headed by Dr. S. Sacchidanand Professor and H.O.D Bangalore Medical college and research centre. There was a psychodermatology capsule by Dr. Chandrashekar H. Professor & HOD, Dept. of Psychiatry, BMCRI. There were a series of case presentations and interesting sessions on dermatosurgery and cosmetology, presented by resident.There were focus sessions on paediatric dermatology, STD & Leprosy. An informative guest lecture on 'An overview of acne update' was presented by Dr. Leelavathy .B,(Associate Prof. BMCRI).

B. Clinical and Therapeutic Approach to Facial Melanosis – An Update.

Dr. Hari Kishan Kumar.Y, MD(DVL)

Associate Prof. of Dermatology, Raja Rajeswari Medical College & Hospital.

Specialist Consultant – BGS Global Hospital.

Facial melanoses (FM) are a common presentation in Indian patients, causing cosmetic disfgurement with considerable psychological impact. Facial melanoses (FM), a common presentation of Indian patients, are complex diagnostic (and even greater therapeutic) problems consisting of few somewhat well defined clinical entities, several of which have overlapping features and some of which have defied classification. The pigment-producing cells of the skin are called melanocytes and their activity is the major determinant of the color of the hair and skin. Within the epidermis, melanocytes reside in the basal layer in a ratio of about 10 keratinocytes to 1 melanocyte. However, each melanocyte via its dendrites supplies melanin to about 30 nearby keratinocytes.

When ultraviolet rays penetrate the skin it triggers the production of melanin as a defense mechanism. Melanin moves along arm-like structures called dendrites in a special container called a melanosome. After the complete synthesis of melanin, melanosomes filled with this pigment are injected in the interior of keratinocytes. Once inside keratinocytes, melanosomes tend to spread through the cytoplasm, over the upper part of the nucleus, so as to protect it from ultraviolet radiations. Inflammation is the cause of Hyperpigmentation, external Causes include Sun Exposure, Skin trauma, burn, cosmetic treatment, surgery, Acne wound, psoriaisis and eczema. Internal Causes includes Inflammatory hormones, Pregnancy, Oral Hormone Therapy, Menopause, other hormones—primarily the sex steroids, progesterone and estrogen and High amounts of Omega 6, low amounts of Omega 3.

Hypermelanosis are a group of disorders characterized by abnormally darker skin that results from increased melanin production from a normal number of melanocytes or from increased proliferation of active melanocytes. Hypermelanoses may result from increased melanin in the epidermis (epidermal hypermelanoses) or the presence of melanin in the dermis(dermal hypermelanoses).

Possible mechanisms for increased epidermal melanin without an increase in the number of melanocytes include the following: Increased melanosome production and transfer to keratinocytes; Increased melanosome size; Decreased keratinocyte turnover, resulting in overloading of the keratinocyte with melanosomes. In dermal hypermelanoses, melanosomes are formed in the epidermis by epidermal melanocytes & are transferred to the dermis, where they are found mostly within macrophages(melanophages). This phenomenon is called “epidermal melanin incontinence”. Hyperpigmentation in the skin can result from: Increased production of the melanin pigment, or pigment incontinence. Accumulation of a large number of melanocytes, or Deposition of other (non-melanin) pigments or substances in the skin.

CAUSES OF FACIAL MELANOSES:

Melasma (Chloasma)
Riehl's melanosis
Post Inflammatory Hyperpigmentation
Solar lentigines
Phototoxic Dermatitis
Erythema Dyschromicum Perstans
Poikiloderma of Civatte
Peribuccal Pigmentation of Brocq
Erythromelanosis follicularis faciei
Drug induced facial hyperpigmentation
Facial hypermelanosis secondary to systemic disorders:
Addisons disease, Acanthosis nigricans

MELASMA (CHLOASMA)

Melasma is derived from Greek word melas (black) while chloasma is derived from the word chloazein (green), and since the pigmentation is

brown-black, melasma is the preferred term. The exact etiology of melasma is not known but several factors have been implicated. UVR (UVA and UVB) and visible light cause peroxidation of lipids in cellular membranes, leading to generation of free radicals, which stimulate melanogenesis. Elevated levels of estrogens and progesterone (as occurring in pregnancy) are important. Progesterone may be more important, as melasma develops in postmenopausal woman who are given progesterone and not when given estrogen supplementation. Melasma is several times commoner in patients with thyroid disease than in controls and MSH may be important as melasma frequently begins as well as worsens during pregnancy as also after profound emotional stress.

Genetic factors are indicated because more than 30% of patients have a family history of melasma and melasma has been reported in identical twins without affecting other siblings. Constituents of cosmetics have been frequently incriminated since the commonest site of affliction is face of women. Drugs (phenytoin, griseofulvin, and NSAIDs) can cause melasma-like pigmentation.

Melasma is characterized by symmetrical hyperpigmented macules, which may be blotchy, irregular, arcuate, or polycyclic and rarely have a linear or a starburst distribution. Depending on the location of melanin (vide supra), melasma is classified into:
Epidermal type: in which the pigment is brown and margins of the lesions are well defined and geographical.

Dermaltype: in which the pigment is grey-brown and the margins of the lesions are poorly defined.
Mixed or epidermo-dermal type: in which melanin is present both in epidermis and dermis.
The face is most commonly affected though rarely pigmentation may extend on to V of the neck or may be confined to the forearms. On the face, three patterns of melasma are recognized:
Centrofacial: the most frequent (63%) pattern, with pigmentation on cheeks, forehead, upper lip, nose, and chin.

Malar: constituting 21%, with pigmentation present only on cheeks and nose.
Mandibular: the least common (16%), with pigmentation on ramus of the mandible.

ERYTHEMA DYSCHROMICUM PERSTANS (EDP)

Syn: Ashy dermatosis of Ramirez, erythema chronicum figuratum melanodermicum. Although Ramirez first described what is today labelled EDP, it is Sulzberger who is credited with coining the term EDP. Though EDP has been reported from many countries including India, it is most common in Latin America and Asia. It occurs in both sexes, but causes greater concern in women. Though it can affect any age group, characteristically lesions start in the 1st - 2nd decade of life.

The etiology of EDP is unknown, The relation of EDP to lichen planus (LP) is uncertain, both have several clinical, histological, and immunohistochemical similarities and often coexist making some authors consider EDP a variant of LP. The pigmentation in EDP is due to presence of melanin in the melanosome complexes in dermis (frequent) and in epidermis (sometimes). EDP presents as numerous asymptomatic, gradually enlarging and coalescing, persistent, macules of variable sizes. Initially having an erythematous hue and an elevated dusky border (not always noted), lesions eventually become pigmented. Initially localised, lesions eventually cover extensive areas of face, trunk, and limbs.

LICHEN PLANUS PIGMENTOSUS

Though the exact etiology of lichen planus pigmentosus (LPP) is not known, cosmetics including fragrances, hair dyes, and mustard oil have been incriminated. LPP is characterized by generally asymptomatic (sometimes itchy), diffuse (less frequently reticular, blotchy, or perifollicular) hyperpigmented dark-brown to slate-grey to black macules present mostly over exposed areas and flexures. The lesions lack the erythematous border of EDP. The clinical

association of this entity with lesions of classical LP in about a third of patients and demonstration of colloid bodies on histopathology prompted Bhutani et al., to consider LPP a macular variant of LP and very similar to EDP. Though the mucous membranes are characteristically spared, some patients may have LP-like lesions.

RIEHL'S MELANOSIS

Syn: Pigmented cosmetic/ contact dermatitis
Etiology Riehl's Melanosis (RM) is probably a pigmented contact dermatitis (CD) to antigens present in cosmetics and textiles with anecdotal reports of air borne CD to musk ambrette and other plants. Cosmetic allergens, include red and yellow pigments, chromium hydroxide, aniline and azo dyes, bactericidal agents (carbanilides, ricinoleic acids), hair dyes, red kumkum, and fragrances. Textile allergens include optical whiteners, dyes, textile finishes, mercury compounds, formaldehyde, and rubber components. Sometimes occupational allergens like coal tar, pitch, asphalt, mineral oil, and chromates have been incriminated.

Incidence of RM is not known and though most reports are from Japan, cases have been reported from Europe, South America, India, and South Africa. In general, it is most pronounced in darkly pigmented races. Women appear to have a greater predilection, with majority of patients being young-middle aged women. Clinical features RM is characterized by diffuse/patchy/ rarely reticular pigmentation, often with satellite perifollicular pigmented macules and scaly follicular hyperkeratosis. Pigmentation is brown (almost black on the forehead and temples of dark skinned patients). It is sometimes preceded by mild erythema (often imperceptible in dark skinned) and pruritus. Sites of involvement depend on the allergen responsible – lesions due to cosmetics begin on forehead and temples spreading to involve rest of the face, even the chest, neck, scalp, hands, and forearms, while those due to textiles more often involve anterior aspect of thighs and axillae (sparing the vault). Patients may show positive patch test to cosmetics or their ingredients.

POST INFLAMMATORY HYPERPIGMENTATION (PIH)

- PIH can occur in various disease process, such as allergic reactions, infections, trauma and phototoxic eruptions, Acne excoriee, lichen planus, SLE, chronic dermatitis, cutaneous T-cell lymphoma and erythroderma.
- The distribution depends on the location of the original inflammatory dermatosis. The colour of the lesion ranges from light brown to black with a lighter brown appearance if the pigment is within the epidermis and a darker gray appearance if lesions contain dermal melanin.
- Epidermal PIH involves increased melanin pigment in the basal layer of the epidermis. Dermal PIH involves the upper dermis with pigment incontinence due to increased numbers of melanophages in the papillary dermis.

ERYTHROMELANOSIS FOLLICULARIS FACIEI

- Affects middle aged men and women Cause is not known.
- Manifests as gradually progressive reddish brown pigmentation and telangiectasias surmounted with pale tiny follicular papules from which vellus hairs are lost.
- Pigmentation involves periauricular areas and sometimes extensing to side of the neck.
- HPE shows characteristic enlargement of sebaceous glands and hair follicles with the latter containing lamellar horny masses. The overlying flattened epidermis contains excess melanin and there is an inconspicuous lymphocytic infiltrate around dilated vessels.

POIKILODERMA OF CIVATTE

- Common in middle aged women
- Photodynamic substances in cosmetics may be the cause
- Characterised by reddish brown

reticulate pigmentation, telangiectasia and atrophy in

irregular, symmetrical patches in convexities of cheeks and sides of neck, sparing the area under the chin.

PIGMENTATION DUE TO DRUGS AND HEAVY METALS:

- Amiodarone – slate grey
- Antimalarials – yellow brown
- Bleomycin- Diffuse flagellate hyperpigmentation
- Minocycline – grayish discolouration
- Bismuth – Blue-grey discolouration
- Gold – blue grey deposits
- Mercury – slate-grey discolouration
- Silver –diffuse slate grey discolouration

ERYTHROSE PERIBUCCALE PIGMENTAIRE DE BROCQ [EPP]

Epidemiology Middle aged women are most frequently affected. Etiology Photodynamic substances in cosmetics are probably responsible. A similar hyperpigmentation has been reported in patients with subsiding perioral dermatitis due to topical steroids.

Clinical features EPP is characterized by diffuse brown-red pigmentation present symmetrically around the mouth, with sparing the vermillion border.

PERIORBITAL MELANOSIS

Periorbital melanosis (POM) (or dark circles) is an ill- defined entity 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, and infraorbital swelling have been incriminated. Familial periorbital hyperpigmentation is determined by an autosomal dominant gene and in one study POM was found to be an extension of pigmentary demarcation lines over the face (PDL-F). Pigmentary demarcation lines (PDL), also known as Futcher's lines or Voigt's lines, are abrupt transition lines from areas of deeper pigmentation to areas with less pigmentation. These are most often observed in darker races and are considered to be normal variants of pigmentation. Clinical features POM is characterized by variegated brown to almost black discoloration around the eyes.

ADDISON'S DISEASE

Another cause of FM is Addisonian pigmentation. Etiology Addisonian pigmentation typically occurs in primary adrenal insufficiency due to autoimmune adrenalitis, tuberculosis or other granulomatous diseases, metastatic malignant disease, sarcoidosis, amylodosis, and congenital adrenal hypoplasia.

Pathogenesis Excessive production of ß-MSH and ACTH by the pituitary due to low circulating levels of adrenocortical steroids

ACANTHOSIS NIGRICANS

Acanthosis nigricans is characterized by hyperpigmented, velvety plaques of body folds may also involve the face as well. Symptomatic treatments include topical retinoids and keratolytics.

APPROACH TO A PATIENT WITH FM

DIAGNOSIS

Some of the well-defined causes of FM include melasma, RM, LPP, EDP, and EPP and poikiloderma of Civatte. However, it is not possible to slot all patients into these clinical entities due to overlap in features. In case of any unexplained hyperpigmentation, evaluation of adrenal function is essential.

TREATMENT

FM causes cosmetic disfigurement with significant emotional impact. Its treatment includes removal of provoking factors, vigorous photoprotection, and some form of active pigment reduction either with topical agents or physical modes of treatment. There is no universally effective specific therapy — existing agents have varying degrees of efficacy and relapses are frequent.

Photoprotection

Photoprotection is essential, because photodarkening can occur with just a couple of hours of sun exposure.

Life style modifcation
This entails avoiding peak hours of sunlight (in tropics, between 11 AM - 4 PM), using shady side for activities and making use of sunshades like parasols and broad rimmed hats.

Sunscreens
Opaque sunscreens containing zinc oxide, 10% (and SPF of 30) have dual benefit of camouflaging FM and preventing photo-induced darkening. Addition of benzophenones has added benefit.

Avoidance of provoking factors
Avoiding triggers is necessary in melasma, RM, and other causes of FM

Hyperpigmentation Treatments
Therapeutic goals include:

- Inhibiting the formation of melanosomes
- Promoting the degradation of melanosomes
- Retarding the proliferation of melanocytes.
- Sun exposure is an important etiologic factor, therefore all patients should use daily, broad-spectrum, 15 SPF-minimum sunscreens and minimize sun exposure.
- The same treatment principles hold for PIH and melasma.
- Treatment of melasma in pregnant women is routinely deferred until after delivery.

Hydroquinone (HQ) 2%-4%

- Widely used for melasma therapy.
- Patch testing elsewhere on the body, e.g., the upper inner arm, should be done to confirm nonallergenicity prior to attempting a trial of bleaching agents.
- Side-effects include irritant and allergic contact dermatitis, PIH, nail bleaching, and rarely, ochronosis-like pigment.
- Under no circumstances should monobenzylether of hydroquinone or other ethers of HQ be used to treat melasma as they can lead to a permanent loss of melanocytes with the development of a disfiguring confetti-like leukoderma.

Retinoids

- Tretinoin (0.05%-0.1%) reduces pigmentation by inhibiting tyrosinase transcription, and interrupting melanin synthesis.
- Typically takes at least 24 weeks to see clinical improvement.
- May increase pigmentation secondary to irritation.
- May cause erythema and peeling.
- Other retinoids include adapalene, tazarotene and topical isotretinoin

Mequinol:

- A derivative and alternative to hydroquinone is 4-hydroxyanisole or mequinol.
- Although the two agents are related, mequinol is thought to be less irritating to the skin than HQ.
- The drug is available by prescription in a 2% concentration and is typically formulated with 0.01% tretinoin, a retinoic acid and penetration enhancer.
- The mechanism by which mequinol causes depigmentation may involve a competitive inhibition of tyrosinase; however, the exact pathway is unknown.

Arbutin:

- Extracted from the dried leaves of the bearberry shrub or pear, cranberry, or blueberry plants, arbutin is another derivative of HQ, but without the melanotoxic effects.
- Arbutin causes depigmentation by inhibiting not only tyrosinase activity but also melanosome maturation. Although its efficacy is dose-dependent, higher concentrations of arbutin can lead to a paradoxical hyperpigmentation.
- Synthetic forms of arbutin, alpha-arbutin and deoxyarbutin, exhibit greater ability to inhibit tyrosinase than the naturally occurring compound.

Niacinamide:

- Niacinamide is the physiologically active derivative of vitamin B3 or niacin.
- *In-vitro* studies show that niacinamide significantly decreases melanosome transfer to keratinocytes without inhibiting tyrosinase activity or cell proliferation, and niacinamide may also interfere with the cell-signaling pathway between keratinocytes and melanocytes to decrease melanogenesis.
- One of the advantages of niacinamide is its stability being unaffected by light, moisture, acids, alkalis, or oxidizers.

N-acetyl glucosamine:

- N-acetyl glucosamine (NAG) is an amino sugar that is a precursor to hyaluronic acid and is found throughout nature and human tissues.
- Its depigmenting ability originates from the inhibition of tyrosinase glycosylation, a step necessary in the production of melanin.
- Glucosamine itself has been reported to decrease melanogenesis; however, formulating a topical agent has been difficult due to its instability. More recently, focus has now shifted to the development of NAG-containing cosmeceuticals given its greater stability, good skin penetration, and overall tolerability.
- NAG is typically used in 2% concentrations as monotherapy or in combination with niacinamide, which may lead to a greater clinical effect given that there are two different mechanisms of depigmentation at work.

Azelaic Acid (15%-20%)

- A reversible inhibitor of tyrosinase.
- May have cytotoxic and antiproliferative effects on melanocytes.
- Was shown to be as effective as HQ 4% without HQ's side effects
- Adverse effects include pruritus, mild erythema, scaling, and burning.

Kojic Acid (2%)

- Produced by the fungus Aspergilline oryzae and is a tyrosinase inhibitor.
- Generally equivalent to other therapies but may be more irritating.
- May be effective if a patient has difficulty tolerating other first-line therapies..

Ascorbic acid:

- L-ascorbic acid (AA) or vitamin C is a naturally occurring antioxidant obtained from certain fruits and vegetables.
- AA causes skin lightening by interacting with copper ions at the tyrosinase active site and by reducing oxidized dopaquinone, a substrate in the melanin synthetic pathway.
- In addition to skin lightening, other advantages of AA include not only antioxidant effects but some studies also demonstrate anti-inflammatory and photoprotective properties.
- However, early formulations of AA were unstable so esterified derivatives, such as ascorbyl-6-palmitate and magnesium ascorbyl phosphate, were created.
- AA is typically used in 5 to 10% concentrations and can be formulated with other depigmenting agents, such as hydroquinone, which is generally well tolerated in skin of color given the good safety profile of AA.

Licorice:

- Licorice root extract (*Glycyrrhiza glabra*, *Glycyrrhiza uralensis*) is a common ingredient found in many skin-lightening cosmeceuticals, and is also used in the treatment of a wide variety of diseases even outside the scope of dermatology due to its anti-inflammatory, antiviral, antimicrobial, and anticarcinogenic properties.

- Some of the active ingredients in licorice root extract include glabridin, which inhibits tyrosinase and possesses anti-inflammatory effects, and liquiritin, which does not inhibit tyrosinase but causes depigmentation by melanin dispersion and removal.

Soy:

- The activation of protease-activated receptor 2 (PAR-2) cell receptors found on keratinocytes mediates the transfer of melanosomes from melanocytes to surrounding keratinocytes.
- Soy proteins, such as soybean trypsin inhibitor (STI) and Bowman-Birk inhibitor (BBI), inhibit the activation of these cell receptors, and as a result, phagocytosis of melanosomes into keratinocytes is reduced leading to reversible depigmentation.
- Soy is now being formulated alone or in combination with other agents including retinol and sunscreen into cosmeceuticals, particularly moisturizers, to help reduce the signs of photodamage as well as PIH in all skin types.
- **Combination Therapy**
- Combination therapy is more effective than single agents used alone.
- Kligman formula (HQ 5%, tretinoin 0.1% and dexamethasone 0.1%) is the most widely used combination therapy for melasma worldwide.
- The combination of azelaic acid with 0.05% tretinoin or 15-20% glycolic acid may produce earlier, more pronounced lightening.
- Kojic acid 2% combined with HQ 2% was shown to be superior to glycolic acid 10% and HQ 2%.
- Glycolic acid 5% with either HQ 4% or kojic acid 4% for 3 months proved equally effective with reduction of pigmentation in 51% of patients.
- A new combination of HQ 4%, tretinoin 0.05%, and fluocinolone acetonide 0.01% (Tri-Luma®) proved effective with 77% of patients showing complete or nearly complete clearing in this multi-centre, randomized, double-blind, control trial.

Physical therapies

Chemical peels

Glycolic acid (GA), found in sugarcane, is a naturally occurring alpha-hydroxy acid (AHA) that induces epidermolysis, disperses basal layer melanin, and increases dermal collagen synthesis.

- GA concentrations range from 20 to 70%, and neutralization with water or sodium bicarbonate is required to terminate the peel.

Salicylic acid (SA), derived from willow tree bark, is a beta-hydroxy acid that induces keratolysis by disrupting intercellular lipid linkages between epithelioid cells.

- Superficial SA peels utilize concentrations ranging from 20 to 30% without the need for neutralization.
- Superficial chemical peels can also be obtained using trichloroacetic acid (TCA) or Jessner's solution, and both agents have been efficacious in the treatment of melasma.

Dermabrasion:

- Patients with resistant melasma especially with a prominent dermal component have been successfully treated with local or full-face dermabrasion upto upper or mid dermis using a 16-mm diameter coarse grit diamond fraise with 97% of the patients maintaining the improvement for a mean of 5 years.
- Less than 1% patients developed hypertrophic scars or permanent hypopigmentation.

LASERS:

- Several lasers have been reported to be effective in patients with FM, they should not be the first line therapy (unpredictable response, frequent relapses despite initial improvement, risk of postinflammatory hyper and hypopigmentation).
- Lasers may, however, be used in selected resistant cases, after proper counselling and preferably after a test patch.
- A combination of pulsed CO2 laser (to remove epidermal pigment) and alexandrite laser

(to remove dermal pigment) gives a better reduction than with either used alone.

- Q-switched alexandrite laser has also

been effectively used in other causes of FM in combination with 15- 25% TCA and Jessner's solution.

- Similarly Erbium: YAG laser (2.94µ at 5.1 to 7.6J/cm2) has been found effective women with melasma unresponsive to topical therapy and chemopeeling.

Cosmetic camouflage:

- Cosmetic camouflage may be useful to conceal pigmentary disorders, vascular lesions, scars, and chronic skin conditions that are not amenable to medical or surgical treatments.
- These coverage techniques can help alleviate the patient's distress regarding their appearance and significantly improve quality of life.
- Camouflage can be particularly useful in darker skinned individuals where pigmentary changes may be more noticeable and when highly visible parts of the body are affected by the disease, such as the face, neck, and hands.
- The characteristics of a good cosmetic cover include a natural appearance and non-greasy feel, and the cover should also be waterproof, long lasting, and noncomedogenic with easy application.
- There are four basic foundations: oil-based for dry skin, water-based for dry-to-normal skin, oil-free for oily skin, and water-free, which mixes oils with waxes to form thicker creams that can incorporate higher amounts of pigment to match the patient's normal skin color.
- Cosmetic covers can be applied for subtle coverage up to full concealment, and color correctors use the lesion's color opposite to neutralize its intensity.

Emerging therapies:

- Demand for newer, more effective depigmenting agents, hence continued research is constantly fueled.
- Undecylenoyl phenylalanine 2% has been shown to safely and effectively treat solar lentigines in a recent randomized, double-blind, vehicle-controlled clinical trial.
- There have also been case reports and clinical studies that have shown that topical 5% methimazole, aloesin, and dioic acid can successfully treat hyperpigmentation secondary to a variety of etiologies.
- Other agents that have shown some depigmenting properties but require further research include 4-(1-phenylethyl)1,3-benzenediol, paper mulberry, ellagic acid, quinolines, piperlonguminine, luteolin, calycosin, emblica, and multivitamins.

CONCLUSION

Management of FM is challenging requiring withdrawal of the 'trigger', vigorous use of sunscreens, an array of depigmenting agents of which HQ is considered gold standard. Although in vivo and in vitro studies have shown the efficacy of several other agents, their place in the management of FM is still not defined.

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C. Eczema Herpeticum: a less known dermatological emergency

- Dr. Sahana

Abstract

Eczema herpeticum also known as Kaposi's varicelliform eruption is an extensive cutaneous vesicular eruption, caused by herpes simplex virus (HSV) arising from a pre-existing skin disease, most commonly atopic dermatitis. We report a 5 year old male child with eczema herpeticum which was misdiagnosed as impetigo. It is a dermatological emergency as it can lead to disseminated infection and death if not treated early.

Key words: Eczema herpeticum, herpes simplex virus, acyclovir

Introduction

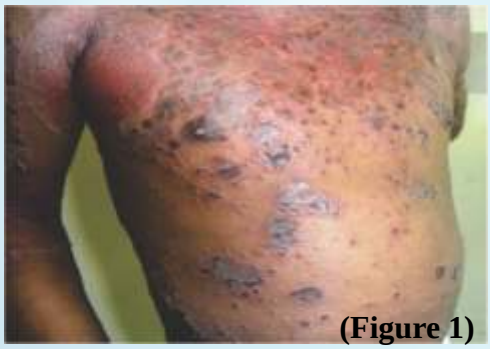
Eczema herpeticum also known as Kaposi's varicelliform eruption was initially described by Moriz Kaposi in 1887. He described a widespread eruption of lentil shaped vesicles in patients with pre-existing dermatitis. [1] All reported cases of childhood eczema herpeticum are caused by HSV-1 infection. The term eczema herpeticum was proposed by Lynch in 1945, and to avoid confusion it is now used to describe only to cutaneous HSV infection of atopic dermatitis. [2] Eczema herpeticum is potentially life threatening complication. We report a 5 year old male child who was diagnosed as eczema herpeticum in a background of chronic atopic dermatitis.

Case report

A-5-year old male term child presented with fever, multiple blisters with erosions, crusting and excoriations on trunk and extremities from 3 days. The lesions were painful. He was a known case of chronic atopic dermatitis from infancy. The child was on regular treatment with petroleum jelly, topical steroid and tacrolimus with moderate improvement of atopic eczema. Initially the child was diagnosed as impetigo and treated with antibiotics but there was no improvement.

On examination the child had fever with a temperature of 38.6oc. Other vitals were normal. Cutaneous examination revealed multiple vesicles, erosions and punched out ulcers on chest, back, extensor and flexor aspect of upper limbs, thighs and lower legs [figure 1 and2]. Few erythematous scaly plaques were seen on flexures and extremities. Laboratory investigations showed normal hemogram. Serum chemistry profile was normal. Tzanck smear from the vesicular fluid showed multinucleated giant cells [figure 3]. Virus isolation was not done as the patient refused.

Based on clinical features and a positive tzanck test a diagnosis of eczema herpeticum was considered. The child was started on oral acyclovir with complete resolution of lesions within 10 days [figure 4].



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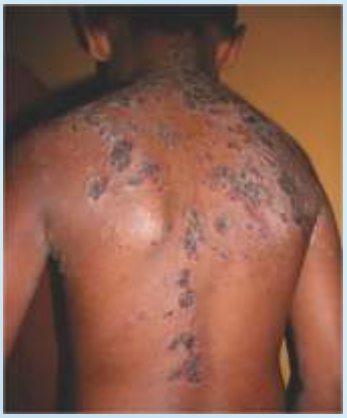

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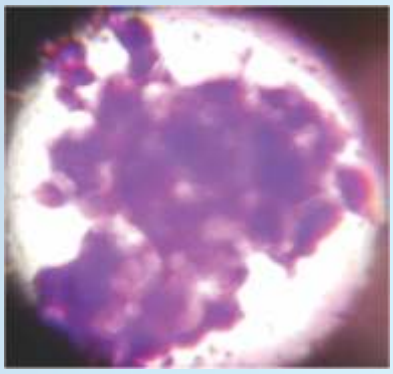

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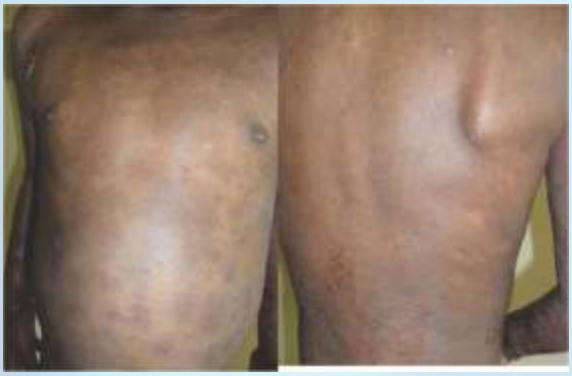

(Figure 4)

Discussion

Children with atopic dermatitis have an impaired skin barrier that predisposes to recurrent bacterial and viral infection of the skin. Eczema herpeticum occurs as a rare complication and is seen in less than 3% cases of atopic dermatitis. Eczema herpeticum usually occurs as a primary HSV infection in children. It is seen in other chronic skin conditions like psoriasis, ichthyosis vulgaris, burns, seborrheic dermatitis, pemphigus, darier disease and others. [3]

The pathogenesis is still not clearly established. It is postulated that patients with atopic dermatitis seem to have a predisposition to HSV infection because of defects in specific proteins necessary for skin barrier function and innate immunity. Recently it seen that there is decreased natural killer cells which allows HSV to proliferate to have a suppressive effect on immune mechanisms like IL-2 receptors resulting in disseminated HSV infection. [4] Early onset of atopic dermatitis, extensive skin involvement, eczematous lesions on head and neck and high immunoglobulin E levels are associated with increased risk of eczema herpeticum. [5] Clinically it presents with abrupt onset of fever, malaise, lymphadenopathy with widespread monomorphic dome shaped vesicles, papulovesicles, pustules rupturing to form punched out ulcers on an erythematous base most commonly on the face, trunk and extremities. Secondary bacterial infection can occur which often leads to misdiagnosis. Complications like keratoconjunctivitis, fluid loss, viremia and rarely death can occur. [6]

It is often difficult to recognize the infection with severe atopic dermatitis. Cutaneous herpes infection cannot be easily distinguished from eczema infected with staphylococcal aureus or streptococcus. Diagnosis can be confirmed by tzanck test by the presence of multinucleated giant cells which is a simple bed side test. Other diagnostic modalities include viral culture, PCR,

direct immunoflorescence, histological examination and serology. [7]

Eczema herpeticum must be differentiated from impetigo, eczema vaccinatum and primary varicella infection. It is often misdiagnosed as impetigo as it happened in our case. Systemic acyclovir 30mg/kg/day for 5-7 days is the treatment of choice. [8] Prognosis is good if treated in the first few days of illness. Mortality is often due to delay in diagnosis. Recurrences are seen in 20% of children. [9]

Atopic dermatitis is a very common disorder seen in dermatology outpatient department. High index of suspicion must be present to examine an atopic dermatitis child developing secondary herpes infection. Early recognition of this condition and timely treatment can prevent complications.

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D. Sensitive skin: An approach for diagnosis and management - Dr. Prabhakar M. Songolli

Sensitive is tolerant to prolonged use of cosmetics and toiletries. Synonyms include intolerant skin reactive skin, irritable skin .Subjective symptoms appear within minutes of application of a cosmetic in the form of pricking, tingling, burning .

Two classifications are popular

1. **Pons Guiraud** classification: Very sensitive, environmentally sensitive and cosmetically sensitive
2. **Muiziddin** classification: Delicate skin, reactive skin and stingers

Sensitive skin is common among young adults especially in females with fluctuations during cycles. Common in Asian population .Face especially **naso labial** folds are the commonest region. Hygienic factors contribute to genital involvement. Harsh winter,air conditioning are contributory factors. Prolonged use of **topical steroids** aggravate the problem. Atopic individual have more incidence of sensitive skin.

Thin stratum corneum, increased sweat,more of neutral lipids, increased epidermal innervations **high baseline TEWL** all play an important role in this common condition

Epidermis has increased **nerve receptors** over keratinocytes and nerve endings which release more neurotransmitters with unique processing in cortex. These include TRP,ET(Pain),heat and cold (TRPM8,TRPA1) receptors. Physical factors interact with these receptors to evoke symptoms.

Skin in patients with atopic dermatitis, acne and rosacea have impaired barrier function which plays key role in sensitive skin

Clinically sensitive skin is dry, less supple, erythematous and has more telangiectasiases.

Testing for sensitive skin

1. Test for development of sensitive skin: Eg;**Lactic acid** sting test on nasoloabial fold
2. Bio engineering tests to measure skin response.: Eg:**Evaporimetry** to measure TEWL
3. **In vivo** testing on sensitive skin population: Eg - Cumulative irritancy test
4. **In vitro test** to demonstrate irritancy: Collagen swelling test

Management of Sensitive skin

Ideal product to be used in sensitive should be free from potent irritants and sensitizers. Vehicle like propylene glycol should be substituted with poly ethylene glycol(PEGs) and non irritant surfactant is to be used

Powder cosmetics which is easily washable with water is preferred. Fresh cosmetics with less than 10 ingredients are chosen. Inorganic sunscreens are better tolerated. Nail polishes are to be avoided.

All cosmetics are to be patch/photo patch tested. Sting test is carried out. Usage test is done 2 cms

later to outer canthus. One cosmetic is introduced per week.

Since 50 % of patients complain of sensitive skin efforts are on to find molecules to neutralize irritation.One such chemical is trans 4 tert-butylcyclohexanol which neutralizes capsacin induced burning .Bacterial lysate of Bifidobacterium longum reduced irritation human volunteers which is an encouraging finding

Since sensitive facial skin is a common problem, research is going on to design least irritant cosmetic acceptable which provokes neural receptors minimally to majority of patients

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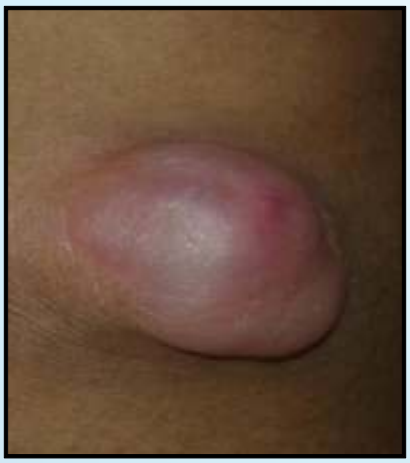
E. Case report with Quiz

Dr. Praveen Kumar S., Dr. Nethavathi A.R., Dr. A.L. Shyam Prasad, Dr. T.K. Sumathy
M.S. Ramaiah Hospitals

A 35 year old male presented with a solitary, asymptomatic swelling on the medial aspect of left arm since 6 months. On clinical examination a solitary fleshy firm, non-tender 5x5 cms swelling, was present over the medial aspect of the left arm(Fig. 1).There was no history of any preceeding trauma. Excision biopsy with histopathological examination showed a well encapsulated tumor in the dermis (Fig.2). The cells in tumor islands were basaloid in the periphery with enucleated cells present in the centre (Fig. 3) there was no evidence of calcification.

The patient was otherwise healthy and there was associated no co-morbities. Other routine laboratory investigations were within normal limit.



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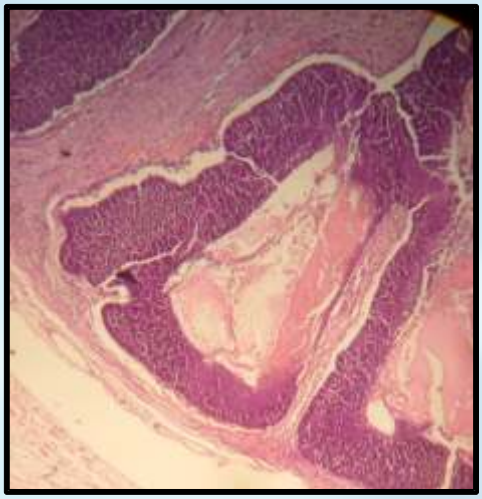

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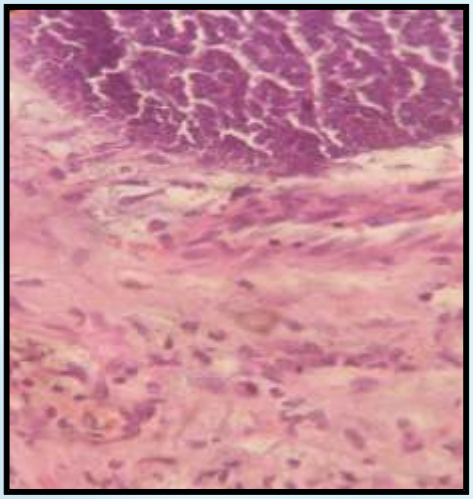
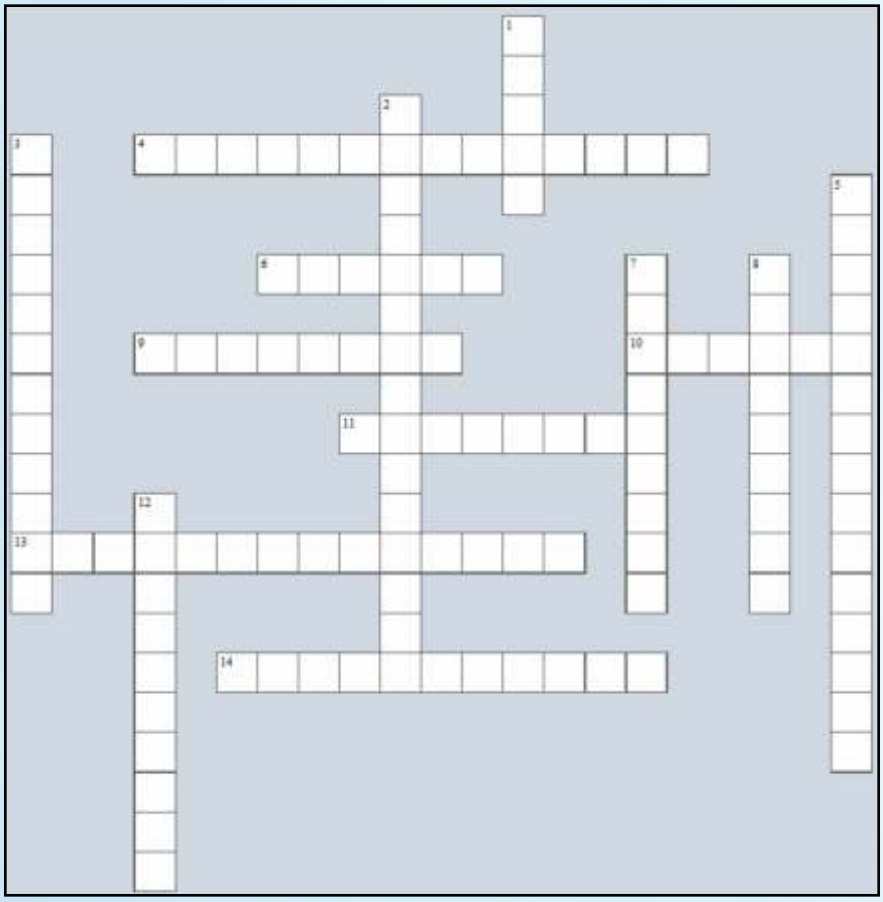


(Figure 3)

(Answers to Quitz on Page No.24)

F. Crossword, Dr. K.N. Shivaswamy



Across

4. Splashes of coffee in milk having distorted border (6,8)
6. I have a leaf on the back and garlic in the nail, can't pass exams (6)
9. I grow bamboo and dislike retinoids (8)
10. I can't see, can't stand, can't hear but have thick nerves (6)
11. Lines on the skin where naevi and dermatoses follow (8)
13. Multiple trichoepitheliomas (6,7)
14. Dirty fellow with hand full of grains (6,5)

Down

1. Fusion of vertebral bodies and acne (5)
2. PPKD with periodontitis (8,7)
3. Tons of fibrofolliculoma, trichodiscoma and fibrous papule over face and neck (4,4,4)
5. I have mickey mouse ear and can't come out during day light (8,6)
7. Number and arrangement of chromosomes (9)
8. Two or more cell lines derived from a single zygote (9)
12. Difficult to eat with hands (keratoderma) and difficult to swallow (5,5)

(Answers to Crossword on Page No.25)

G. DERMACARTOONS

Dr. Namrata (Resident Dermatology, M.S. Ramaiah Memorial Hospital

A)

B)

(Answers to Quitz on Page No.26)

F. Answers to Case report & quiz - Pilomatricoma

Discussion:

Pilomatricoma (synonyms : Benign calcifying epithelioma of Melherbe, Trichomatricoma, Pilomatrixoma) is a benign tumor originating from outer root sheath cells or hair matrix cells.Mutation in CTNNB1gene is present, indicating misregulation of beta catenin gene.

Clinical features:

It may present from infancy, but commonly seen in patients below 20 years of age, with the female preponderance. Familial cases are recorded.

Clinicaly presents as a solitary, dermal or subcutaneous tumor, usually 3-30mm, present over the head, neck or upper extremities.Palms, soles and genital region are usually spared.It is firm to stony hard in consistency with skin over the swelling being normal. Histologically, tumor islands are present in the deep dermis and subcutaneous tissue with basaloid cells arranged in the periphery and towards the centre, there are enucleated "ghost cells" or "shadow cells". Calcification can be noted. Malignant transformation is rare.

Surgical excision is the treatment of choice.

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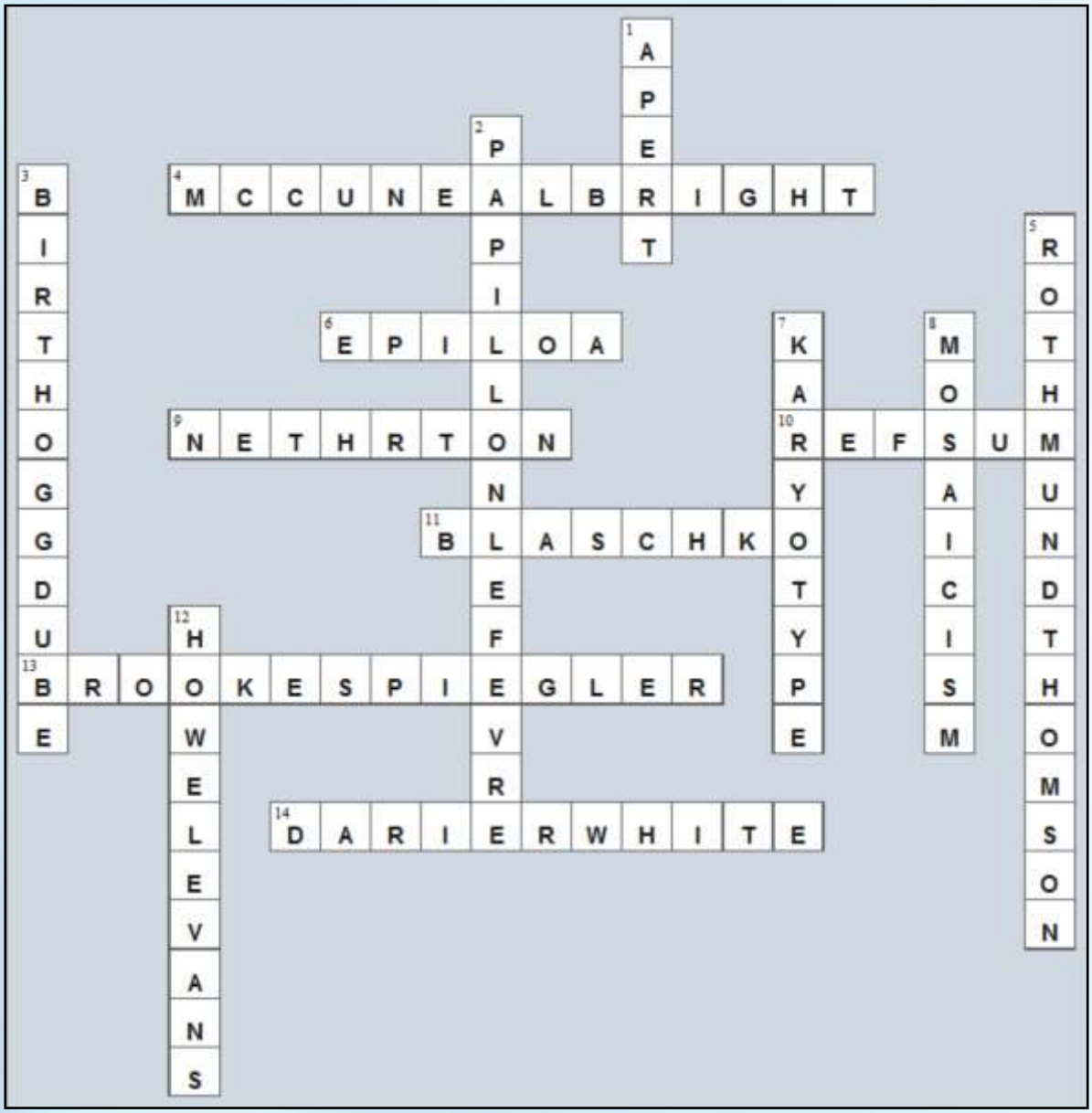
Answers to Crossword



Answers to Dermacortoons

UGLY DUCKLING SIGN

In 1998, Grov et al introduced the ugly duckling sign / concept, the observation that all naevi in the same indivdual look alike.

Only when one of them develop into a maligmant melanoma, it looks distinctively different from other naevi.

DECK CHAIR SIGN / FOLDED LUGGAGE SIGN

Seen in papulo erythroderma of Offuji

When the rash spans the creases of the skin as if the person has sat in the sun on a deck chair

H. NEWS & EVENTS

CME on DERMA BASICS on 4th August 2013
at M S Ramaiah Medical College

The department of Dermatology M S Ramaiah Medical College in association with IADVL Karnataka branch had organized one day CME (DERMA BASICS) for postgraduate students on Basics of Dermatology, first of its kind, at M. S. Ramaiah Memorial Hospital auditorium on Sunday 4th August 2013.

There was a good response for the CME from postgraduates from all over the state and a few practicing dermatologists as well, amounting to a total of 90 delegates.

Dr. A. C. Ashok, Principal and Dean MSRMC the chief guest, Dr. Venkataram Mysore, president of IADVL KN branch, Dr. B. S Chandrashekar, Secretary IADVL KN branch, Dr A L Shyam Prasad and Dr T K Sumathy were the dignitaries on the dais.
The scientific session started at 9am and was focused mainly on basics of dermatology for postgraduate students. Both in house and outside speakers have transferred their knowledge to postgraduates with an active participation from students as well.

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