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REVIEW ARTICLE

A Review on Various Aspects of Facial Skin Disorders

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ABSTARCT
Skin diseases endanger patients' well-being, mental health, capacity to function, and social participation, a measure of disability defined generally by the WHO as a person's ability to be involved and engaged in interpersonal relationships. Dermatologic QoL is particularly worse for members of vulnerable populations, such as the elderly, women, and children. Seniors were far more likely to be disabled as a result of a skin issue. A working classification was adopted to discuss the most prevalent facial dermatoses in our study and the prevalent dermatoses affecting the facial skin were categorized as disorders of pilosebaceous unit, eczema, hypersensitivity, infections, miscellaneous, pigmentary disorders, combination, tumours. Of all the aforementioned disease classes, the most common facial dermatoses were of infectious origin which constituted 21.7% cases which is in accordance with the cross-sectional study conducted by in Pigmentary diseases and hypersensitivity disorders.
Keywords: *Skin disorders; Dermatoses; Population study; Infectious disease; Hypersensitivity.*

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INTRODUCTION

Hypermelanoses are a category of diseases marked by unusually darker skin caused by increased melanin synthesis from a normal amount of melanocytes [1].Increased melanin in the epidermis can cause epidermal hypermelanoses, that in the dermis can cause dermal hypermelanoses, or both - mixed hypermelanoses. In dermal hypermelanoses, melanosomes are generated in the epidermis by epidermal melanocytes and transported to the dermis, where they are usually located inside macrophages (melanophages). The term for this occurrence is 'epidermal melanin incontinence' [2].The majority of facial melanoses are more frequent in darker skin. The role of light and photosensitizing compounds is critical.

PERIORBITAL MELANOSIS

Periorbital hyperpigmentation, which is also known as Idiopathic cutaneous hyperchromia of the orbital region (ICHOR), periorbital melanosis, dark circles, or infraorbital pigmentation are more common in darker people. It might be caused by primary or secondary causes. Secondary periorbital hyperpigmentation is generally caused by a combination of factors, including dermal melanocytosis, hereditary or constitutional pigmentation, post-inflammatory hyperpigmentation (PIH) secondary to atopic and/or allergic contact dermatitis, periorbital edema, and increased subcutaneous vascularity, age-related shadowing caused by skin laxity and tear through, excessive sun exposure, medicines, hormonal reasons, and pigmentary demarcation lines extension. ICHOR is distinguished by darkening of the orbital skin and eyelid on both side, which is not caused by systemic or local disease [3-5].

The vascular type (41.8%) of periorbital hyperpigmentation, which is defined by the presence of erythema affecting the inner aspects of lower eyelids, with prominent capillaries/telangiectasia or blue discoloration owing to prominent blue veins was found to be the commonest cause of peri-orbital pigmentation in a study conducted by Ranu *et al* [6] on 200 patients; constitutional form (38.6%) characterized by brown-black hyperpigmentation of the lower eyelid skin following the contour of the

orbital rim, PIH (12%) and shadow effects due to an overhanging tarsal muscle or a deep tear trough for11.4 percent of the cases. Earlier research by Watanabe et al. and Malakar *et al*. studied skin biopsies from the lesions and discovered that the dermal melanocytosis and melanin pigment in upper dermal macrophages, partially explains the condition's resistance to various therapies [9].

MELASMA

Melasma (a name derived from the Greek word melas, which means black) is a type of common acquired hypermelanosis that affects only sun-exposed regions of the body, mostly the face but also the neck and forearms.Melasma affects women more than males. According to a study done by Nouveau S et al in 1204 Indian women in 2016 found that, melasma occurred in 20–30% of 40–65 years old women. According to reports, men account for 10% of cases and exhibit the same clinical and histopathological features as women [11]. Melasma affects people of all races, however it is more prevalent in people having darker complexions of (Fitzpatrick's skin types IV–VI) Hispanic, East Asian and people of Southeast Asian ancestry who dwell in places with high levels of ultraviolet radiation (UVR) [6-9].

The specific aetiology of melasma is unknown, although two most common factors- Sunlight and genetic variables have been linked to its etiopathogenesis. Melasma flares up after a period of sun exposure, and it diminishes over time when the sun is avoided. As evidenced by familial involvement and greater rates among Hispanics and Asians, genetic factors also play a crucial role. Other risk factors include pregnancy, thyroid dysfunction, oral contraceptives, estrogen- progesterone treatments, cosmetics, phototoxic, and anti-seizure medications. Melasma is characterised by hyperpigmented macules that are symmetrical. It can be blotchy, uneven, arcuate, or polycyclic and has a linear pattern on rare occasions or a starburst like distribution.

Melasma comes in a variety of clinical forms and patterns are mixed in most of the patients.The most prevalent pattern is centrofacial, which consists of lesions on the forehead, cheeks, nose, upper lip, and chin. The cheeks and nose are the most commonly affected areas in the malar pattern. The mandibular pattern is made up of lesions on the mandible's ramus. It's still unknown why some parts of the face are more likely to develop lesions while others aren't. Hormone receptors and blood vessels play a role, but other variables such as sebaceous gland density and activity, phototoxicity and antioxidants are also involved [10]. Grichnik *et al* discovered that a stem cell factor can boost melanocyte number, size, and dendricity [11].

LICHEN PLANUS PIGMENTOSUS

Lichen planus pigmentosus (LPP) is a rare lichen planus variant that mostly affects young to middle-aged individuals with skin phototypes III–V [12-14]. It is distinguished by focal or merging gray-blue patches or faint plaques that appear on exposed parts, particularly the neck and surrounding thorax. It's usually symmetrical on both sides [15]. According to a study done by Tasleem Arif *et al* in 2017 , LPP was seen in 525 (7.37%) patients out of 7124 patients.LPP can occasionally resemble melasma. Some patients have complained about photosensitivity. Because the hyperpigmentation is mostly dermal, the lesions do not amplify under the Wood's light.If palpable lesions are present and sampled, LPP, as a subset of lichen planus, may demonstrate typical lichenoid pathology. A macular lesion with a relatively flat epidermis, lack of rete pattern, localised basal cell vacuolization with occasional necrotic keratinocyte and civatte body, dermal melanophages, and a sparse lymphohistiocytic inflammation is seen histopathologically. Squamatized epidermis foci are occasionally seen, indicating prior vacuolar injury to the basal layer in a rather old lesion. Histology of LPP shows sparse inflammation only, necessitating a comprehensive examination of the sample. As a result, clinicopathological correlation is used to diagnose LPP [15].

EPHELIDES OR FRECKLES

Ephelides are little pigmented macules that appear on the face and, to a lesser extent, other sun-exposed parts of light-skinned people. Ephelides are irregular, distinct, little macules that are commonly spread throughout the nose and malar regions and are around 1-2 mm in size. The pigmentation changes with the seasons and the amount of sun exposure [16]. According to a study done by TasleemArif et al in 2017, ephelides was found in 491 (6.89%) patients of 7124 patients.The basal epidermis is hypermelanized, with a normal epidermal architecture, according to histological findings. The number of melanocytes has not increased. In comparison to normal skin, electron microscopy investigations have revealed enhanced melanin synthesis by the damaged melanocytes [16].

VITILIGO

The oldest credible mention to the ailment may be found in the classic Tarikh-e-Tib-e- Iran from the

Aushooryan period (2200 BC) [17]. Pharonic medicine in the Ebers Papyrus (1550 BC) reported two forms of disorders altering skin colour: (a) tumours, most likely leprosy, and (b) just colour change, most likely vitiligo. The latter was thought to be curable [18]. Vitiligo is a frequent, acquired skin discoloration characterised by well-defined ivory or chalky white macules that are flush with the skin's surface. Although vitiligo can affect any portion of the skin or mucous membranes, the illness has a preference for typical hyperpigmented areas such the face, groins, axillae, aerolae, and genitalia. Furthermore, as a result of Koebner's phenomenon, lesions may form in other locations such as the ankles, elbows, and knees, which are susceptible to recurrent injury / friction. [22] Vitiligo is divided into four types: focal, segmental, generalised, and universal, according to a standard classification system [19-22]. Lerner divided vitiligo into three categories:a) segmental, localised, partial, or focal vitiligo corresponding to a dermatome / adjacent dermatomes; b) vitiligo vulgaris generalised, involving the hands, face, axillae, and limbs; and c) complete, total, or universal vitiligo spanning the entire or nearly the whole of the body surface. Pityriasis versicolor, pityriasis alba, nevus depigmentosus, idiopathic guttate hypomelanosis, postinflammatory hypopigmentation, tuberous sclerosis, Waadenberg's syndrome, leprosy, leukoderma, piebaldism and Vogt-Koyanagi-Harada syndrome are some of the differential diagnoses for vitiligo [23-28].
Vitiligo is treated with a variety of topical and systemic drugs, as well as phototherapy, laser therapy, and surgical therapy. Corticosteroids, calcineurin inhibitors, and vitamin- D analogues are examples of topical treatments. Phototherapy is a successful therapeutic method. Narrowband UV-B with a wavelength of 311-312nm is commonly utilised, usually two to three times a week. The excimer laser is used to treat vitiligo patches that are small and stable. Tacrolimus and systemic corticosteroids can be used together to treat segmental vitiligo, which is resistant to most therapies [29]. Surgical treatment options are used for segmental or localized vitiligo that is restricted to a small area. Vitiligo is one of the most prevalent depigmentary skin disorders affecting approximately 0.5 to 1% of the population [30].

NEVUS ANEMICUS

Nevus anemicus (NA) is a vascular abnormality characterised by hypopigmented confluent and mottled macules or patches [31]. It usually involves the trunk, but lesions on the face and extremities have been observed. The prevalence of nevus anemicus has been estimated to be 1% to 2% in India. It may have some associated conditions [32].Other hypopigmented lesions such as nevus depigmentosus (ND), ash-leaf macules, hypomelanosis of Ito, vitiligo, pityriasis alba, and leprosy must be distinguished clinically. Diascopy with a glass slide shows blending of the borders of the lesion with the surrounding normal skin. On Wood's lamp examination, no accentuation will be seen. Dermoscopic features of this condition are specific, making diagnosis easier, which are reduction in blood vessels in the lesional skin with diffuse erythema and linear telangiectatic vessels in the surrounding skin [35]. For naevus anaemicus, which is a mild and benign condition, no therapy is required or accessible. If required, cosmetic camouflage can be applied [33, 34].

DISORDERS OF PILOSEBACEOUS UNIT ACNE VULGARIS

It is a chronic inflammatory disorder of the pilosebaceous unit that affects about 85% of young individuals between the age group of 12 and 24 years [35, 36]. Acne is more common in males later in adolescence and is more serious also. Females have a milder, but still persistent, condition. The causes of acne vulgaris are multifaceted, and the following variables influence acne severity: Kurokawa et al. gives the opinion that high-glycemic index food and milk increase the tissue level of DHT; hence, this group of food items has an influence on exacerbation of acne lesions. 70% of females are affected by acne flare-ups before or sometimes even during menstruation [37]. In adults and female patients aged > 25 years suffering from persistent acne, polycystic ovarian syndrome (PCOS) [38] and other endocrine disorders play a major role. Acne affects the regions of skin with the maximum number of sebaceous follicles, with prevalence and severity rates of 92 %, 45 %, and 61 %, respectively, on the face, chest, and back [39]. Acne affects the cheeks the most on the face, followed by the nose, the forehead, and the chin. Other areas affected are ears, pre-auricular areas, retro-auricular areas, and even neck.

Table 1. Comparison of acne distribution for different age groups

Age group	Location of lesions	Type of lesions	Sex
Neonates	Cheeks, chin, eyelids, forehead	Papules and pustules, no comedones	Both
Infants	Full face	Comedones, papules, nodules, scars	Male
Preadolescent	Forehead, upper cheeks, nose	Predominanthly comedonal, occasional papule	Both
Adolescent	Full face, seborrheic areas of torso	All types of lesions	Both
Adults	Chin, upper lip, jaws	Papules, excoriated papules	Female

Major clinical signs of acne vulgaris are greasy skin, seborrhea and presence of comedowns [43]. Persistent acne and late-onset acne are the two kinds of adult acne. Persistent adult acne is a kind of adolescent acne that lasts longer than 25 years. Late- onset adult acne is that acne which appears for the first time after the age of 25 [40,41].Different types of comedones are open comedones, closed comedones, sand paper comedones, macrocomedones, secondary comedones, submarine comedones [42].

TABLE 2 : CLINICAL GRADING SYSTEM OF ACNE (PILLSBURRY'S CLASSIFICATION)

GRADING	DESCRIPTION
Grade-1 (Mild)	Open or closed comedones, occasional papules or pustules without scarring.
Grade-2 (Moderate)	Comedones, papules, few pustules; with mild scarring.
Grade-3 (Severe)	Predominantly pustules, nodules or abscesses with moderate scarring.
Grade-4 (Very severe)	Predominantly cysts, abscesses or scars; with severe scarring

SENILE COMEDONES

Senile comedones, is also referred to as FRS (Favre Racouchot Syndrome) [43-45]. It is characterized by the presence of a diffuse yellowish hue with the presence of large, open comedones symmetrically distributed on the temporal and periorbital areas of the face [46]. The differential diagnoses are milia, syringomas, trichoepitheliomas [47].But, HPE is distinctive [47].For treatment, we can combine both medical and surgical modalities. Surgical techniques include comedone extraction, simple or multiple-stage excision, curettage, laser resurfacing and dermabrasion [48], In the study done by Gaurav et al senile comedones were found in 10.6% of 540 patients.

INFECTIONS

TINEA FACIEI

Trichophyton mentagrophytes and Trichophyton rubrum are the organisms that cause Tinea Faciei. Infections spread either directly from organisms or indirectly from pre-existing lesions in other parts of the body. Erythematous patches and limited scaling with imprecise boundaries were found in the patients. There may or may not be typical annular lesions present. A minor photosensitivity of the lesional region has been observed [49]. So can be confused with LE and PMLE. KOH examination and biopsy are used to diagnose it because of its variable shape over the face. The major pathogens of tinea faciei in children are zoophilic dermatophytes, such as zoophilic strains of *T. interdigitale*, *M. canis*, and Trichophyton species of Arthrodermabenhamiae [50]. According to a study done by Nagaral et al in Chithradurga in 2019, it was found that among 426 subjects of dermatophytosis, the prevalence of Tinea facei was 8.6%.

TINEA VERSICOLOR

Malassezia, a dimorphic fungus, causes this widespread fungal illness. Pityriasis versicolor, Tinea flavea, Liver spots, and Chromophytosis are some of the other names for it. 1% to 50% is the prevalence seen [51]Dark-skinned folks show it more. M. globosa, M. furfur, and M. sympodialis are the most common causes [52].Lesions appear less pigmented as the fungus-secretes tyrosinase inhibitors, while hyperpigmentation is caused by fungus- induced melanosome expansion. Scales stained with KOH reveal fungal hyphae and many spores, giving them a spaghetti and meatballs resemblance. In vitiligo, there is no scaling or surface changes, only total pigmentloss. Children have been shown to have more cases of facial TV [53, 54].

IMPETIGO

There are two types of clinical patterns of impetigo: bullous and nonbullous. S. aureus is the cause of

bullous impetigo. In developing countries, Group a Streptococcus is still the most prevalent cause of nonbullousimpetigo. Nonbullous Impetigo: It accounts for more than 70% of all cases of this kind of pyoderma. It can affect both children and adults of various ages. The first lesion is a transitory vesicle or pustule that swiftly transforms into a honey-colored crusted plaque with a diameter of more than 2 cm. There may be erythema in the vicinity. Bullous impetigo is characterized by the fast transition of vesicles to flaccid bullae and is more prevalent in newborns and older infants. The Nikolsky sign is missing. Bullae have a clear yellow fluid that becomes dark yellow and murky with time, and their margins are well defined without an erythematous halo. The bullae are shallow, and they rupture and disintegrate after a day or two, sometimes creating thin, light-brown to golden- yellow crusts [55].

MOLLUSCUM CONTAGIOSUM

Molluscum contagiosum (MC) is a viral skin illness caused by the molluscum contagiosum virus (MCV), a DNA virus belonging to the Poxviridae family and the poxvirus genus. The time it takes for MC to incubate varies from 2 weeks to 6 months. 61 According to a study conducted by Karthikeyan *et al* in South India, it was found that 54 (2.5%) of 551 cases were MC cases. A lesion begins as a small papule and grows to a diameter of 5–10 mm in 6–12 weeks. Small lesions might clump together to form a plaque (agminate form) [56]. The number ranges from a few to a large number. In an adult, the occurrence of many lesions indicates immunosuppression. Discrete, dome-shaped, umbilicated, waxy papules are the most common lesions. They come in a variety of colours, including flesh tones, pink tones, and white tones. In all well-formed lesion, a central punctum may be seen. The manner of infection, the kind of clothing worn, and the climate all affect the distribution [57].

HERPES LABIALIS

Infections with the herpes simplex virus (HSV) are widespread all over the world and are caused by two closely related strains of HSV. Mucocutaneous infections are the most common clinical symptoms, with HSV type 1 (HSV-1) being predominantly linked with orofacial illness and HSV type 2 (HSV-2) with genital and perigenital infection [58].Primary oral herpes symptoms include ulcerative lesions involving the hard and soft palate, tongue, buccal mucosa, and surrounding tissues, which are similar to those of aphthous stomatitis. Fever, malaise, salivation, myalgias, ache when swallowing, irritability, and cervical adenopathy are all prevalent symptoms [58].The perioral facial region, namely the lips, is impacted by reactivation of virus from these primary infections, with the outer one-third of the lower lip being the most usually affected. The nose, chin, and cheeks are impacted in fewer than 10% of instances [58].The typical herpes lesions have been categorised into phases depending on their characteristics: prodromal, erythema, and papule (developmental stages); vesicle, ulcer, and hard crust (disease stages); and dry flaking and residual swelling (resolution stages). Within 5–15 days, the lesions normally disappear [58].Emotional stress, sickness, sun exposure, trauma, exhaustion, menses, chapped lips, and seasonal fluctuations, trigeminal nerve surgery, oral trauma, epidural morphine injection, and abrasive, laser, and chemical facial cosmetic treatments are all known to trigger oral herpes recurrences [58].

WARTS

Warts are a benign skin growth caused by infection with the human papillomavirus. More than a hundred serotypes have been discovered. Flat warts, produced by serotypes 3,10,28, and 49, are the most prevalent clinical type of facial wart. According to a study done by Sruthi et al in 2016, plane warts was seen in 18% cases and filiform warts in 4% cases out of 100 cases. Filiform warts on the face and neck are frequent in males, and they are unevenly distributed and sometimes grouped. They might also manifest themselves on the scalp [59]. Plane warts are quite prevalent, particularly among children. They are occasionally found in adult women, but they are uncommon in men. The face, dorsum of the hand, and shins are the most typically affected areas in clinical practise. They are frequently numerous and have a flat topped smooth textured papule with a light brown tint. The phenomena of Koebnerization can be observed [60].

HERPES ZOSTER

The reactivated varicella zoster virus causes a segmental painful skin condition [61]. The virus stays latent in the dorsal root ganglia after the first chicken pox infection, and reactivation causes herpes zoster. Shingles are another name for it. The word zoster means girdle. People in their forties and fifties are the most typically afflicted, with a small female predominance. Herpes Zoster occurring in the elderly is common and is estimated to be 1,000,000 cases annually, which increases over the age of 60 years. Trigeminal dermatome is present in 15% of instances. Pain and paraesthesia are frequently the

presenting complaints of the patient. Then erythematous patches, purulent and crusty vesicles develop. Nasocilliary involvement is indicated by a vesicle on the side of the nose. In Herpes Zoster Ophthalmicus, the patient suffers ocular problems as well as unilateral forehead involvement.

ECZEMA

SEBORRHEIC DERMATITIS

Seborrheic dermatitis (SD) is a chronic inflammatory skin disease that affects 1– 3% of the adult population. SD can be provoked by emotional stress, despair, exhaustion, or seasonal changes (more during winter seasons). Sunlight has shown to both benefit and worsen SD. It is caused by yeasts from the Malassezia genus [61, 62]. Infantile and adult seborrheic dermatitis are the two kinds of seborrheic dermatitis. The infantile form is usually seen between 3 - 8 months of age. SD is commonly characterised by poorly defined scaling and erythematous patches. The level of erythema, greasy scale, and pruritus varies, and in darkly pigmented people, the affected skin may seem hypopigmented. The nasal sidewall, nasolabial and melolabial folds, eyebrows, glabela, scalp, and central chest are among places where sebaceous gland activity is significant. Male patients are more likely to get the condition than female patients, and it is most common in newborns, adolescents, and people over the age of 50. Immunocompromised patients have a greater rate of occurrence, ranging from 30% to 83 % in HIV-positive and AIDS patients, and 18–50 % in Parkinson's disease patients [63].

ATOPIC DERMATITIS

Atopic eczema (AE, or atopic dermatitis) is a pruritic, persistent, recurrent eczematous eruption that commonly begins in childhood [64]. The etiology of AE is unknown, however they are most probably to be multifactorial, comprising of genetic,socioeconomic, and environmental factors [65, 66].AE is a huge global public health issue that affects 1% to 20% of the world's population. Adults with AE have a prevalence of 1% to 3%, whereas children have a frequency of 10% to 20% [67-69]. Atopic eczema (AE) is the most frequent kind of eczema inchildren.Clinically, acute eczema is characterised by a vibrant red infiltrate with oedema, vesicles, oozing, and crusting in its acute form; while in the subacute and chronic forms, lichenification, excoriations, papules, and nodulespredominate.Pruritus is seen commonly on the face ,scalp, and extensor surfaces of the limbs in newborns and young kids with atopic dermatitis. The flexor surfaces, neck, wrists, and ankles are commonly affected in older children and adults. In them, the existence of lesions in extensor areas suggests a poor prognosis for final cure [70].

PITYRIASIS ALBA

Pityriasis alba (PA) is a benign inflammatory dermatosis that affects roughly 5% of all children. It is most commonly observed in tropical places; as a result, it is more visible in people with darker complexions, but it is prevalent in all skin types. Its cause is unclear; but infectious causes, vitamin deficits, excessive and unprotected sun exposure are thought to be contributing causes. Xerosis is another key risk factor. Water absorption-desorption tests have revealed deficiencies in hygroscopicity and water-holding capacity in the stratum corneum of PA lesions, suggesting that the disease is caused by a dermatitic alteration and that hypopigmentation is a postinflammatory mechanism [71].There are two forms of PA: endemic, which affects babies and children in underdeveloped countries with low socioeconomic status, and atopic-dermatitis-related PA, which is linked to postinflammatory hypopigmentation [72].

CONTACT DERMATITIS

Contact dermatitis is an eczematous inflammatory skin condition. It is caused by chemical or metal ions which exert toxic effects without stimulating a T-cell response (contact irritants) or by tiny reactive chemicals that alter proteins and induce innate and adaptive immune responses (contact allergens) [73-76]. CD constituted 0.75% of the total dermatological OPD cases in a study conducted by Sadagopan et al in 2017.Irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD) are the two types of contact dermatitis. ACD is divided into the three types which are scalp dermatitis, aerosolized allergens and directly applied allergens. In India Bindi dermatitis is most common and it is caused due to PTBP or nickel, thiomersal , colophony [77]ICD lesions develop following exposure to a drug, the effects can build up over time, resulting to persistent skin damage and lesions. Chemicals are linked to ICD due to their irritating or toxic properties [78-80].

HYPERSENSITIVITY DISORDERS

URTICARIA

Urticaria is a transitory, circumscribed edematous and itchy swelling of the dermis. [84] Other names given

are hives, weals, and nettle rash [81,82]. It may be divided into two categories based on the length of time it lasts and the circumstances that cause it. On the basis of duration, it is divided into ′acute′ and ′chronic′ categories. This is a random division that ranges from 6 to 8 weeks [83, 85]. When cutaneous mast cells are activated, they release a variety of mediators, including histamine, which causes increased permeability of capillaries and venules, resulting in uriticaria. This notion is supported by the clinical response of urticaria to antihistamines.

Each episode begins with itching. After that, erythematous macules and weals form, which are transitory and vanish within a few hours to 24 hours. The weals can occur anywhere on the body, and range in sizes from a few mms to several cms, but they are very prevalent on the face because the skin there is frequently exposed to allergens and other irritants. They can be linked to angio-oedema The skin lesions disappear completely without any trace [83].Angioedema is a kind of urticaria in which the subcutaneous tissue is affected [85]. The eyes, lips, genitalia, tongue, and throat may all develop skin lesions. The skin lesions might persist anywhere from a few hours to few days [86]. Angio-oedema can manifest as an emergency in dermatology and requires prompt treatment. Heriditary angio-oedema is an uncommon disorder that is passed down through the generations as an autosomal dominant characteristic [87].

INSECT BITE REACTION

Bites and stings caused by members of the phylum Arthropoda are frequently referred to as "bug bites." Chilopoda, Diplopoda, Insecta, and Arachnida are the four medically relevant groups of arthropods. When exposed to the bites for the first time, there is no reaction. The host's reaction to salivary or contactant proteins produces eruption after further exposures [88]. According to a study done by Karthikeyan et al in a dermatology outpatient clinic in Pondicherry, it was discovered that children below 14 years of age had a prevalence of 5.3%.Most commonly present with erythematous, occasionally edematous papules that might be single, clustered, or widespread depending on the arthropodimplicated.There is frequently a pruritic eruption at the bite or sting site, as well as other common symptoms such local erythema, wheals, and urticaria. Scratching may cause skin excoriation in those with highly pruritic lesions, and subsequent bacterial infection may be present.Insect bite responses are treated symptomatically, with topical corticosteroids and antihistamines for moderate reactions, a brief course of systemic corticosteroid for severe reactions, antibiotics if infected and anaphylaxis treatment if it occurs [89].

POLYMORPHIC LIGHT REACTION

PMLE is an idiopathic eruption that is stimulated by UVR and has a clinical pattern that differs from patient to patient. It is induced by both UVA and UVB wavelengths. In temperate locations, young women under 30 years of age are commonly affected, with a frequency of 10 to 20% [90]. The prevalence of PMLE is approx. 10–20% in the generalpopulation.It's a type 4 delayed hypersensitivity reaction occurring to skin endogenous photo antigen [91]. UVR causes a natural skin particle to transform into an allergen, stimulating an immunological response.The morphology of the lesions varies. The itchiness of the skin lesions is limited to sun-exposed regions. The papular variety is the most frequent, consisting of erythematous papules atop a patchy erythematous base. The plaque kind, in which superficial urticarial plaques are visible, is the second most prevalent variety. The urticarial plaques becoming vesicles is the third common type.

ID REACTION

Autoeczematization, also known as an id reaction, is a widespread eczematous reaction that develops days or weeks after exposure to an initial stimulus and is caused by the release of antigen (s). Whitfield 96 initially characterised AE in 1921, claimingthat the id reaction was caused by skin sensitization following a primary stimulus [92]. One research found that 17% of individuals with dermatophyte infections had an id reaction, often tinea pedis, with vesicles on the palms [93]. Tinea capitis is one of the most prevalent causes of AE in children, and it's sometimes mistaken for a drug response.AE manifests itself clinically as vesicular dissemination that clusters to create papules or nummular patches on the legs, feet, arms, and/or trunk. Other kinds of eczema and other vesicular eruptions are possible differential diagnosis.To reduce the inflammatory response that causes the id reaction, the underlying ailment should be managed. Antifungal, antibiotic, antiviral, or antiparasitic medications should be used to treat infectious causes of ID reaction. Systemic or topical corticosteroids, as well as moist compresses, are used to treat the id reaction [94, 95].

TUMOURS

SEBORRHEIC KERATOSIS

This benign tumour is frequent in individuals who have excess sun exposure [96].Seborrheic wart, basal cell papilloma are other names for it. It usually begins around the age of 40 or older, and it is inherited as an autosomal dominant trait with a familial propensity.According to Nair PA et al, the maximum incidence of SK was seen during 51- 60 years of age group in 25.49% cases.It is commonly observed on the face across the forehead, temple, and preauricular region, sometimes seen in trunk or hands. The number of lesions varies from fewer than 20 in most people to multiple lesions in others. The classic lesion has a characteristic stuck-on look. D/ds are nevi, lentigo maligna, actinic keratosis, malignant melanoma. Variants are DPN, stucco keratosis, inverted follicular keratosis [97]. Smith et al found it to be 88%, Beauregard and Gilchrest 61.2%, Priya Cinna and Thappa found it be50.6%.

DERMATOSIS PAPULOSA NIGRA (DPN)

Dermatosis papulosa nigra (DPN) is a condition defined by the appearance of many hyperpigmented, sessile to filiform, smooth-surfaced papules measuring 1 to 5 mm on the face of darkly pigmented people [98]. DPN has a strong familial predisposition and has been reported to have an incidence ranging from 10% to 75% in selected study populations. It's found in children on rare occasions. Women are twice as likely as males to be affected. There is no link between DPN and any systemic illness or condition [98]. In the study done by Chopra et al in Punjab in 2000 DPN was found in 7.2% in 214 patients.DPN has an unknown cause. It appears at a younger age than SKs, but generally is comparable to SKs and is most likely a form of seborrheic keratosis (SK) [98]. The malar eminences and forehead are covered with pigmented papules that are symmetrically distributed. Lesions on the neck, chest, and back occur less often. The papules often arise throughout adolescence and subsequently grow in size and quantity, increasing in the sixth decade [98].

BASAL CELL CARCINOMA

The most common malignancy in humans is non-melanoma skin cancer (NMSC). According to Gloster et al, nonmelanoma skin cancers (NMSCs) comprise 1%–2% of cutaneous neoplasms in Indians, in contrast to one-third in Caucasians [99]. Basal cell carcinomas (BCCs) account for 75–80% of NMSCs, with squamous cell carcinomas accounting for the remaining 25%. (SCCs). Although BCC seldom metastasizes, it can locally invade (hence called “Rodent ulcer”) and hence rarely results in death. But it can cause severe morbidity if not detected and treated properly [100].BCC becomes more common as people get older, and males have a greater prevalence than women [100]. The most prominent environmental factor of BCC is sun exposure. Majority of BCC occurrences occur on the face [100].Nodulo ulcerative type of BCC is observed across the nose, forehead, cheeks, and periocular regions. [105]Intermittent intense sun exposure, light skin colour, tendency to burn instead of tan, history of blistering sunburns as a child, therapeutic ionising radiation, immunosuppression in organ transplant recipients, HIV, and genetic syndromes: nevoid basal cell carcinoma, xeroderma pigmentosum, and Bazex syndrome are all risk factors for the development of BCC [100].

ANGIOFIBROMA

Cutaneous angiofibromas can appear anywhere on the body and when they appear on face, they're known as fibrous papules or adenoma sebaceum. Tuberous sclerosis complex (TSC) is a neurocutaneous syndrome characterized by the development of multiple hamartomas distributed at various body sites, such as brain, skin, retina, kidney, heart, and lungs [101]. Facial angiofibromas are one of the most visible clinical manifestations of TSC and they usually appear in infancy or early adulthood. They are papular lesions that primarily affect the nasolabial folds, cheeks, and chin bilaterally and symmetrically or as mosaic pattern unilaterally [102, 103]. On the surface of the papule, they may have little telangiectatic vessels.According to a study conducted by Jyoti et al in 2015, the incidence of angiofibromas ranged from 88% to 100% in patients with TSC.

ACROCHORDON

Acrochordons also known as fibroepithelial polyps, skin tags, and papillomas are benign skin neoplasms. They have a high prevalence, affecting 46% of the general population, and are more common as patients get older. These little pedunculated polyps, occur around the neck, axilla, and groin regions and are skin-colored to hyper-pigmented. Patients may be bothered by them because of related symptoms such as discomfort, itching,and rubbing against clothing, or simply because of their look. Acrochordons have been linked to insulin resistance and obesity, as well as a hereditary tendency to develop such lesions [104-106].

MISCELLANEOUS

ALOPECIA AERATA

Alopecia areata (AA) is a non-scarring alopecia that affects the scalp and/or body hair and is characterised by hair loss without any clinical signs of inflammation. Dermatologists view it as one of the most frequent types of hair loss, accounting for 25% of all alopecia cases [107]. Cornelius Celsus was the first to describe it, and Sauvages created the name AA in 1760 [108].It accounts for 0.7% of new dermatological cases in India [109, 110]. The frequency in the general population was estimated to be 0.1-0.2%, with a lifetime risk of1.7% [110].Hair loss in well-defined, localised patches, that spread circumferentially is a typical AA manifestation. It might appear as a single patch or a cluster of patches.The most prevalent location (90 %) is the scalp, however it can infect any portion of the body. The degree and pattern of hair loss can be used to classify AA [111]. It can be patchy AA, alopecia totalis (AT) affecting the entire scalp as well as body hair such as eyebrows, eyelashes, beard, axillary hair, and pubic hair, and alopecia universalis (AU) affecting the full body hair [112].

HIRSUTISM

Hirsutism is the presence of terminal coarse hairs in females that are distributed in a male-like pattern. It affects about 5% to 10% of women [113, 114].It is a sign of higher androgen levels [115, 116].Idiopathic hirsutism (IH) affects around 20% of people.

LICHEN SPINULOSIS

The dermatosis lichen spinulosus (LS) is a rare dermatosis which is more frequent in children and teenagers, and rare in adults [117]. It might be caused by hereditary factors [118-121]. It appears as a cluster of sandpaper-textured projected lesions [122].The lesions frequently appear in different areas of the skin and last for a few weeks to months.

LICHEN NITIDUS

Pinkus initially described lichen nitidus in 1907. It is an uncommon asymptomatic chronic inflammatory illness, with well-defined clinical and histological features [123]. It mostly affects children and young adults, and it is neither gender or ethnicity specific [124, 125]. Shiny, flat-topped, fleshy pink to dark brown papules characterise Lichen nitidus. Their diameter ranges between 1 and 2 mm. Neck, trunk, limbs, abdomen, and penile shaft are all common sites for lesions. Mucous membranes, palms, soles, and nails involvement is uncommon, but it has been reported [126]. Occasionally pruritus is seen in some cases. The Koebner phenomenon is a distinctive feature of lichen nitidus, as shown in lichen planus, however it is not always present.

PYOGENIC GRANULOMA

Pyogenic granuloma (PG), also known as lobular capillary hemangioma, is a benign vascular tumour that affects the skin and mucous membranes. It can also be seen subcutaneously or intravascularly on rare occasions. PG can appear on its own, in injury sites, or inside capillary abnormalities [127]. PG affects people of all ages and genders. They are reddish exophytic vascular nodules that can be tiny or big, smooth or lobulated, and develop quickly. Larger lesions get lobulated and may grow into pediculated, mushroom-like tumours. PGs have a proclivity for bleeding extensively. The hands, lower lips, and gingiva are the most commonly afflicted areas [127].

PSORIASIS

In the general population, psoriasis affects 2 to 3% of the population. Psoriasis that only affects the face is extremely rare. The three clinical types of facial psoriasis are hairline psoriasis, sebo psoriasis, and true facial psoriasis.Psoriasis that extends beyond the hairlines and into the forehead is known as hairline psoriasis. In sebopsoriasis, lesions extend towards the eyebrows and beard regions [128]. True facial psoriasis is characterised by symmetrical, well delineated scaly plaques that can appear on any part of the face.

IMMUNE THROMBOCYTOPENIC PURPURA

Primary ITP is defined as a platelet count of $<100 \times 10^9$/L in the absence of other causes of thrombocytopenia [129].Normally, otherwise healthy platelets are destroyed in ITP, usually in reaction to an unknown stimuli. This can happen on its own (primary ITP) or in combination with other problems (secondary). Secondary ITP can be caused by a variety of factors such as autoimmune conditions (including antiphospholipid antibody syndrome and Systemic lupus erythematosus),infections caused by

viruses(including cytomegalovirus, varicella-zoster, hepatitis C and HIV), helicobacter pylori infection, medications, lymphoproliferative disorders [130, 131]. ITP in children is most usually caused by a viral infection, although it can also develop after immunization [132]. The clinical picture might range from no symptoms to a potentially fatal intracranial haemorrhage. Petechiae or bruising is the most prevalent symptom. Up to a quarter of the people had nosebleeds. Gastrointestinal bleeds and haematuria are less prevalent. Menorrhagia can affect older girls. Intracranial bleeding is extremely uncommon [132]

MILIA

These are keratin cysts in the subepidermal layer of the skin. They can happen as a result of subepidermal blistering, such as epidermolysis bullosa.

XANTHELASMA PALPEBRUM

Xanthelasma Palpebrarum is an eyelid condition characterised by symmetrical soft, yellowish brown velvety papules on the inner canthi of the upper and lower lids, which are numerous, progressive, permanent, and coalescent. It is one of the most prevalent xanthomas seen in in clinical practice, occuring at an average age of 30-50 years. The specific reason is unknown, however various causes have been suggested, including lipid abnormalities, hormone factors, acetylated LDL, and macrophages. Xanthelasma is made up of xanthoma cells, which are foamy histiocytes with intracellular fat deposits, mainly in the upper reticular dermis.

NEVI

Acquired nevi are common in both sexes and might arise throughout adolescence or early adulthood. People with white skin are more likely to be impacted than those with black complexion. UV radiation and mutations in the BRAF gene have been proposed as etiopathogenic contributors

CONCLUSIONS

Facial dermatoses became less common as people became older. Females were more impacted than males. Infections were the most common face dermatoses. Patients with pigmentary illnesses and hypersensitivity disorders made up 17.4% of the total. The most common cause of pigmentation was melasma. UV exposure was determined to be the most important exacerbating factor for melasma, followed by familial history. In our analysis, acne vulgaris was the second most frequent condition. The majority of the Acne vulgaris patients were in the Grade- II group, followed by the Grade- III category.

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