"A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS"

By

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Dissertation submitted to the

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In partial fulfillment of the requirement for the degree of

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IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

Under the Guidance Of

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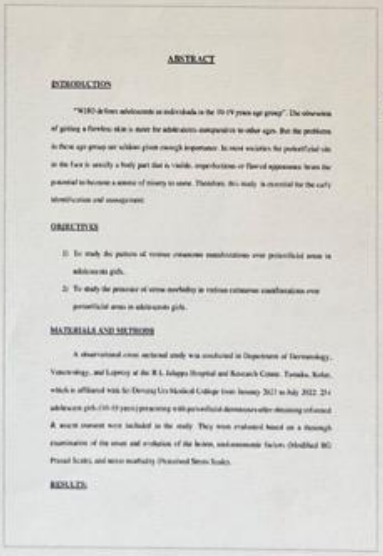

ABSTRACT

INTRODUCTION

OBJECTIVES

1) To study the pattern of various cutaneous manifestations over pomicficial areas in adolescents girls.
2) To study the presence of stress morbidity in various cutaneous manifestations over pomicficial areas in adolescents girls.

MATERIALS AND METHODS

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LIST OF ABBREVIATIONS

DPN	Dermatosis papulosa nigra
SCC	Squamous cell carcinoma
BCC	Basal cell carcinoma
TSC	Tuberous sclerosis complex
NF	Neurofibromatosis
MDT	Multidrug therapy
HSV	Herpes simplex virus
IgG	Immunoglobulin G
HPV	Human papillomavirus
MCV	Molluscum contagiosum virus
KOH	Potassium hydroxide
SD	Seborrheic dermatitis
ISD	Infantile seborrheic dermatitis
ASD	Adult seborrheic dematitis
SA	Salicyclic acid
ICD	Irritant contact dermatitis
ACD	Allergic contact dermatitis

AD	Atopic dermatitis
PMLE	Polymorphic light eruption
UVR	Ultraviolet radiation
TH2	T helper 2 cells
HCQ	Hydroxychloroquine
PV	Pemphigus vulgaris
PF	Pemphigus foliaceous
HLA	Human leukocyte antigen
EBA	Epidermolysis bullosa acquisita
LR	Lichenoid reaction
LP	Lichen planus
PUVA	Psoralen +Ultraviolet A radiation
UVB	Ultraviolet B rays
TLR	Toll like receptors
CO2 Laser	Carbondioxide laser
POD	Perioral dermatitis
PDGF	Platelet derived growth factor
OPD	Out patient department

TGF-β	Transforming growth factor-Beta
VEGF	Vascular endothelial growth factor

ABSTRACT

INTRODUCTION

"WHO defines adolescents as individuals in the 10-19 years age group". The obsession of getting a flawless skin is more for adolescents comparative to other ages. But the problems in these age group are seldom given enough importance. In most societies the periorificial site in the face is usually a body part that is visible, imperfections or flawed appearance bears the potential to become a source of misery to some. Therefore, this study is essential for the early identification and management.

OBJECTIVES

1) To study the pattern of various cutaneous manifestations over periorificial areas in adolescents girls.
2) To study the presence of stress morbidity in various cutaneous manifestations over periorificial areas in adolescents girls.

MATERIALS AND METHODS

A observational cross sectional study was conducted in Department of Dermatology, Venereology, and Leprosy at the R L Jalappa Hospital and Research Centre, Tamaka, Kolar, which is affiliated with Sri Devaraj Urs Medical College from January 2021 to July 2022. 254 adolescent girls (10-19 years) presenting with periorificial dermatoses after obtaining informed & assent consent were included in the study. They were evaluated based on a thorough examination of the onset and evolution of the lesion, socioeconomic factors (Modified BG Prasad Scale), and stress morbidity (Perceived Stress Scale).

RESULTS:

Out of the total 254 cases enrolled, most of them were in 18-19 years of age (44.1%). Most common dermatoses was appendageal disorders (25.2%) followed by pigmentary disorders (22.8%) and infections (18.5%). Moderate stress was observed in about 62.6% of the cases.

CONCLUSION:

Adolescents often develop facial dermatoses. In this age group, certain dermatoses are of aesthetic significance. Early detection, careful treatment, and patient education are crucial to preventing late disfiguring consequences and psychological aftereffects.

KEYWORDS: Adolescents, facial dermatoses, psychological comorbidities, symptoms and topical treatment application.

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INTRODUCTION

INTRODUCTION

Puberty marks the beginning of a key developmental stage called adolescence, which ranges from 10-19 years. Considering the differences between 12 and 24-year-old selves, both biologically and cognitively, as well as psychosocially and emotionally, a significant amount of growth occurs between those two ages[1]

In addition to physical changes like pubic hair growth, genital changes in males, voice changes, an increase in height, and the start of menstruation, puberty is also accompanied by emotional and chemical changes (menarche).[2]

Adolescent's dermatological issues are mostly caused by changes in hormone levels, particularly androgens leading to acne, hair issues, and excessive sweating. It is estimated that 80% of people in this age range are affected by it, or possibly 100% of young people if lesions of low intensity are taken into consideration.[3]

During this stage, the skin undergoes significant changes that put the skin's appendages at risk for certain dermatological issues. When a very apparent skin condition attracts undesired peer attention, adolescent self-esteem is readily undermined. Dermatological conditions have an increased influence on adolescents' quality of life, which causes significant physical and psychological morbidity given that they make up one-fifth of the Indian population. The periorificial location on the face, which is often a conspicuous body feature in most countries, can be a source of suffering for certain people because of flaws in appearance.[4]

Acne, seborrhoea, viral, fungal, bacterial infections, neurodermatitis, contact dermatitis, , and venereal disease are the most typical dermatoses that affect adolescents. Acne, dermatitis, atopic eczema, herpes infection, molluscum contagiosum, papulosquamous

disorders, pigmentary disorders, appendageal disorder, urticaria/dermographism, keloid, papular urticaria, scabies, dermatophytosis, contact dermatitis, and verrucae are some of the skin conditions that can manifest over the periorificial area.

Numerous external and internal factors have a role in the etiology of adult acne. Endocrine abnormalities, persistent induction of innate immunity, and genetic predispositions are examples of the former, whereas cosmetics, stress, and cigarette use are examples of the latter. With significant psychological, social, and emotional effects and up to a 40% frequency of mental comorbidity, adult acne is a chronic illness that seems to affect adult patients quality of life more than their younger counterparts.[5]

Perioral dermatitis is a inflammatory condition that most frequently affects young females. The perioral region is where the illness is most frequently found, however it can also affect the paranasal and periocular skin. Hence, the term “Periorificial dermatitis” was later designated to this condition. This may be brought on by topical steroid usage on the face, thus the patient should stop applying steroid creams as the first line of therapy. Oral antibiotics like tetracycline, topical metronidazole & calcineurin inhibitors are further therapeutic options. Although perioral dermatitis frequently responds well to treatment, it can sometimes be persistent and recurring.[6]

Therefore, periorificial areas will benefit from early detection of these illnesses as it will decrease patient morbidity. These factors make the study of different cutaneous manifestations over periorbital regions common in the age range of 10 to 19 years significant from a social and therapeutic standpoint. The significance of our study is due to the dearth of data on teenage periorificial dermatoses in the literature.

AIMS & OBJECTIVES

AIM AND OBJECTIVES

AIM :

To study clinical and epidemiological parameters of cutaneous manifestations over the periorificial areas in adolescent girls.

OBJECTIVES :

1) To examine the distribution of different cutaneous symptoms over periorificial regions in adolescent females.
2) To investigate if females in adolescence exhibit stress morbidity in diverse cutaneous symptoms over periorificial regions.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

- With a case of "light sensitive seborrhoeid" in 1957, which is thought to be the first closest description of the ailment, the disorder appears to have suddenly appeared.[7]
- Without specific clinical criteria, the illness in adults began to be identified as perioral dermatitis by 1964.
- The disorder in adolescence was identified in 1970. Since then, it has been hotly contested whether all lesions around the mouth constitute perioral dermatitis. It has been suggested that the term for this disorder be changed to periorificial dermatitis.
- One of the early "definitive" descriptions of "perioral dermatitis" was provided by British dermatologist Darrell Wilkinson (1919–2009), who also noticed that the ailment wasn't necessarily linked to the use of fluoride steroid creams.[8]

CLASSIFICATION:

S. NO	CONDITIONS	DISEASES	
1.	Genodermatoses	Tuberous sclerosis, Neurofibromatosis	
2.	Infections	Bacterial	Impetigo, Folliculitis, Erythrasma, Hansen disease.
		Viral	Herpes labialis, Varicella, Wart, Molluscum contagiosum.
		Fungal	Tinea, Candidiasis, Pityriasis versicolor
3.	Infestations	Scabies, Pediculosis, Paedrus dermatitis	
4.	Eczematous disorders	Contact dermatitis, Intertrigo, Seborrheic dermatitis, Pityriasis alba, Atopic dermatitis, periocular dermatitis	
5.	Photodermatoses	Polymorphic light eruption, chronic actinic dermatitis, Photo toxic, Photoallergic.	
6.	Immunobullous disorders	Pemphigus Vulgaris, Pemphigus foliaceous, Epidermolysis bullosa	
7.	Papulosquamous disorders	Psoriasis, Lichen planus, Lichenoid reaction	
8.	Pigmentary disorders	Hypopigmentary & Depigmentary	Vitiligo, Pityriasis alba, Pityriasis versicolor.
		Hyperpigmentary	Nevus of ota, Lentigines, Freckles, Lichen planus pigmentosus, Melanocytic nevi, Acanthosis Nigricans.
9.	Disorders of appendages	Acne, Rosacea, Perioral dermatitis, Miliaria, Milia.	
10.	Hair disorders	Alopecia areata.	
11.	Metabolic disorders	Vitamin deficiency, Xanthelasma palpebrarum, Hartnup disease, Porphyria.	
12.	Skin tumours	Keloid, Hypertrophic scar, Syringoma, Seborrheic keratosis, DPN, SCC, BCC, Malignant melanoma.	
13.	Miscellaneous Condition	Epidermal cysts, Xanthelasma palpebrum	

TABLE 1: CLASSIFICATION OF DERMATOSES.[9]

The following categories of periorificial dermatoses in teenage females are those that occur more frequently:

1. GENODERMATOSES

Genodermatoses are a hereditary condition that affect many systems. Because genetic variability is so prevalent, a lot of work must go into molecular diagnostics. Families with xeroderma, Griscelli, have recurrent mutations in unrelated families. Oligonucleotide arrays are anticipated to ultimately displace the majority of other techniques for regular mutation scanning of the more prevalent illnesses**,** and planned sequencing is utilized more and more for uncommon disorders.[10]

I. TUBEROUS SCLEROSIS [11]

Tuberous sclerosis complex (TSC) is a genetic condition with autosomal dominant trait. It is characterized by an elevated propensity for hamartoma development. TSC is often discovered in childhood or infancy, and afflicted people may experience seizures, cutaneous symptoms, or developmental delays.

EPIDEMIOLOGY:

With an incidence of 1 in 20,000, tuberous sclerosis complex affects around 1 in 6000 to 1 in 10,000 live births. All ethnic groups are affected by the clinical presentation, which often involves numerous organs.

ETIOLOGY:

Mutations in the genes TSC1 (9q34) and TSC2 (16p13.3), which encode hamartin and tuberin, respectively, cause tuberous sclerosis complex.

PATHOPHYSIOLOGY:

A hereditary disorder called tuberous sclerosis can impact practically all organ systems. It involves changes to the TSC1 and TSC2 genes, which produce proteins that control the body's ability to divide cells and expand. Hamartomas may develop in the brain, skin, heart, kidney, liver, and lungs when this equilibrium is upset by gene alterations, impairing the functionality of these organs.

TREATMENT:

- Psychiatric and behaviour management.
- Anti-seizure medications.

II. NEUROFIBROMATOSIS [12]

A neurocutaneous illness called neurofibromatosis is characterized by tumors on the skin and in the nerve system. The two autosomal dominant kinds of neurofibromatosis that are most prevalent are type 1 and 2. Neurofibromas, axillary freckling, cafe-au-lait spots and optic gliomas are the symptoms of NF 1, commonly known as von Recklinghausen's disease. Meningiomas & bilateral vestibular schwannomas are seen in NF 2. Neurofibromatosis types 1 and 2 are treated with clinical surveillance and, if necessary, medical intervention.

EPIDEMIOLOGY:

Approximately 96% of all instances of neurofibromatosis are type 1. One in every 3000 newborns is affected. Both racial and gender differences are present. Half of the patients have a hereditary mutation, while the other half have a spontaneous mutation.

ETIOLOGY:

A loss of function mutation in the neurofibromin 1 (NF1) gene, either acquired or de novo, results in neurofibromatosis type 1. This gene codes for neurofibromin and is found on band 17q11.2.

TREATMENT:

- Symptomatic lesions may be removed surgically.
- It has been demonstrated that imatinib reduces plexiform neurofibroma size.

2. INFECTIONS

Skin infections make up a sizable component of dermatological illnesses. Regarding frequency, etiologic organisms, and clinical symptoms, infections of the skin and subcutaneous tissues are quite varied.[13]

I. BACTERIAL

The most typical superficial bacterial skin infection is impetigo. It can be bullous or non-bullous, and Staphylococcus aureus or Streptococcus pyogenes are commonly the culprits.

IA. IMPETIGO

Children who live in hot, humid settings are more likely to have impetigo. This condition usually affects face, but it can also happen in any other area of the body where there has been trauma, such as an insect sting, abrasion, or laceration.

EPIDEMIOLOGY:

S. aureus is the most frequent cause of non-bullous impetigo, accounting for 70% of cases. The summer and fall seasons see the highest prevalence. It is predominantly seen in preschool-aged children, hence also known as school sores.

PATHOPHYSIOLOGY:

There are two types of impetigo: Primary and secondary. The bacterial directly invades the healthy skin in primary impetigo whereas it develops at site of trauma/injury in case of secondary impetigo. These lesions spread to other sites as well as to other individuals either by self-inoculation or due to direct contact. Some of the predisposing factors are overcrowding, immunosuppressed states, malnutrition, diabetes, , childcare attendance, & poor personal cleanliness.

Triggering factors:

- Trauma
- Scratching
- Viral infection: Herpes & varicella
- Burns
- Insect bites.[14]

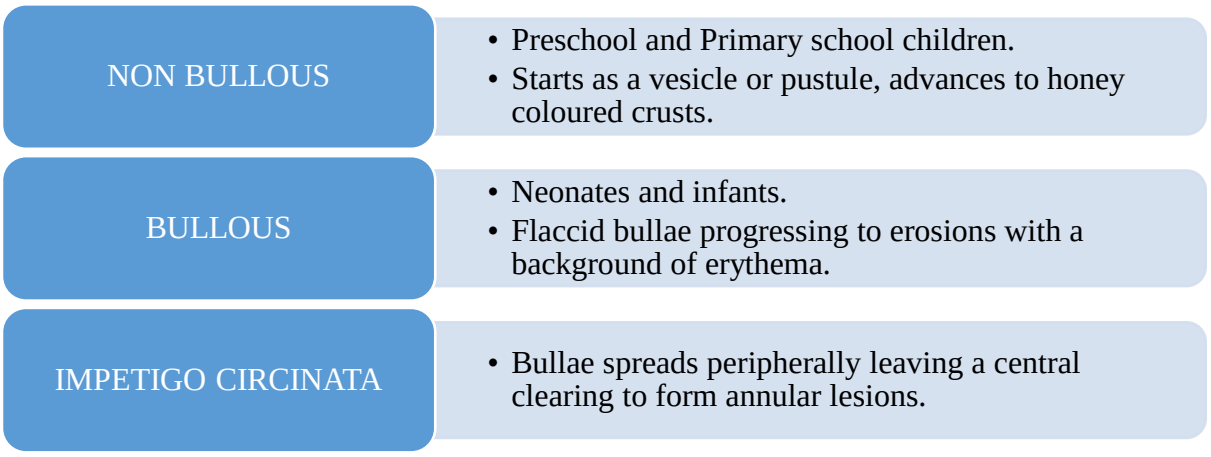


FIGURE 1: TYPES OF IMPETIGO.[15]

DIFFERENTIAL DIAGNOSIS:

- Localised SSSS
- Contact dermatitis
- Herpes simplex
- Immunobullous disorder

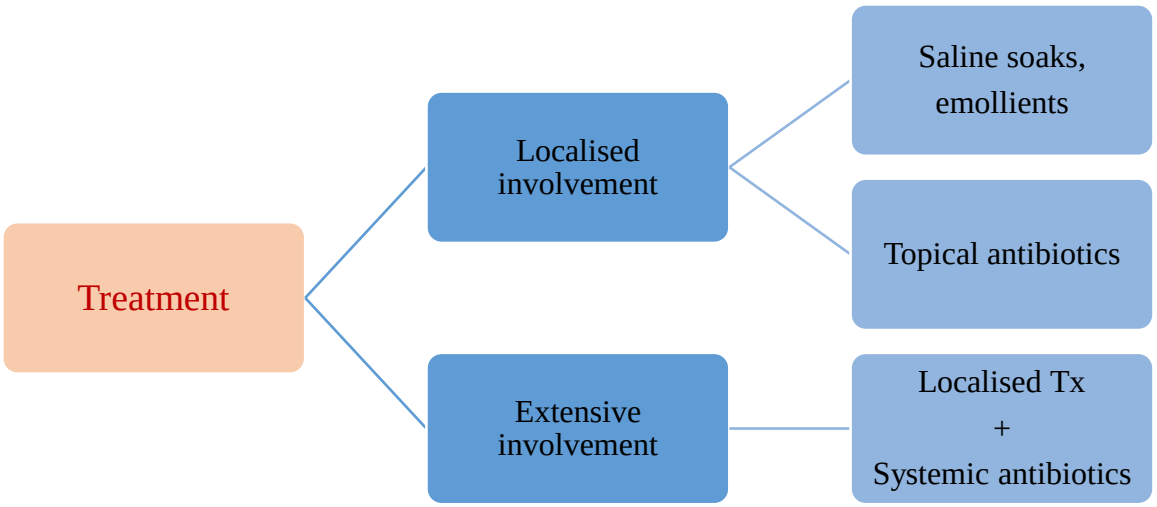


FIGURE 2: TREATMENT OF IMPETIGO.[16]

IB. FOLLICULITIS

Folliculitis is defined as a “bacterial infection of the hair follicle”. Even though this ailment is often benign, early detection can help individuals with this condition live better. Folliculitis is often brought on by a bacterial infection of the deep or superficial hair follicle. However, this illness may also be brought on by some fungi, viruses, or even non-infectious organisms. The most prevalent type of folliculitis is superficial bacterial folliculitis, which is often brought on by the bacterium Staphylococcus aureus.

TYPES : [17]

SUPERFICIAL FOLLICULITIS	Bockhart’s impetigo
	Chronic folliculitis of the legs
	Gram-negative folliculitis a) Post acne treatment b) Hot tub folliculitis
DEEP FOLLICULITIS	Sycosis barbae

TABLE 2: TYPES OF FOLLICULITIS

IC. ERYTHRASMA

Burchardt's teacher, Von Barensprung in 1862 devised the term erythrasma and the bacteria Microsporum minutissimum. Erythrasma is caused by the aerobic gram positive non spore forming bacillus, Corynebacterium minutissimum, a normal resident flora. Favoured sites of infection are inguinal, intergluteal, interdigital, axillary and submammary areas. It is characterized by asymptomatic, well circumscribed red brown plaques. C. minutissimum

develops in the stratum corneum when there is moisture and occlusion. Due to coproporphyrin III, in wood's lamp examination it exhibits an coral-red fluorescence. Although this responds to topical and oral treatments, it frequently reoccurs. Erythromycin, clindamycin, or fusidic acid are examples of topical treatments.[18]

ID. HANSEN DISEASE

Another name for leprosy is Hansen disease. Mycobacterium leprae and Mycobacterium lepromatosis, both of which largely affect the peripheral nerves and skin, are often to blame for this persistent granulomatous infection. Although the exact transmission of leprosy is unknown, it is thought to occur via the respiratory system. People with lepromatous infections who are untreated typically have a large number of bacilli. To prevent microbial resistance, multiple drug therapy (MDT) is typically used as the main form of leprosy treatment. The MDT therapy successfully eradicates M. leprae and swiftly renders the patient non-infectious.[19]

II. VIRAL

Diseases caused by viral infections are Herpes labialis, Varicella, Wart, Molluscum contagiosum.

IIA. HERPES LABIALIS

Herpes labialis is a moderate, self-resolving herpes simplex virus type-1 infection (HSV-1). It is also known as fever blisters or cold sores.

EPIDEMIOLOGY:

20% to 40% of individuals will have moderate, self-limiting painful blisters around the mouth as a result of herpes simplex virus type 1 infection.

It affects the vermillion border of the lip most commonly with prodromal symptoms such as burning, tingling, pain and profound grouped vesicles which later rupture and form superficial erosions, gets crusted and subsides in a few days, healing without scarring.

DIFFERENTIAL DIAGNOSIS:

- Aphthous stomatitis
- Stevens-Johnson syndrome
- Herpangina.

TREATMENT:

Treatment with a neutral cream (zinc oxide or zinc sulphate), an anaesthetic cream, or an antiviral cream, if used quickly, has a slight favourable impact on the duration of symptoms. Antiviral medication (cream or oral) can offer some protection if taken prior to exposure to the trigger factor (sunlight). Herpes labialis relapses can be prevented over the long run using oral antiviral treatment.[20]

II B. VARICELLA

The varicella-zoster virus (VZV), which causes chickenpox or varicella, is an infectious illness It starts as tiny, itchy blisters that later produces scab. Usually, it begins on the face, back, and chest before spreading. Prodromal symptoms such as fever, headaches, pharyngitis, and myalgia often lasts for 5-7 days. Pneumonia, meningitis, and cutaneous

bacterial infections are all complications. Adults are more severely affected by the illness than children. Ten to 21 days after exposure, symptoms appear, but the usual incubation time is only two weeks.

Production of host immunoglobulins G, M, and A is triggered by exposure. Immunity is provided by IgG antibodies, which last a lifetime.

Symptomatic alleviation is the only kind of treatment. Those who are ill typically have to stay at home as they are contagious as a precaution. Wearing gloves and cutting your nails short can help minimize scratching and lower your chances of getting secondary infections which leads to scarring. Calamine lotion used topically might reduce itching. Warm water washing on a daily basis will aid in preventing subsequent bacterial illness. Fever can be reduced with acetaminophen.[21]

II C. WART

Warts are benign lesions primarily affecting skin & mucosa caused by human papillomavirus (HPV), of which there are over 100 different varieties. HPV may develop everywhere.

EPIDEMIOLOGY:

Warts are a frequent medical condition, particularly among White people. This affects 10% of the world's population. The incidence among school going children reaches upto 10%-20%. Immunosuppressed individuals and meat workers are more prone to them. Any age can experience a wart. Although uncommon in early childhood and infancy, frequency rises among school-aged children and reaches a peak between the ages of 12 and 16. White people get warts twice as often as Black people or Asian people do. The Heck disease, or

focal epithelial hyperplasia, is more common among Inuit and American Indian populations. The ratio of men to women is almost equal.

ETIOLOGY:

The HPV virus has more than 100 subtypes, but only a few of them can result in skin warts at particular anatomical places. However, the HPV may spread to any area of the body through skin-to-skin contact. Palmoplantar warts, plane warts, and genital warts are most frequently observed. Direct or indirect contact is a simple way to spread warts, especially if the usual epithelial barrier has been compromised.

PATHOPHYSIOLOGY:

Only a handful of the 100 HPV subtypes have the potential to cause cancer. The HPV strains 6, 11, 16, 18, 31, and 35 are among these subtypes. Immunocompromised people and those with genital warts are more likely to develop malignant transformation. Additionally, HPV strains 5, 8, 20, and 47 have the potential to cause cancer in people with epidermodysplasia verruciformis.[22]

TYPES:[23]

MORPHOLOGICAL	• Verruca vulgaris • Plantar warts • Plane warts • Filiform warts • Anogenital warts • Oral warts • Butcher's warts
HISTOPATHOLOGICAL	• Inclusion wart • Papillomatosis,foci of vacuolization, parakeratosis • Diffuse vacuolization in upper dermis • Dysplastic vacuolization

TABLE 3: TYPES OF WARTS

DIAGNOSIS:

The existence of a virus is confirmed by immunohistochemical detection of HPV structural proteins, however this method has low sensitivity. Southern blot hybridization is more accurate. For testing, viral DNA is amplified using polymerase chain reaction.

TREATMENT : [24]

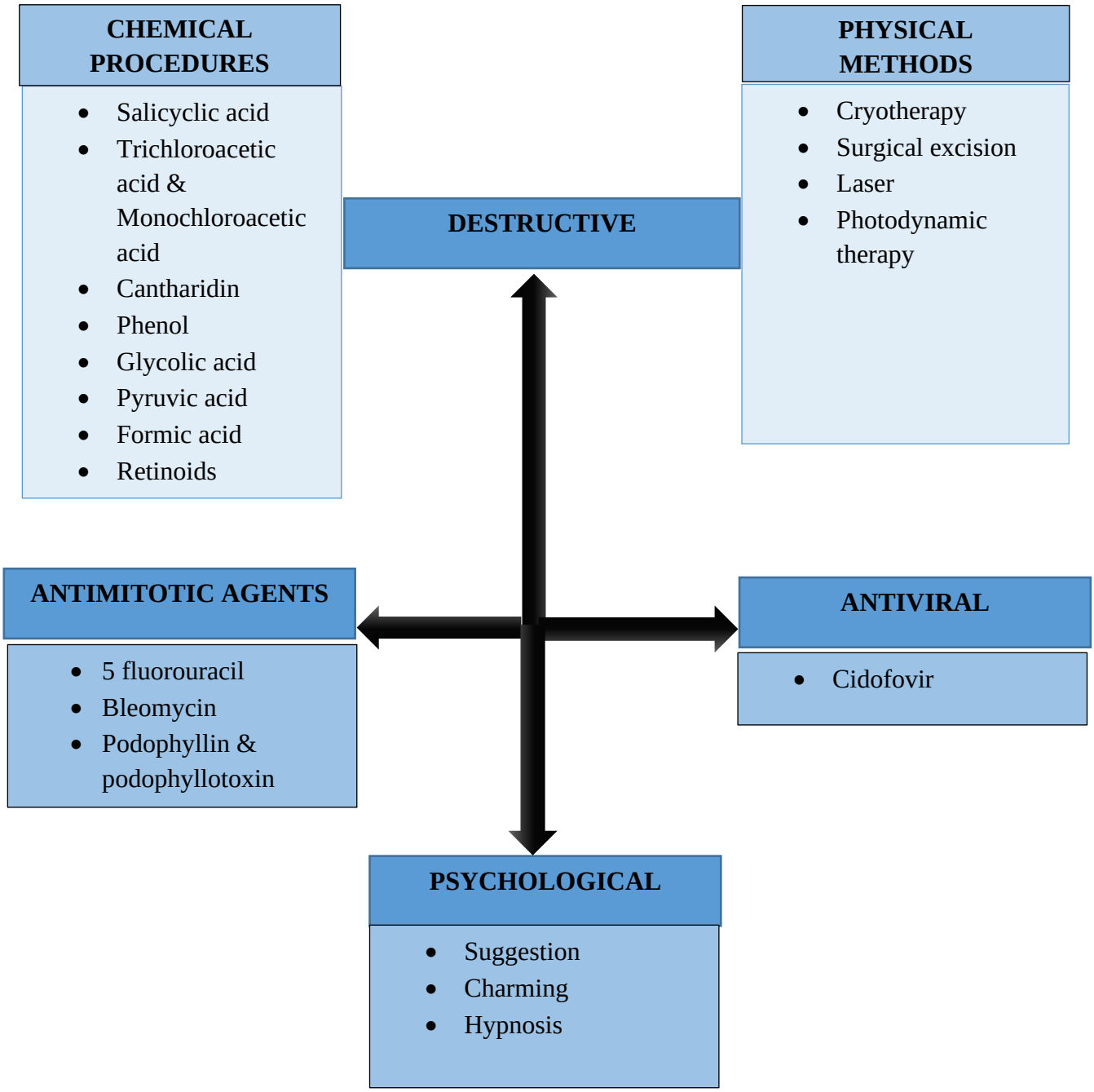


FIGURE 3: TREATMENT OF WARTS

II D: MOLLUSCUM CONTAGIOSUM

Water warts, also known as Molluscum contagiosum, are a benign cutaneous ailment. The usual lesion is spherical, pinkish-purple, and dome-shaped.

EPIDEMIOLOGY:

A common medical problem is molluscum contagiosum. There will be roughly 122 million instances in 2020. Although it happens everywhere, it tends to happen more frequently in hot, humid places. Molluscum contagiosum is typically diagnosed in children aged between 2-5 years, although it can also affect immunocompromised patients, sexually active teens, and adults.

ETIOLOGY:

Molluscum contagiosum virus (MCV) which is a dsDNA poxvirus is responsible for this condition. The molluscum contagiosum virus has four recognized subtypes, and they are the majority of MCV-1 instances (98%) involve youngsters, whereas MCV-2 is mostly responsible for skin lesions in HIV-positive individuals. Asia and Australia both have MCV-3 and MCV-4. Molluscum contagiosum viral cultures are not yet possible.

PATHOPHYSIOLOGY:

Incubation period is around 2 weeks to 6 months. This virus affects the basal keratinocytes in the epidermis. The replicated mature virions are present intracellularly and are enclosed by an lipid and protein rich sac. This protects the virus from the host immune response and the skin lesions develops.

DIAGNOSIS:

Clinical examination is the foundation for the diagnosis of molluscum contagiosum. Lesions are solid, dome-shaped, flesh-to-white, pearly papules with a central umbilication from which one might exude a gooey substance which is termed as molluscum body, histopathologically it is called Henderson Patterson body.

Mollusca are seen on the face, trunk, limbs, and axillary regions of youngsters. Molluscum contagiosum can be severe, usually in those with impaired immune systems.

Treatment:

People commonly seek therapy for purely aesthetic reasons, in order to avoid social shame, or due to worries about spreading the condition to others. The patient's (or their parents') preferences will determine the course of treatment. Although several therapies for molluscum contagiosum have been suggested, none had been shown to be successful. The three main types of therapy available today are systemic, topical, and manual removal.[25]

III. FUNGAL

IIIA. TINEA

Tinea is defined as a “superficial fungal infection caused by dermophyte affecting the skin”. Several names are designated for infection affecting various parts of the body such as the scalp “tinea capitis”, face “tinea faciei”, beard “tinea barbae”, body “tinea corporis”, hands “tinea manuum”, groin “tinea cruris”, and feet “tinea pedis”.

EPIDEMIOLOGY:

Tinea corporis is ubiquitous as its prevalence is all over the world. The most frequent cause of superficial fungal infections is dermatophytes. Occlusive clothing, excessive heat leading to increased moisture, and high relative humidity all have a role to play in their etiology. Tinea corporis may also be more common in certain groups of people, such as children. The two most prevalent dermatophytic infections in prepubescent children are tinea capitis and corporis. Additionally, children are more susceptible to get zoophilic infections.

Contact with animals, such as cats and dogs, can lead to the transmission of zoophilic illnesses.

ETIOLOGY:

Dermatophytoses are caused by the organism ‘s ability to bind to keratinized skin tissue. Dermotophytic genera causing tinea corporis are “Trichophyton, Epidermophyton, and Microsporum”. T. Rubrum is responsible for 80 to 90% of the strains.

DIAGNOSIS:

Tinea corporis is often diagnosed clinically after obtaining a comprehensive medical history and conducting a thorough physical examination. The diagnosis can, however, be verified by certain tests. A potassium hydroxide (KOH) preparation used to analyze skin scrapings under a microscope will show septate and branching long, slender hyphae.

TREATMENT

The treatment varies depending upon the type of infection. Some of the treatment options available are topical antifungals (Imidazole, Triazole, Allylamines, Benzylamines, Hydroxypyridine) and Systemic antifungals (Terbinafine, Itraconazole, Fluconazole, Griseofulvin).

III B. CANDIDIASIS

Candida, a fungus, is the source of the opportunistic infection known as candidiasis. Eukaryotic creatures known as fungi include yeasts, molds, and dimorphic fungus. Yeast in the form of Candida. In those with impaired immune systems, secondary infections like candidiasis are most frequent. These are frequent occupants of the vaginal canal, gastrointestinal system, oral cavity, and other areas. Only in the presence of suitable

circumstances do they become pathogenic. It may impact the vagina, penis, oral cavity, or other areas of the body.

EPIDEMIOLOGY:

Candidiasis is more common in young children and the elderly. Oral candidiasis in children who use inhaled steroids is more common. It frequently occurs to women during pregnancy. The first sign of HIV infection may be thrush. Both men and females can have thrush.

Although Candida albicans is the most common cause of candidiasis, non-Candida species have become much more common in recent years. The most prevalent Candida species were *C. albicans, C. lusitaniae, C. parapsilosis, C. glabrata, C. kefyr, C. famata, C. africana,* and *C. orthopsilosis,* in that order. People with weakened immune systems are more susceptible to invasive and disseminative candidiasis, which has been on the rise internationally.[26]

ETIOPATHOGENESIS: [27]

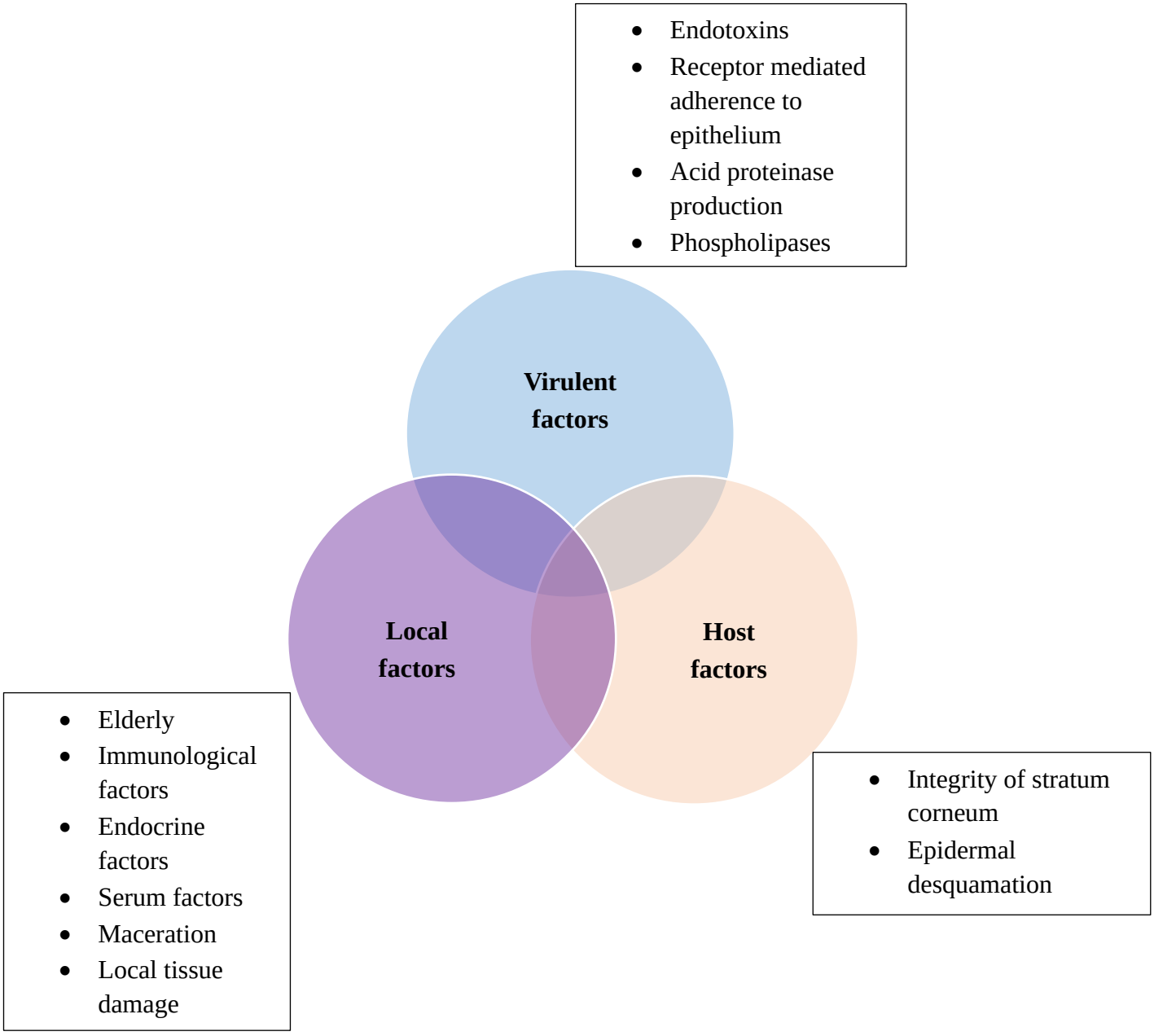


FIGURE 4: ETIOPATHOGENESIS OF CANDIDIASIS

TREATMENT:

Candida infections are treated with antifungal medications such as miconazole, clotrimazole, amphotericin B, and nystatin,. To treat mild to moderate genital Candida infections, antifungal vaginal cream can be used. These topical antifungals can be used for a

duration of 1, 3 & 7 day treatment. Additional options include econazole or fluconazole 150 mg orally as a one-time treatment.

Systemic candidiasis needs the administration of oral/parenteral antifungal drugs, such as amphotericin B, fluconazole, and caspofungin.[28]

III C. PITYRIASIS VERSICOLOR

Tinea versicolor is also been used alternatively for pityriasis versicolor. It is defined as a “common, benign, superficial cutaneous fungal infection caused by Malassezia species”.

EPIDEMIOLOGY:

Pityriasis versicolor has been documented all over the world, however it is more frequently observed in hot & humid climates. The incidence ranges from 50% in tropical nations to 1.3% in cold climatic place. There is no gender & ethnic preponderance. It is more common in teens which is most likely attributed to an increased sebum production. This allows Malassezia to flourish in a more lipid-rich environment.

ETIOLOGY:

Malassezia also known as Pityrosporum is a dimorphic fungus which is a normal flora of skin. This causes Pityriasis versicolor. Malassezia species that have been isolated commonly are Malassezia furfur, globosa, sympodialis but several other species have also been recognised such as equin, japonica, nana, obtuse, restricta, yamatoensis, cuniculi, caprae.

EVALUATION:

Pityriasis versicolor is typically easily diagnosed based on its unique clinical appearance “hyperpigmented or hypopigmented, finely scaling patches or plaques”. The coppery-orange fluorescence of pityriasis versicolor may be demonstrated using a Wood lamp.

A potassium hydroxide microscopic examination confirms the diagnosis, revealing the typical “Spaghetti and meat ball appearance”. As the typical KOH mount lacks colour contrast, methylene blue / Swartz-Medrik stain can be used. Malassezia species are notorious for being difficult to culture in the laboratory due to their stringent culture requirements.

TREATMENT:

Topical medicines are the first-line treatment for pityriasis versicolor. Non-specific antifungal medications (selenium sulfide 2.5%, zinc-pyrithione & sulfur with salicylic acid) eliminate necrotic tissue and prevents further spread, whereas certain antifungal therapies have fungicidal / fungistatic effects. Imidazoles such as (ketoconazole 2%, miconazole, isoconazole), allylamine (terbinafine 1%) & ciclopirox olamine 1%, are some of them.

In the case of extensive, severe, stubborn, or recurring pityriasis versicolor, oral medicines are considered a second-line therapeutic option. Systemic medicines such as itraconazole and fluconazole are favoured over oral ketoconazole, which is no longer authorized due to the possibility of hepatotoxicity.[29]

3 : INFESTATIONS

I. SCABIES

Scabies is an parasitic infectious skin disorder caused by a mite infestation. Sarcoptes scabiei var hominis mite burrows beneath the skin, causing intense itching. This itching is constant, especially at night.

EPIDEMIOLOGY:

Each year, an estimated 300 million people worldwide are infested with scabies. It is a major health problem in many underdeveloped nations, and the World Health Organization designated it as a neglected skin condition in 2009.

Scabies is common in the following countries: Africa, South America, Australia, and Southeast Asia. The high frequency is associated with poverty, low nutritional status, and poor cleanliness.

It is quite frequent among children and young adults. Cases in these nations have a high morbidity rate because to complications and subsequent infections. Abscesses, lymphadenopathy, and post-streptococcal glomerulonephritis are examples of these.

In industrialized nations, scabies outbreaks can occur sporadically or as institutional outbreaks in schools, nursing homes, hospitals, jails, retirement homes, other overcrowded regions.

ETIOLOGY:

Sarcoptes scabiei var. Hominis is the mite that causes scabies. Direct contact or fomite transfer via clothing or bed linens can potentially result in disease spread.[30]

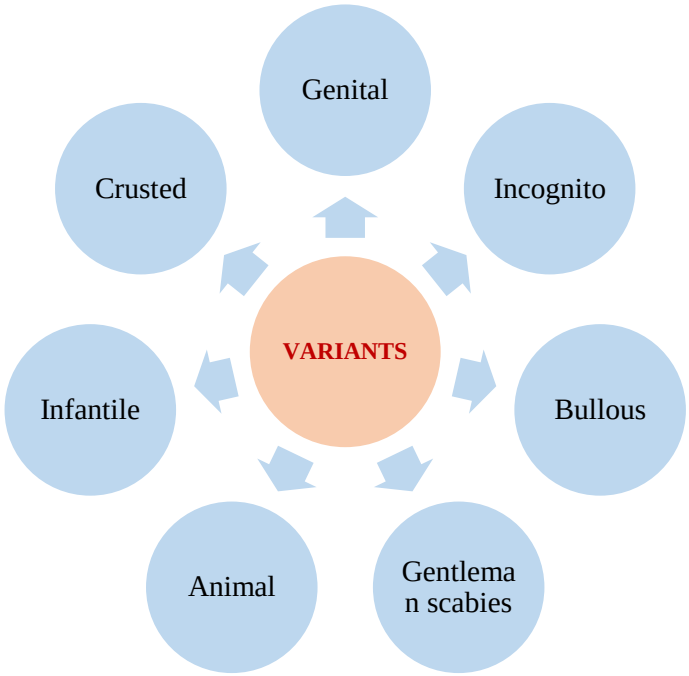


FIGURE 5: VARIANTS OF SCABIES.[31]

DIAGNOSIS:

Scabies can be diagnosed by seeing mites in stratum corneum skin scrapings. Because of the significant possibility of sample mistake, this approach frequently misses the proper diagnosis.

During the physical examination, other procedures, such as non-invasive videodermatoscopy, might be used. Videodermatoscopy employs a video camera linked to digital systems and outfitted with optic fibers, lenses with magnifications of up to 1000x, and a light source or immersion liquid.

In Dermoscopy, the burrow structure of scabies, sometimes known as the "jet in contrail," may be seen.

A skin biopsy might be done to confirm the diagnosis which shows mite and its product if the diagnosis is equivocal.

TREATMENT: [32]

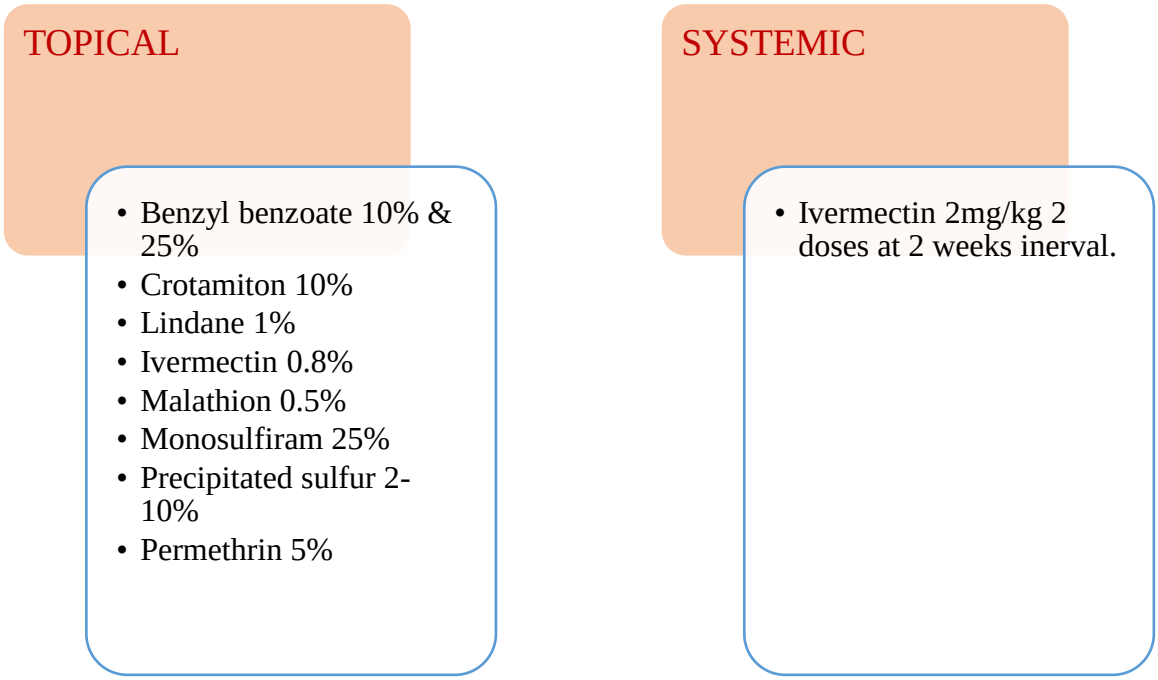


FIGURE 6: TREATMENT OF SCABIES.

II PEDICULOSIS

Lice are obligate, parasitic insects which causes pediculosis (louse infestation). They spread by direct skin-to-skin or fomite-to-skin contact. Human parasites include Pediculus humanus capitis (head louse), Pediculus humanus (body louse) & Pthirus pubis (pubic louse).

Scratching is caused by itching and leads to secondary bacterial infection. This can eventually develop to issues including impetigo and pyoderma. Humans can contract epidemic typhus, trench fever and relapsing fever through body lice.

The pharmacologic treatment of lice relies on two mechanisms: neurotoxicity, which results in louse paralysis, and suffocating via topical administration. It is critical to understand that existing treatments kill lice but do not consistently remove eggs.[33]

III PAEDRUS DERMATITIS

Paederus dermatitis is a rare, irritating contact dermatitis characterized by the abrupt emergence of erythematobullous lesions on exposed body parts. The sickness is caused by an insect of the genus Paederus. This beetle does not bite or sting, but accidental touching or crushing causes the release of coelomic fluid, which includes paederin, a strong vesicant agent. Paederus is a huge genus with about 650 species that are found on all continents except Antarctica. The irritation is first removed by cleaning the affected region with soap and water. Cool damp soaks should be applied to the blistered area, followed by a powerful topical steroid.[34]

4: ECZEMATOUS DISORDERS

I. SEBORRHEIC DERMATITIS

Seborrheic dermatitis (SD) is a common inflammatory skin condition that often affects the scalp, face, and body folds. Infants and adults can develop seborrheic dermatitis, a chronic, recurrent, and often moderate type of dermatitis. The degree of severity can range from little, asymptomatic scalp scaling (dandruff) to more pervasive involvement. Although seborrheic dermatitis has been linked to human immunodeficiency virus (HIV) infection,

Parkinson disease, a variety of other neurologic conditions, and usage of neuroleptic drugs, affected persons are often in good health.

EPIDEMIOLOGY:

Seborrheic dermatitis is more commonly seen in adolescence and adulthood than in newborns. Approximately 3% of people have clinically severe seborrheic dermatitis, with the third and fourth decades seeing the highest frequency. With moderate instances included, the true frequency is probably much greater. Men experience it more often than women.

The frequency of seborrheic dermatitis is estimated to be 5% globally, while dandruff, which is a non-inflammatory variant, is almost 50%. All races and ethinicity worldwide are impacted by SD. This condition has an bimodal prevalence, peaking during the first three months of life and then again during the fourth decade following adrenarche.

ETIOLOGY:

Certain factors are linked to the etiology of SD. This includes the genetic individual susceptibility, Malassezia spp. fungus as anormal skin flora & skin composing of lipids. SD is characterised by increased sebum production as well as inflamed skin. Both the quantity of yeast and the amount of sebum generated don’t seem to be an important variables.

DIAGNOSIS:

The most significant clinical aspect of seborrheic dermatitis is the location of lesions, with lesions more frequently appearing on the scalp and face, which have dense concentrations of sebaceous glands. ISD often has no symptoms, however it frequently coexists with atopic dermatitis. On the other hand, individuals with ASD present with burning and itching, particularly with the scalp lesions, although they are not associated with atopic dermatitis.

A non-inflammatory variety of SD known as sicca or pityriasis capitis is the mildest form. It predominantly affects the “scalp” and "beard region" and has shedding of tiny, light-colored skin flakes (scaling), which are frequently mistaken for "dandruff" when viewed against a backdrop of black clothes.

Adult SD most frequently affects the face, scalp, and chest, with lesions forming there in 88%, 70%, and 27% of cases, respectively. The Centro-facial area which comprises the central forehead, medial aspect of eyebrows, malar region, nasolabial folds, postauricular area, the external ear canal are all affected by seborrheic dermatitis. Blepharitis which is a eyelid inflammation is a frequent finding seen.[35]

TREATMENT:[36]

NON SCALP SEBORRHEIC DERMATITIS		SCALP SEBORRHEIC DERMATITIS	
Antifungal	Ketoconazole Ciclopiroxolamine Sertaconazole Metronidazole Itraconazole Lithium Gluconate	**Antifungal**	Ketoconazole shampoo Ciclopiroxolamine shampoo
Corticosteroids	Hydrocortisone	**Corticosteroids**	Clobetasol propionate
Immunomodulator	Calcineurin inhibitors	**Keratolytics**	Lipohydroxy acid Propylene glycol
Combination	Promiseb cream	**Combination**	Promiseb Scalp wash Ciclopiroxolamine + SA Ciclopiroxolamine + Zinc pyrithione

TABLE 4: TREATMENT OF SEBORRHEIC DERMATITIS

II. CONTACT DERMATITIS

Contact dermatitis is an eczematous inflammatory skin condition. It is induced either by contact with chemicals or metal ions that are harmful but not eliciting a T-cell response, or by tiny reactive compounds that change proteins and generate innate and adaptive immunological responses (contact allergens).

Allergic contact dermatitis [ACD] and Irritating contact dermatitis [ICD] are the two types of contact dermatitis. Irritant contact dermatitis is a generic skin response to direct

chemical injury that releases inflammatory mediators mostly from epidermal cells, whereas allergic contact dermatitis is a delayed (type 4) hypersensitivity reaction to external contact antigens.[37]

EPIDEMIOLOGY:

Females, newborns, old age, and those with atopy are more prone for ICD. It has been claimed that irritating contact dermatitis accounts for up to 80% of instances of occupational dermatitis. Age, work, and a history of atopic dermatitis are all risk factors for allergic contact dermatitis. People of Fitzpatrick type I & II (Red hair & pale complexion) are more prone to get CD. Due to the continual usage of jewelry and scents, females are more prone to acquire contact dermatitis.

ETIOLOGY: [38,39]

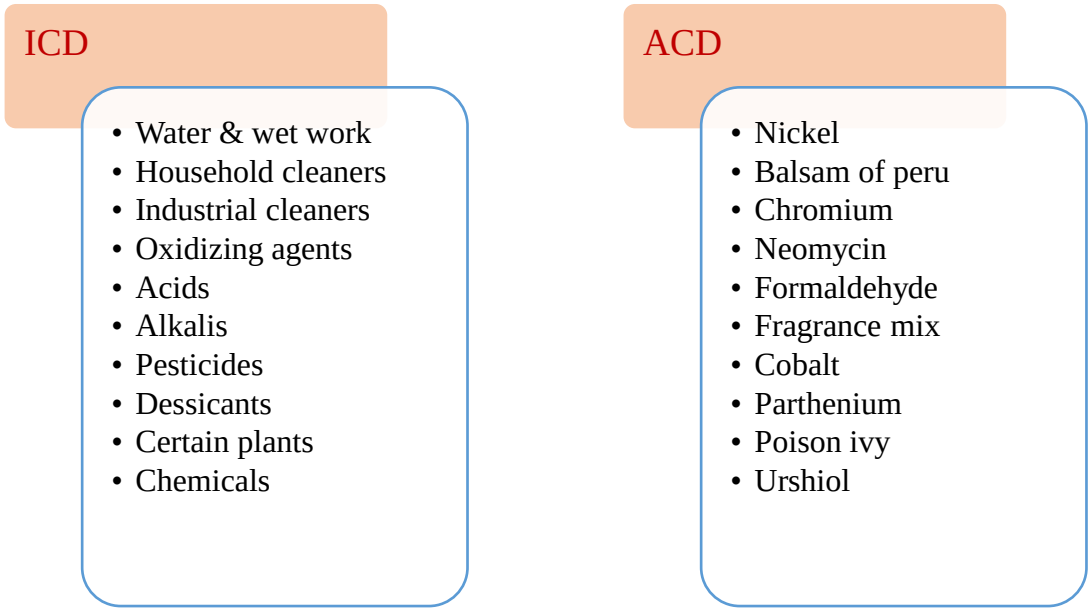


FIGURE 7: ETIOLOGY OF CD

DIAGNOSIS:

In order to diagnose contact dermatitis, history about their profession, any topical or oral medications, their hobbies must be obtained. Gold standard diagnostic test is “Patch testing” as the exact cause can be determined. A cotton tip with a small amount of dimethylglyoxime and hydroxide solutions is used. This is rubbed onto the metal surface of the jewelry in case of nickel allergy sufferers. If pink colour emerges onto the applicator, it is indicative of a positive nickel test. Women who are nickel allergic can perform this test even at home to check for nickel presence in their jewelry.

TREATMENT: [40]

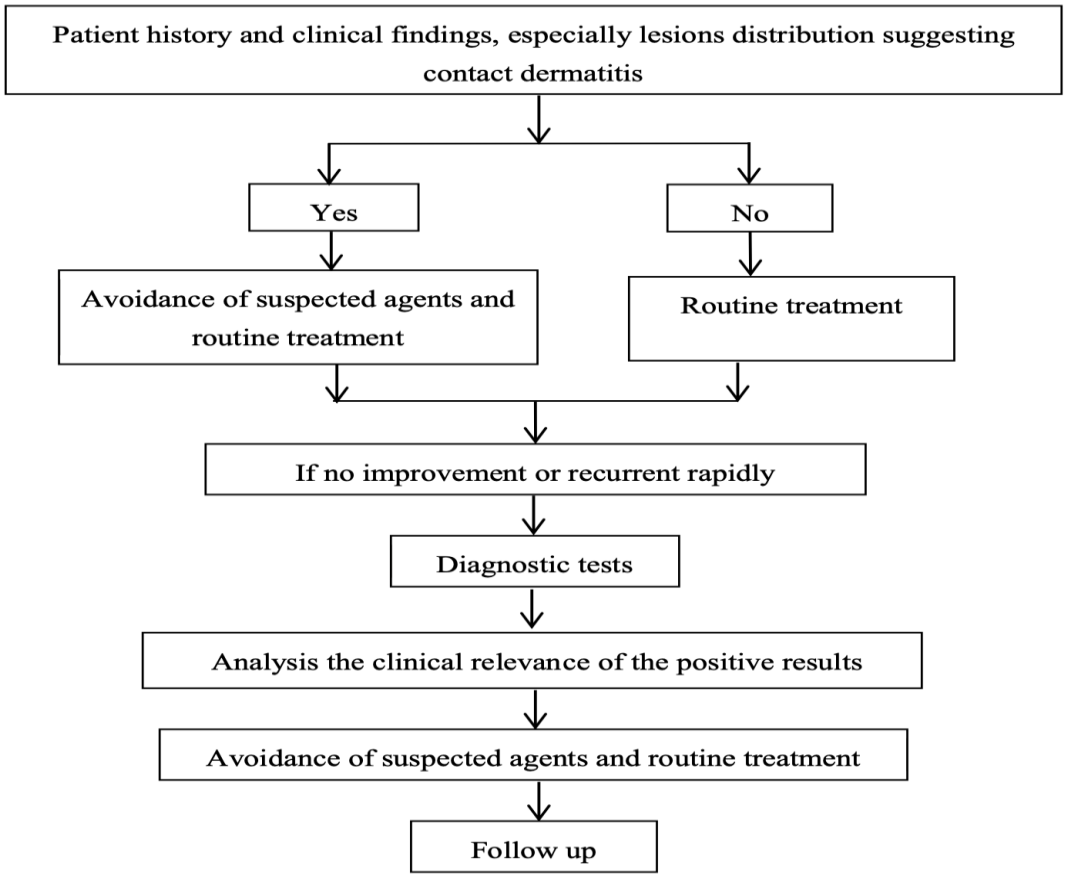


FIGURE 8: TREATMENT OF CONTACT DERMATITIS

To minimize inflammation, high potent topical corticosteroids can be administered. In order to prevent risk of skin atrophy, high-potent corticosteroids are not recommended on areas with thin skin, such as the face, genitals, or intertriginous regions. To treat pruritus, antihistamines are indicated. In extreme instances, systemic steroids are used, but they should be decreased carefully to avoid recurrences. Certain triggers such as harsh soaps, dyes, fragrances & friction should be avoided. Emollients are substances that hydrate the skin. Topical calcineurin inhibitors which is an immunomodulator helps to suppress calcineurin and can be used to treat ACD.[41]

III. PITYRIASIS ALBA

Pityriasis alba is a widespread, benign skin condition that primarily affects children and teenagers. The term is derived from its appearance referring pityriasis to thin scales, and alba alludes to its light hue. The majority of individuals with pityriasis alba have been associated with atopy which is one among the several factors in the diagnostic criteria of AD. It is characterized by "ill-defined macules and patches (or thin plaques), which are round or oval, frequently with minor scaling, and occasionally with mild pruritus". Most predominant site is on the face (particularly the cheeks), upper trunk & limbs.

EPIDEMIOLOGY:

Pityriasis alba is frequently seen in children of age 3-16 years, accounting for 90% of all the cases. An estimated 5% of American children may be impacted. No evidence of racial preponderance is seen. However, it is more apparent in individuals with darker shades of skin due to its classical hypopigmented feature. Pityriasis alba has no seasonal variation, however scaling may be seen greater in winter season (dry air) and lesions are more visible in the

spring and summer (sun exposure and darkening of the perilesional skin). Spontaneous resolution is achieved in about a year.

PATHOPHYSIOLOGY:

Pityriasis alba has the microscopic appearance of a moderate, persistent, nonspecific dermatitis with diminished melanin synthesis. There have been several nonspecific histopathologic characteristics documented. Hyperkeratosis, parakeratosis, acanthosis, spongiosis, and dermal perivascular infiltrate with pigment incontinence are examples of these. Although no particular diagnostic criteria exist, several characteristics features are diagnostic. These include uneven/significantly reduced basal layer melanin, no change in normal melanocyte count, and a reduction in the number of active melanocytes with a reduction in the number and size of melanosomes.

DIAGNOSIS:

The lesions may be exacerbated yet are nonfluorescent when examined with a Wood's light. This observation contrasts with vitiligo, which has brighter fluorescence and margins with greater demarcation. A negative results for fungus in KOH preparation will be observed. This is one of the differentiating feature with that of tinea versicolor / tinea corporis where both of which will show fungal components. Skin biopsy is not always necessary.

TREATMENT:

Sun exposure should be avoided in the afflicted regions, as surrounding skin pigmentation affects the aesthetic look. Topical low-potent steroids may alleviate symptoms like erythema and pruritis while also hastening repigmentation. Sunscreens protects the affected lesions from sunlight and also prevents darkening of the surrounding skin. Topical

calcineurin inhibitors “0.1% tacrolimus ointment & 1% pimecrolimus cream” have also been shown to be efficacious; however, they are rarely used due to their high cost. Topical vitamin D analog was also shown to be effective. PUVA and targeted phototherapy with a 308-nm excimer laser are two alternative therapeutic options that are normally reserved for severe patients.[42]

IV. ATOPIC DERMATITIS/

The most prevalent chronic inflammatory eczematous skin condition is atopic dermatitis (AD). This chronic condition linked with pruritus typically begins in childhood and manifests as dry skin, eczematous lesions, and lichenification. AD is thought to be linked to other IgE-related illnesses such as allergic rhinitis, food allergies & asthma.

EPIDEMIOLOGY:

In wealthy nations, atopic dermatitis affects 10% to 30% of children and 2% to 10% of adults. In recent decades, the incidence has climbed two to thrice. Atopic dermatitis is more common in temperate climate places, which may be attributed to less exposure to sun & low humidity. Based on the age onset, atopic dermatitis is classified into three subtypes:

- Early-onset AD (Birth - 2 years): This is the most prevalent type accounting for around 60% of cases.
- Childhood phase: This is seen in 2 to 12 years of individuals.
- Late-onset AD: Symptoms appear after puberty.
- Senile onset AD: This is a rare subtype that manifests in adults beyond the age of 60.[43]

ETIOPATHOGENESIS:[44]

Atopic dermatitis has a complicated etiology,

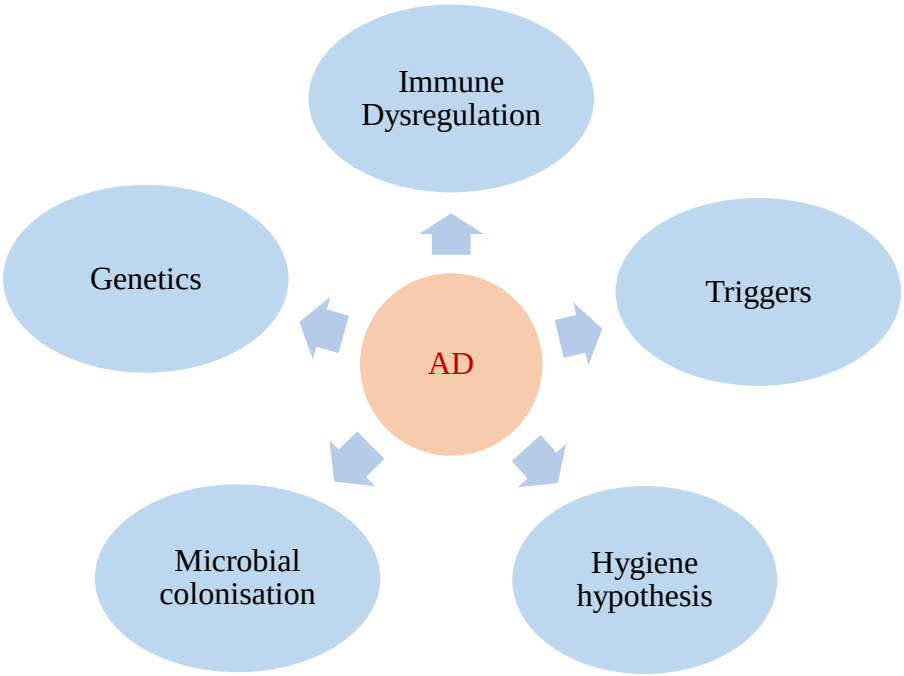


FIGURE 9: ETIOPATHOGENESIS OF ATOPIC DERMATITIS

AD is a component of the atopic trio “Atopic dermatitis, allergic rhinoconjunctivitis, and asthma”. This manifests either concurrently or sequentially which is termed as the "Atopic march."

TREATMENT:[45]

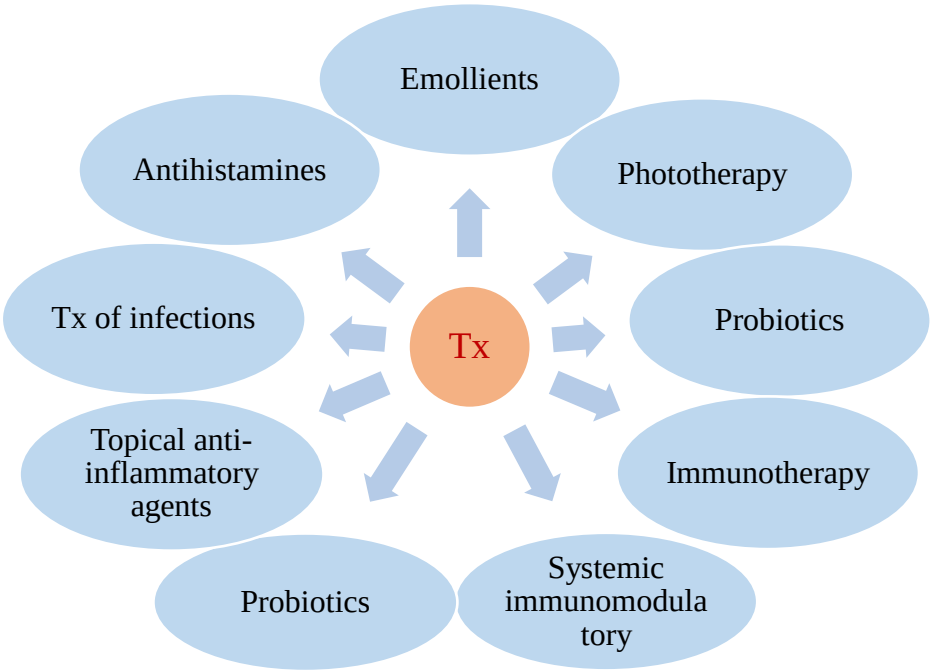


FIGURE 10: TREATMENT OF AD

VI. INTERTRIGO

Intertrigo is defined as "superficial inflammatory skin disorder that affects the flexural surfaces of the skin". This condition is triggered or aggravated by hot climates, friction, inadequate ventilation, wetness, and maceration. Intertrigo in Latin is described as inter "between" & terere "to rub".

The primary causes of intertrigo are mechanical forces and subsequent infections. Heat from maceration is essential in promoting this process. The skin folds are subjected to continual frictional stresses, which cause irritation sometimes leading to erosions of the irritated skin. Furthermore, moisture accumulates in the damaged intertriginous tissues, creating a breeding ground for other secondary infection.

Intertrigo may afflict people of all ages, from infancy to senior age. Environmental and genetic variables both play a role in their pathophysiology. Irritation to the epidermis caused by friction between the skin folds is one of the variable.[46]

5 : PHOTODERMATOSES

I. POLYMORPHIC LIGHT ERUPTIONS

The most prevalent kind of immunologically induced photosensitive dermatoses is polymorphic light eruption. Robert Willan was the first to describe features of PMLE at eighteenth century and used the term “ECZEMA SOLARE”. In the spring or summer, exposure to sunlight causes an itchy rash that, as long as additional exposure is avoided, goes away quickly. Juvenile spring eruption (vesicles on young boys' ears) and PMLE sine eruption are examples of variations (pruritus on sun-exposed skin without visible skin changes). The terms "polymorphic light eruption" and "polymorphous light eruption" are interchangeable.

EPIDEMIOLOGY:

Around the world, polymorphic light eruptions are not universally common. It may happen to those who are affected every time they walk outside or just sometimes. All skin types are affected, however lighter skin is more frequently affected than darker skin. It can start at any age, but ladies between the ages of 20 and 40 are the ones that experience it the most frequently. Polymorphic light eruptions are more common at high altitudes than at sea level. [47]

PATHOPHYSIOLOGY: [48]

There are various theories proposed,

- Delayed type hypersensitivity/ Type IV hypersensity: Cell mediated immune response to neoantigen which is sunlight induced.
- Genetic predisposition leads to immunosuppression by UVR causing raised recognition of antigen.
- Reduced TH2 Skewing and Decreased Langerhans cell migration.
- Arachidonic acid metabolism is abnormal.

PHYSICAL EXAMINATION:

In temperate climes, the spring or early summer is when the first signs of polymorphous light eruption often show following initial exposure to bright sunshine. An itchy rash develops hours to days later on newly exposed skin, such as the forearms, backs of hands, lower legs, and feet. The face is seldom affected.

If more UV exposure is avoided, the rash lasts for a few days before going away without leaving any scars. Depending on latitude, polymorphic light eruption tends to return every year and, in some people, remains all year long.

TREATMENT:[49]

TREATMENT:[49]

Finding the cause of the eruption and making an effort to reduce it are necessary for management.

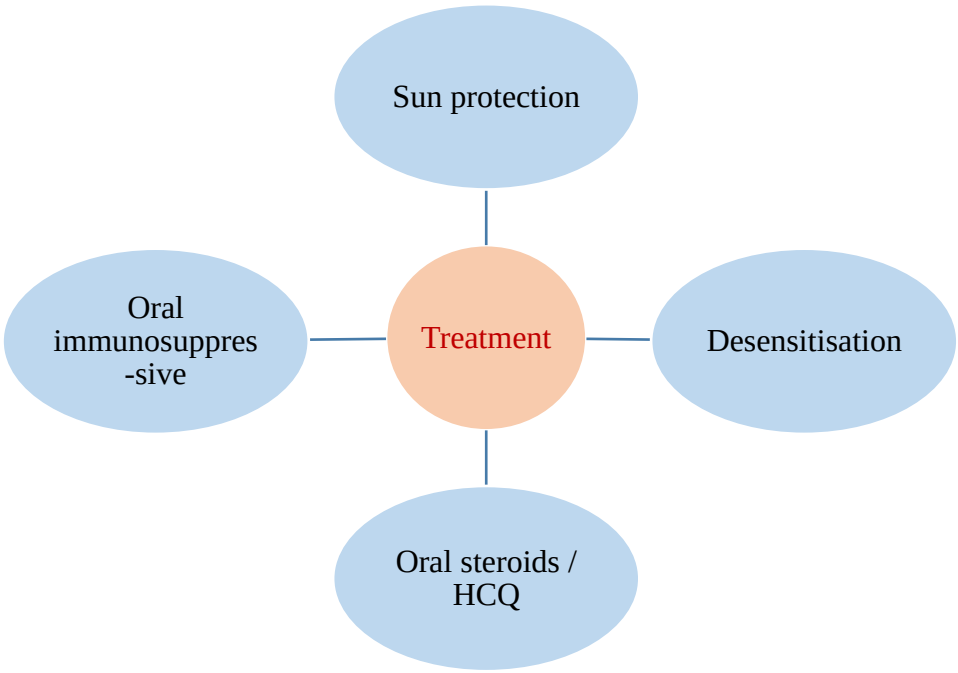


FIGURE 11: TREATMENT OF PMLE

6 : IMMUNOBULLOUS DISORDERS

Immunobullous illnesses are cutaneous blistering disorders produced by pathogenic antibodies attaching to protein targets in the skin. There are several immunobullous illnesses with distinct shape that is related to the structural features of the target protein.[50]

I. PEMPHIGUS VULGARIS

PV is an autoimmune illness that causes blisters on the cutaneous and mucosal surfaces. Pemphigus is derived from the Greek word meaning blister, pemphix. Although PV

has not been demonstrated to be communicable as previously assumed, potential triggers for PV in people with other autoimmune illnesses have been found. PV is caused by autoantibody formation against desmoglein 1 & 3. The reported annual incidence is between 0.1 and 0.5 per 100,000 individuals. PV is four times more prevalent in women than in men. Biopsy was used to evaluate the patient. Systemic corticosteroids & immunosuppressive drugs are the main stay of treatment.[51]

II. PEMPHIGUS FOLIACEOUS

Pemphigus is a potentially fatal autoimmune vesiculobullous condition defined by the "presence of circulating antibodies against desmogleins, which are critical components of epidermal intercellular adhesion integrity". However, unlike pemphigus vulgaris (PV), which is characterized by mucosal lesions, pemphigus foliaceus (PF) is characterized by solely skin involvement and no mucosal symptoms. The presence of HLA-DR4, DR-14, and DR-1 has been linked to susceptibility to pemphigus foliaceus. Pemphigus foliaceous is less frequent than Pemphigus vulgaris globally. Pemphigus foliaceous affects both men and women equally, and the average onset age is 50 to 60 years. Autoantibodies in all types of Pemphigus foliaceus appear to be pathogenic, according to experimental studies. These autoantibodies identify desmoglein-1, a 160-KDa desmosomal cadherin found in the skin of the upper torso.[52]

III. EPIDERMOLYSIS BULLOSA

The uncommon subepidermal blistering disorder known as epidermolysis bullosa acquisita (EBA) is brought on by antibodies binding to type VII collagen, which is present in the anchoring fibrils that connect the extracellular matrix proteins of the dermis to the epidermal basement membrane. It can be classified as either inflammatory or classical EBA. Skin fragility and tight bullae or erosions at the sites of mechanotrauma, such as the hands,

elbows, and knees, are characteristics of classic EBA. Tense bullae and mucosal ulceration are the hallmarks of inflammatory EBA, which resembles mucous membrane pemphigoid and BP. There may be significant mucosal involvement, affecting the pharynx, larynx, and oesophagus, leading to dysphagia, dysphonia, and oesophageal strictures. Such patients must be managed using a multidisciplinary approach.[53]

7 : PAPULOSQUAMOUS DISORDERS

Skin conditions known as papulosquamous diseases have scale-covered plaques or papules that are crimson or purple. These represent a typical category among the diverse range of skin conditions that affect kids. Children may develop them owing to autoimmune conditions, viral or bacterial infections, or hereditary causes.[54]

I. PSORIASIS

A persistent inflammatory and proliferative skin disorder, psoriasis. Erythematous plaques with silvery scales, especially on the scalp and lumbosacral area, are its defining features. Although the actual cause is uncertain, T cells are thought to be the primary mediators of this autoimmune illness. HLA antigens are often associated with psoriasis, especially across a range of racial and ethnic groupings. Psoriasis can occur at any age. It has been determined that onset ages are bimodal. The average age at which psoriasis initially manifests itself might range from 15-20 years and a second peak occurring between the ages of 55-60.[55]

II. LICHENOID REACTION

Lichen planus, which affects the skin and mucous membranes, is clinically and histopathologically comparable to lichen reaction (LR). LR has a known cause and can be brought on by other substances or systemic drug exposure. All racial groups experience LR,

which is most frequently seen in people between the ages of 20-40 years, however it can happen at any age. The typical clinical sign of LR is a rash with tiny (3-5 mm) glossy, elevated, reddish-purple papules that itches. The rash often affects the back of the body, the anterior area of the arm and hand, and it occurs rapidly.[56]

III. LICHEN PLANUS

LP is an "inflammatory papulosquamous condition of the skin and mucous membranes with no recognized origin". The wrists, lower back, and ankles are the most typical locations for its pruritic, violaceous papules and plaques to form.[56] Lichenoid drug eruption, also known as drug-induced lichen planus, is commonly photographed but may be difficult to identify from idiopathic LP.

EPIDEMIOLOGY:

In adults across the world, cutaneous LP affects 0.2% to 1% of them. More frequently reported in 1% to 4% of the population is oral LP. Overall, a female preponderance is seen with male to female ratio of 1:1.5 and most instances appear between the ages of 30 and 60. Children make up fewer than 5% of all LP cases, thus it is uncommon among them.

ETIOPATHOGENESIS:[57]

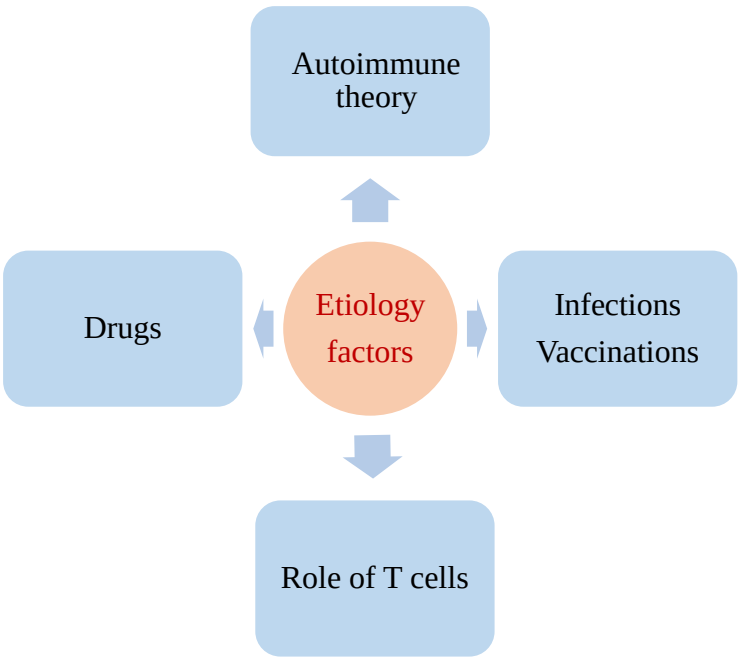


FIGURE 12: ETIOLOGICAL FACTORS

DIAGNOSIS:

Dermoscopy often enables visualization of Wickham striae while in the clinic. The typical discovery is a network of white lace like pattern with crimson globules at the edges. The presence of oral LP lesions in close proximity to dental fillings should trigger patch testing to check for metal allergies. The most effective method to confirm the existence of LP is a biopsy with microscopic investigation. As previously mentioned, lesions have a variety of features that often enable a precise diagnosis.

TREATMENT:

The main aim of the treatment for cutaneous LP is to lessen pruritus and shorten the time it takes for the condition to resolve, which normally takes 1 to 2 years. First line of therapy for localized LP would be administration of Super potent topical steroids (clobetasol)

BD for 2 - 4 weeks. In unresponsive cases, Intralesional steroid injections may be considered. For diffuse LP, first line of therapy would be oral corticosteroids (prednisone 30-60mg) daily tapered over 2 - 6 weeks. Second-line treatments would be Metronidazole, methotrexate, acitretin, sulfasalazine (increasing by 500 mg every three days for 3 to 6 weeks, until 2.5 grams daily is attained), PUVA, UVB, calcineurin inhibitors. Trimethoprim-sulfamethoxazole, tetracyclines, ciclosporin, griseofulvin, azathioprine, terbinafine, mycophenolate mofetil, etanercept, adalimumab, or low-molecular-weight heparin are examples of third-line treatments.[58,59]

8: PIGMENTORY DISORDERS

I. HYPOPIGMENTARY & DEPIGMENTARY

The most frequent skin lesions seen in daily clinical practice are hypopigmented macules. The term "hypopigmentation" refers to diminished pigmentation, which translates to much less melanin than in healthy skin. Contrast this with depigmentation, which refers to the full lack of melanin as a result of a large loss of melanocytes. Clinically, it might be difficult to distinguish between depigmented illnesses and hypopigmented disorders.

Depigmentation is described as the loss of color (pigment) in the eye's retina, mucous membranes, hair, or skin. Melanin, a coloring substance, is deposited and gives color to the skin, mucous membranes, hair, and retina. Melanocytes are specialized cells that create melanin.

I A. VITILIGO

The clinical manifestation of vitiligo, a common acquired hypopigmented cutaneous disorder which is caused by the loss of melanocytes from the basal layer of epidermis, is the appearance of distinct white patches all over the body. Although there are several hypotheses

about the pathophysiology of vitiligo, the actual cause is still unclear. It is a specific type of autoimmune disease.

EPIDEMIOLOGY:

The most frequent cause for depigmentation is determined to be vitiligo. Although it can manifest at any age, from childhood to maturity, the second and third decades are considered to have the highest occurrence. The age of onset often varies by gender. It affects all races equally and has a prevalence of 0.5% to 2% among all persons, including adults and children, globally.[61]

PATHOPHYSIOLOGY:[62]

The multifactorial polygenic condition that is vitiligo has a complicated pathophysiology. It is frequently linked to both hereditary and non-genetic variables. However, a number of hypotheses regarding its pathophysiology have been put forth, but the precise etiology is still unclear.

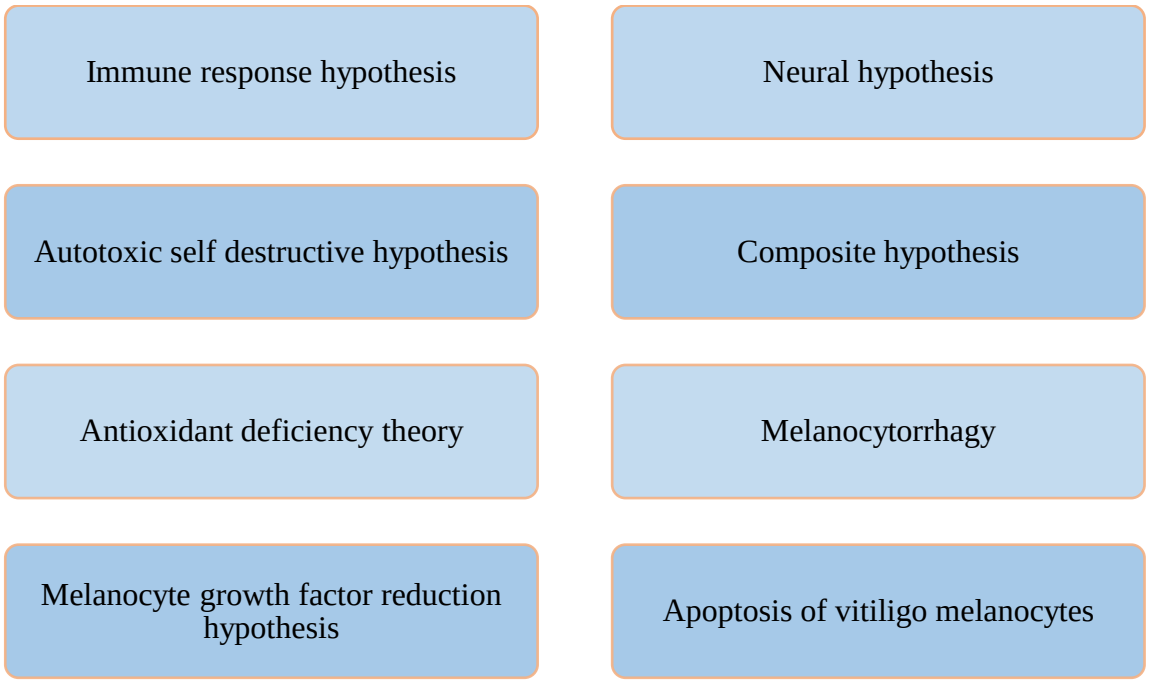


FIGURE 13 : PATHOPHYSIOLOGY OF VITILIGO

CLASSIFICATION:[63]

Localized	Focal	• Segmental distribution • ≧ 1 macule in one area
	Unilateral/segmental	• ≧ 1 macule in segmental distribution • Abruptly stops in midline
	Mucosal	• Mucous membranes alone
Generalized	Acrofacialis	• Face + distal extremities
	Vulgaris	• Widely distributed scattered patches
	Mixed	• Acrofacialis + vulgaris
Universalis	Complete or nearly complete depigmentation	
	Special forms	• Trichrome vitiligo • Quadrichrome vitiligo • Pentachrome vitiligo • Inflammatory vitiligo

TABLE 5: CLASSIFICATION OF VITILIGO

TREATMENT:

The use of corticosteroids, calcineurin inhibitors, and vitamin-D analogs are all examples of topical therapy techniques. One option for treatment is phototherapy. In most individuals with early-stage and localized illness, it causes repigmentation. Narrowband UV-B is often utilized, typically twice or three times a week with a wavelength of 311-312 nm. Due to the harmful side effects of psoralen photochemotherapy, it has mainly supplanted it. Vitiligo is treated with the excimer laser in small, stable patches.[64]

IB. PITYRIASIS VERSICOLOR

Tinea versicolor, another name for pityriasis versicolor, is a most common cutaneous superficial fungal infection. It is one of the illnesses caused by Malassezia. Fine scaly

macules that are either hyper- or hypopigmented are clinical signs of pityriasis versicolor. The trunk, neck, and proximal extremities are the areas that are most often impacted. Pityriasis versicolor is frequently diagnosed solely only on clinical evidence. In iffy situations, the use of an woods lamp and a microscopic examination of scales that have been sopped in potassium hydroxide may be beneficial. Induction treatment has good results for treating pityriasis versicolor. However, because to the high likelihood of recurrence, long-term maintenance therapy is frequently necessary.[65,66]

II. HYPERPIGMENTARY

It takes a lot of complexity to make pigment. Inflammation, UV protection, and several other processes all include it. There are reasons of hyper-pigmentation outside underlying illness. Examples include tattoos, tans, tanning creams, and adverse drug reactions.

II A. NEVUS OF OTA

The trigeminal nerve distribution area is predominantly affected by the benign melanosis known as nevus of Ota. The trigeminal nerve's first and second divisions, or the ocular V1 and maxillary V2, are most frequently affected. There is concomitant ocular hyperpigmentation. Ocular dermal melanosis, often termed as nevus of Ota.

EPIDEMIOLOGY:

The Asian community, which accounts for 0.014% to 0.034% of the population, is more frequently affected. The majority of the time, lesions are present at birth, however hormonal changes can also cause lesions to develop throughout puberty or during pregnancy. At a ratio of 5:1, females are more frequently afflicted than men. White folks are less likely

to experience it than persons of Asian and African origin. Although White people are far more likely than other races to get malignant melanoma.

Nevus of Ito can also appear in children or teenagers during puberty. Asian ethnicities and races with dark complexion are more frequently impacted. Nevus of Ota and nevus of Ito are occasionally discovered to be related.

PATHOPHYSIOLOGY:

The ophthalmic V1 and maxillary V1 divisions of the trigeminal nerve, which run along the distribution of the eye and its adnexa, respectively, are hyperpigmented. The eyes and periocular area have a blue pigmentation, which is indicative of hyperpigmentation. There are too many melanocytes in the dermis, and they can develop into malignant melanoma.

EVALUATION:

The nevus of Ota and Ito cannot be verified by a conclusive diagnostic test. The primary methods of diagnosis are clinical examination and history. Dermatoscopy reveals homogenous pigmentation that is bluish to slate grey. A skin biopsy is required if the surrounding skin becomes ulcerated or if papule growth is accompanied by a change in pigmentation. Deeper variations impact the periorbital regions, the temple, and the forehead, whereas superficial variants often affect the cheeks. To rule out ocular issues, yearly periodic re-evaluations with an ophthalmologist should also be carried out.[67]

DIFFERENTIAL DIAGNOSIS:

- Café-au-lait macules
- Nevus spilus

- Mongolian spots
- Vascular malformations
- Ecchymoses
- Drug-induced hyperpigmentation.[68]

TREATMENT:

To rule out any uveal melanomas, ophthalmological screening and recording of vision, intraocular pressures, a thorough slit-lamp examination, dilated fundus examination and gonioscopy, should be performed.

Dermatologic care is necessary for aesthetic reasons or if a malignant change occurs. The lesions can be removed via Mohs micrographic surgery, and the periocular tissues can then be rebuilt. A combination of surgical excision and radiation, transpupillary thermotherapy, or chemotherapy may be considered for more severe lesions or metastases. Lasers can be used to cure skin lesions cosmetically. For nevi of Ota and Ito, pulsed Q-switched laser surgery is the preferred course of action. By selectively photothermally and photomechanically destroying cutaneous melanocytes and melanophages, it reduces pigmentation.[67]

II B. LENTIGINES

Lentigines are also termed as “Liver spots” is a benign hyperpigmented lesion mostly seen on the parts of the body exposed to the sun, The faces and backs of the hands are frequent locations. Age-related increases in the lesions' frequency make them widespread among people in their middle and later years. They come in sizes ranging from 0.2 to 2 cm. These irregularly shaped, dark-colored flat lesions typically have distinct edges. These

lesions are brought on by a noticeable rise in pigment cells in the skin's superficial layers. If a lesion develops a very uneven border, changes in colour, or changes in thickness, a biopsy should be taken to rule out malignancy.

Since lentigos are mostly benign, no treatment is required. Cryotherapy, hydroquinone preparations (bleaching preparations), retinoid cream, chemical peels, and lasers are a few effective cosmetic procedures. Protective precautions should be used to prevent overexposure to sunlight. These include sun protection products and protective attire like caps and long sleeves.

II C. FRECKLES

Freckles are tiny brown patches that often appear in sun-exposed areas. Freckles are often not harmful. They develop as a result of melanin, the pigment responsible for skin and hair color, being produced in excess (pigmentation). In general, ultraviolet (UV) radiation stimulation causes freckles.

Ephelides and solar lentigines are the two types of freckles. Ephelides are the sort that most people often associate with freckles. Dark skin patches called solar lentigines appear on adults. This comprises age spots, sunspots, and freckles. The two varieties of freckles might have a similar appearance but differ in other respects, such as how they grow.[69,70]

II D. ACANTHOSIS NIGRIGANS

The skin darkening condition known as acanthosis nigricans typically affects intertriginous regions. The skin may thicken along with this hyperpigmentation, which typically appears in skin fold locations like the back of the neck, the axilla, and the groin.

Although acanthosis nigricans is most frequently linked to diabetes and insulin resistance, it can occasionally be an indicator of an underlying cancer. Additionally, some

drugs such oral contraceptives and systemic glucocorticoids as well as hormone abnormalities and their treatment may cause it.[71,72]

9: DISORDERS OF APPENDAGES

I. ACNE

Acne vulgaris (AV) is defined as "a chronically recurring, self-limiting, inflammatory condition of the pilosebaceous unit". Cutibacterium acnes causes acne vulgaris to appear throughout adolescence when dehydroepiandrosterone is naturally circulating in the blood (DHEA). It is a fairly common skin condition that often affects the seborrheic areas such as face but can also affect the upper arms, torso, and back. It can appear with both inflammatory and non-inflammatory lesions.

EPIDEMIOLOGY:

Adolescence is when acne may first emerge, and it can last into the early thirties. Males are more likely than females to get acne. Populations in urban areas are more affected than those in rural areas. 20% of those who are impacted get severe acne that leaves scars. It seems that certain races are more impacted than others. Severe acne is more common in Asians and Africans, but moderate acne is more prevalent in white people. People with darker complexion in general also prone to acquire hyperpigmentation. Neonatal can also get acne, although it often goes away on its own.[73,74]

PATHOGENESIS:[75]

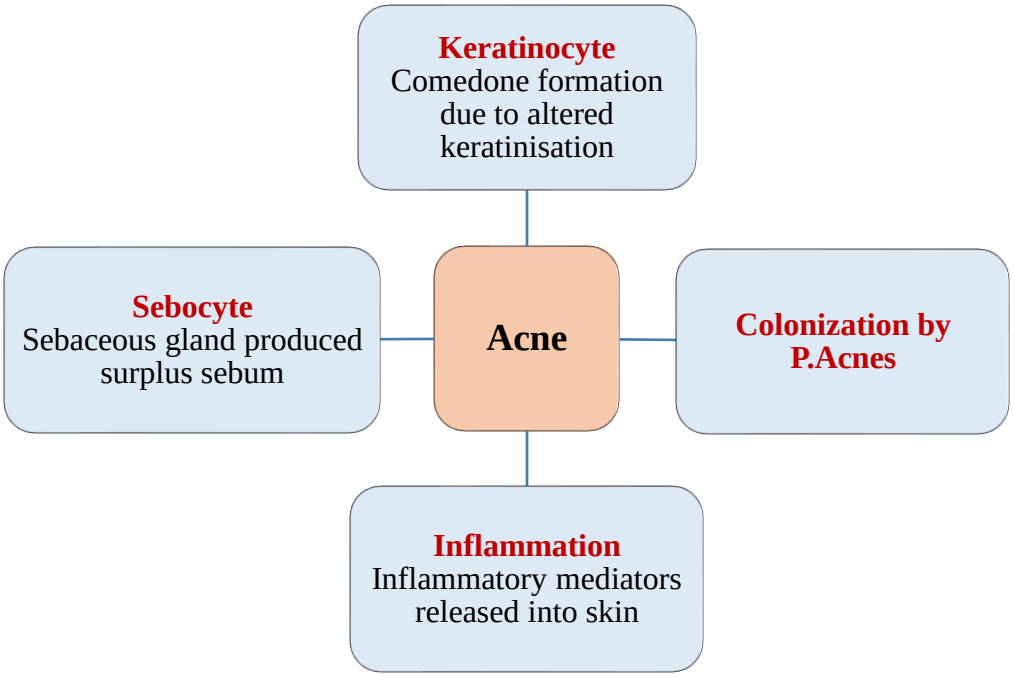


FIGURE 14: PATHOGENESIS OF ACNE VULGARIS

CLINICAL FEATURES: [77]

The back, upper trunk, and deltoid region of the body all have acne-prone centrofacial regions. Comedones are the first polymorphic lesions that acne shows as:

Grade 1	Comedones
Grade 2	Erythematous tiny papules
Grade 3	Pustules
Grade 4	Nodules and cysts

TABLE 6: GRADING OF ACNE

VARIANTS:[78]

1. Adult acne	2. Acne fulminans
3. Occupational acne	4. Gram negative folliculitis
5. Radiation acne	6. Acne aestivalis
7. Acne mechanica	8. Acne cosmetica
9. Tropical acne	10. Drug induced acne
11. Pomade acne	12. Chloracne
13. Acne conglobate	14. Acne mallora

TABLE 7: VARIANTS OF ACNE

THERAPY:[79]

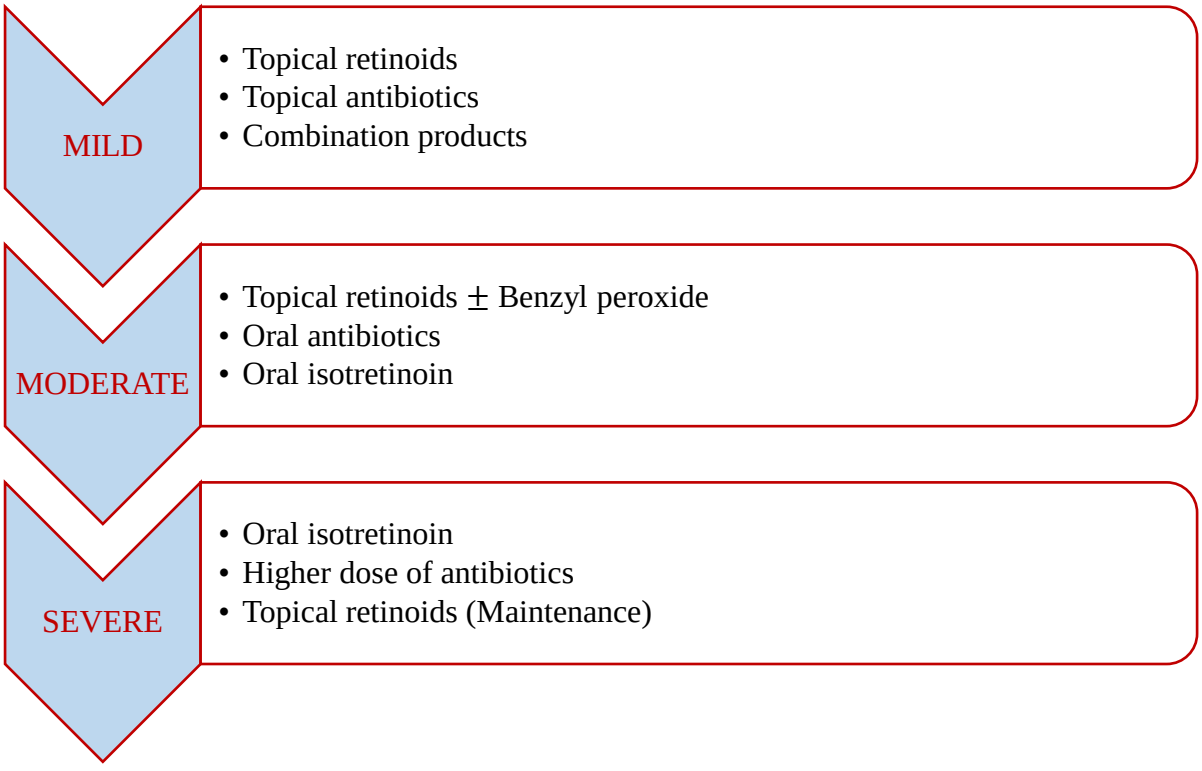


FIGURE 15: TREATMENT OF ACNE

COMPLICATIONS OF ACNE:

- Poor facial aesthetics
- Scars
- Psychological issues: Lack of self-esteem, anxiety & Depression.[80]

II. ROSACEA

Rosacea is defined as "Chronic inflammatory condition characterised by recurrent flushing, erythema, telangiectasia, papules, or pustules on the nose, chin, cheeks, and forehead". Rosacea can be classified into four different clinical subtypes: erythematotelangiectatic, papulopustular, phymatous, and ocular. The subtypes do not compete with one another.[81]

EPIDEMIOLOGY:

Since the majority of rosacea diagnoses are made purely on clinical judgment, many individuals, particularly those with moderate illness, may go untreated. The prevalence of rosacea is thought to be greater than 5% of the global population. It affects more than 10% of White people, is more prevalent in adults between the ages of 30 and 50. Women are more afflicted than men, and is more often characterised in people having pale skin (Fitzpatrick I and II).

ETIOLOGY:

It is unclear how exactly rosacea is caused. Known etiological causes for the onset of rosacea include genetics, immunological response, microbes, environmental variables, and neurovascular dysregulation. In addition, ultraviolet (UV) exposure may contribute to the

genesis of the condition in addition to its established action as a rosacea trigger. A greater disease incidence in the patients with a positive family history of rosacea provides evidence of a genetic susceptibility. Additionally, distinct HLA loci have been established in individuals having rosacea. Demodex mites are among the microorganisms that appear to be involved with rosacea since they are more prevalent on skin that has the condition, while it is unclear whether this is a cause or effect of rosacea.

PATHOPHYSIOLOGY:

Among the pathophysiological pathways proposed for rosacea are immune system activation, Demodex mite infestation, and neurovascular dysregulation.

It has been shown that rosacea patients' lymphatic and blood vessels enlarge when exposed to hot temperatures, spicy foods, and alcohol. The theorized mechanism for the erythema and flushing involves increased expression of transient receptor protentional vanilloid 1 (TRPV-1) and ankyrin 1 on keratinocytes & sensory neurons as well as release of vasoactive peptides after exposure to triggers.

Another recognized pathomechanism for rosacea is the activation of the innate and adaptive immune systems by upregulation of toll-like receptor 2 (TLR-2) and Th1/Th17, respectively. Mast cell activity is boosted by TLR-2 activation due to an increase in LL-37 synthesis.[82]

DIAGNOSIS:

Rosacea is predominately a clinical diagnosis. A thorough history about the potential triggers must be asked for. Patients having ocular symptoms should have an Ophthalmic evaluation.

TREATMENT:[83,84]

The first step in treating this condition is to counsel them on how to recognize & avoid triggers that includes certain foods, alcohol, UV radiation, and seasonal changes. All rosacea sufferers should use an pH-balanced facial cleansers rather than soaps, a broad-spectrum sunscreen with ≥ SPF 30 and moisturizers on a daily basis.

TOPICAL TREATMENT

Erythema: Oxymetazoline hydrochloride 1% cream, ivermectin 1% cream, Brimonidine tartrate 0.33% gel, azelaic acid 15% gel, foam or 20% cream, metronidazole 0.75% and 1% gel or cream.

SYSTEMIC TREATMENT

Flushing: Propranolol, carvedilol, clonidine.

Inflammatory papules and pustules: Doxycycline, minocycline, azithromycin, isotretinoin.

Phyma (inflamed): Doxycycline, tetracycline, isotretinoin.

Procedures/Interventions: Intense pulsed light therapy, Nd:YAG laser, CO2 laser, surgery & electrosurgery.

Ocular Involvement: Fusidic acid gel, metronidazole gel, cyclosporine eye drops.

PERIORAL DERMATITIS

POD is a “benign inflammatory condition that most frequently affects young females, characterised by pink, scaly areas around the mouth or tiny, inflamed papules and pustules”. The perioral region is where the illness is most frequently found, however it can also affect the periocular and paranasal skin. Hence the term “Periorificial dermatitis” is designated for this condition. This may be brought on by topical steroid usage on the face, thus the patient should stop applying steroid creams as the first line of therapy. Adolescent females are more likely to develop CD in the periorificial area.

EPIDEMIOLOGY OF PERIORAL DERMATITIS

Perioral dermatitis commonly affects young females. There is no significant gender or racial preponderance in the pediatric population.[85]

CLINICAL FEATURES

Around the lips, eyes, and nose, erythematous papules usually bilateral but may also be unilateral in appearance are seen in these patients. In addition, there may be vesicles/pustules & scaling,. Usually, the vermillion edges of the lips remain unaffected. Periocular region, nasolabial area & perioral areas are usually affected. Unusual involvement of the ears, neck, scalp, vulva, and extremities has also been documented.

Although skin biopsy should be taken into consideration in the event of an unusual presentation or poor response to therapy. In vast majority of individuals with POD, diagnosis is usually made with clinical features.[86]

ETIOLOGY OF PERIORAL DERMATITIS:[87]

Perioral dermatitis has an unkown specific etiology.

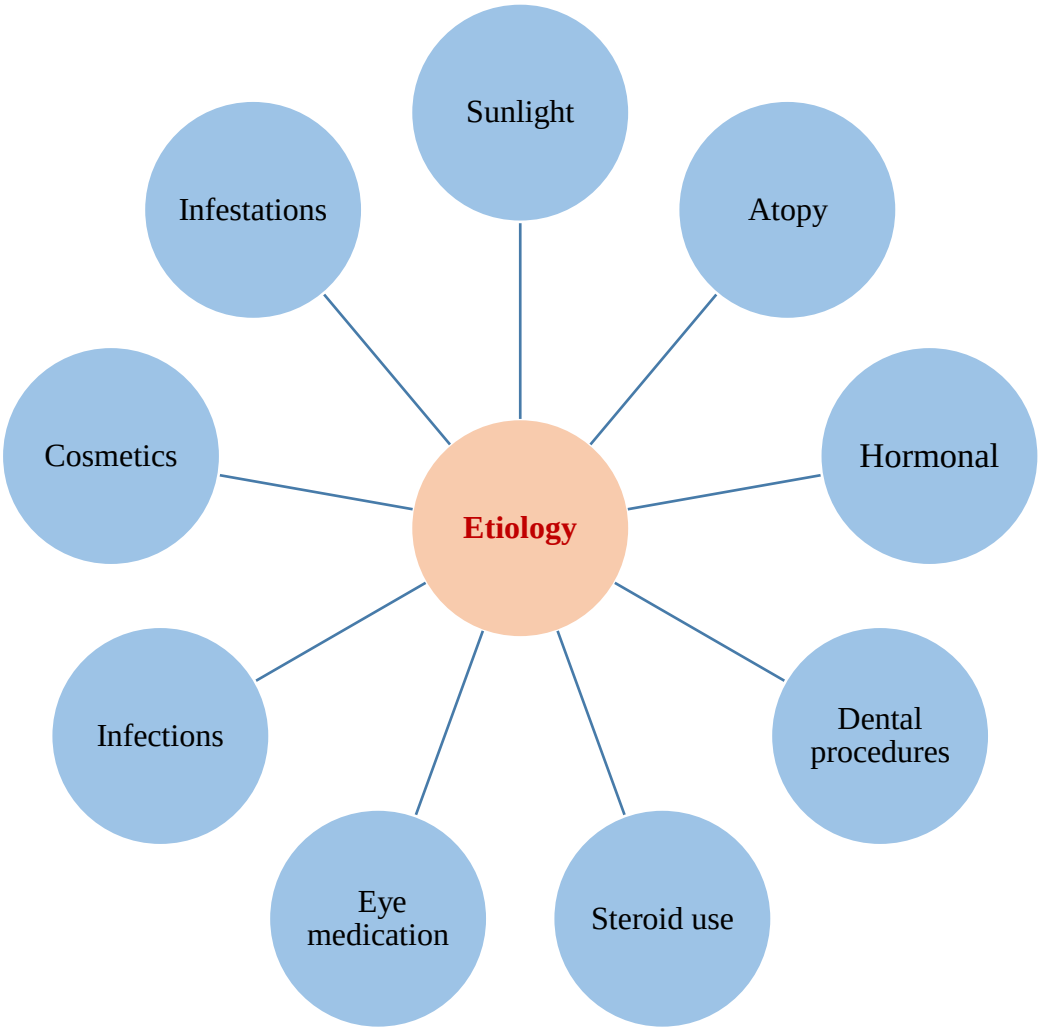


FIGURE 16: ETIOLOGICAL FACTORS OF POD

PROGNOSIS:

After stopping the problematic substance, such as topical steroids or skincare products, the rash may fully clear up in some people. However, perioral dermatitis frequently develops into a chronic, recurrent illness that needs long-term care.

COMPLICATIONS:

- Emotional pain brought on by the disease's protracted nature
- Poor quality of life brought on by often disfiguring facial lesions
- Atopic dermatitis' itching might make it difficult to fall asleep.
- Depression and anxiety are linked to POD. This can be connected to the chronic itchiness and sleep issues that are typical of atopic dermatitis sufferers.[88]

THERAPIES:[89]

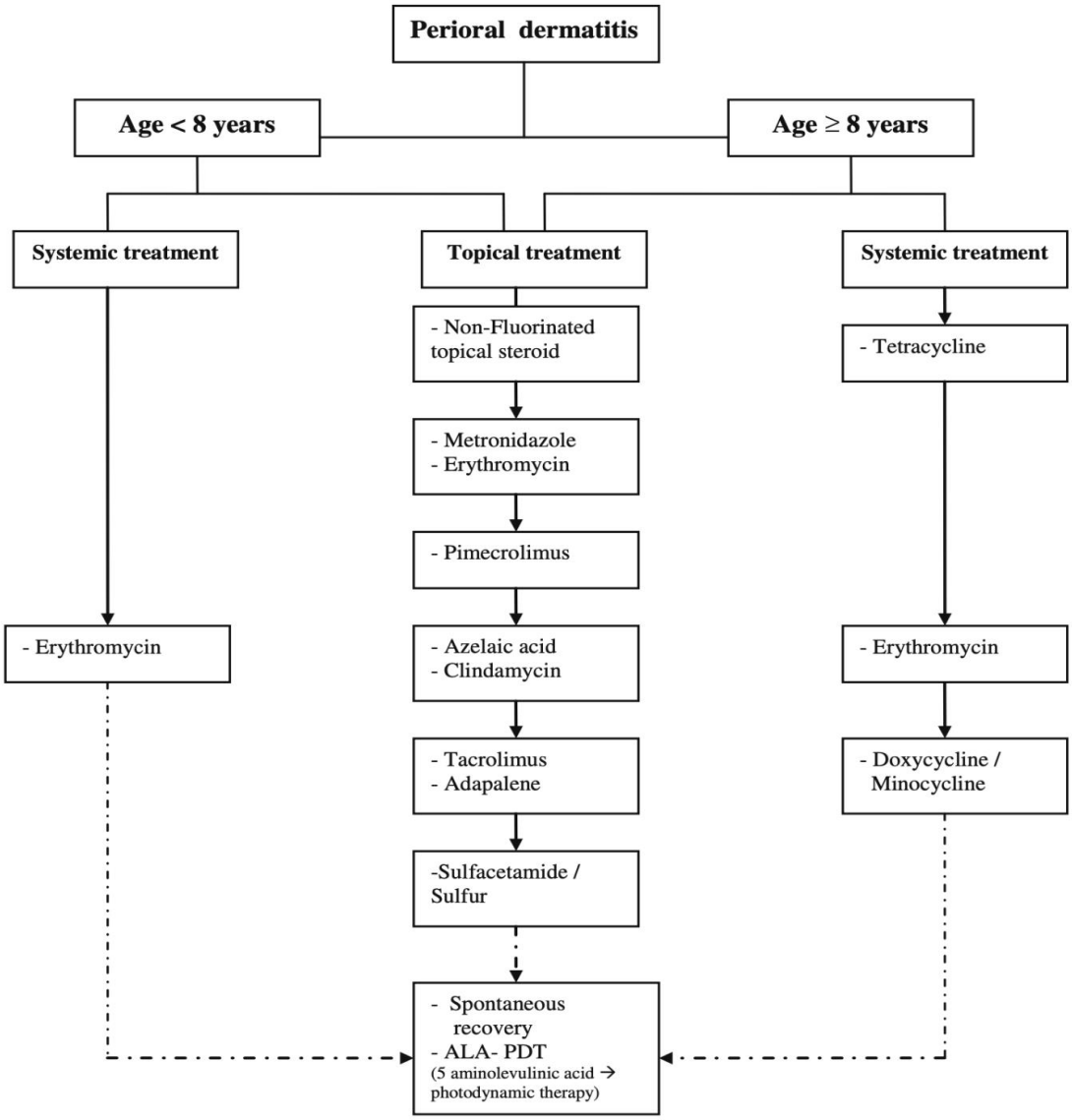


FIGURE 17: TREATMENT OF POD

III. MILIARIA

Eccrine miliaria, often known as miliaria, is a skin condition that is frequently observed. Eccrine sweat is forced back into the dermis or epidermis as a result of clogged eccrine sweat glands and ducts. This backflow causes sweat-filled vesicles to develop under the skin as a rash. This skin ailment also known as "sweat rash," "prickly heat,"/"heat rash". In warm, muggy settings, it occurs most frequently in the summer. The three primary forms of miliaria are crystallina, rubra, and profunda, and they are distinguished from one another by the degree of sweat duct blockage that results in clinical and histological variations. Usually, the rash is self-limiting and goes away without any medical intervention.[90]

IV. MILIA

Milia (plural: milium) are tiny, firm white papules that can appear anywhere on the body, although they most frequently appear on the face, upper torso, extremities, and genital region. Milia are benign, temporary keratine dermal cysts (prepuce).

Milia are divided into major and secondary categories. Congenital milia, mostly located across the scalp, eyelids, nose , cheeks, and palate, make up the great majority of primary milia cases (Epstein pearls). Yet another proportion of primary milia may develop in children and adults with specific uncommon genodermatoses. While pharmaceutical side effects, skin trauma, or underlying skin pathologies might cause secondary milia.[91]

10: SKIN TUMOURS

I. KELOID

Keloids are the outcome of aberrant wound healing brought on by inflammation or skin damage. Genetic as well as environmental factors have a role in the keloid formation. Darker colored people of African and Hispanic heritage have an higher incidence. The

etiology of keloids is thought to be mediated by excessively active fibroblasts that produce large quantities of collagen and growth factors.

EPIDEMIOLOGY:

Compared to Caucasians, those with dark complexion who are of African, Asian, or Hispanic heritage experience increased rates of keloid formation. In these people with darker pigmentation, the incidence ranges from 4.5% to 16%. Pregnancy and puberty have considerably increased incidence rates. Although no unique gene has been found, a positive family history has been attributed to an increase in the likelihood of keloid formation. Rubinstein-Taybi syndrome and Goeminne syndrome are two rare genetic diseases that can increase the likelihood of developing keloids.

ETIOLOGY:

Keloid growth is influenced by both hereditary and environmental factors. Following any degree of skin damage, such as surgery, piercings, acne, tattooing, bug bites, burns, lacerations, abrasions, immunizations, and any other procedure causing cutaneous inflammation, predisposed people may develop a keloid. Keloid development may also be facilitated by increased strain in a wound.[92]

EVALUATION:

Diagnosis is predominantly clinical. Biopsy is not required unless the diagnosis is not definitive. In such cases, a biopsy is necessary as it is diagnostic.

TREATMENT:

Treatment methods included corticosteroids, cryotherapy, surgical excision, radiotherapy, and laser treatment. There have also been reports of other treatments, such as

topical imiquimod after excision, intralesional bleomycin, intralesional botox, intralesional 5-fluorouracil, and silicone gel sheeting, being effective either alone or in conjunction with the before mentioned modalities in treating keloid scars.

PATHOPHYSIOLOGY:[93]

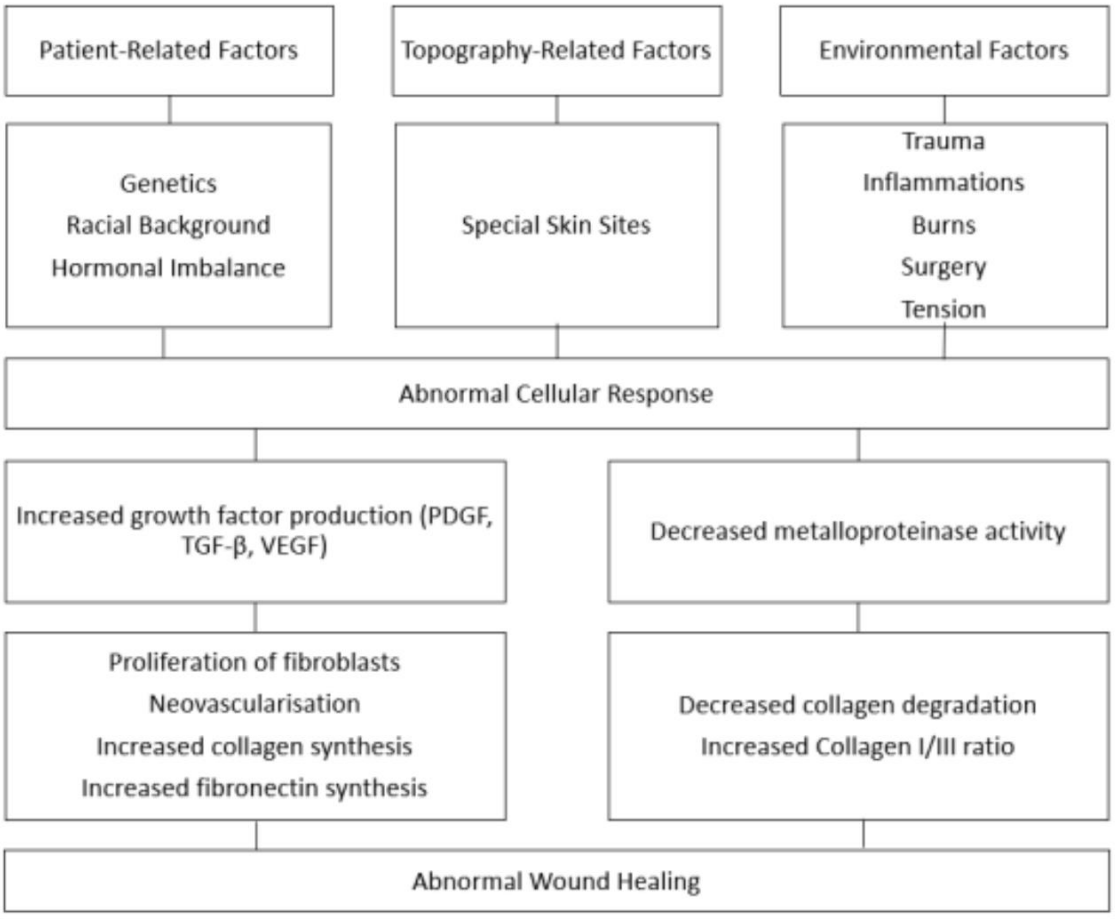


FIGURE 18: PATHOGENESIS OF KELOID

II. HYPERTROPHIC SCAR

An unwanted variation in the wound healing process is hypertrophic scarring. Keloid scars, a different kind of wound healing, are frequently confused with hypertrophic scarring, although this is incorrect.[94]

EPIDEMIOLOGY:

Teenagers and expectant mothers may be more prone to developing hypertrophic scars. The likelihood of developing keloid scars is around 15% higher in those with darker skin tones, whereas albinos have the lowest rate of hypertrophic scarring. According to estimates, hypertrophic scars develop in 70% of profound (full thickness) burn victims.

ETIOLOGY:

Both keloids and hypertrophic scars are connected to a number of risk factors. Tension on the wound is a very significant and a frequent risk factor in clinical practice. The parts of the body that experience the highest skin stress are where hypertrophic scars and keloid scars form. These areas includes the trunk, upper limbs, or the presence of any scar over any bony prominence are examples of these. In contrast, regions with low skin tension, including the top eyelids, seldom form hypertrophic scars. But any scar can become keloid in those who are predisposed to it.

TREATMENT:

It has been demonstrated that laser therapy, such as long-pulsed Nd:YAG laser or pulsed dye laser, is effective as an treatment. These lasers are highly effective in hypertrophic scar as they help in lightening the scars by targeting blood vessels. Scars that are hypertrophic or keloid may be treated with cryotherapy. Although one may anticipate that a traumatic procedure like cryotherapy would leave scars, medical professionals think that cold damage causes a different set of inflammatory mediators to be produced than other forms of injury, including burn injury. Keloids have been effectively treated and avoided using 5-fluorouracil.

III. SEBORRHEIC KERATOSIS

Seborrheic keratosis is a typical epidermal tumor that affects middle-aged and elderly people more frequently but has also been seen in 2nd decade individuals. One of the most frequent forms of skin tumors encountered by general practitioners and dermatologists in an outpatient environment are these lesions.

EPIDEMIOLOGY:

The most prevalent benign skin tumor, seborrheic keratosis, affects nearly 80 million Americans. Patients over 50 are more likely to get seborrheic keratosis, and it becomes more common with age. Seborrheic keratosis lesions can appear in young individuals even though they are more prevalent in middle-aged and elderly people. There is no distinction in prevalence between genders. However, populations with lighter skin tones seem to have a higher incidence of seborrheic keratosis.

PATHOPHYSIOLOGY:

The benign clonal proliferation of epidermal keratinocytes causes seborrheic keratosis. The development of a large number of seborrheic keratosis lesions is thought to have a hereditary component. The precise familial inheritance is unknown, though. The precise pathophysiology of this skin disorder is still unknown, however there may be a connection with the oncogenes PIK3CA and/or fibroblast growth factor receptor 3 (FGFR3). In cases of spontaneous seborrheic keratosis, activating mutations in the tyrosine kinase receptor fibroblast growth factor receptor-3 (FGFR3) are frequent and are thought to be responsible for the development of this benign tumor. Seborrheic keratosis can be classified into a number of subtypes, such as acanthotic, hyperkeratotic, clonal, adenoid, irritated, and melanoacanthoma.

EVALUATION:

A dermatoscopic examination would assist further distinguishing between benign characteristics and dysplastic/malignant tumors. For SK, the dermatoscope often reveals cysts, comedo-like holes, fissures & ridges. The diagnosis and workup of these lesions might be more challenging if there are several seborrheic keratoses present or if there are overlapping lesions. Seborrheic keratosis patients should undergo rigorous screening since there is a higher risk of missing co-existing melanoma or pigmented basal cell cancer.

TREATMENT

These lesions have been treated with alpha-hydroxy acids, urea ointment, imiquimod cream, topical gels and creams used to treat hyperkeratotic skin disorders, and vitamin D analogs (calcipotriol). Overall, topical hydrogen peroxide treatment was well tolerated and successful in removing seborrheic keratosis. Erythema, scaling, and hyperpigmentation were among the relatively moderate adverse effects of therapy. Another non-surgical alternative for people seeking treatment for seborrheic keratosis is laser therapy. Ablative and non-ablative laser therapy are the two types of lasers that are used to treat seborrheic keratosis. Non-ablative lasers (755 nm alexandrite laser) have also been used for this purpose. Ablative laser treatment uses (YAG and CO2 lasers).[95]

IV. MALIGNANT MELANOMA

A melanoma is a tumor that develops when melanocytes undergo malignant transformation. Melanomas can develop in various other organs where the neural crest cells migrate, including the GIT and brain, despite the fact that they often occur on the skin because melanocytes are produced from the neural crest. Excisional biopsy should be performed on suspicious lesions so that a pathologist can make a diagnosis. The only

effective treatment for early-stage melanoma is by surgery, such as extensive local excision with sentinel lymph node biopsy. First, think about the surgical margins before executing the extensive local excision.[96]

V. SYRINGOMA

Syringomas are benign tumors of the sweat duct. The eyelids are where they are most frequently found in groups, although they can also appear elsewhere on the face, in the armpits, above the umbilicus, or in the vulva. Syringomas are benign growths that develop from eccrine ducts' intraepidermal region. On the basis of the presentation, the body's distribution of the tumor, the absence of concomitant symptoms, and family history, syringomas are frequently identified clinically. A skin biopsy is necessary for a certain diagnosis so that the tissue may be viewed under a microscope. Carbon dioxide lasers, dermabrasion, surgical excision, electrocoagulation, and chemical peels are often used destructive therapy modalities. Many of these techniques take a long time and need numerous therapy sessions.[97]

MATERIALS & METHODS

METHODOLOGY

Source of data:

From January 2021 to July 2022, this study was carried out in the Department of Dermatology, Venereology, and Leprosy at R L Jalappa Hospital and Research Center, Tamaka, Kolar, which was affiliated with Sri Devaraj Urs Medical College.

Study Design:

Observational cross-sectional study.

Sample size: Purposive sampling

Sample size calculation

$$\text{Sample size} = \frac{Z_{1-\alpha/2}{}^2 p(1-p)}{d^2}$$

Here

$Z_{1-\alpha/2}$ = Is standard normal variate (at 5% type 1 error ($P<0.05$) it is 1.96 and at 1% type 1 error ($P<0.01$) it is 2.58). As in majority of studies P values are considered significant below 0.05 hence 1.96 is used in formula.

p = Expected proportion in population based on previous studies or pilot studies.

d = Absolute error or precision – Has to be decided by researcher.

Ethical consideration:

Ethical approval was obtained from the Institutional Ethics Committee (IEC) on December, 2020 from Sri Devaraj Urs Medical College, Tamaka, Kolar. All subjects enrolled in the study adhered to ethical guidelines set by the Ethics Committee. All participants

adhering to the inclusion parameters set by the study protocol were included in the study after due Informed and written consent. The researchers maintained strict anonymity of the participants with strict confidentiality of the data collected from the participants. The results obtained from the data were handled with confidentiality and the researchers will discard the entire data gathered after the publication of the thesis.

Method of collection of data (including sampling procedure)

Based on the most frequent Cutaneous symptoms in the periorificial area in the age range of 10 to 19 years, the sample size for the current study was calculated, with an alpha error of 5% and a 95% confidence interval taking an absolute error of 10% into account. 254 was the predicted sample size. The percentage of cutaneous infections and infestations in the periorificial area among teenage females aged 10 to 19 who visited the dermatology OPD was used to determine the sample size.

Statistical analysis:

All the data was entered initially into Microsoft Excel 2020 and then these spreadsheets were used for analysis and statistical analysis was done using SPSS version 20.0. Descriptive statistics were calculated as frequency, percentage, mean and standard deviation, depicted using various tables, graphs, diagrams, etc. Statistically significant was concluded by Online calculators. The significance test utilized was the chi-square. Statistics are deemed significant at p 0.05.

Inclusion criteria:

The study covered all teenage females in the age range of 10 to 19 who presented with cutaneous symptoms across the periorificial region.

Exclusion criteria:

- Patients with congenital disorders who may have skin manifestations at birth or appear thereafter and which continue to persist in the adolescent period.
- Patients and/or their guardians not giving consent for the participation in the study, patient with drug reactions and seriously ill patients.

Methods of data collection:

- Adolescent girls between the ages of 10 and 19 who visit the Department of Dermatology, Venereology, and Leprosy at the R L Jalappa Hospital and Research Centre, Tamaka, Kolar, affiliated with Sri Devaraj Urs Medical College, were evaluated based on a thorough clinical history, including the onset and evolution of the lesion, socioeconomic factors (Modified BG Prasad Scale), and stress morbidity (Perceived Stress Scale).
- A thorough medical history was gathered, including the patient's name, age, sex, the kind and length of their ailment, and any risk factors such drug use, topical application of medications, cosmetics use, and so on.
- Based on the history and clinical examination, a diagnosis was determined.
- When necessary, tests such the KOH mount, Tzanck smear, Gram's stain, Wood's lamp inspection, and biopsy were performed.
- The individual lead investigator was responsible for covering the cost of the necessary blood tests.
- The information so gathered was placed into a case record form that was especially created, and it was then submitted to statistical analysis such as percentage and the Chi-square test.

RESULTS

OBSERVATION AND RESULTS

1. PERIORIFICIAL DERMATOSES

We conducted this study to shed light on the different aesthetic issues that teenagers confront as a result of physiological and social causes. These varied face blemishes can have a greater psychological and physiological impact on this age group than a dermatologist would predict. During the study's duration, 254 female patients were enrolled. Over half of all teenagers are between the ages of 18 and 19. The clinical and epidemiological characteristics of cutaneous manifestations across the periorificial regions in teenage females were established in the following study.

SOCIODEMOGRAPHIC DISTRIBUTION

Age, seasonal variations were recorded as sociodemographic profile in the present study.

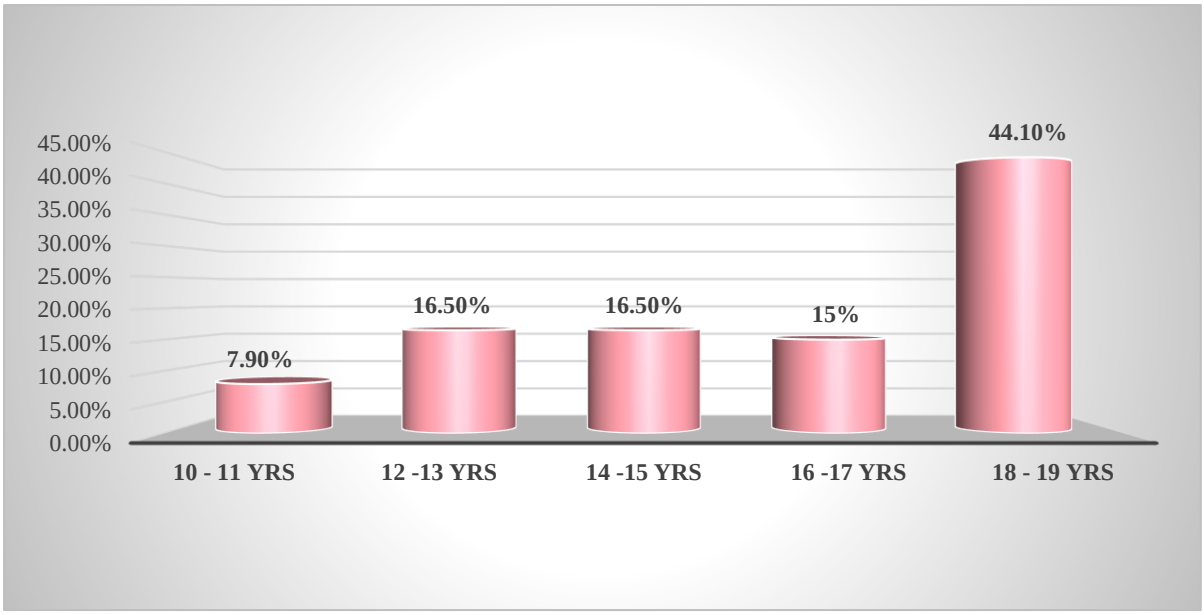
1. AGE DISTRIBUTION

The maximum number of patients were seen in 18-19 years (44.1%) of age group which is followed by 12-13 years (16.5%) & 14-15 years (16.5%).

TABLE:8 AGE WISE DISTRIBUTION OF PERIORIFICIAL DERMATOSES (n=254)

AGE GROUP (IN YEARS)	TOTAL	PERCENTAGE (%)
10-11	20	7.9 %
12-13	42	16.5 %
14-15	42	16.5 %
16-17	38	15.0 %
18-19	112	44.1 %

GRAPH: 1 AGE WISE DISTRIBUTION OF PERIORIFICIAL DERMATOSES (n=254)



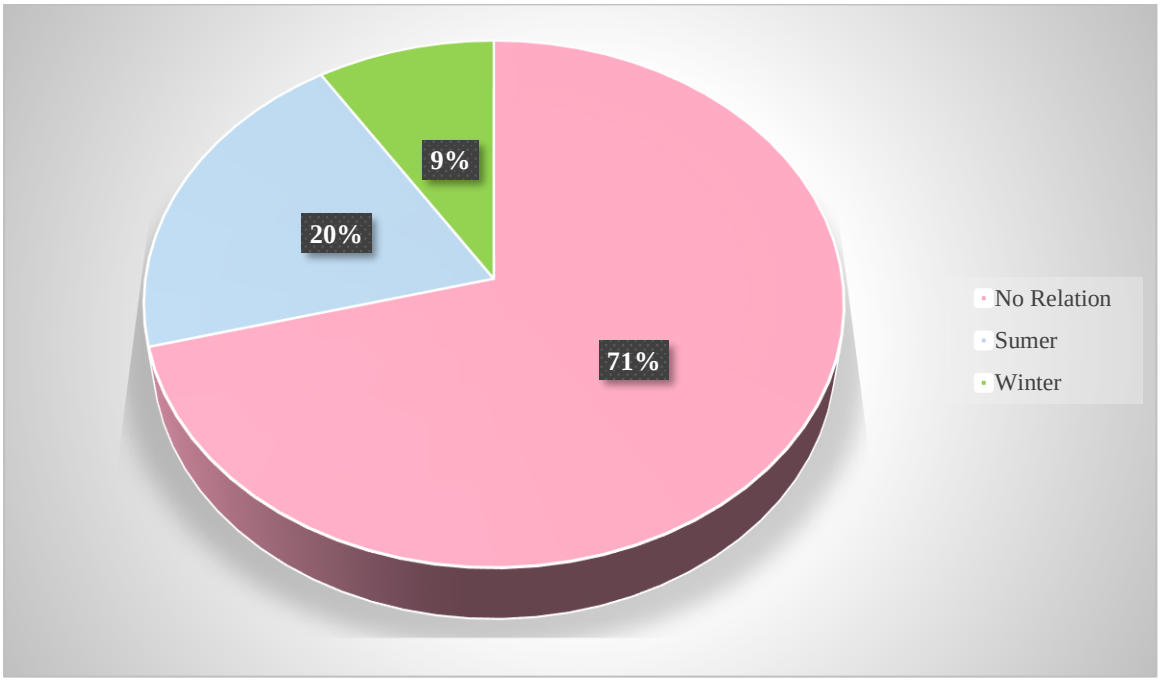
2. SEASONAL VARIATIONS

180 patients (70.9 %) has no seasonal variation. Summer exacerbation was observed in 20.1% and winter exacerbation observed in 9%.

TABLE: 9 SEASONAL VARIATIONS OF PERIORIFICIAL DERMATOSES (n=254)

SEASONAL VARIATION	TOTAL	PERCENTAGE
No relation	180	70.9 %
Summer	51	20.1 %
Winter	23	9.0 %

GRAPH: 2 SEASONAL VARIATIONS OF PERIORIFICIAL DERMATOSES (n=254)



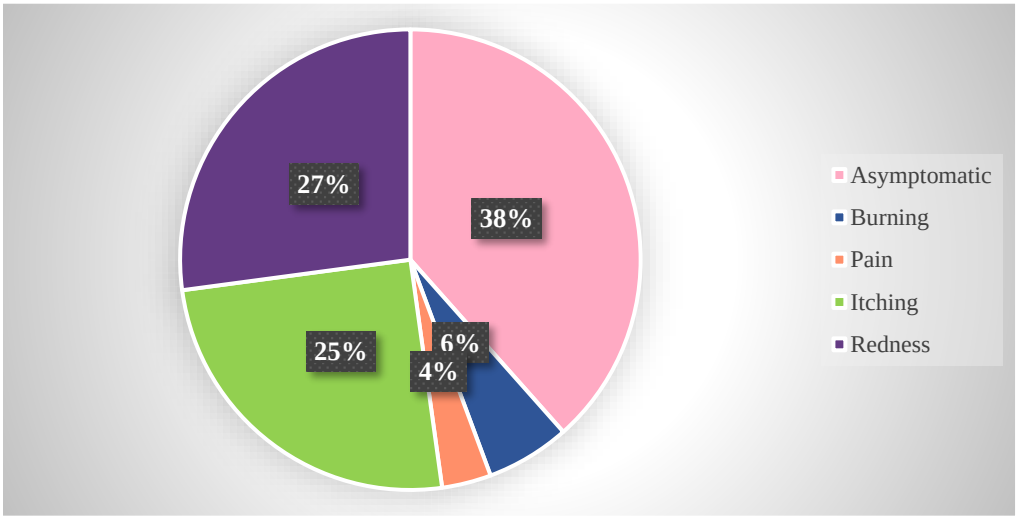
3. SYMPTOMS IN PERIORIFICIAL DERMATOSES

Most of the patients of about 144 (56.7%) were asymptomatic followed by itching in 70 (27.5%).

TABLE: 10 SYMPTOMATIC DISTRIBUTION OF PERIORIFICIAL DERMATOSES (n=254)

SYMPTOMS	TOTAL	PERCENTAGE
ASYMPTOMATIC	144	56.7 %
BURNING	22	8.7 %
PAIN	13	5.1 %
ITCHING	94	37.0 %
REDNESS	7	2.7 %

GRAPH: 3 SYMPTOMATIC DISTRIBUTION OF PERIORIFICIAL DERMATOSES



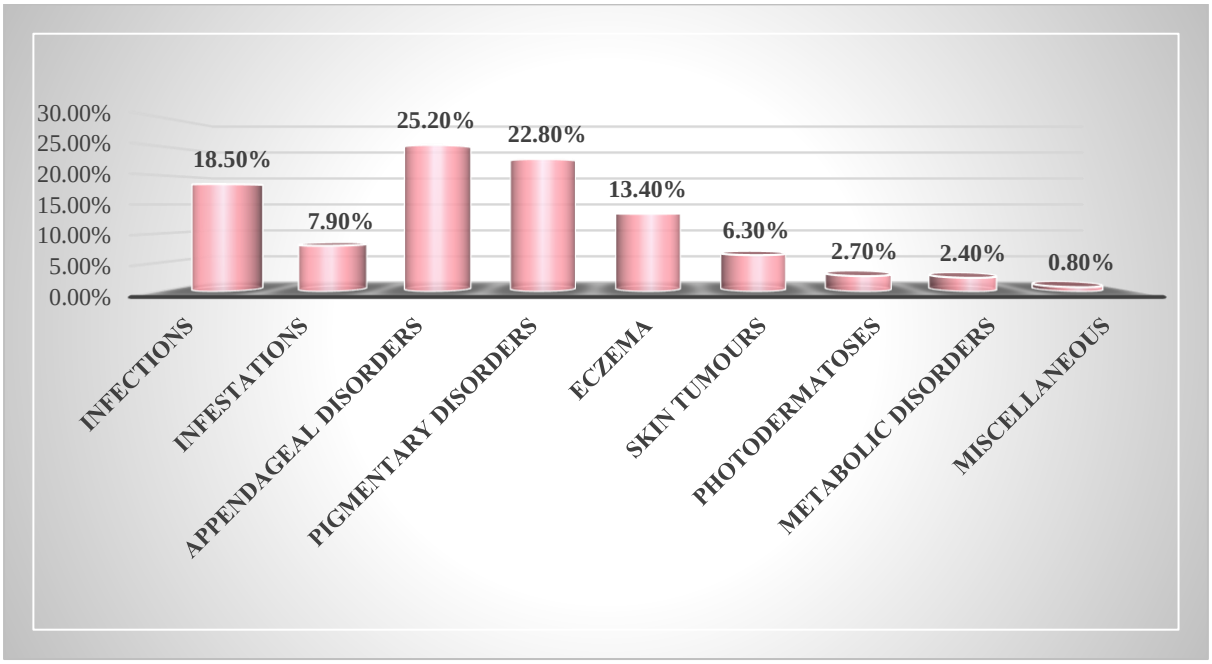
4. INCIDENCE OF VARIOUS PERIORIFICIAL DERMATOSES

Disorders of appendages were the most common seen in 25.2%, followed by pigmentary disorders in 22.8%, infections seen in 18.5%.

TABLE: 11 INCIDENCE OF PERIORIFICIAL DERMATOSES (n=254)

DERMATOSES	TOTAL	PERCENTAGE
Infections	47	18.5 %
Infestations	20	7.9 %
Pigmentary disorders	58	22.8 %
Appendageal disorders	64	25.2 %
Eczema	34	13.4 %
Photodermatoses	7	2.7 %
Skin tumours	16	6.3 %
Metabolic disorder	6	2.4 %
Miscellaneous	2	0.8 %

GRAPH: 4 INCIDENCE OF PERIORIFICIAL DERMATOSES (n=254)



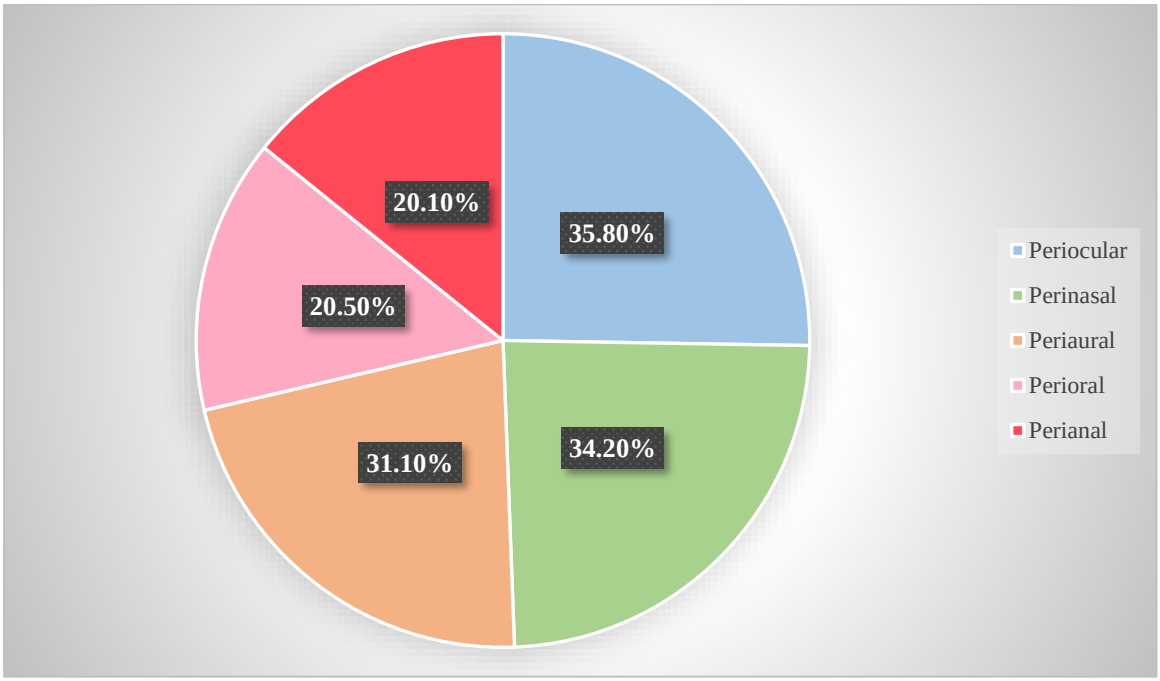
4. DISTRIBUTION OF PERIORIFICIAL DERMATOSES

The most common site to be involved was periocular area accounting for 35.8%, followed by Perinasal and Periaural area seen in 34.2% and 31.1% respectively.

TABLE: 12 DISTRIBUTION OF PERIORIFICIAL DERMATOSES (n=254)

SITES	PERCENTAGE
Periocular	35.8 %
Perinasal	34.2 %
Perioral	20.5 %
Periaural	31.1 %
Perianal	20.0 %

GRAPH: 5 DISTRIBUTION OF PERIORIFICIAL DERMATOSES (n=254)



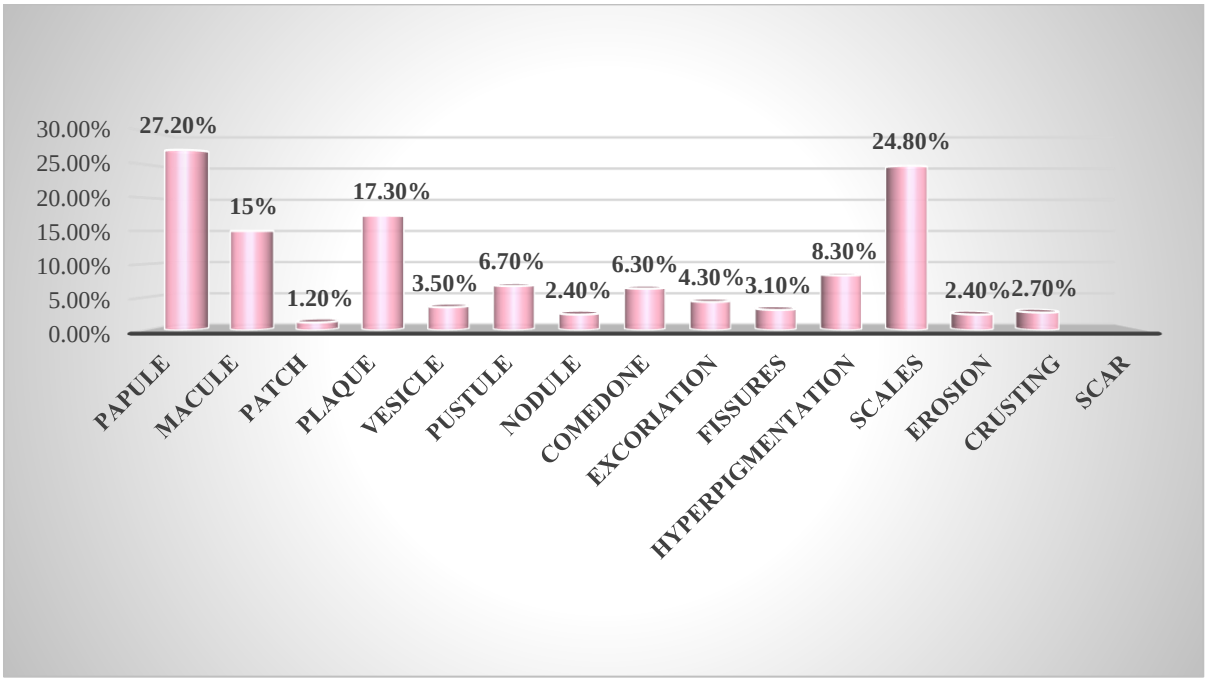
5. LESION MORPHOLOGY

The predominant lesion from the total study case was papules seen in 27.2%, followed by scales seen in 24.8%.

TABLE: 13 DISTRIBUTION OF LESION MORPHOLOGY OF PERIORIFICIAL DERMATOSES (n=254)

LESION	PERCENTAGE
Macule	15 %
Papule	27.2 %
Patch	1.2 %
Plaque	17.3 %
Vesicle	3.5 %
Pustule	6.7 %
Nodule	2.4 %
Comedone	6.3 %
Excoriation	4.3 %
Fissures	3.1 %
Hyperpigmentation	8.3 %
Scales	24.8 %
Erosion	2.4 %
Crusting	2.7 %
Scar	5.9 %

GRAPH: 6 DISTRIBUTION OF LESION MORPHOLOGY (n=254)



6. STRESS MORBIDITY

Severity of stress among the 254 periorificial dermatoses were separated into mild moderate and severe in the present study. Maximum number of cases was seen with moderate severity (63%). Mild severity cases seen in 30%. Remaining 7% of patients has severe stress.

TABLE: 14 STRESS MORBIDITY OF PERIORIFICIAL DERMATOSES (n=254)

SEVERITY	NO. OF PATIENTS	PERCENTAGE (%)
MILD	77	30.3%
MODERATE	159	62.6%
SEVERE	18	7.1%
TOTAL	254	100%

GRAPH: 7 STRESS MORBIDITY OF PERIORIFICIAL DERMATOSES (n=254)

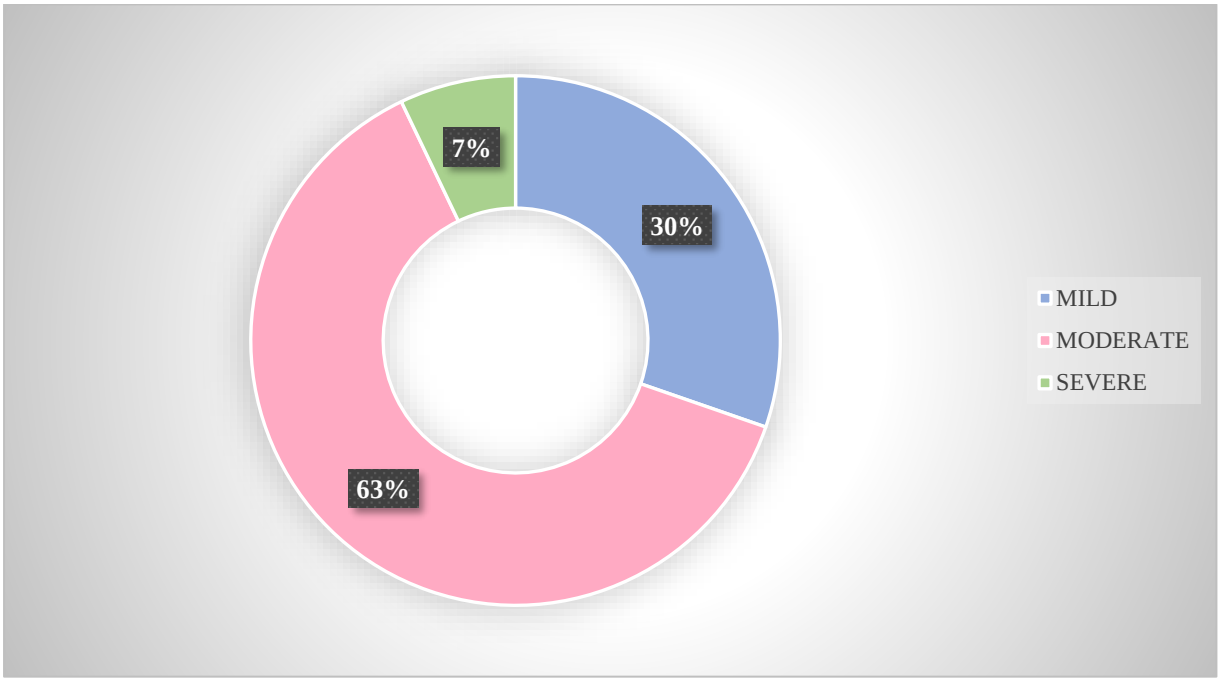
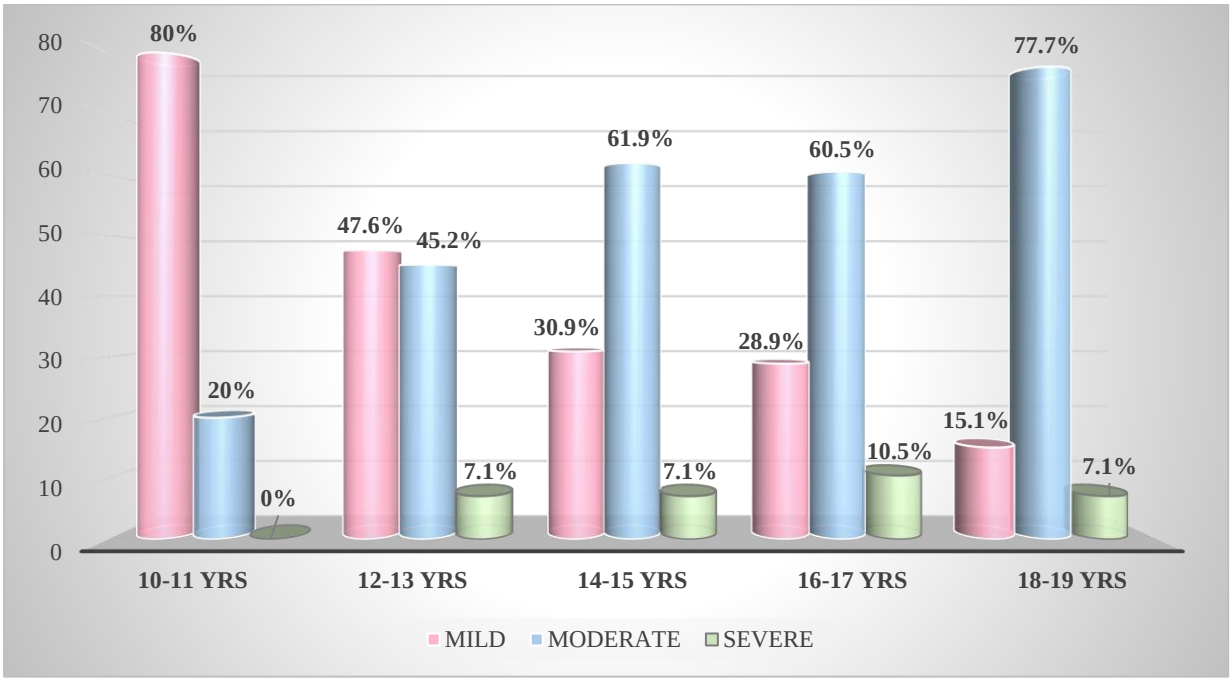


TABLE: 15 AGE WISE DISTRIBUTION OF STRESS MORBIDITY

The age distribution among the 254 cases of varying severity is depicted in the table and figure below. The age range of 12 to 13 years had the greatest number of individuals with severe stress.

AGE GROUP (IN YEARS)	SEVERITY		
	MILD	MODERATE	SEVERE
10-11	16	4	0
12-13	20	19	3
14-15	13	26	3
16-17	11	23	4
18-19	17	87	8
TOTAL	77 (30.3%)	159 (62.6%)	18 (7.1%)

GRAPH: 8 AGE WISE DISTRIBUTION AMONG STRESS MORBIDITY



1. INFECTIONS

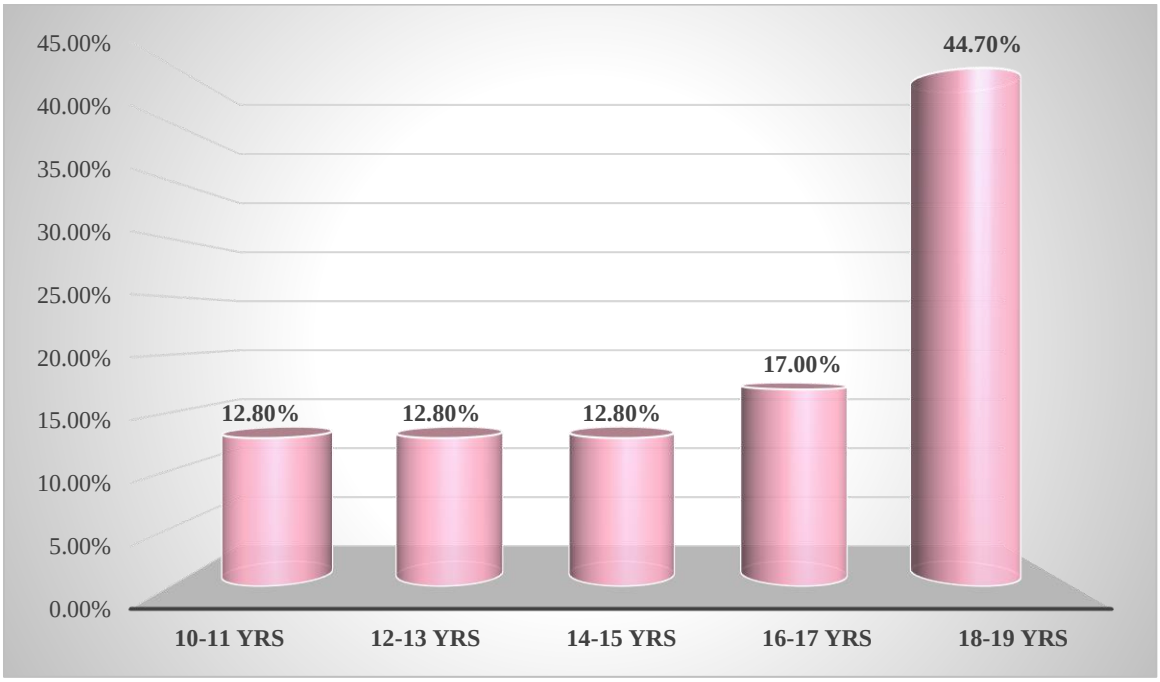
AGE-WISE DISTRIBUTION OF INFECTIONS :

Out of 254 patients, 18.5 % were affected by infections. Majority of the patients belonged to the age group 18-19 years seen in 21 patients (44.7%), which was followed by 16-17 years seen in 8 patients (17%).

TABLE: 16 AGE WISE DISTRIBUTION OF INFECTIONS IN PERIORIFICIAL DERMATOSES (n=47)

AGE GROUP (IN YEARS)	TOTAL	PERCENTAGE (%)
10-11	6	12.8 %
12-13	6	12.8 %
14-15	6	12.8 %
16-17	8	17.0 %
18-19	21	44.7 %

GRAPH: 9 AGE WISE DISTRIBUTION OF INFECTIONS IN PERIORIFICIAL DERMATOSES (n=47)



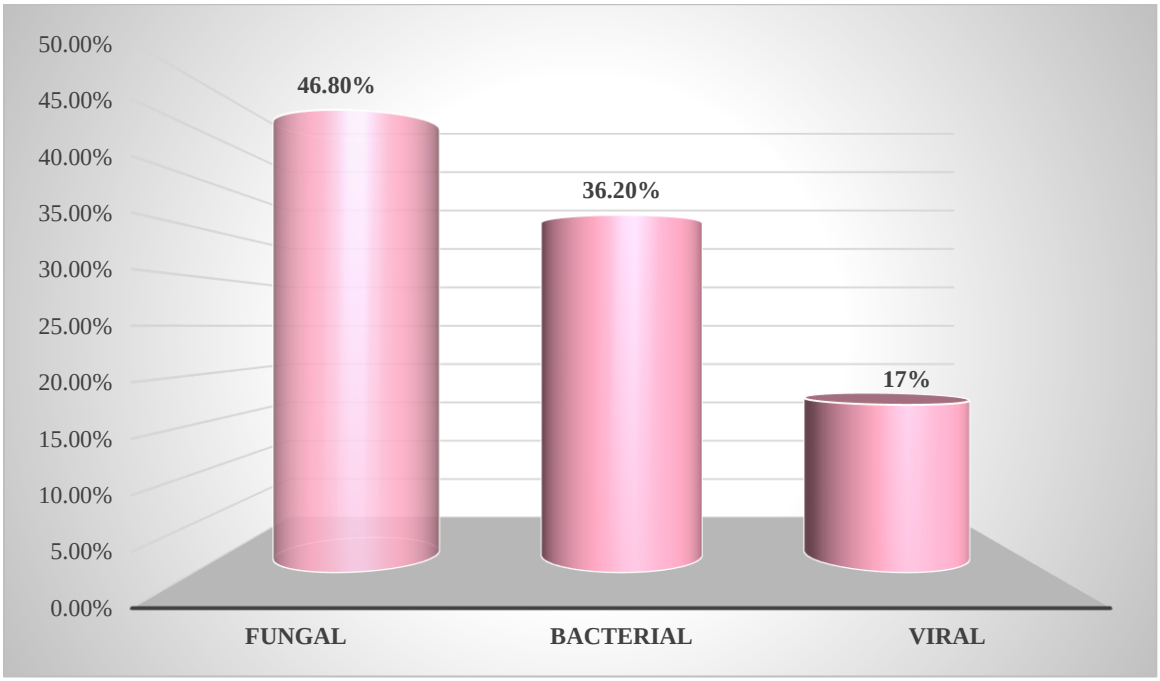
INCIDENCE WISE DISTRIBUTION (n= 47)

Among the 47 patients, most common infections were fungal infections seen in 46.8%, followed by bacterial infections and viral infections seen in 36.2% & 17% respectively. Duration of symptoms ranges from 2 days to 5 months.

TABLE: 17 INCIDENCE WISE DISTRIBUTION OF PERIORIFICIAL DERMATOSES (n=47)

INFECTIONS	TOTAL	PERCENTAGE
BACTERIAL	17	36.2 %
VIRAL	8	17.0 %
FUNGAL	22	46.8 %

GRAPH: 10 INCIDENCE WISE DISTRIBUTION OF PERIORIFICIAL DERMATOSES (n=47)

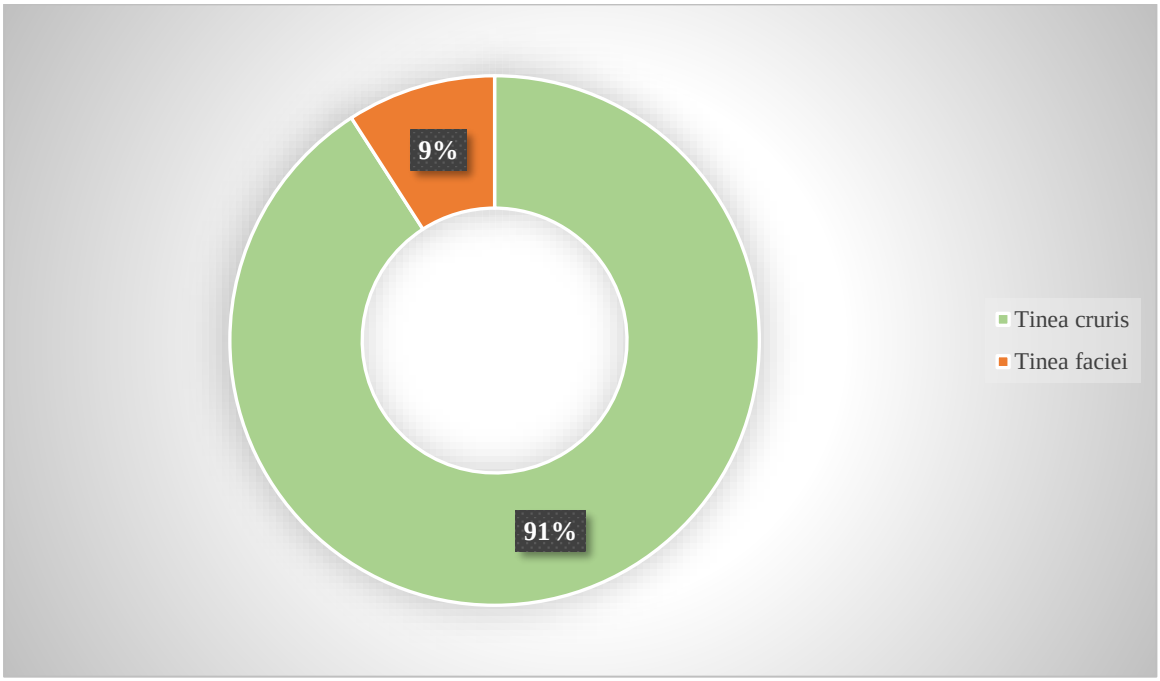


FUNGAL INFECTIONS:

Tinea cruris was the most common fungal illness, affecting 20 individuals (90.9%), whereas tinea faciei affected just two (9.1%). Itching was the most prevalent symptom noticed in these individuals. The most typical location was in the Perianal region.

Both individuals with tinea faciei and six of the twenty patients with tinea cruris had additional body region involvement. Summer aggravation affected 50% of the patients. Tinea was found in 27.3% of patients' families. Four patients (18.2%) had topical steroid application.

GRAPH: 11 DISTRIBUTION OF FUNGAL INFECTIONS (n=22)



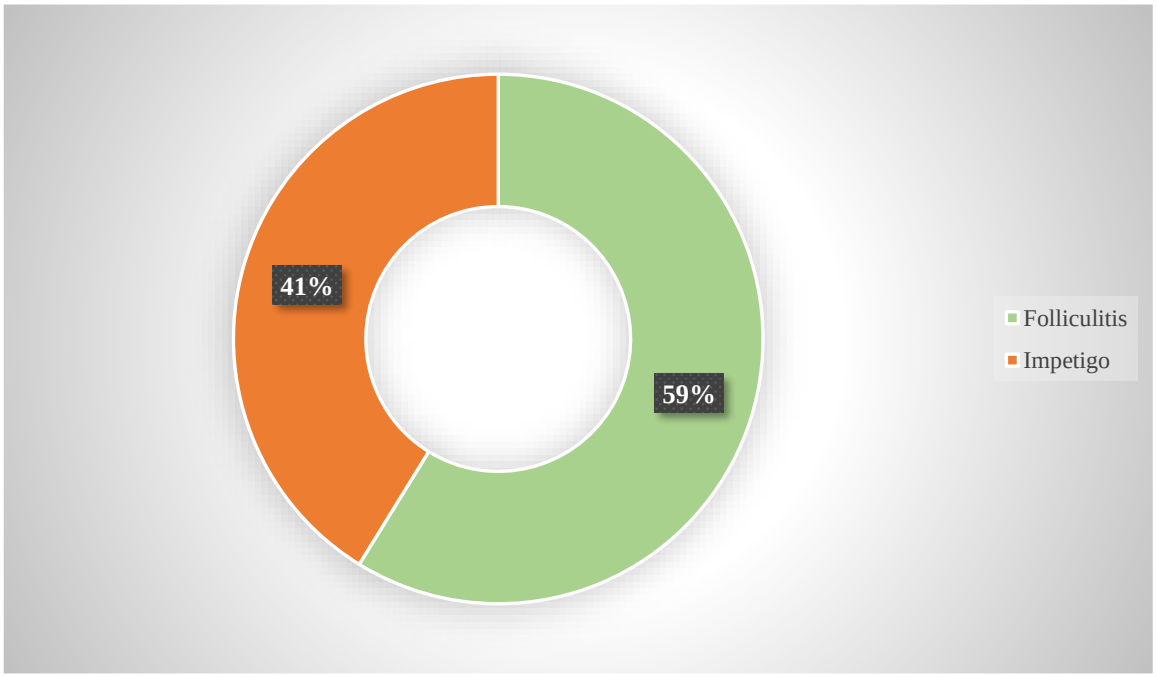
BACTERIAL INFECTIONS:

Folliculitis is observed in 58.8% of individuals with bacterial infections, followed by impetigo in 41.2%. The most usually impacted age group in folliculitis was 16-19 years of

age, whereas the affected age group in impetigo was 10-11 years of age. Itching was the most common symptom.

In folliculitis, the perioral area was the most common location for impetigo, followed by the perianal area. Summer aggravation affected two of the ten folliculitis patients. Two of the seven impetigo patients had a favorable family history.

GRAPH: 12 DISTRIBUTION OF BACTERIAL INFECTIONS (n=17)



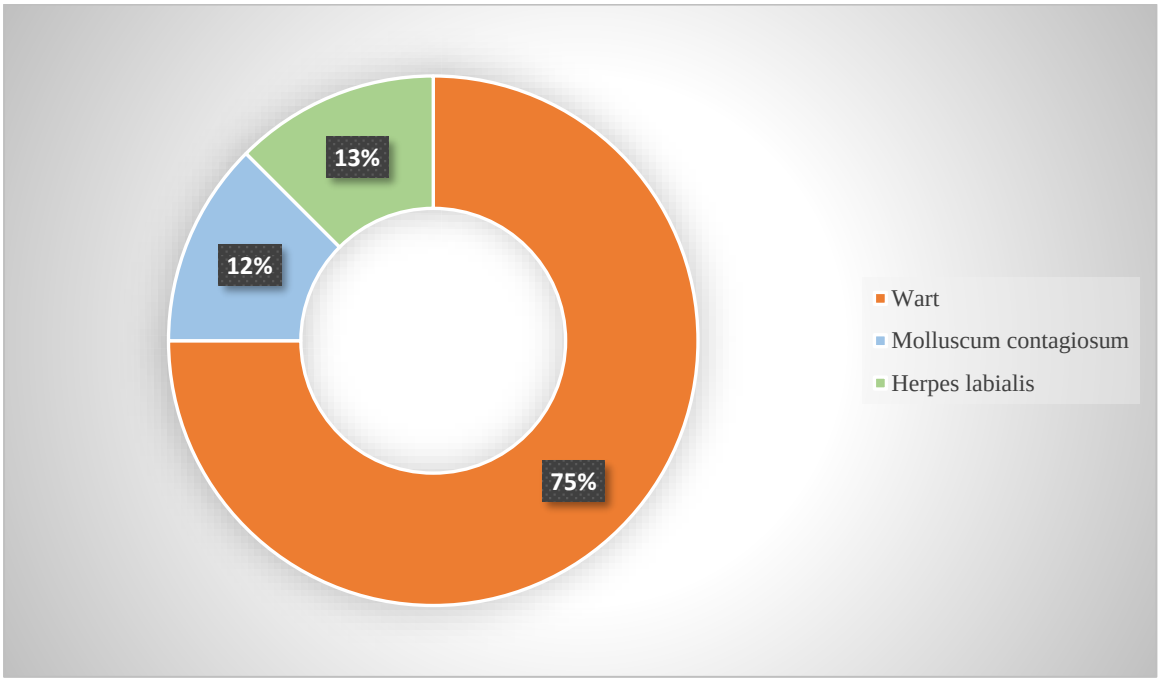
VIRAL INFECTIONS:

In this investigation, warts were the most frequent viral infection (75%), followed by molluscum contagiosum (12.5%) and herpes labialis (12.5%). The majority of them were between the ages of 18 and 19.

The most common place identified in warts is the periocular (33.3%) and perinasal (33.3%) area, whereas periocular and perioral areas were observed in molluscum

contagiosum and herpes labialis, respectively. Out of the six individuals with warts, one had a family history.

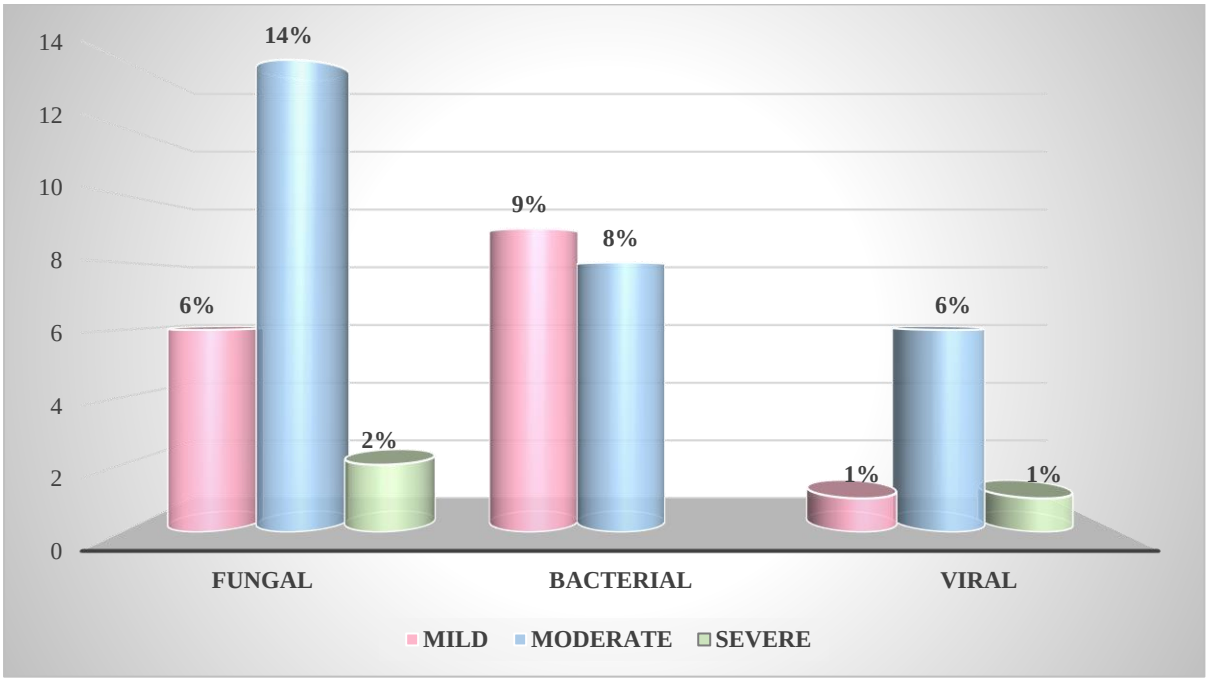
GRAPH: 13 DISTRIBUTION OF VIRAL INFECTIONS (n=8)



STRESS MORBIDITY IN INFECTIONS

The image below depicts several bacterial, fungal, and viral infections in 47 instances of mild, moderate, and severe severity. Among all infections, the number of moderate cases was higher.

GRAPH: 14 DISTRIBUTION OF STRESS MORBIDITY IN INFECTIONS (n=47)



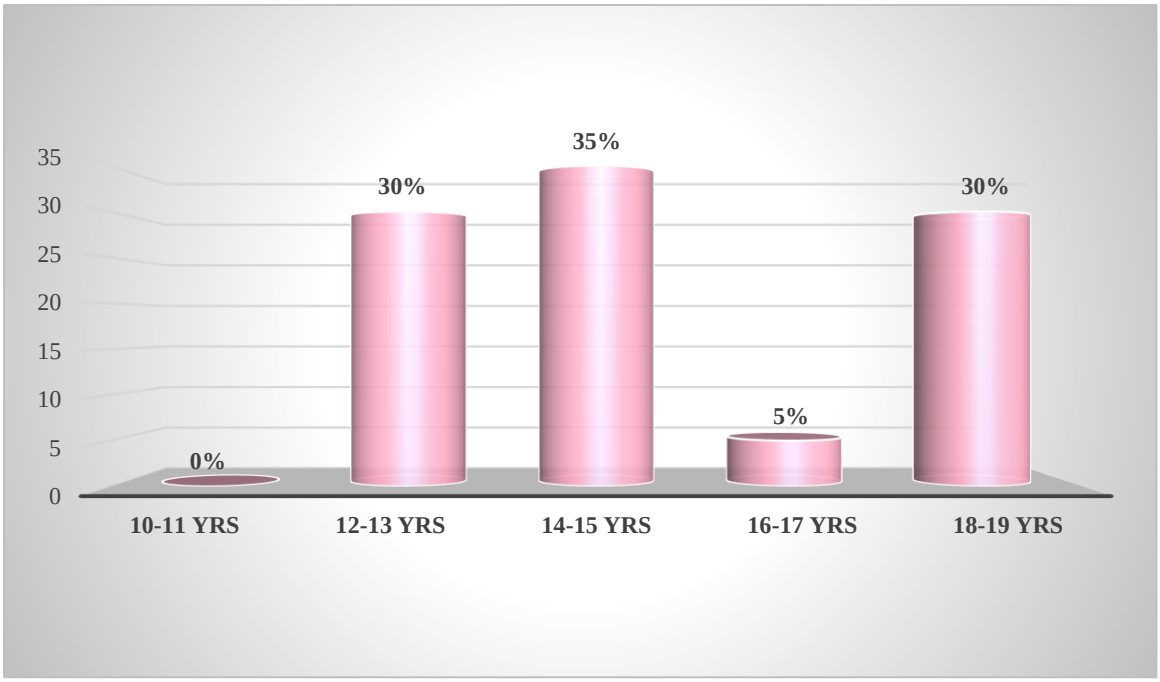
2. INFESTATIONS

In the current study, approximately 35% of the infestations found in 14 - 15 years of total 20 cases of infestations. In 16 to 17 years, over 5% were seen.

TABLE: 18 AGE WISE DISTRIBUTION OF INFESTATIONS IN PERIORIFICIAL DERMATOSES (n=20)

AGE GROUP	TOTAL	PERCENTAGE
10-11	0	0%
12-13	6	30%
14-15	7	35%
16-17	1	5%
18-19	6	30%

GRAPH: 15 AGE WISE DISTRIBUTION OF INFESTATIONS IN PERIORIFICIAL DERMATOSES (n=20)



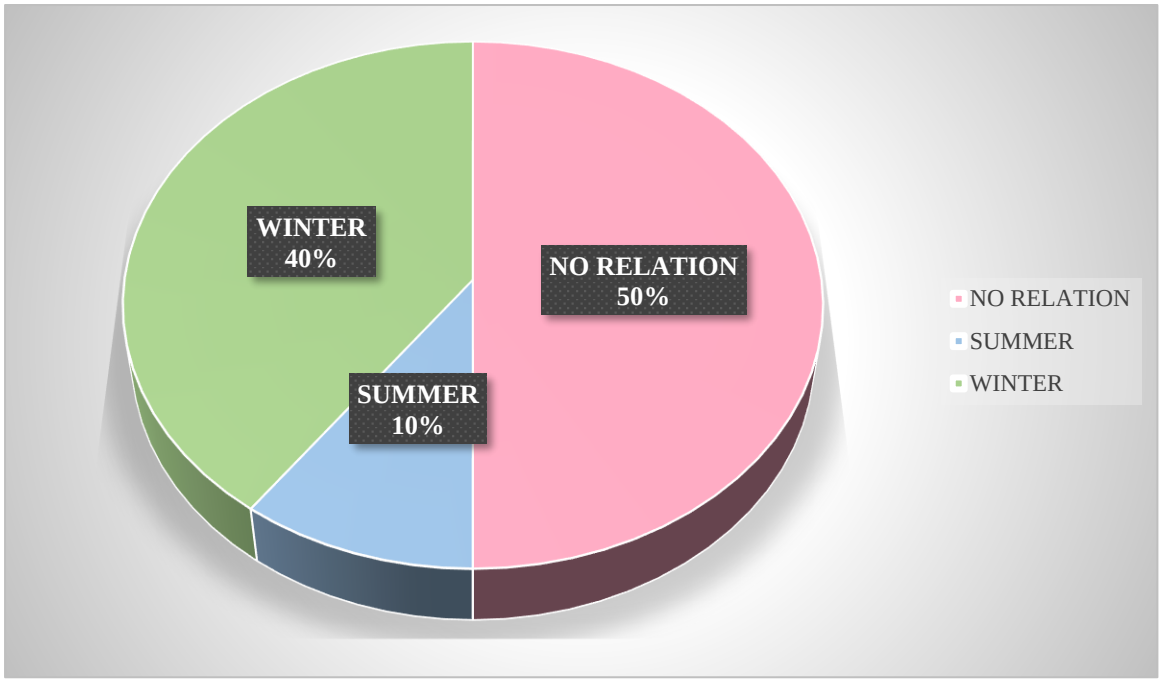
SEASONAL VARIATION IN SCABIES

Scabies are more prevalent in the winter season (40%). About half of the cases had no seasonal relationship with scabies fluctuation.

TABLE: 19 SEASONAL VARIATIONS IN SCABIES OF PERIORIFICIAL DERMATOSES (n=20)

SEASONAL VARIATION	TOTAL	PERCENTAGE
NO RELATION	10	50%
SUMMER	2	10%
WINTER	8	40%

GRAPH: 16 SEASONAL VARIATION IN SCABIES OF PERIORIFICIAL DERMATOSES (n=20)



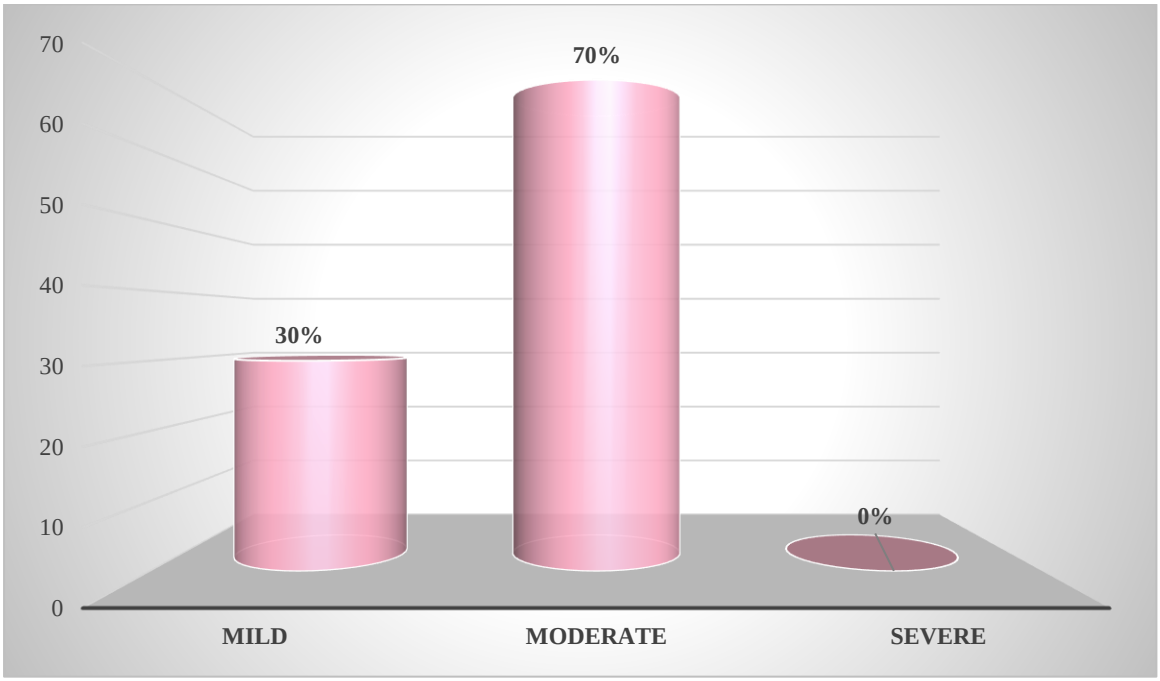
STRESS MORBIDITY IN SCABIES

The image below depicts scabies among mild, moderate, and severe stress morbidity in 20 instances. Among all infestations, the number of moderate cases was higher.

TABLE: 20 STRESS MORBIDITY AMONG SCABIES (n=20)

SEVERITY	TOTAL	PERCENTAGE
MILD	6	30%
MODERATE	14	70%
SEVERE	0	0%

GRAPH: 17 STRESS MORBIDITY AMONG SCABIES (n=20)



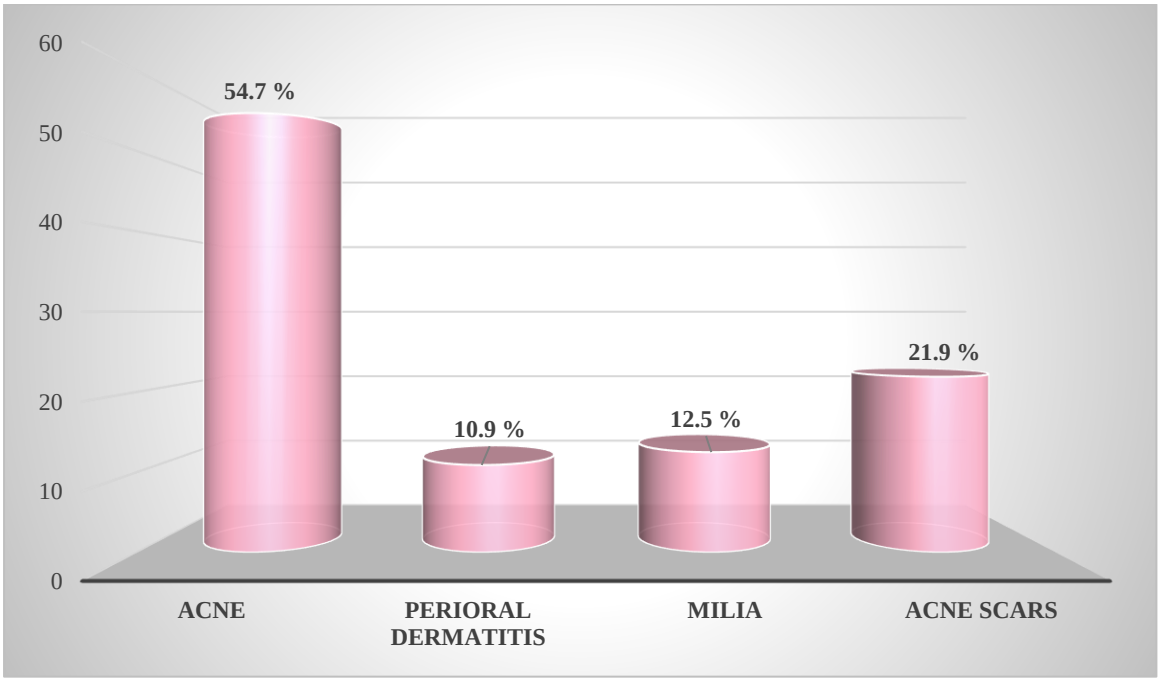
3. APPENDAGEAL DISORDERS

Distribution of appendageal disorders is seen among 64 cases. In this 55% were seen with acne. 22% seen with acne scars.

TABLE: 21 AGE WISE DISTRIBUTION OF APPENDAGEAL DISORDERS (n=64)

CONDITIONS	TOTAL	PERCENTAGE
ACNE	35	54.7%
PERIORAL DERMATITIS	7	10.9%
MILIA	8	12.5%
ACNE SCARS	14	21.9%

GRAPH: 18 AGE WISE DISTRIBUTION OF APPENDAGEAL DISORDERS (n=64)



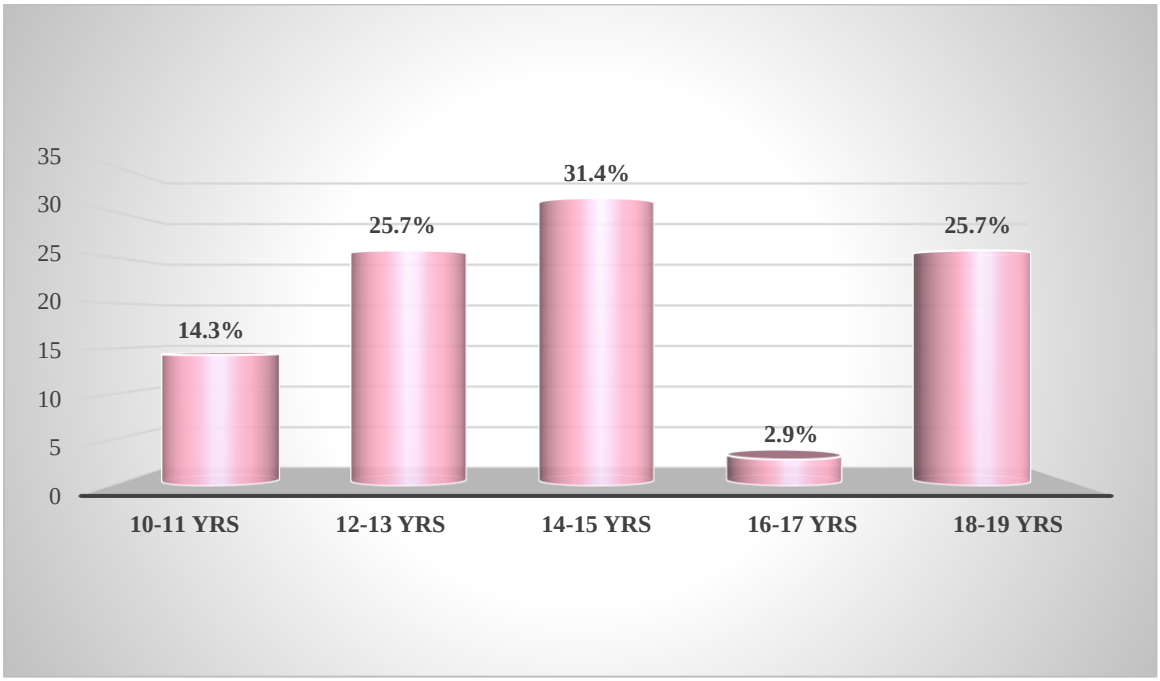
AGE GROUP DISTRIBUTION OF ACNE

In the current study, acne was seen maximum (31%) in 14 - 15 years of age and minimum (3%) in 16 – 17 years of age.

TABLE: 22 AGE WISE DISTRIBUTION OF ACNE (n=35)

AGE GROUP (IN YEARS)	TOTAL NO OF PATIENTS	PERCENTAGE (%)
10-11	5	14.3%
12-13	9	25.7%
14-15	11	31.4%
16-17	1	2.9%
18-19	9	25.7%

GRAPH: 19 AGE WISE DISTRIBUTION OF ACNE (n=35)



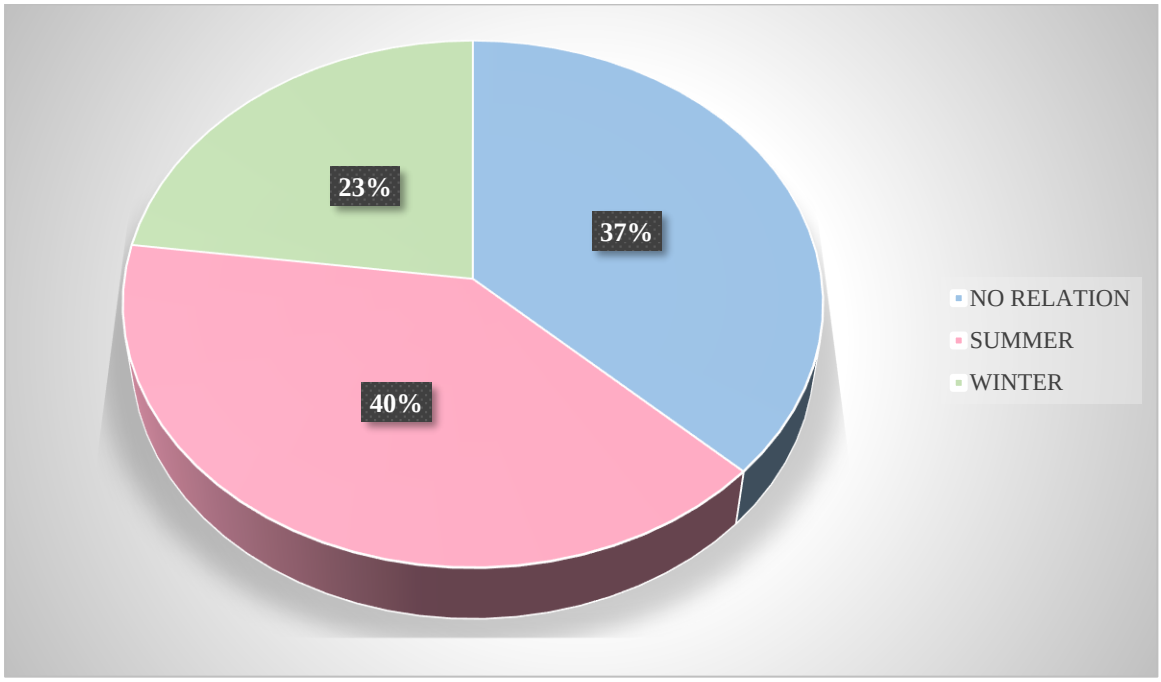
SEASONAL VARIATION

Acne are more prevalent in the summer season (40%). About 37% of the cases had no seasonal variation.

TABLE: 23 SEASONAL VARIATION IN DISTRIBUTION OF ACNE (n=35)

SEASONAL VARIATION	TOTAL NO. OF PATIENTS	PERCENTAGE (IN YEARS)
NO RELATION	13	37.1%
SUMMER	14	40.0%
WINTER	8	22.9%

GRAPH: 20 SEASONAL VARIATION IN DISTRIBUTION OF ACNE (n=35)



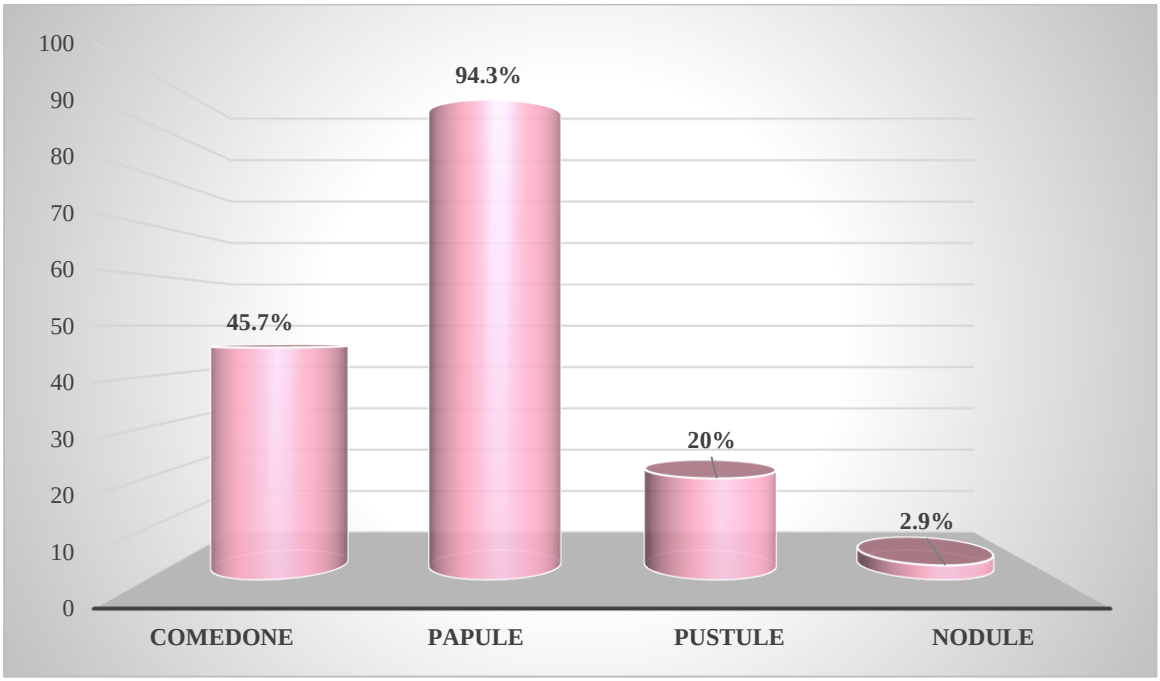
LESION MORPHOLOGY

Distribution of lesion morphology seen among 35 cases of acne. In this 94% were seen with papule lesions. 46% seen with comedone.

TABLE: 24 LESION MORPHOLOGY AMONG ACNE CASES (n=35)

LESION	NO. OF CASES	PERCENTAGE
COMEDONE	16	45.7%
PAPULE	33	94.3%
PUSTULE	7	20%
NODULE	1	2.9%

GRAPH: 21 LESION MORPHOLOGY AMONG ACNE CASES (n=35)



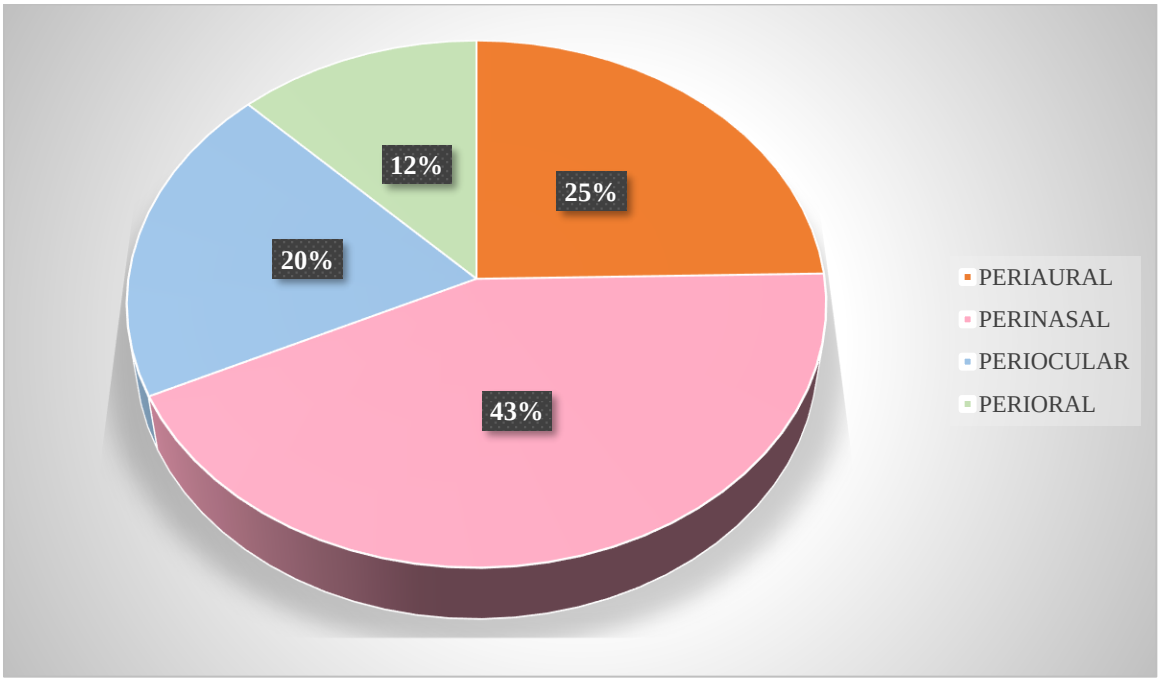
SITES OF DISTRIBUTION

About 100% of acne were present in perinasal region of face. About 57% of acne seen in periaural region.

TABLE: 25 SITES OF DISTRIBUTION AMONG ACNE CASES (n=35)

SITES	NUMBER OF CASES	PERCENTAGE
PERIAURAL	20	57.1%
PERINASAL	35	100%
PERIOCULAR	16	45.7%
PERIORAL	10	28.6%

GRAPH: 22 SITES OF DISTRIBUTION AMONG ACNE CASES (n=35)



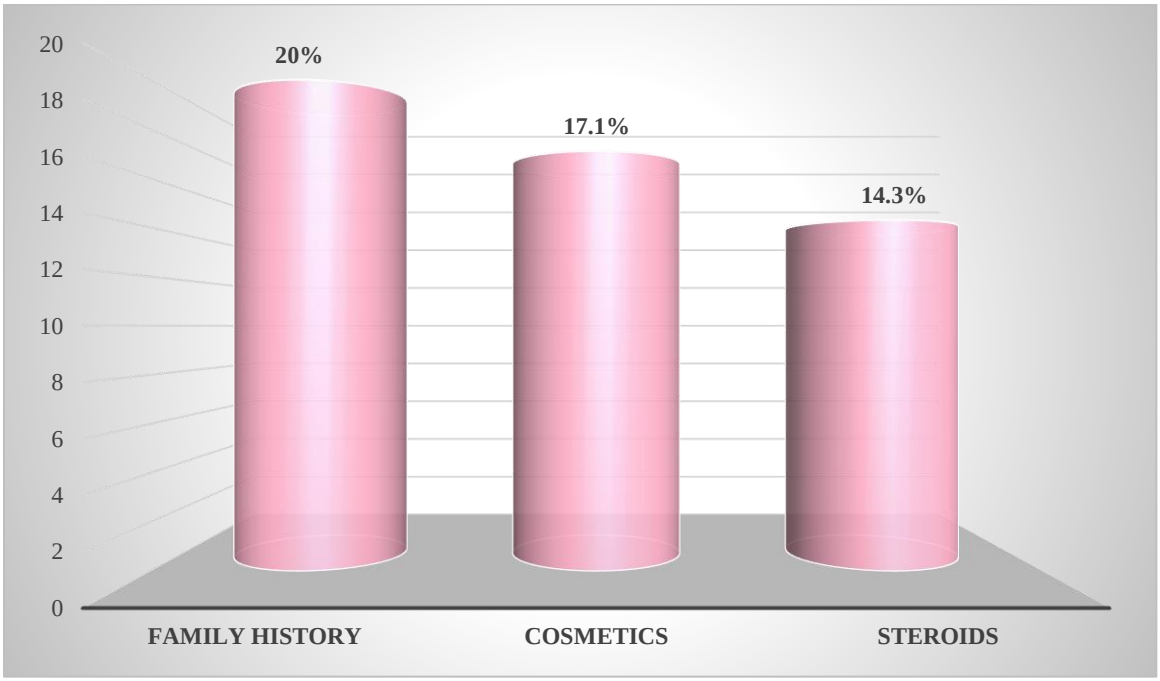
AGGRAVATING FACTORS

Family history, cosmetics, and steroids were all the factors that aggravated acne. Family history is responsible for over 20% of acne cases. Cosmetics are responsible for around 17% of acne cases.

TABLE: 26 AGGRAVATING FACTORS AMONG ACNE CASES (n=35)

FACTORS	TOTAL NO OF CASES	PERCENTAGE (%)
FAMILY HISTORY	7	20%
COSMETICS	6	17.1%
STEROIDS	5	14.3%

GRAPH: 23 AGGRAVATING FACTORS AMONG ACNE CASES (n=35)



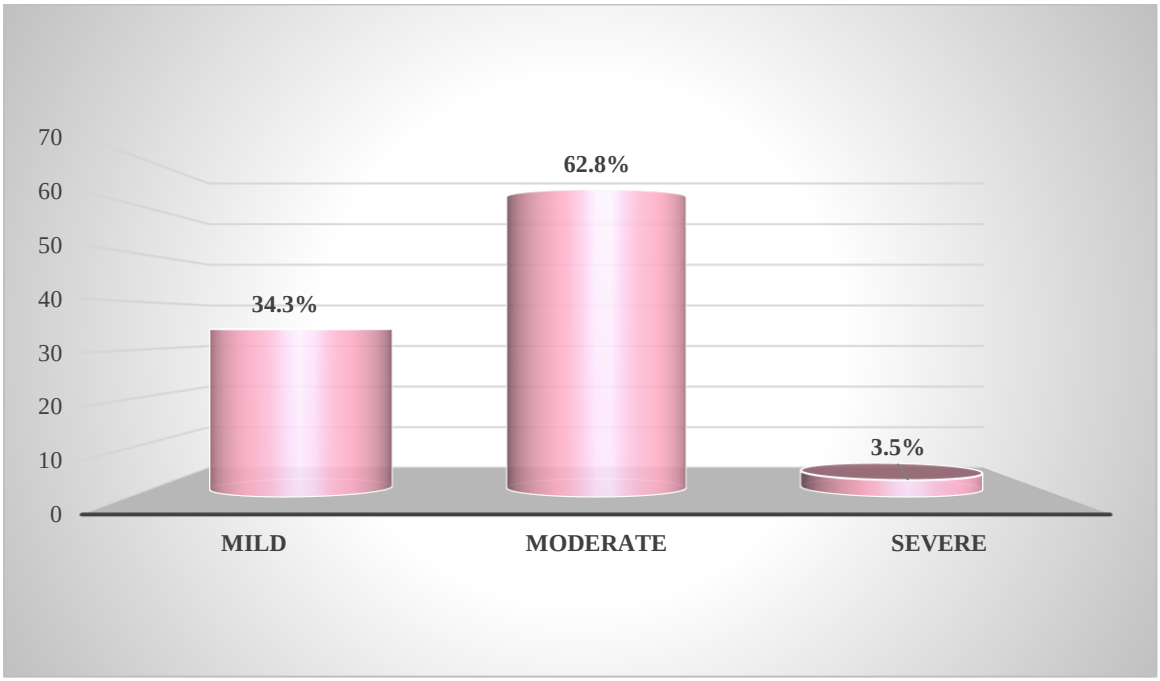
STRESS MORBIDITY IN ACNE

The image below depicts acne distribution among mild, moderate, and severe stress morbidity in 35 instances. Among all stress morbidity, the number of moderate cases was higher which accounts for 62.8%

TABLE: 27 STRESS MORBIDITY IN ACNE (n=35)

SEVERITY	TOTAL	PERCENTAGE
MILD	12	34.3%
MODERATE	22	62.8%
SEVERE	1	2.9%

GRAPH: 24 STRESS MORBIDITY AMONG ACNE CASES (n=35)



ACNE SCARS

Maximum number of subjects 50% belonged to the age group 18-19 years followed by 16-17 years which is 42.9%. Duration of the scars ranged from 2 months to 2 years. Perinasal area was involved in all the cases (100%) whereas periaural and periocular area was seen in 71.4% and 50% respectively. 64.3% of the patients has moderate stress and 35.7% of them has severe stress. Topical steroid application was seen in only 2 cases (14.3%).

MILIA

Majority 87.5% of the subjects were in the age group 18-19 years. 75% of them showed moderate stress whereas 25% of them showed mild stress. Periocular area was involved in all (100%) of the subjects.

PERIORAL DERMATITIS

Majority (42.8%) of them belonged to 10-11 years of age. 71.4% of the subjects had no seasonal variation. Most common symptom was itching (100%). 71.4% of the subjects had moderate stress followed by 28.6% had mild stress.

4. PIGMENTARY DISORDER

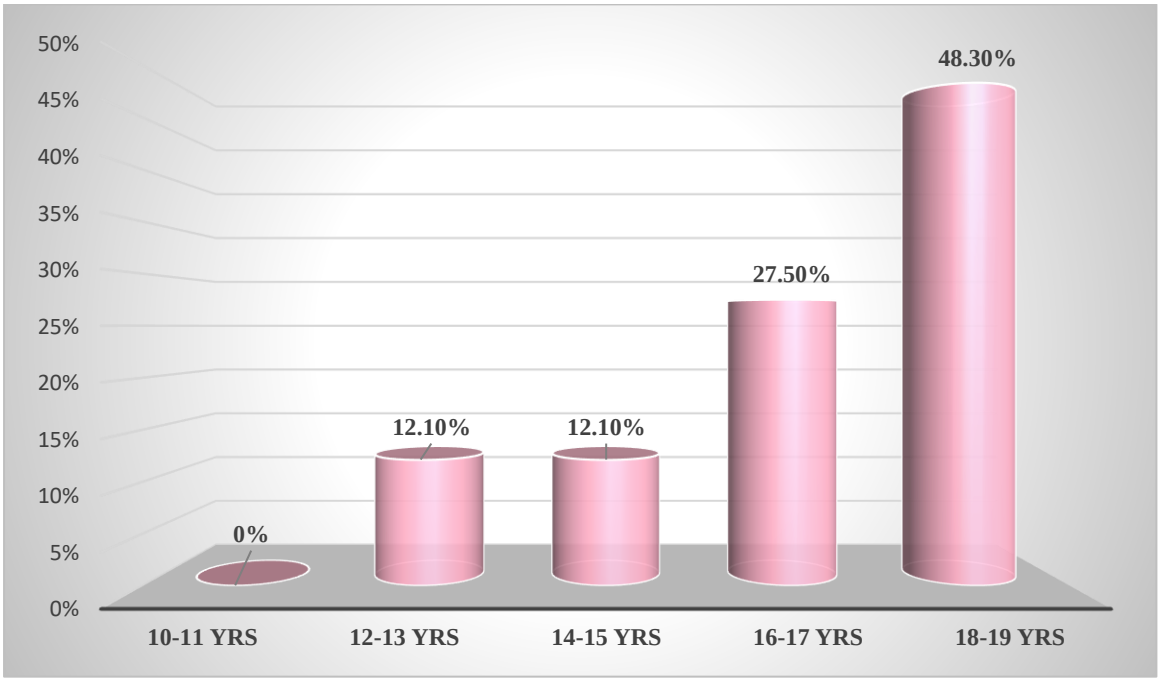
AGE DISTRIBUTION

Among 58 cases of Pigmentary disorder, 48% were seen in 18 to 19 years of age group. Nearly, 28% were seen in 16 to 17 years age group.

TABLE: 28 AGE WISE DISTRIBUTION OF PIGMENTARY DISORDERS (n=58)

AGE GROUP (IN YEARS)	TOTAL NO OF CASES	PERCENTAGE (%)
10-11	0	0%
12-13	7	12.1%
14-15	7	12.1%
16-17	16	27.5%
18-19	28	48.3%

GRAPH: 25 AGE WISE DISTRIBUTION OF PIGMENTARY DISORDERS (n=58)



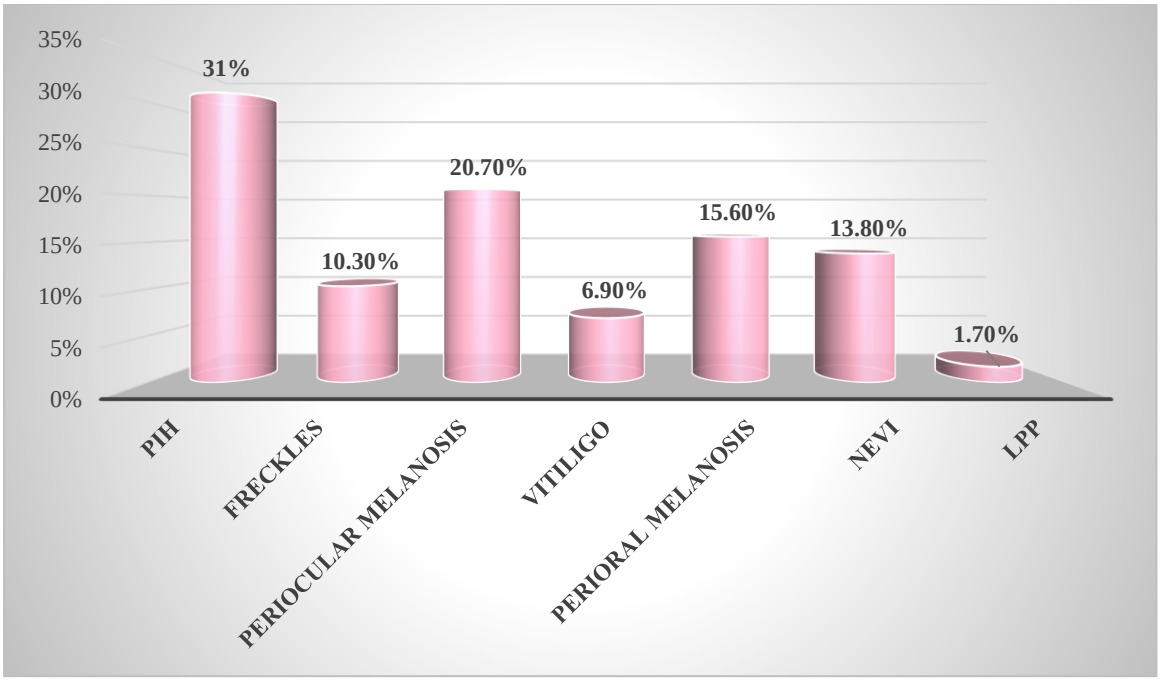
INCIDENCE OF PIGMENTARY DERMATOSES (n=58)

TABLE: 29 INCIDENCE WISE DISTRIBUTION OF PIGMENTARY DISORDERS (n=58)

DERMATOSES	TOTAL NO OF PATIENTS	PERCENTAGE (%)
POSTINFLAMMATORY HYPERPIGMENTATION	18	31.0%
FRECKLES	6	10.3%
PERIOCULAR MELANOSIS	12	20.7%
VITILIGO	4	6.9%
PERIORAL MELANOSIS	9	15.6%
NEVI	8	13.8%
LPP	1	1. 7%

Most Pigmentary disorders (31%) was caused due to post-inflammatory hyperpigmentation. About 21% was due to periocular melanosis.

GRAPH: 26 INCIDENCE WISE DISTRIBUTION OF PIGMENTARY DISORDERS (n=58)



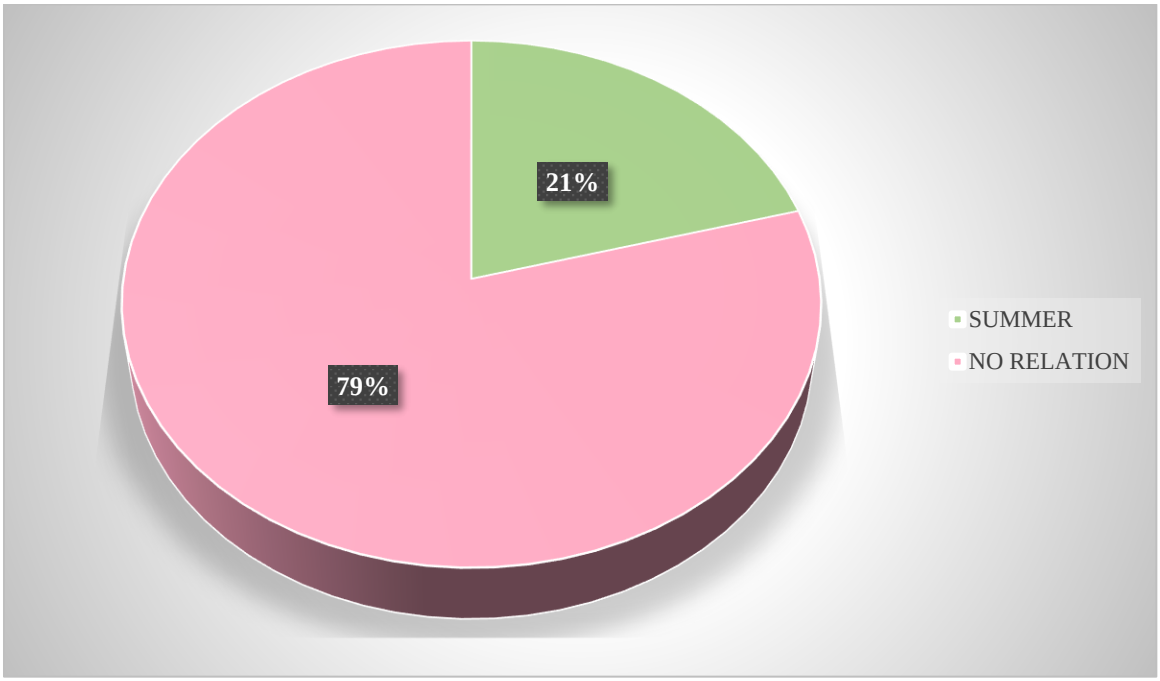
SEASONAL VARIATION IN PIGMENTARY DERMATOSES

Pigmentary dermatoses were less prevalent in the summer season (21%). About 79% of the cases had no seasonal relationship with Pigmentary dermatoses formation.

TABLE: 30 SEASONAL VARIATION IN PIGMENTARY DISORDERS (n=58)

SEASONAL VARIATION	TOTAL NO. OF CASES	PERCENTAGE (%)
SUMMER	12	20.7%
NO RELATION	46	79.3%

GRAPH: 27 SEASONAL VARIATION IN PIGMENTARY DISORDERS (n=58)



DISTRIBUTION OF SITES

TABLE: 31 SITES DISTRIBUTION IN PIGMENTARY DISORDERS (n=58)

SITES	FRECKLES	NEVI	PIH	POM	POcM	VITILIGO	LPP	TOTAL
PERINASAL	6	2	14	0	0	0	0	22 (37.9%)
PERIOCULAR	6	2	12	0	12	2	1	35 (60.3%)
PERIORAL	0	2	5	9	0	1	0	17 (29.3%)
PERIAURAL	0	1	14	0	0	1	0	16 (27.6%)
PERIANAL	0	1	0	0	0	0	0	1 (1.7%)

Maximum involvement by the pigmentary disorder was periocular area (60.3%), followed by perinasal area (37.9%) and perioral area (29.3%).

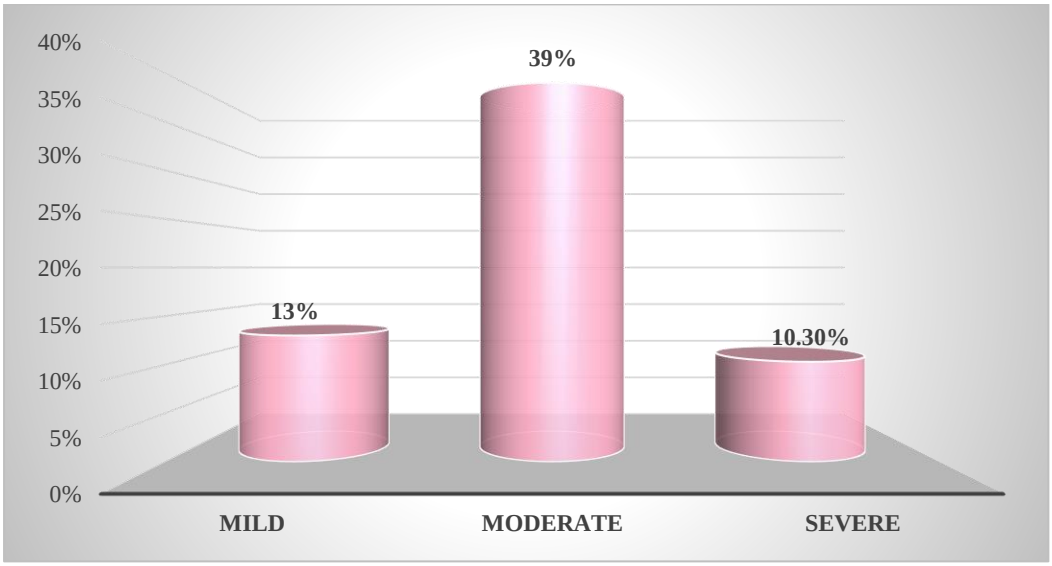
STRESS MORBIDITY

The table below depicts different dermatoses among mild, moderate, and severe stress morbidity in 58 instances. Among all stress morbidity, the number of moderate cases was higher which was seen in 39 cases (67.2 %).

TABLE: 32 STRESS MORBIDITY IN PIGMENTARY DISORDERS (n=58)

DERMATOSES	**SEVERITY**		
	MILD	**MODERATE**	**SEVERE**
VITILIGO	0	2	2
PIH	4	14	0
NEVI	6	2	0
LPP	0	0	1
FRECKLES	1	3	2
POcM	1	11	0
POM	1	7	1
TOTAL	13 (22.4%)	39 (67.2%)	6 (10.3%)

GRAPH: 28 STRESS MORBIDITY IN PIGMENTARY DISORDERS (n=58)



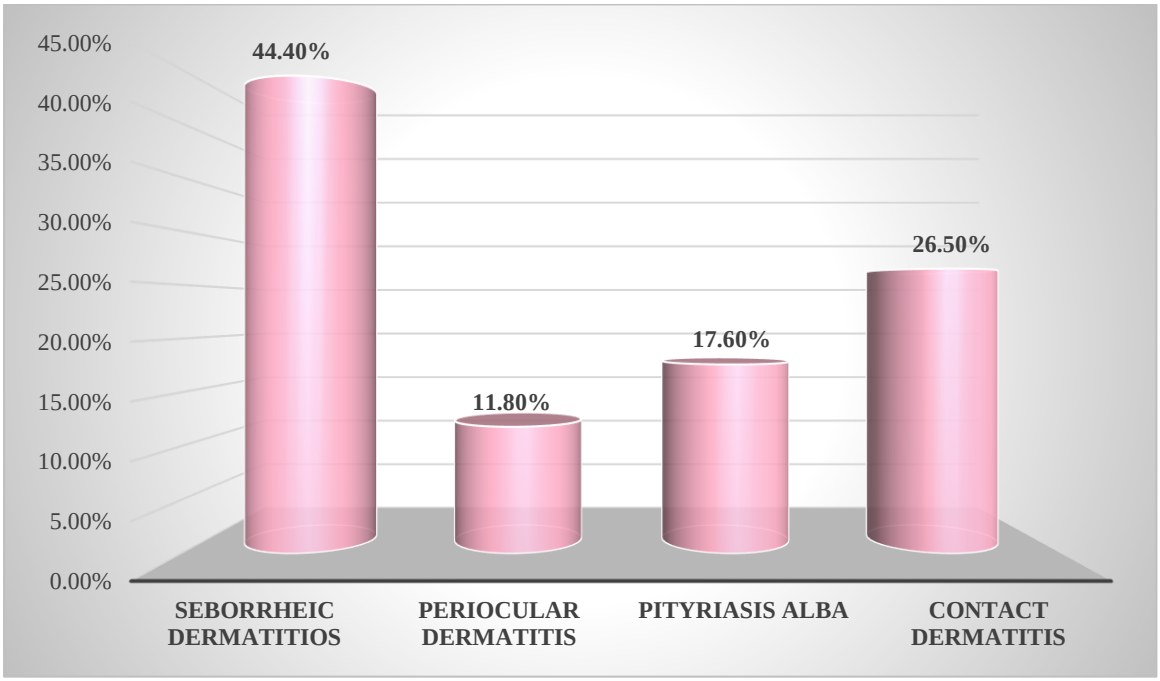
ECZEMA (n=34)

Seborrheic dermatitis were found in 15 of the 34 instances (44%). There were 9 incidences of contact dermatitis (26.5%). Pityriasis alba was found in 6 instances (17.6%). Periocular dermatitis was detected in four instances (11.8%).

TABLE: 33 INCIDENCE OF DERMATOSES IN ECZEMA (n=34)

DERMATOSES	TOTAL	PERCENTAGE
SEBORRHEIC DERMATITIS	15	44.1%
PERIOCULAR DERMATITIS	4	11.8%
PITYRIASIS ALBA	6	17.6%
CONTACT DERMATITIS	9	26.5%

GRAPH: 29 INCIDENCE OF DERMATOSES IN ECZEMA (n=34)



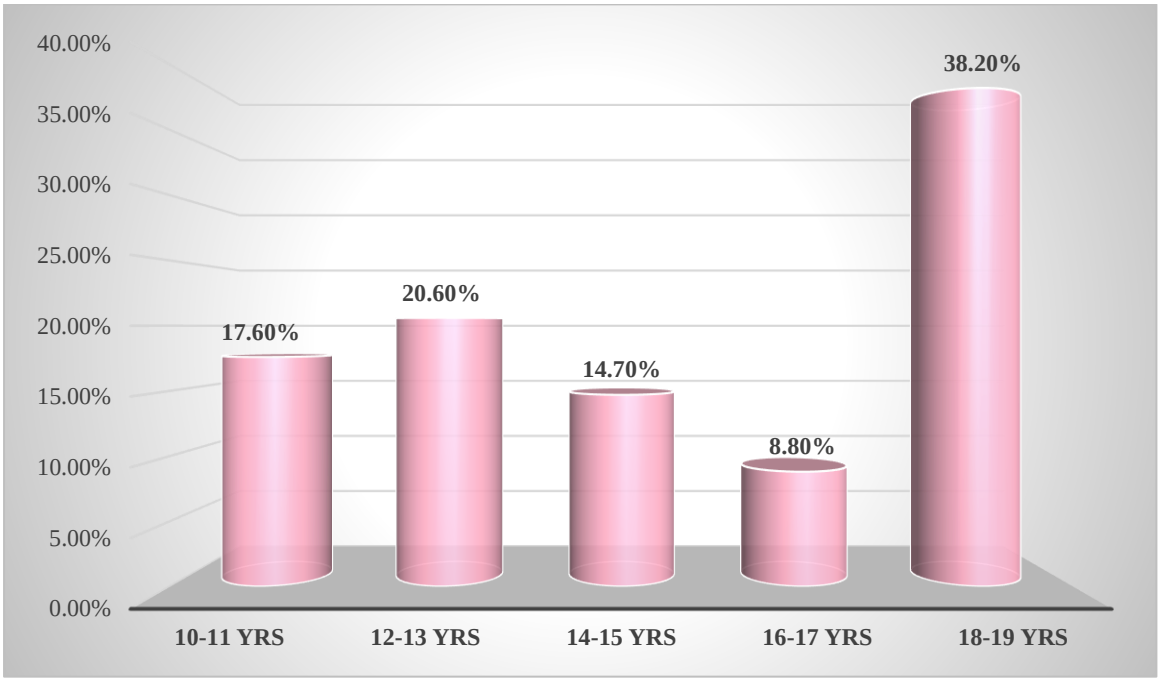
AGE-WISE DISTRIBUTION

Among 34 cases of Eczema, 38% were seen in 18 to 19 years of age group. Nearly, 21% were seen in 12 to 13 years age group.

TABLE: 34 AGE WISE DISTRIBUTION AMONG ECZEMA CASES (n=34)

AGE GROUP (IN YEARS)	TOTAL NO OF CASES	PERCENTAGE (%)
10-11	6	17.6%
12-13	7	20.6%
14-15	5	14.7%
16-17	3	8.8%
18-19	13	38.2%

GRAPH: 30 AGE WISE DISTRIBUTION AMONG ECZEMA CASES (n=34)



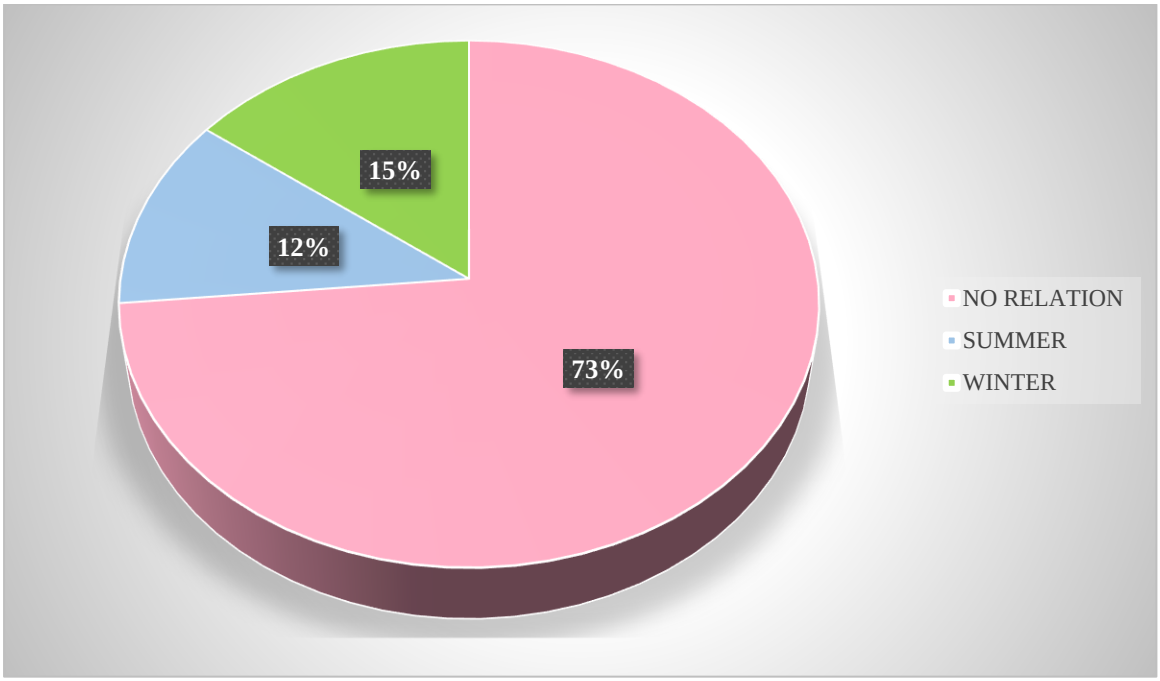
SEASONAL VARIATION

Eczema are equally prevalent in the summer season (12%) and winter season (15%). About 74% of the cases had no seasonal relationship.

TABLE: 35 SEASONAL VARIATION AMONG ECZEMA CASES [n=34]

SEASONAL VARIATION	TOTAL NO OF CASES	PERCENTAGE (%)
NO RELATION	25	73.6%
SUMMER	4	11.8%
WINTER	5	14.7%

GRAPH: 31 SEASONAL VARIATION AMONG ECZEMA CASES (n=34)



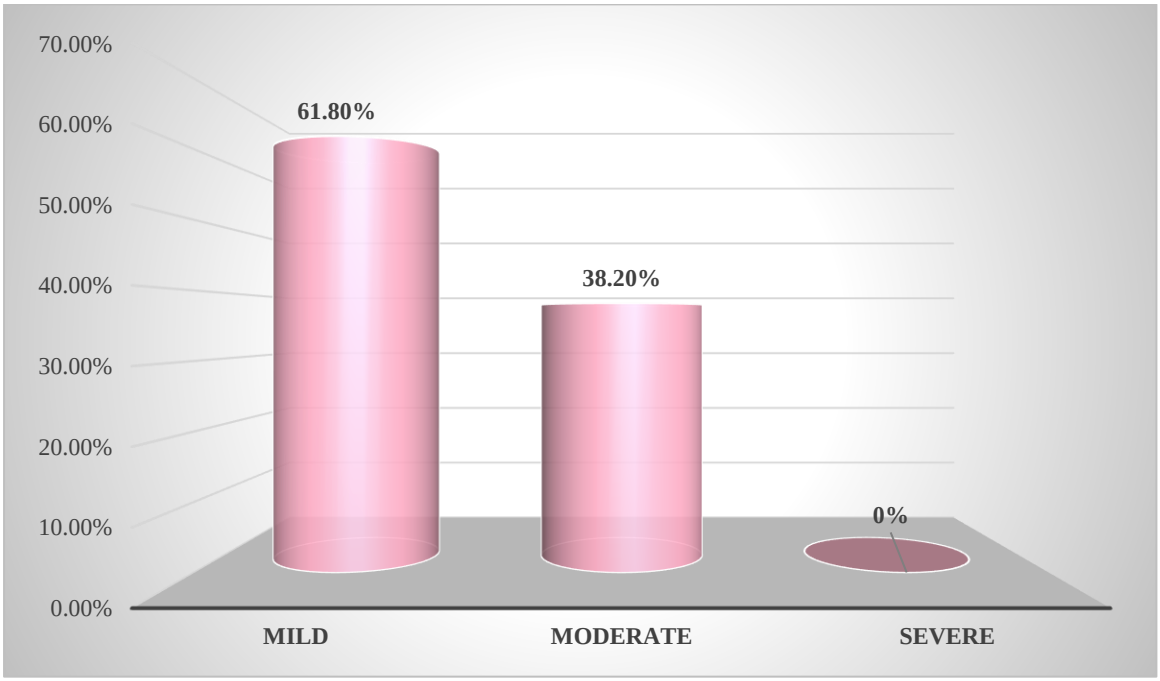
STRESS MORBIDITY

The table and figure below depicts eczema distribution among mild, moderate, and severe stress morbidity in 34 instances. Among all stress morbidity, the number of mild cases was higher.

TABLE: 36 STRESS MORBIDITY AMONG ECZEMA CASES (n=34)

DERMATOSES	SEVERITY		
	MILD	MODERATE	SEVERE
SEBORRHEIC DERMATITIS	11	4	O
PERIOCULAR DERMATITIS	2	2	0
PITYRIASIS ALBA	6	0	0
CONTACT DERMATITIS	2	7	0
TOTAL	21 (61.8%)	13 (38.2%)	0 (0%)

GRAPH: 32 STRESS MORBIDITY AMONG ECZEMA CASES (n=34)



5. SKIN TUMOURS

DPN and Keloid instances were found in 31% and 69% of skin tumour cases, respectively.

KELOID:

The majority of keloid cases (64%), were between the ages of 18 and 19. Approximately 9% of those who complained had a family history of keloids. Keloids were most prevalent in the periaural (82%) followed by perinasal (18%). 73% of the cases had moderate stress.

DPN:

All of the patients were between the ages of 18 and 19. Approximately 20% of those who complained had a family history. The number of moderate cases was larger, accounting for 80% of all stress morbidity.

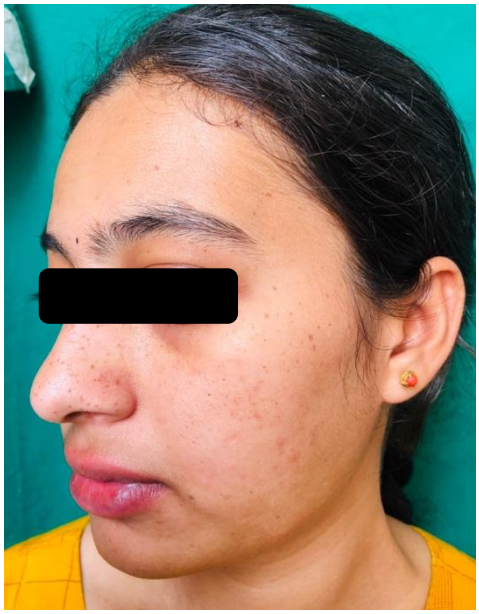
6. PHOTODERMATOSES

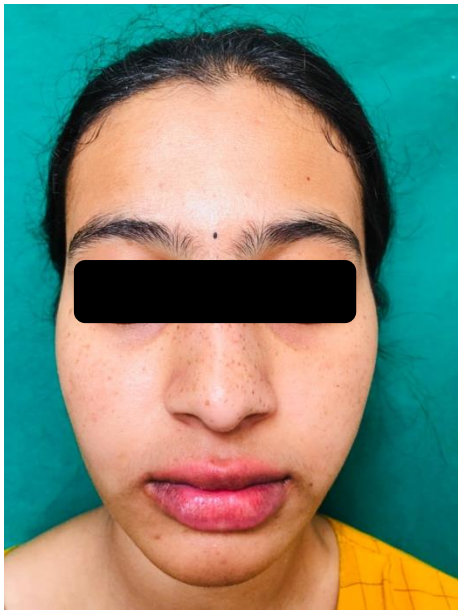
The majority of instances (43% of PMLE patients) were between the ages of 18 and 19. Approximately 71% of cases are found during the summer season. The periocular region was found in 100% of photodermatoses patients. Moderate stress was observed in all the patients.

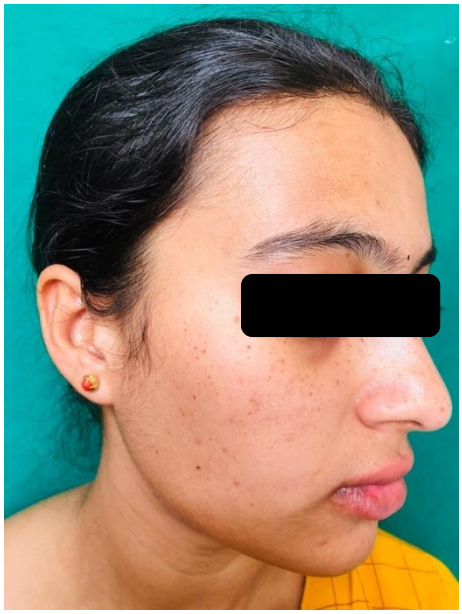
7. METABOLIC DISORDER

With metabolic problems, 33% of cases occurred between the ages of 12-13, 14-15 and 18-19. The perioral region was seen in all instances with metabolic abnormalities. The number of moderate and mild cases accounts for a 1:1 ratio among all stress morbidity.

PHOTOGRAPHS

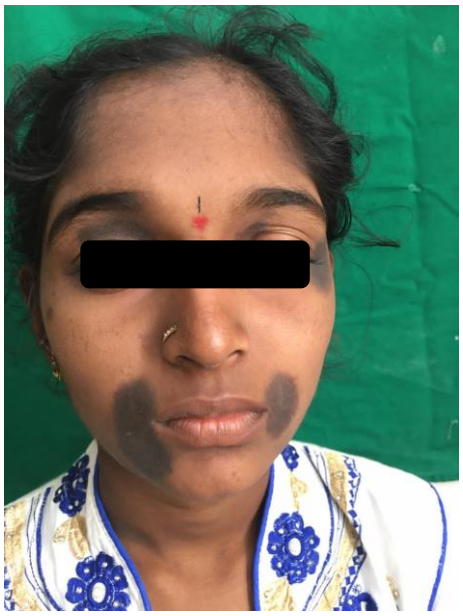






PHOTOGRAPH 1 :

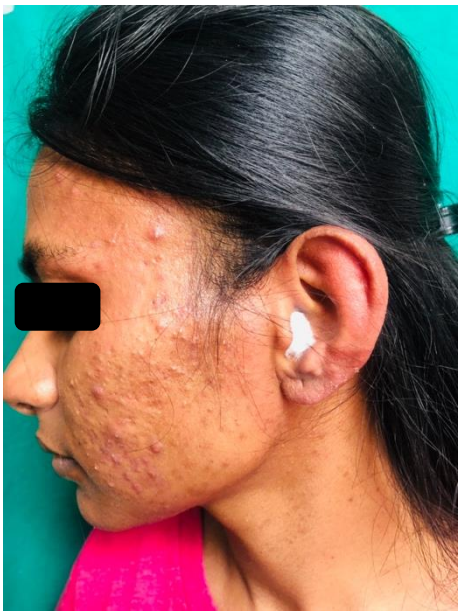
FRECKLES

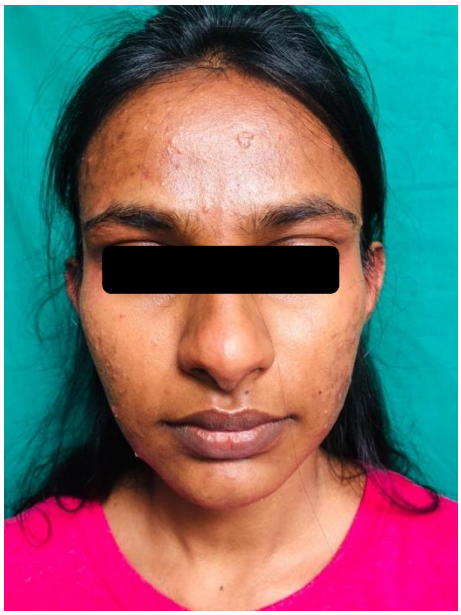


PHOTOGRAPH 2 :

LICHEN PLANUS

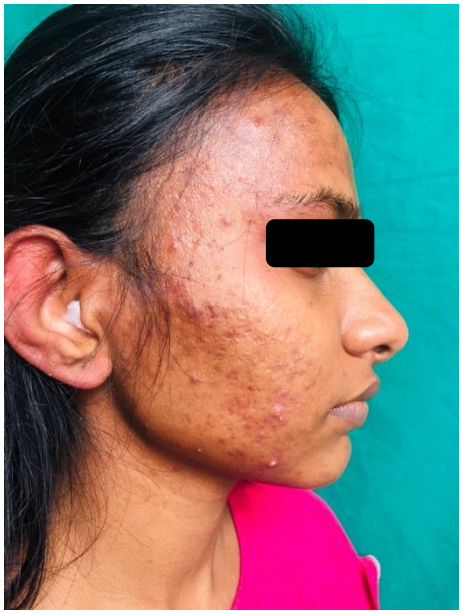
PIGMENTOSUS

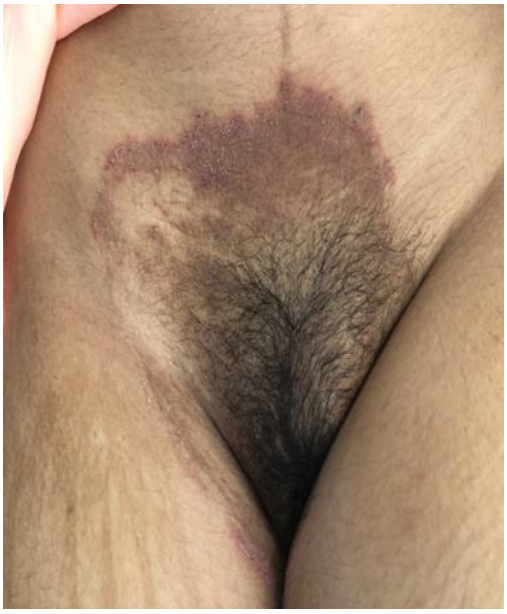


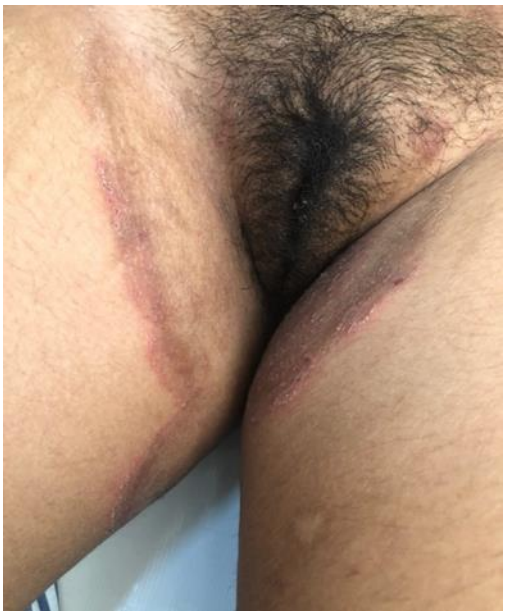


PHOTOGRAPH 3 :

ACNE VULGARIS







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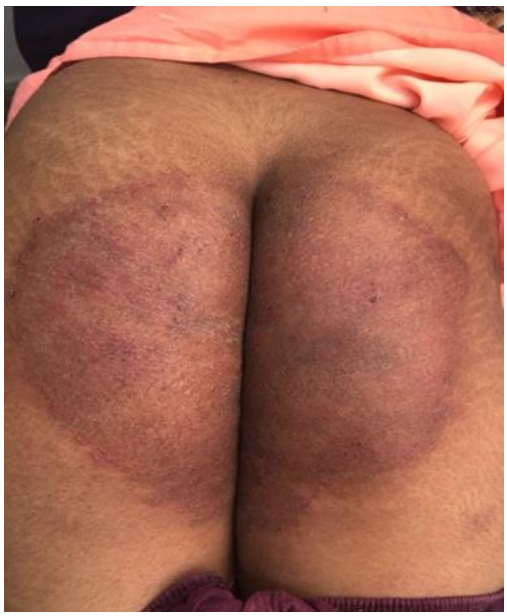
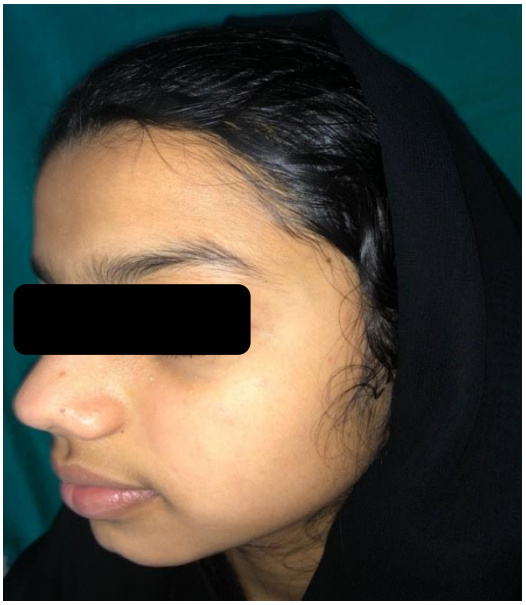
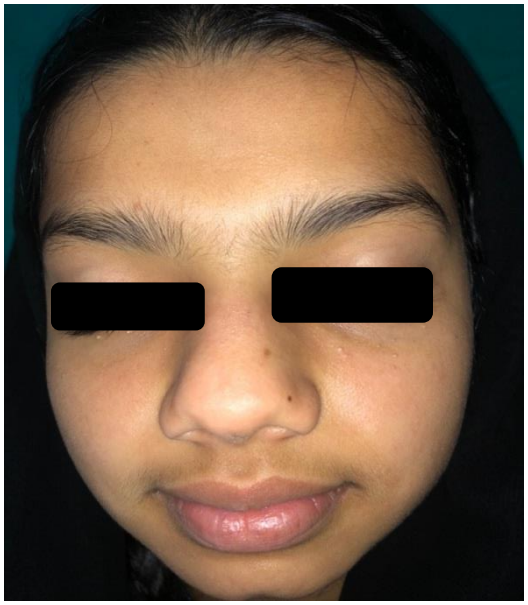
TINEA CRURIS







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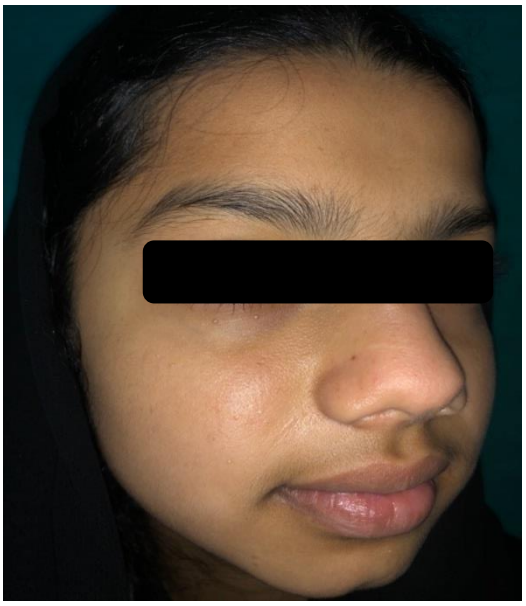
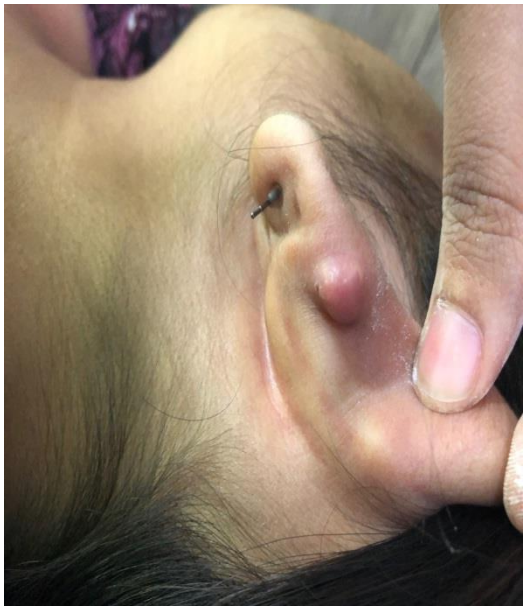
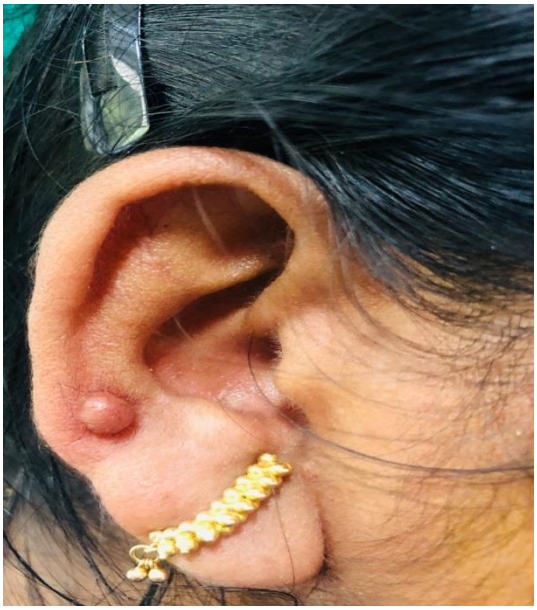
MILIA





PATIENT 1



PATIENT 2

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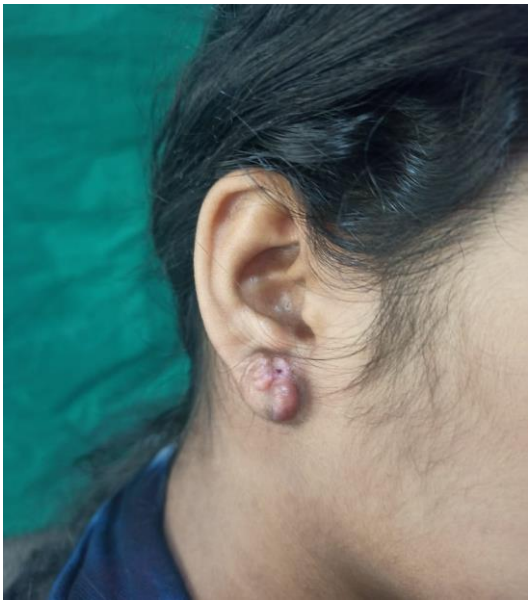
KELOID



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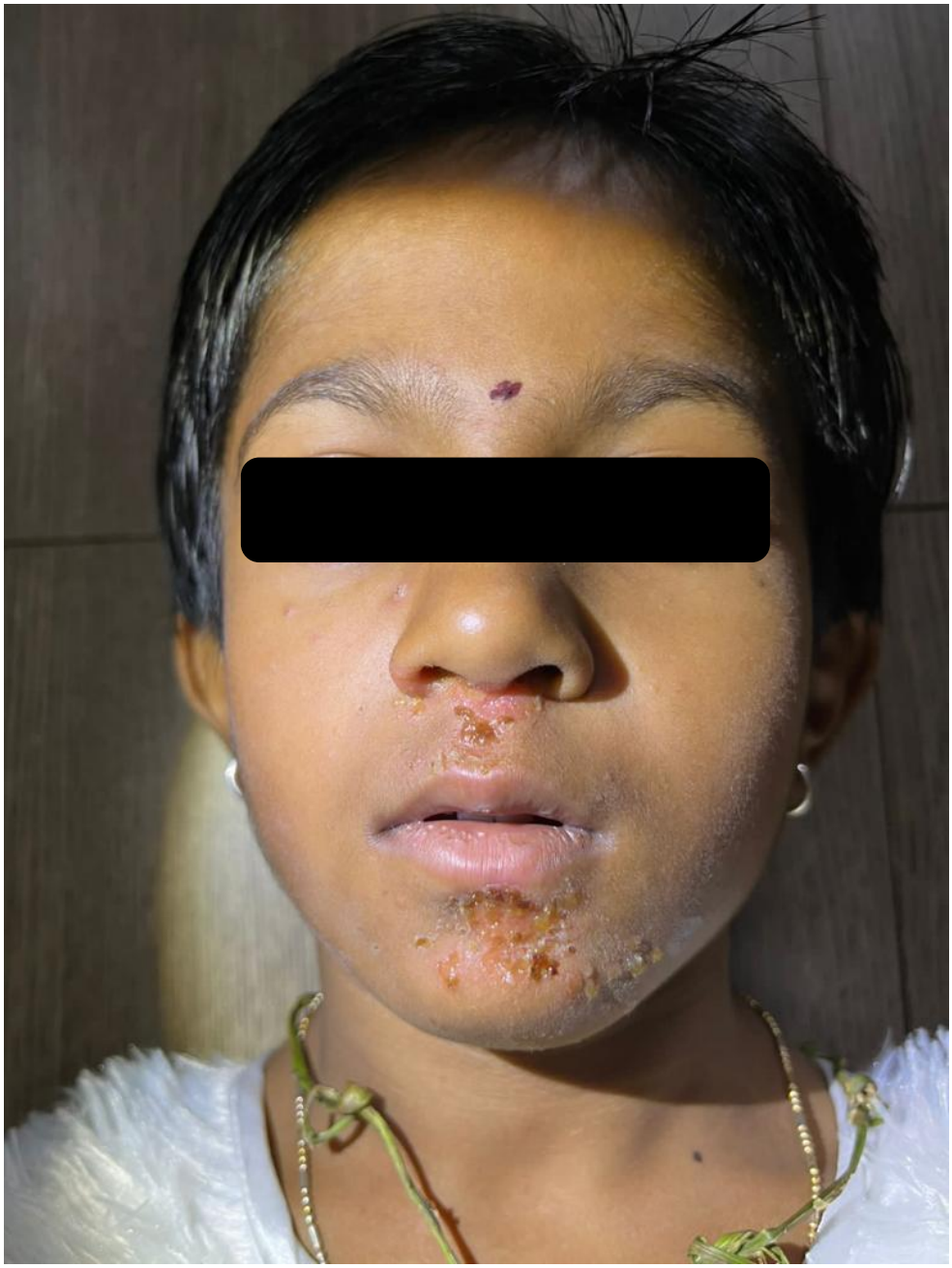
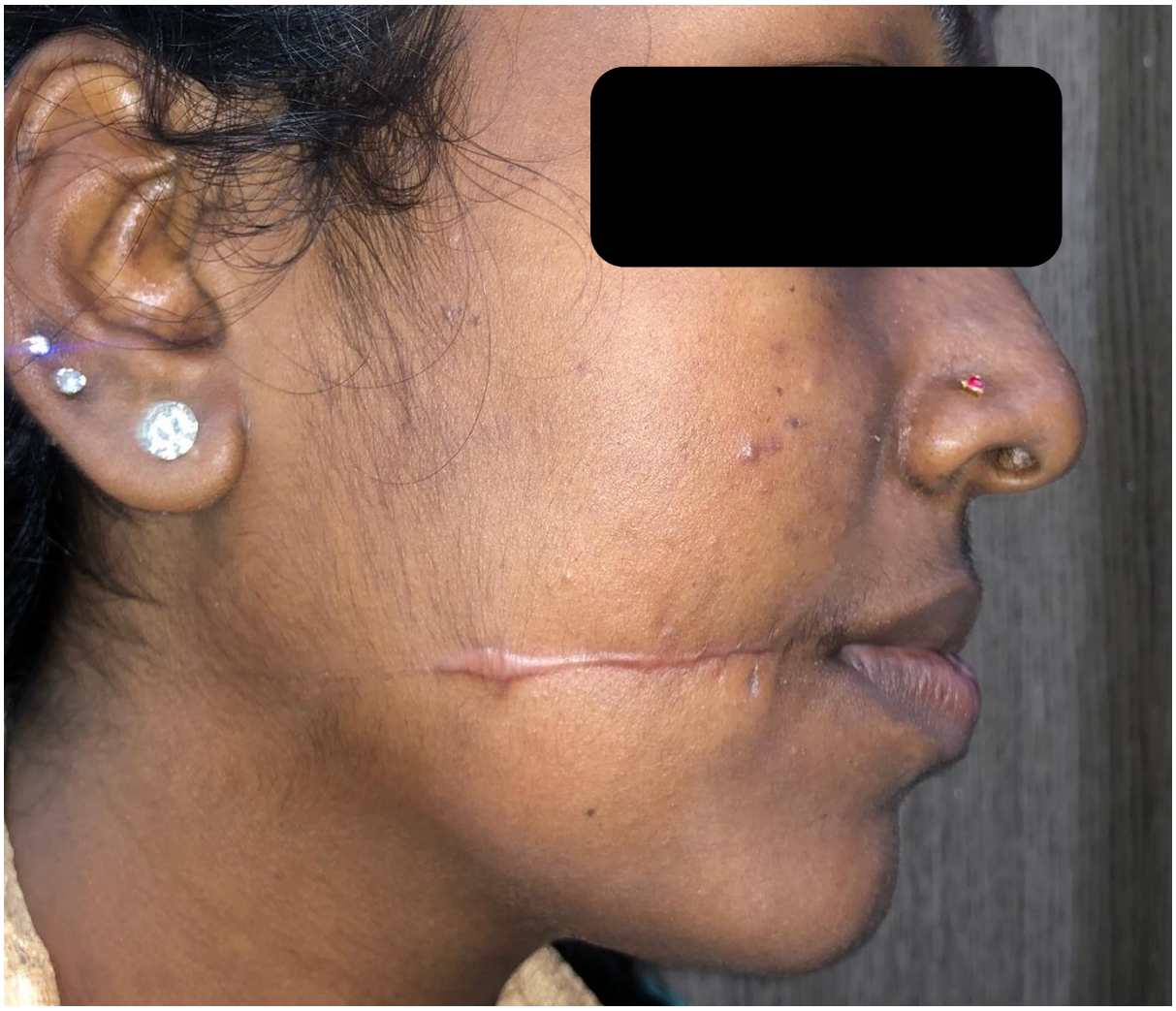


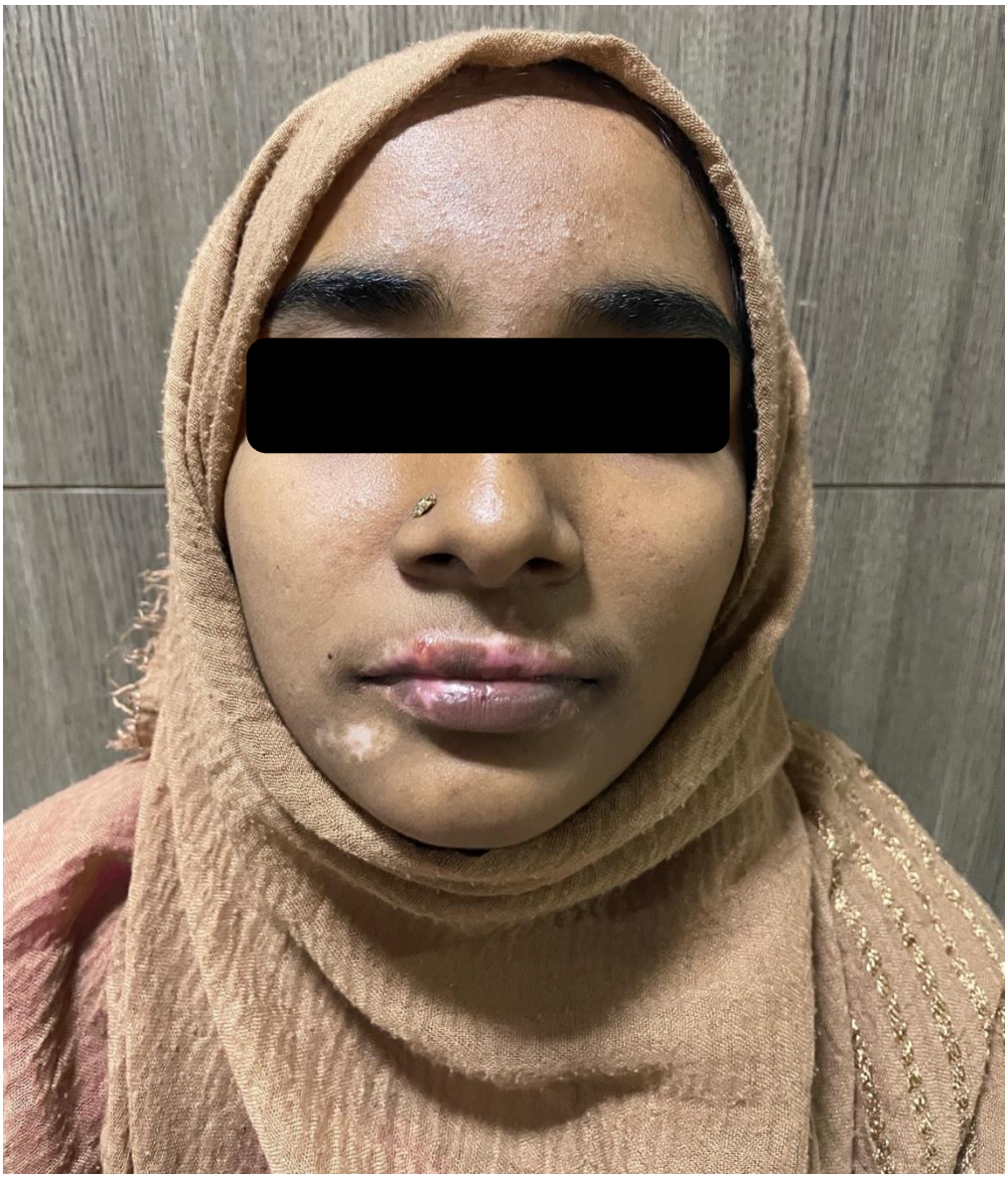
PHOTOGRAPH 7 :

IMPETIGO



PHOTOGRAPH 8 :

POST TRAUMATIC SCAR



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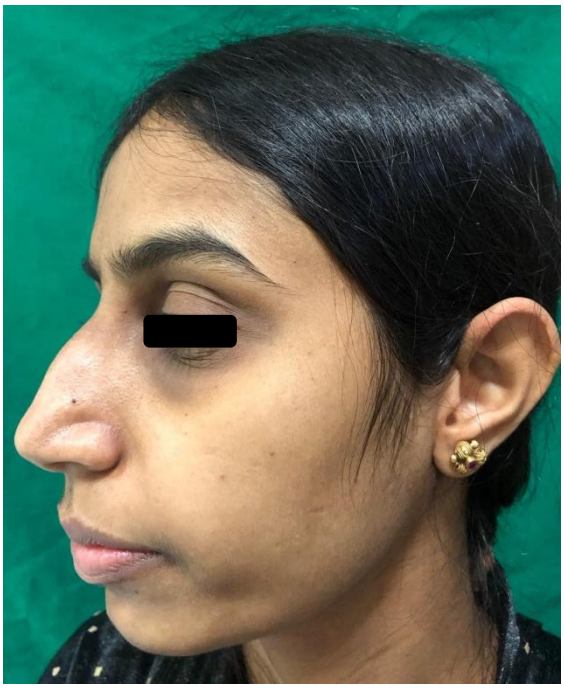
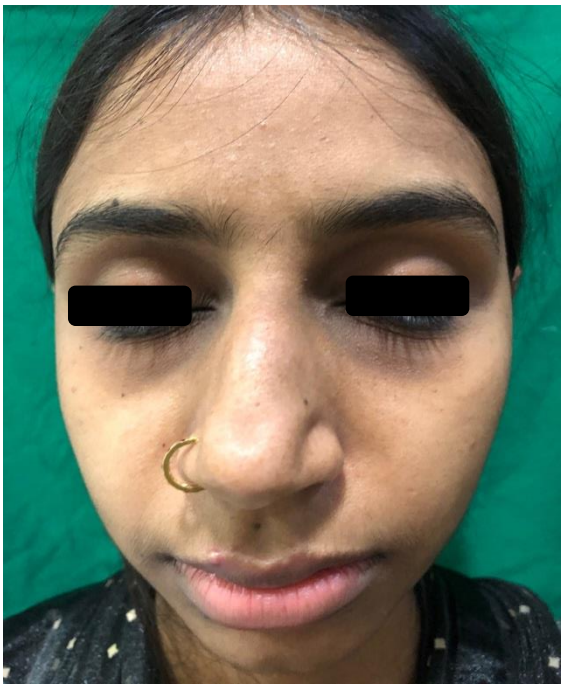
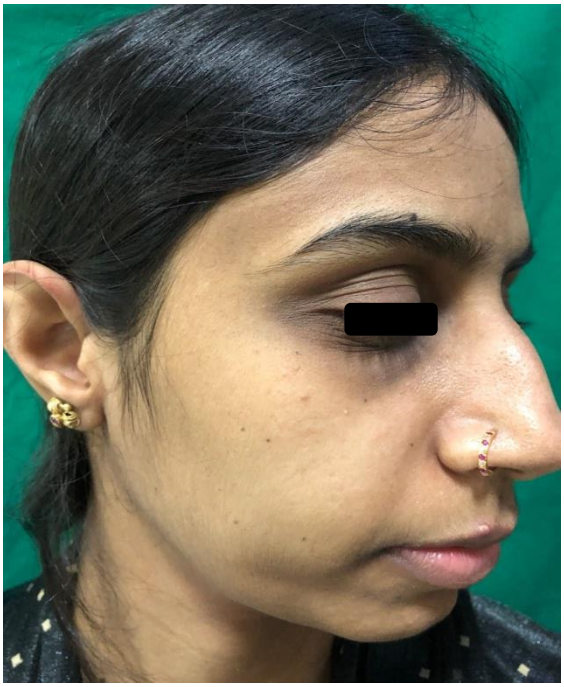
VITILIGO



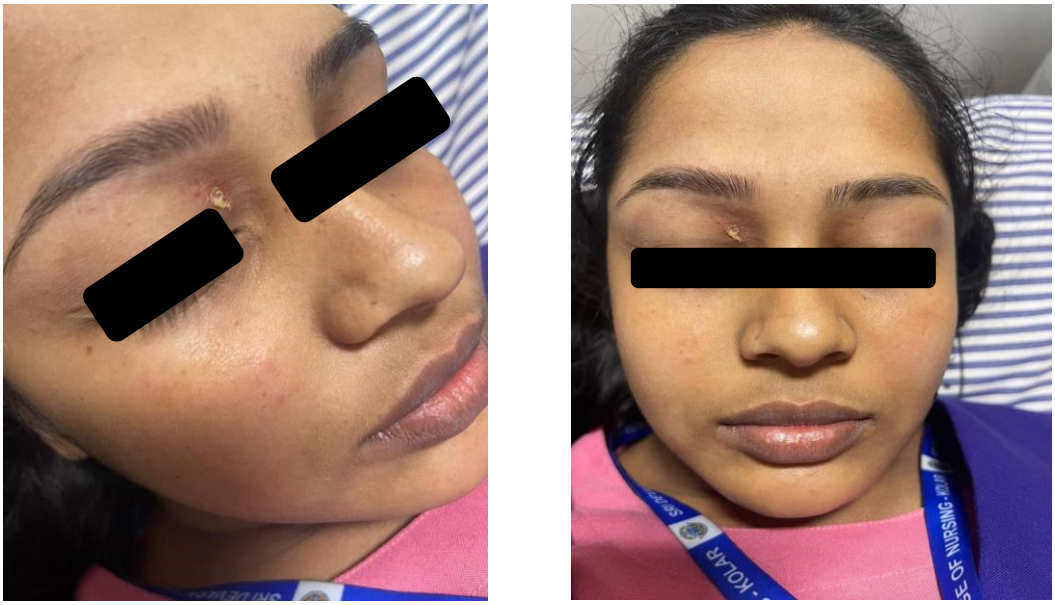




PHOTOGRAPH 10 :

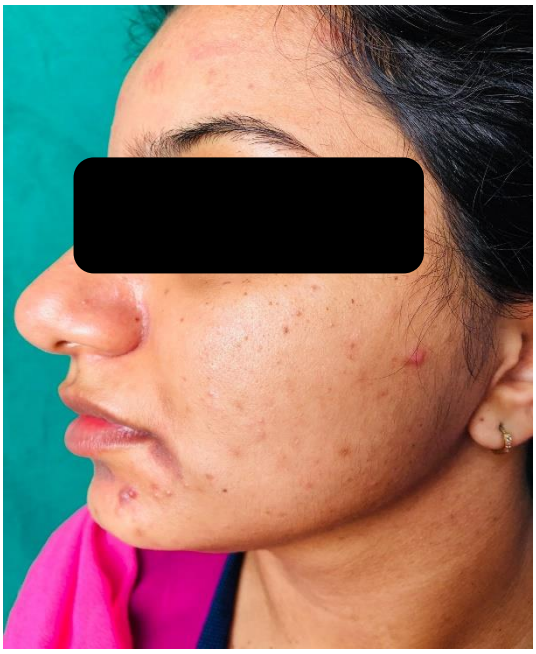
PERIORBITAL

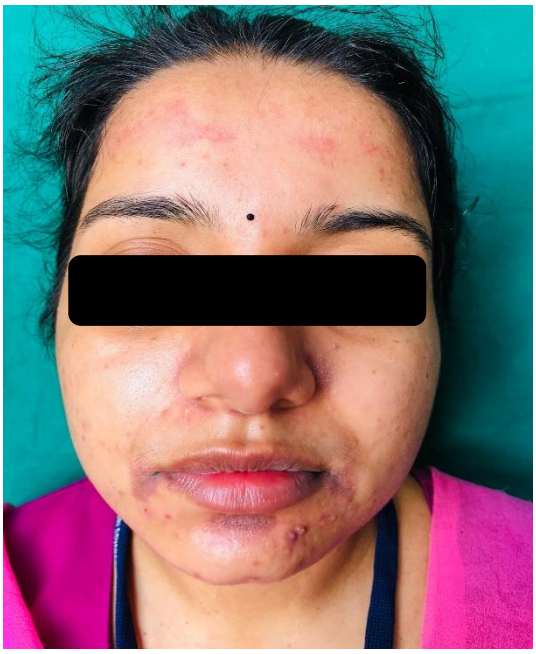
MELANOSIS

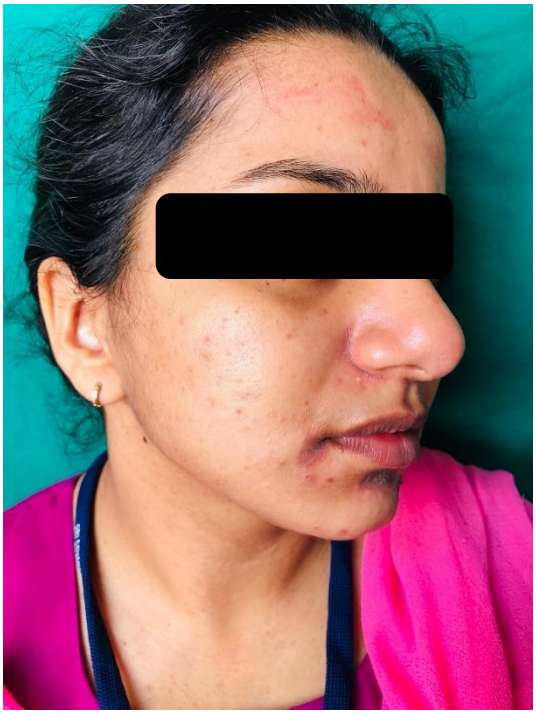


PHOTOGRAPH 11 :

XANTHELASMA PALPBERUM



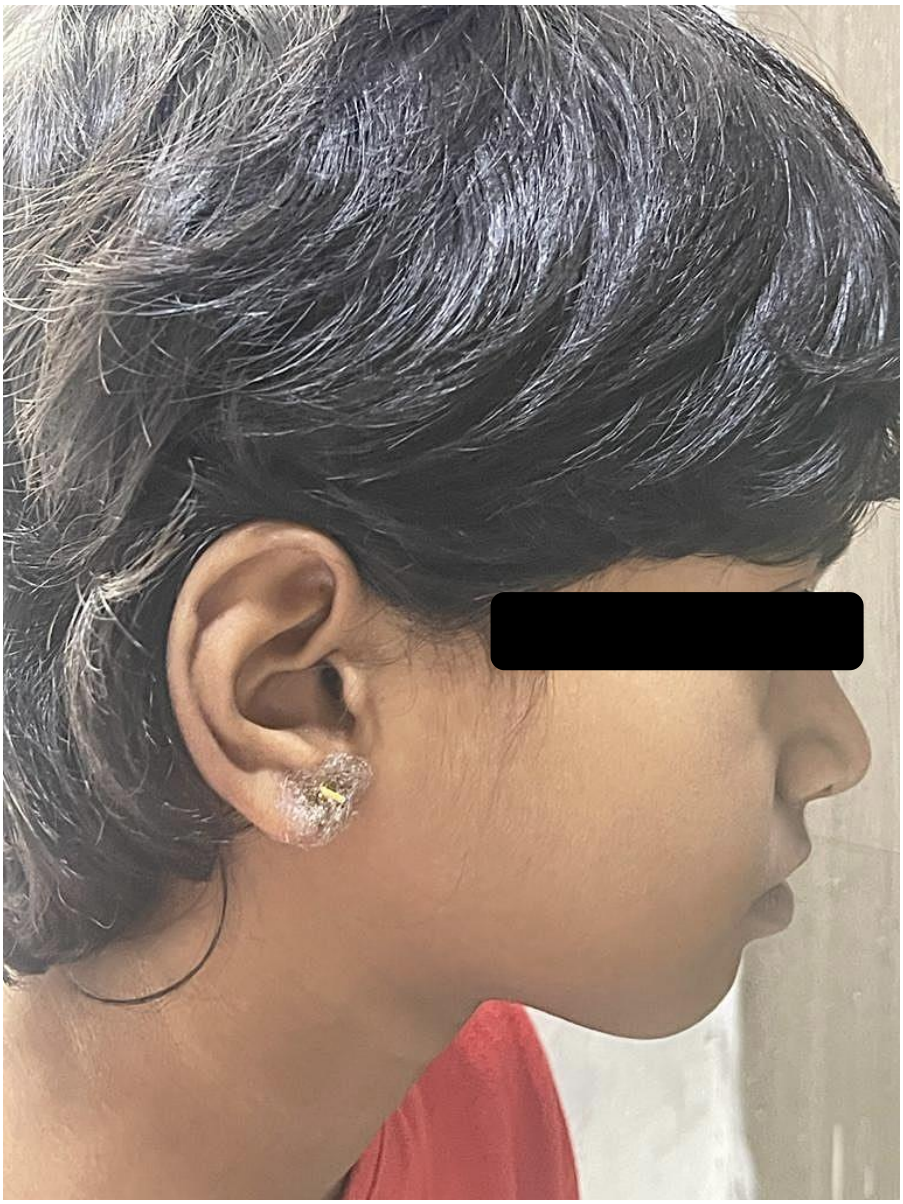




PHOTOGRAPH 12 :

PERIORAL

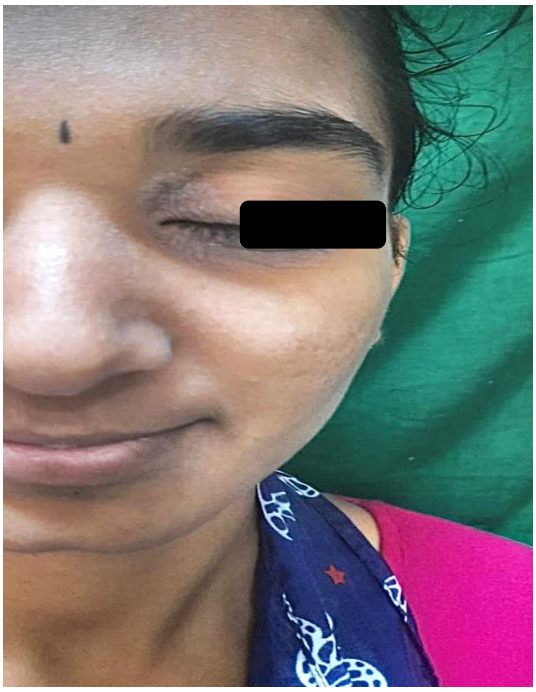
DERMATITIS

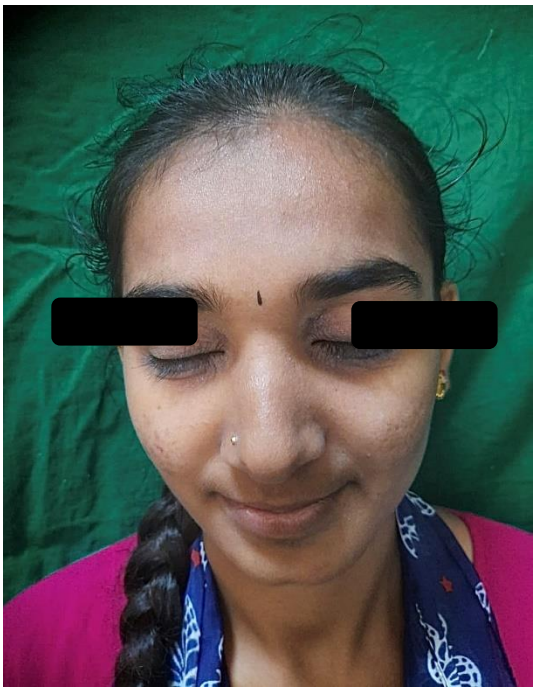


PHOTOGRAPH 13 :

CONTACT

DERMATITIS

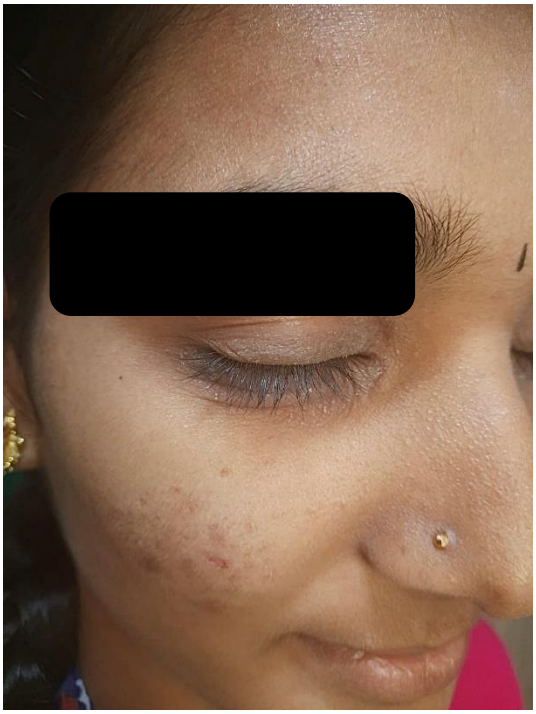


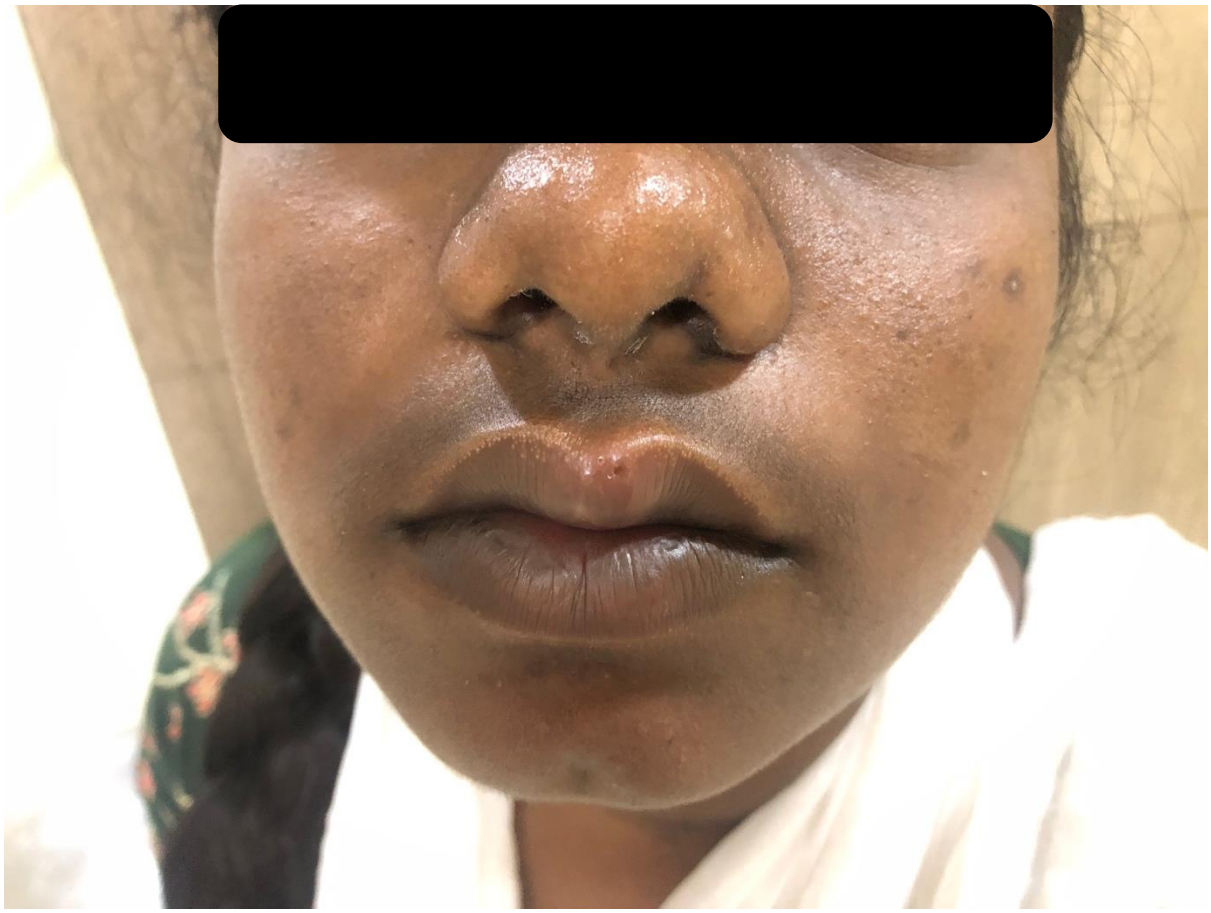


PHOTOGRAPH 14 :

PERIOCULAR

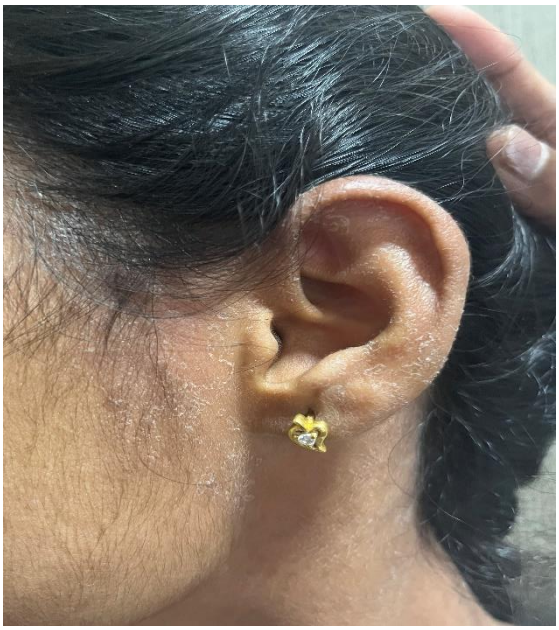
DERMATITIS

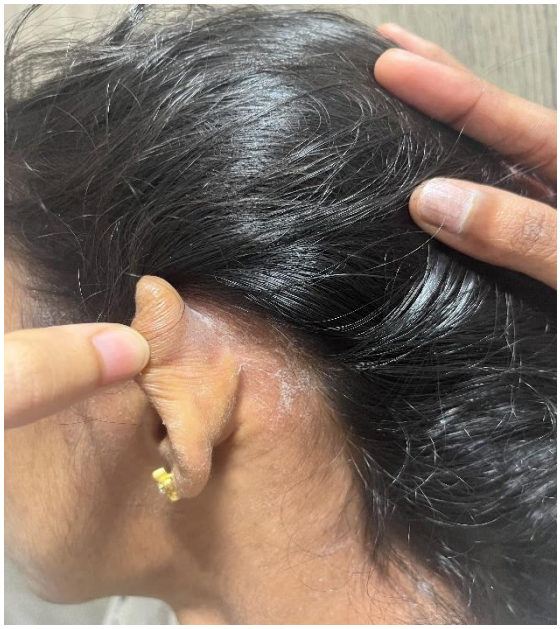




PHOTOGRAPH 15 :

HERPES LABIALIS

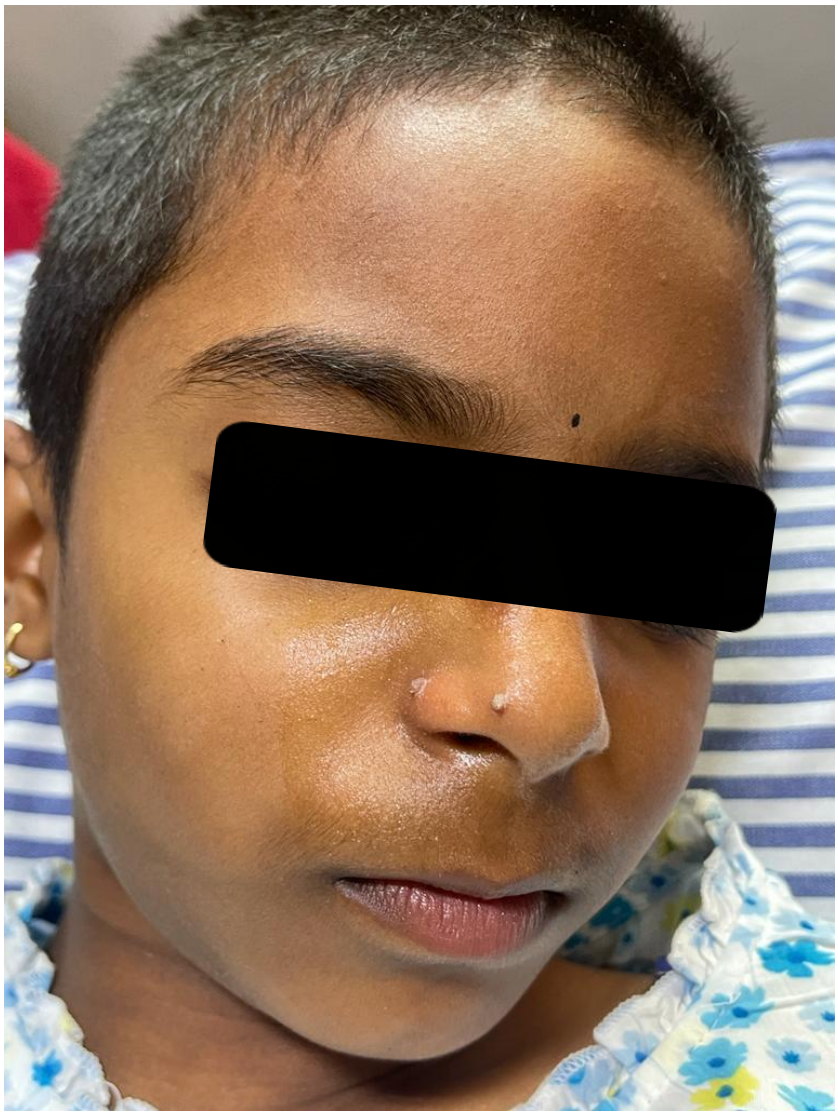




PHOTOGRAPH 16 :

SEBORRHEIC

DERMATITIS



PHOTOGRAPH 17 :

VERRUCA VULGARIS

DISCUSSION

DISCUSSION

AGE DISTRIBUTION

During the study's duration, 254 female patients were enrolled. Over half are between the ages of 18 and 19 (44.1%) which was similar to a study done by Amulya et al.[98] This might be because patient in this age group are more concerned about their appearance and seek professional help from clinicians.

SEASONAL VARIATION

Approximately 71% of the cases had no seasonal variation whereas 20% had summer exacerbation and 9% had winter exacerbation.

SYMPTOMS IN PERIORIFICIAL DERMATOSES

Most of the patients about 144 (56.7%) were asymptomatic followed by itching in 94 cases (37%) which was similar to a study by Clementson B et al.[99] who stated that only 32 (8%) individuals complained of itching or discomfort, whereas 368 (92%) were asymptomatic.

INCIDENCE OF DERMATOSES

Disorders of appendages were the most common seen in 64 (25.2%), which was in agreement to a study by ravindranath et al. In our study, it was later followed by pigmentary disorders in 58 (22.8%) & infections seen in 47 (18.5%) contrast to the study by ravindranath et al.[100] where infections was followed by eczema and pigementary disorder.

SITE INVOLVEMENT

The most common site to be involved in our study was periocular area accounting for 35.8%, followed by Perinasal and Periaural area seen in 34.2% and 31.1% respectively. The least involved site was perianal area (20%).

According to Ollech et al., the periocular area was the most often affected location (38%), followed by the perinasal and perioral areas (24% and 39%, respectively).[101]

Although the perioral region is the most prevalent site of infection, Tolaymat L et al., discovered that this illness can also affect the periocular and paranasal skin. As a result, it is also known as periorificial dermatitis.[102]

LESION MORPHOLOGY

The predominant lesion in our study was papules seen in 27.2% followed by scaling in 24.8%. Macule and papules were observed in 15% and 17% of cases.

INFECTIONS:

In our study, majority of the cases had fungal infection seen in 22 (46.8%) followed by bacterial infection in 17 (36.2%) & viral infection in 8 (17%). This finding was contrast to a study by Saini et al.,[103] where viral infections was majorily observed in 21.3% followed by fungal infections and bacterial infections seen in 11.4% and 5.7% respectively. The most common affected age group was 18-19 years seen in 21 of the 47 cases accounting for 44.7%.

Tinea cruris was the most common dermatophyte infection in our study seen in 20 cases (90.9%) followed by tinea facei in 2 cases (9.1%) which is in contrast to a study by Gandhi et al.,[104] where most common was tinea corporis followed by tinea cruris. As dermatophyte infection of the non-periorificial areas were excluded from this study, there is a

difference in the incidence of clinical types among the studies. Itching was the most predominant symptom. Summer aggravation was noted in 50% of the cases due to increased perspiration.

In bacterial infections, folliculitis was observed in 58.8% followed by impetigo in 41.2% in our study which was contrast to a study by ravindranath et al where impetigo (72.7%) was most common followed by cellulitis in (23.3%).[100] 28.6% of the impetigo cases had a positive family history.

In viral infections, warts were most common seen in 75% of the cases followed by molluscum contagiosum (12.5%) which is in agreement with a study by ravindranath et al.[100] The majority of them were between the ages of 18 and 19.

INFESTATIONS

In the current study, approximately 35% of the infestations found in 14 - 15 years of total 20 cases of infestations. In 16 to 17 years, over 5% were seen.

Scabies are more prevalent in the winter season (40%) and less prevalent in summer season (10%). About half of the cases had no seasonal relationship with scabies fluctuation. Among all the scabies patients, moderate stress was observed in 70% of them.

A similar finding has been established in an review article by Cox V et al., where children and adults suffer from psychological stress.[105] This may be attributed by itching leading to discomfort as well as by social isolation due to the contagiosity of the disease.

APPENDAGEAL DISORDERS

Distribution of appendageal disorders is seen among 64 cases. In this 55% were seen with acne & 22% seen with acne scars. This finding was in accordance to another study of facial dermatoses by chintada DC et al., where acne was predominant.[106]

In the current study, acne was seen maximum (31%) in 14 - 15 years of age and a minimum (3%) in 16 – 17 years of age. Acne is more prevalent in the summer season (40%). About 37% of the cases had no seasonal variation. Majority (94%) had papular lesions followed by comedones (46%) which was contrast to chintada DC et al.[106] About 100% of acne were present in perinasal region of face. About 57% of acne seen in periaural region. Cosmetics are responsible for around 17% of acne cases. Moderate stress was seen in almost 63% of the individuals followed by severe stress in only 3% of them. A study of acne in correlation with stress by Jusuf NK et al.,[107] observed a similar finding with stress severity where severe stress was seen least in 6.7% of acne patients.

ACNE SCARS

Duration of the scars ranged from 2 months to 2 years. Perinasal area was involved in all the cases (100%) whereas periaural and periocular area was seen in 71.4% and 50% respectively. 64.3% of the patients has moderate stress and 35.7% of them has severe stress.

Machiwala AN et al., found that Acne affects at a mean age of 18.5.[108] FT Lauermann et al., were the first to discover a link between acne scarring and the severity stress. Because there is a clear relationship between acne severity and scarring, timely and effective treatment is the best approach to prevent scarring.[109]

PIGMENTARY DISORDER

Among 58 cases of Pigmentary disorder, 48% were seen in 18 to 19 years of age group. Nearly, 28% were seen in 16 to 17 years of age group. Most Pigmentary disorders (31%) was caused due to post-inflammatory hyperpigmentation. About 21% was due to periocular melanosis. Pigmentary dermatoses were less prevalent in the summer season (21%). About 79% of the cases had no seasonal relationship with Pigmentary dermatoses. Maximum involvement by the pigmentary disorder was over periocular area (60.3%), followed by perinasal area (37.9%) and perioral area (29.3%).

A study by F Teichgräber et al., showed 2% of instances in the pigmentary condition was congenital melanocytic nevi, 1% had vitiligo, and 1% had freckles. This is slightly lower compared to our study.[110]

R Marrone et al., found that pigmentary disorders and skin appendage disorders were more prevalent in 14 – 16 years.[111] Bacterial infections, skin appendage and pigmentary diseases in children aged 15 to 18 found by Bissek AC et al.[112]

ECZEMA

Seborrheic dermatitis were found in 15 of the 34 instances. There were 9 incidences of contact dermatitis. Pityriasis alba was found in 6 instances. Periocular dermatitis was detected in four instances. Among 34 cases of Eczema, 38% were seen in 18 to 19 years of age group. Nearly, 21% were seen in 12 to 13 years age group. Eczema are equally prevalent in the summer season (12%) and winter season (15%). About 74% of the cases had no seasonal relationship. Among all cases in regard to stress morbidity, the number of mild cases was higher. This finding in our study is similar to that of another study done by Pradeep et al.[113]

SKIN TUMORS

DPN and Keloid instances were found in 31% and 69% of skin tumour cases, respectively.

KELOID

The majority of keloid cases (64%) were between the ages of 18 and 19. Approximately 9% of those who complained had a family history of keloids. Keloids were most prevalent in the perinasal (18%) and periaural (82%).

DPN

In DPN instances, all of the patients were between the ages of 18 and 19. Approximately 20% of those who complained had a family history of keloids. The number of mild cases was larger, accounting for 80% of all stress morbidity.

Adults were more likely to have keloids (P = 0.015), skin cancers or malignancies (P = 0.030), or drug eruptions (P = 0.003). DPN and papulosquamous illnesses had a substantial impact on female participants (P = 0.030 and 0.019, respectively) according to AO Akinboro et al.[114]

PHOTODERMATOSES - PMLE

The majority (43%) were of PMLE patients. Additionally, P Lehmann et al. (2011) found that polymorphic light eruption (prevalence 10% to 20%), are quite prevalent.[115]

In Central Europe, Scandinavia, and the US, polymorphous light eruption (PMLE) is the most prevalent photodermatosis, with a prevalence of 10% to 20% according to Gambichler T et al.[116]

In our study, approximately 71% of cases are found during the summer season. The periocular region was found in 100% of photodermatoses patients. An aberrant response to sunlight, often its UV component, is what causes photodermatoses. Moderate stress was observed in these patients.

A similar finding to our study was found by H. Hönigsmann et al., which revealed that PMLE is most common from March to June, however it can also happen at any time among travellers who have visited warm places. It may happen to anyone, including young children.[117]

METABOLIC DISEASE

With metabolic problems, 33% of cases occurred between the ages of 12-15 and 18-19. The perioral region was seen in all instances with metabolic abnormalities. The number of moderate and mild cases accounts for a 1:1 ratio among all stress morbidity.

CONCLUSION

CONCLUSION

This study was done to analyse the many dermatoses affecting the face and their diverse appearances. Early detection and characterisation of facial skin problems were harder in comparison to other skin illnesses. Many of the research explained particular facial dermatoses. This study provides insight into several face dermatoses seen in clinical practise.

We conducted this study to shed light on the different aesthetic issues that teenagers confront as a result of physiological and social causes. These varied periorificial dermatoses might have a greater psychological impact on this age group than a dermatologist would imagine. Eventhough majority of individuals had milder conditions, it had a severe influence over them. Females were more impacted by these dermatoses involving the face as well as the genital area. Hence, timely treatment and educating the patient can ameliorate their self-esteem and confidence.

SUMMARY

SUMMARY

- Patients varied in age from 11 to 19. Teenagers aged 18 to 19 (44.1%), followed by 12-13 (31%), and 14-15 (16.5%). The prevalence was lowest in children aged 10 to 11 (7.9%).
- Duration of symptoms ranges from 2 days to 5 months. Most of the patients about 144 (56.7%) were asymptomatic followed by itching in 94 cases (37%).
- The most common site to be involved was the periocular area accounting for 35.8%, followed by Perinasal and Periaural area seen in 34.2% and 31.1% respectively.
- The most common lesion in the present whole study case was papules, which were observed in 27.2% of the cases, followed by scales in 24.8%. Macule and papules were observed in 15% and 17% of cases.
- The severity of stress among the 254 periorificial dermatoses was separated into mild moderate and severe in the present study. A maximum number of cases was seen with moderate severity (63%). Mild severity cases are seen in 30%. The remaining 7% of patients have severe stress.
- In the present study, Among the 47 patients, most common infections were fungal infections seen in 46.8%, followed by bacterial infections and viral infections seen in 36.2% & 17% respectively.
- Tinea cruris was the most common fungal illness, affecting 20 individuals (90.9%), whereas tinea faciei affected just two (9.1%). Itching was the most prevalent symptom noticed in these individuals. The most typical location was in the Perianal region.
- Folliculitis is observed in 58.8% of individuals with bacterial infections, followed by impetigo in 41.2%. In folliculitis, the perioral area was the most common location for impetigo, followed by the perianal area. Summer aggravation affected two of the ten folliculitis patients. Two of the seven impetigo patients had a favorable family history.

- In this investigation, warts were the most frequent viral infection (75%), followed by molluscum contagiosum (12.5%) and herpes labialis (12.5%). The most common place identified in warts is the periocular (33.3%) and perinasal (33.3%) area, whereas periocular and perioral areas were observed in molluscum contagiosum and herpes labialis, respectively. Out of the six individuals with warts, one had a family history.
- In the current study, approximately 35% of the infestations found in 14 - 15 years of total 20 cases of infestations. In 16 to 17 years, over 5% were seen.
- Scabies are more prevalent in the winter season (40%) and less prevalent in summer season (10%). About half of the cases had no seasonal relationship with scabies fluctuation. Among all infestations, the number of moderate cases was higher.
- Distribution of appendageal disorders is seen among 64 cases. In this 55% were seen with acne. 22% seen with acne scars. In the current study, acne was seen maximum (31%) in 14 - 15 years of age and a minimum (3%) in 16 – 17 years of age.
- Acne is more prevalent in the summer season (40%). About 37% of the cases had no seasonal variation. Distribution of lesion morphology seen among 57 cases of acne. In this 94% were seen with papule lesions. 46% seen with comedone.
- About 100% of acne were present in perinasal region of face. About 57% of acne seen in periaural region. Family history is responsible for over 20% of acne cases. Cosmetics are responsible for around 17% of acne cases.
- Duration of the scars ranged from 2 months to 2 years. Perinasal area was involved in all the cases (100%) whereas periaural and periocular area was seen in 71.4% and 50% respectively. 64.3% of the patients has moderate stress and 35.7% of them has severe stress.
- Among 58 cases of Pigmentary disorder, 48% were seen in 18 to 19 years of age group. Nearly, 28% were seen in 16 to 17 years age group. Most Pigmentary disorders

(31%) was caused due to post-inflammatory hyperpigmentation. About 21% was due to periocular melanosis. Pigmentary dermatoses were less prevalent in the summer season (21%). About 79% of the cases had no seasonal relationship with Pigmentary dermatoses formation. Maximum involvement by the pigmentary disorder was periocular area (60.3%), followed by perinasal area (37.9%) and perioral area (29.3%).

- Dermatoses were found in 15 of the 34 instances of seborrheic dermatitis. There were 9 incidences of contact dermatitis. Pityriasis alba was found in 6 instances. Periocular dermatitis was detected in four instances. Among 34 cases of Eczema, 38% were seen in 18 to 19 years of age group. Nearly, 21% were seen in 12 to 13 years age group. Eczema are equally prevalent in the summer season (12%) and winter season (15%). About 74% of the cases had no seasonal relationship. Among all stress morbidity, the number of mild cases was higher.
- DPN and Keloid instances were found in 31% and 69% of skin tumour cases, respectively.
- The majority of keloid cases (64%) were between the ages of 18 and 19. Approximately 9% of those who complained had a family history of keloids. Keloids were most prevalent in the perinasal (18%) and periaural (82%).
- In DPN instances, all of the patients were between the ages of 18 and 19. Approximately 20% of those who complained had a family history of keloids. The number of mild cases was larger, accounting for 80% of all stress morbidity.
- The majority of instances had 43% of PMLE patients. Additionally, P Lehmann et al. (2011) found that some forms, such as polymorphic mild eruption (prevalence 10% to 20%), are quite prevalent.

- Approximately 71% of cases are found during the winter season. The periocular region was found in 100% of photodermatoses patients. An aberrant response to sunlight, often its UV component, is what causes photodermatoses.
- With metabolic problems, 33% of cases occurred between the ages of 12 and 15 and 18 and 19. The perioral region was seen in all instances with metabolic abnormalities. The number of moderate and mild cases accounts for a 1:1 ratio among all stress morbidity.

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ANNEXURE

ANNEXURE I

PROFORMA

CASE NUMBER

PATIENT PARTICULARS

<table>
<tr><td>NAME:</td><td>UHID:</td></tr>
<tr><td>AGE:</td><td rowspan="2">DATE:</td></tr>
<tr><td>GENDER:</td></tr>
<tr><td>ADDRESS:</td><td rowspan="2">Occupation:</td></tr>
<tr><td>PHONE NUMBER:</td></tr>
</table>

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

Diet: Veg/ nonveg/ mixed
Bowel/ Bladder habits: Regular/ altered.
Sleep: Adequate/ disturbed
Appetite:
Habits: Smoking/ tobacco chewing/ alcoholism

TREATMENT HISTORY:

ON EXAMINATION:

1) GENERAL PHYSICAL EXAMINATION:

Built and Nourishment:

Pallor/ Icterus/ Clubbing/ Cyanosis/ Significant lymph node enlargement/ Edema

Vitals: Temperature-

Pulse -

Blood pressure-

Respiratory rate-

2) SYSTEMIC EXAMINATION :

- CVS :
- RS :
- PER ABDOMEN :
- CNS :
- Others:

3) CUTANEOUS EXAMINATION:

Sites of involvement :

Morphology of the lesions :

4) HAIR EXAMINATION:

5) NAIL EXAMINATION:

6) ORAL / MUCOSAL EXAMINATION:

<u>INVESTIGATIONS:</u>

- Complete haemogram
- RBS
- LFT
- Urine Routine
- KOH mount
- Fungal culture
- Bacterial culture
- Tzanck smear
- Gram stain
- Skin biopsy

FINAL DIAGNOSIS:

STRESS SCALE:

TREATMENT:

Perceived Stress Scale (Children)

The following questions ask you about your feelings and thoughts during the last week. For each question you will be asked to circle the picture that best fits your answer.

Name: Date:

Age: Birthday: I am a: Boy Girl

1. Which one has a lot of something?

2. In the last week, how often did you feel rushed or hurried?

3. In the last week, how often did you have enough time to do what you wanted?

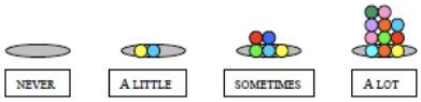
4. In the last week, how often did you feel worried about being too busy?

5. In the last week, how often did you feel worried about grades or school?

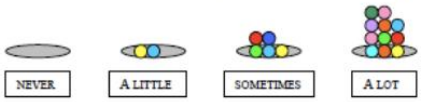
6. In the last week, how often did your mom and/or dad make you feel better?

7. In the last week, how often did your mom and/or dad make you feel loved?

8. In the last week, how often did you feel scared or nervous?

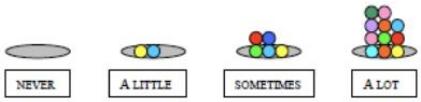


9. In the last week, how often did you feel angry?



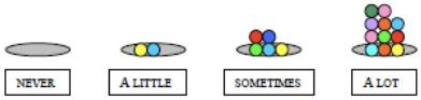
What made you angry?

10. In the last week, how often did you feel happy?

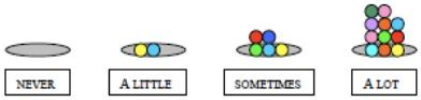


What made you happy?

11. In the past week, how often did you get enough sleep?

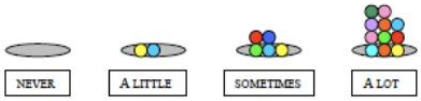


12. In the past week, how often did you have fights with your friends?



13. In the past week, how often did you play with your friends?

14. In the past week, how often did you feel that you had enough friends?



Thank You!

Perceived Stress Scale

The questions in this scale ask you about your feelings and thoughts **during the last month**. In each case, you will be asked to indicate by circling *how often* you felt or thought a certain way.

Name _____ Date _____

Age _____ Gender (*Circle*): **M F** Other _____

0 = Never 1 = Almost Never 2 = Sometimes 3 = Fairly Often 4 = Very Often

1. In the last month, how often have you been upset because of something that happened unexpectedly?	**0**	**1**	**2**	**3**	**4**
2. In the last month, how often have you felt that you were unable to control the important things in your life?	**0**	**1**	**2**	**3**	**4**
3. In the last month, how often have you felt nervous and "stressed"?	**0**	**1**	**2**	**3**	**4**
4. In the last month, how often have you felt confident about your ability to handle your personal problems?	**0**	**1**	**2**	**3**	**4**
5. In the last month, how often have you felt that things were going your way?	**0**	**1**	**2**	**3**	**4**
6. In the last month, how often have you found that you could not cope with all the things that you had to do?	**0**	**1**	**2**	**3**	**4**
7. In the last month, how often have you been able to control irritations in your life?	**0**	**1**	**2**	**3**	**4**
8. In the last month, how often have you felt that you were on top of things?	**0**	**1**	**2**	**3**	**4**
9. In the last month, how often have you been angered because of things that were outside of your control?	**0**	**1**	**2**	**3**	**4**
10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	**0**	**1**	**2**	**3**	**4**

Please feel free to use the *Perceived Stress Scale* for your research.

ANNEXURE II

CONSENT

PARENT/ GUARDIAN CONSENT FORM (10-17 years**)**

Title of the study: A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS

Name of the Participant:
Name of the Principal investigator: Dr.PAVITHRA.T.R
Name of the Institution: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.
Documentation of the informed consent

I Mr./Mrs. _____F/o or M/o or G/o Ms._____ have been explained in my own understandable language, that my daughter/ward will be included in the study which is **"A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS.**

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained that my daughter's clinical findings, investigations, will be assessed and documented for study purpose
4. I have informed the investigator of all the treatments my daughter has been taking or have taken in the past.
5. I agree for my daughter to cooperate with the investigator and will inform her immediately if my daughter suffers unusual symptoms.
6. My daughter has not participated in any research study at any time .
7. I am aware of the fact that my daughter can opt out of the study at any time without having to give any reason and this will not affect my relation with my doctor or the treatment for my daughter's ailment in this hospital.

8.I have understood that all the details found during the study are kept confidential and while publishing or sharing of the findings, the details will be masked. I hereby give permission to the investigators to release the information obtained from me about my daughter as a result of participation in this study

9. I have decided for my daughter to be included in this research study.
10. I am aware that if I have any question during this study, I should contact my principal investigator mobile number for enquiries.

I was free to ask any questions which have been answered and clarified. I do hereby give my consent for my child to be included as a participant in the study.

By signing this consent form I attest that the information given in this document has been clearly explained to me and apparently understood by me. I will be given a copy of this consent document.

Participant's initials:_____

Name and signature/thumb impression of the guardian

_____ _____ _____

Name Signature Date

Name and signature of witness

_____ _____ _____

Name Signature Date

Name and Signature of the investigator or his representative obtaining consent:

_____ _____ _____

Name Signature Date

ಪೋಷಕ / ಗಾರ್ಡಿಯನ್ ಕನ್ಸೆಂಟ್ ಫಾರ್ಮ್ (10-17 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಹದಿಹರೆಯದ ಬಾಲಕಿಯರ ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳ ಮೇಲೆ ಕಟಾನಿಯಸ್ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್‌ಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ.ಪವಿತ್ರ.ಟಿ.ಆರ್.

ಸಂಸ್ಥೆಯ ಹೆಸರು: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ , ಕೋಲಾರ.

ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆ:

ನಾನು ಶ್ರೀ / ಶ್ರೀ. _____ F / o ಅಥವಾ M / o ಅಥವಾ G / o Ms ._____ ನನ್ನ ಮಗಳು / ವಾರ್ಡ್ ಅನ್ನು "ಹದಿಹರೆಯದ ಬಾಲಕಿಯರ ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳ ಮೇಲಿರುವ ಕಟಾನಿಯಸ್ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್‌ಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುವುದು ಎಂದು ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ,

1. ಈ ಒಪ್ಪಿಗೆಯ ರೂಪ ಮತ್ತು ನನಗೆ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ನಾನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.
2. ನನಗೆ ವಿವರಿಸಿದ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆ ಇದೆ.
3. ನನ್ನ ಮಗಳ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ
4. ನನ್ನ ಮಗಳು ಈ ಹಿಂದೆ ತೆಗೆದುಕೊಳ್ಳುತ್ತಿರುವ ಅಥವಾ ತೆಗೆದುಕೊಂಡ ಎಲ್ಲಾ ಚಿಕಿತ್ಸೆಗಳ ಬಗ್ಗೆ ನಾನು ತನಿಖಾಧಿಕಾರಿಗೆ ತಿಳಿಸಿದ್ದೇನೆ.
5. ನನ್ನ ಮಗಳು ತನಿಖಾಧಿಕಾರಿಯೊಂದಿಗೆ ಸಹಕರಿಸಲು ನಾನು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ನನ್ನ ಮಗಳು ಅಸಾಮಾನ್ಯ ಲಕ್ಷಣಗಳಿಂದ ಬಳಲುತ್ತಿದ್ದರೆ ತಕ್ಷಣ ಅವಳಿಗೆ ತಿಳಿಸುತ್ತೇನೆ.
6. ನನ್ನ ಮಗಳು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿಲ್ಲ.
7. ನನ್ನ ಮಗಳು ಯಾವುದೇ ಕಾರಣವನ್ನು ನೀಡದೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹೊರಗುಳಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ಈ ಆಸ್ಪತ್ರೆಯಲ್ಲಿನ ನನ್ನ ಮಗಳ ಕಾಯಿಲೆಗೆ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ ಎಂಬ ಅಂಶದ ಬಗ್ಗೆ ನನಗೆ ತಿಳಿದಿದೆ.
8. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿದ ಪರಿಣಾಮವಾಗಿ ನನ್ನ ಮಗಳ ಬಗ್ಗೆ ನನ್ನಿಂದ ಪಡೆದ ಮಾಹಿತಿಯನ್ನು ಬಿಡುಗಡೆ ಮಾಡಲು ತನಿಖಾಧಿಕಾರಿಗಳಿಗೆ ನಾನು ಈ ಮೂಲಕ ಅನುಮತಿ ನೀಡುತ್ತೇನೆ
9. ನನ್ನ ಮಗಳನ್ನು ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಬೇಕೆಂದು ನಾನು ನಿರ್ಧರಿಸಿದ್ದೇನೆ.
10. ಈ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನನಗೆ ಯಾವುದೇ ಪ್ರಶ್ನೆ ಇದ್ದರೆ, ವಿಚಾರಣೆಗೆ ನನ್ನ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಯನ್ನು ಸಂಪರ್ಕಿಸಬೇಕು ಎಂದು ನನಗೆ ತಿಳಿದಿದೆ.

ಉತ್ತರಿಸಿದ ಮತ್ತು ಸ್ಪಷ್ಟಪಡಿಸಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನಾನು ಮುಕ್ತನಾಗಿದ್ದೆ. ನನ್ನ ಮಗುವನ್ನು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ನಾನು ಈ ಮೂಲಕ ನೀಡುತ್ತೇನೆ.

ಈ ಒಪ್ಪಿಗೆ ಫಾರ್ಮ್ಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ಈ ಡಾಕ್ಯುಮೆಂಟ್ನಲ್ಲಿ ನೀಡಲಾದ ಮಾಹಿತಿಯನ್ನು ನನಗೆ ಸ್ಪಷ್ಟವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ಸ್ಪಷ್ಟವಾಗಿ ನನಗೆ ಅರ್ಥವಾಗಿದೆ ಎಂದು ನಾನು ಧೃಡೀಕರಿಸುತ್ತೇನೆ. ಈ ಒಪ್ಪಿಗೆ ದಾಖಲೆಯ ನಕಲನ್ನು ನನಗೆ ನೀಡಲಾಗುವುದು.

ಭಾಗವಹಿಸುವವರ ಮೊದಲಕ್ಷರಗಳು: _____

ಪೋಷಕರ ಹೆಸರು ಮತ್ತು ಸಹಿ / ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ

_____ _____ _____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ಸಾಕ್ಷಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

_____ _____ _____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ತನಿಖಾಧಿಕಾರಿ ಅಥವಾ ಅವರ ಪ್ರತಿನಿಧಿಯ ಒಪ್ಪಿಗೆ ಪಡೆಯುವ ಹೆಸರು ಮತ್ತು ಸಹಿ:

_____ _____ _____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ

ASSENT CONSENT FORM (12-17 years)

Title of the study: A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS

Name of the Participant:
Name of the Principal investigator: Dr.PAVITHRA.T.R
Name of the Institution: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

Documentation of the informed consent

I Ms./Mrs._____ have been explained in my own understandable language, that I will be included in the study which is **"A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS"**

I have been explained that my clinical findings, investigations will be assessed and documented for study purpose.

I have been explained that my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I have principal investigator's mobile number for enquires.

I, in my sound mind give full consent to be added in the part of this study.

Participant's initials:_____

Name and signature/thumb impression of the patient

_____ _____ _____

Name Signature Date

Name and signature of witness

_____ _____ _____

Name Signature Date

Name and Signature of the investigator or his representative obtaining consent:

_____ _____ _____

Name Signature Date

ಒಪ್ಪಿಗೆ ಪತ್ರ (12-17 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ:

ಹದಿಹರೆಯದ ಬಾಲಕಿಯರ ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳ ಮೇಲೆ ಕಟಾನಿಯಸ್ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್‌ಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ.ಪವಿತ್ರ.ಟಿ.ಆರ್.

ಸಂಸ್ಥೆಯ ಹೆಸರು: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ , ಕೋಲಾರ.

ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆ

ನಾನು ಶ್ರೀ / ಶ್ರೀ. _____ ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಇದು "ಹದಿಹರೆಯದ ಹುಡುಗಿಯರಲ್ಲಿ ಪೀರಿಯೊರಿಫಿಶಿಯಲ್ ಪ್ರದೇಶಗಳಲ್ಲಿ ಚರ್ಮದ ಅಭಿವ್ಯಕ್ತಿಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಸ್ಟಡಿ" ಎಂಬ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಿಕೊಳ್ಳಲಾಗುವುದು.

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದು ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆ ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಗೆ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ವಿಚಾರಣೆಗಾಗಿ ನನ್ನ ಬಳಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ ಇದೆ.

ನಾನು, ನನ್ನ ಉತ್ತಮ ಮನಸ್ಸಿನಲ್ಲಿ ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಮೊದಲಕ್ಷರಗಳು: _____

ರೋಗಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ / ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ

_____	_____	_____
ಹೆಸರು	ಸಹಿ	ದಿನಾಂಕ

ಸಾಕ್ಷಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

_____	_____	_____
ಹೆಸರು	ಸಹಿ	ದಿನಾಂಕ

ತನಿಖಾಧಿಕಾರಿ ಅಥವಾ ಅವರ ಪ್ರತಿನಿಧಿಯ ಒಪ್ಪಿಗೆ ಪಡೆಯುವ ಹೆಸರು ಮತ್ತು ಸಹಿ:

_____	_____	_____
ಹೆಸರು	ಸಹಿ	ದಿನಾಂಕ

PATIENT CONSENT FORM (18 & 19 years)

Title of the study: A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS

Name of the Participant:
Name of the Principal investigator: Dr.PAVITHRA.T.R
Name of the Institution: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

Documentation of the informed consent

I Ms./Mrs. _____ have been explained in my own understandable language, that I will be included in the study which is **"A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS.**

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained that my clinical findings, investigations, will be assessed and documented for study purpose
4. I have informed the investigator of all the treatments I have been taking or have taken in the past.
5. I agree to cooperate with the investigator and will inform her immediately if I suffer unusual symptoms.
6. I have not participated in any research study at any time .
7. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my relation with my doctor or the treatment for my ailment in this hospital.

8.I have understood that all the details found during the study are kept confidential and while publishing or sharing of the findings, the details will be masked. I hereby give permission to the investigators to release the information obtained from me as a result of participation in this study

9. I have decided to be included in this research study.
10. I am aware that if I have any question during this study, I should contact my principal investigator mobile number for enquiries.

I _____ was free to ask any questions which were answered and clarified , I do hereby give my consent to be included as a participant in the study.

By signing this consent form I attest that the information given in this document has been clearly explained to me and apparently understood by me. I will be given a copy of this consent document.

Participant's initials:_____

Name and signature/thumb impression of the participant

_____ _____ _____

Name Signature Date

Name and signature of witness

_____ _____ _____

Name Signature Date

Name and Signature of the investigator or his representative obtaining consent:

_____ _____ _____

Name Signature Date

ರೋಗಿಯ ಕನ್ಸೆಂಟ್ ಫಾರ್ಮ್ (18 ಮತ್ತು 19 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಹದಿಹರೆಯದ ಬಾಲಕಿಯರ ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳ ಮೇಲೆ ಕಟಾನಿಯಸ್ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್‌ಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ.ಪವಿತ್ರ.ಟಿ.ಆರ್.

ಸಂಸ್ಥೆಯ ಹೆಸರು: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ , ಕೋಲಾರ.

ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆ

ನಾನು ಶ್ರೀ / ಶ್ರೀ. _____ ನನ್ನದೇ ಆದ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, "ಹದಿಹರೆಯದ ಹುಡುಗಿಯರಲ್ಲಿ ಆವರ್ತಕ ಪ್ರದೇಶಗಳಲ್ಲಿನ ಚರ್ಮದ ಅಭಿವ್ಯಕ್ತಿಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಸ್ಟಡಿ" ಎಂಬ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಿಕೊಳ್ಳಲಾಗುವುದು.

1. ಈ ಒಪ್ಪಿಗೆಯ ರೂಪ ಮತ್ತು ನನಗೆ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ನಾನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.
2. ನನಗೆ ವಿವರಿಸಿದ ಒಪ್ಪಿಗೆಯ ದಾಖಲೆ ಇದೆ.
3. ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳು ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲ್ಪಡುತ್ತವೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ
4. ನಾನು ಈ ಹಿಂದೆ ತೆಗೆದುಕೊಳ್ಳುತ್ತಿರುವ ಅಥವಾ ತೆಗೆದುಕೊಂಡ ಎಲ್ಲಾ ಚಿಕಿತ್ಸೆಗಳ ಬಗ್ಗೆ ತನಿಖಾಧಿಕಾರಿಗೆ ತಿಳಿಸಿದ್ದೇನೆ.
5. ತನಿಖಾಧಿಕಾರಿಯೊಂದಿಗೆ ಸಹಕರಿಸಲು ನಾನು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ನಾನು ಅಸಾಮಾನ್ಯ ರೋಗಲಕ್ಷಣಗಳನ್ನು ಅನುಭವಿಸಿದರೆ ತಕ್ಷಣ ಅವರಿಗೆ ತಿಳಿಸುತ್ತೇನೆ.
6. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿಲ್ಲ.
7. ಯಾವುದೇ ಕಾರಣವನ್ನು ನೀಡದೆ ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹೊರಗುಳಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ಈ ಆಸ್ಪತ್ರೆಯಲ್ಲಿನ ನನ್ನ ಕಾಯಿಲೆಗೆ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ ಎಂಬ ಅಂಶದ ಬಗ್ಗೆ ನನಗೆ ತಿಳಿದಿದೆ.
8. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿದ ಪರಿಣಾಮವಾಗಿ ನನ್ನಿಂದ ಪಡೆದ ಮಾಹಿತಿಯನ್ನು ಬಿಡುಗಡೆ ಮಾಡಲು ತನಿಖಾಧಿಕಾರಿಗಳಿಗೆ ನಾನು ಈ ಮೂಲಕ ಅನುಮತಿ ನೀಡುತ್ತೇನೆ
9. ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲು ನಾನು ನಿರ್ಧರಿಸಿದ್ದೇನೆ.
10. ಈ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನನಗೆ ಯಾವುದೇ ಪ್ರಶ್ನೆ ಇದ್ದರೆ, ವಿಚಾರಣೆಗೆ ನನ್ನ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಯನ್ನು ಸಂಪರ್ಕಿಸಬೇಕು ಎಂದು ನನಗೆ ತಿಳಿದಿದೆ.

ನಾನು _____ ಉತ್ತರಿಸಿದ ಮತ್ತು ಸ್ಪಷ್ಟಪಡಿಸಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಮುಕ್ತನಾಗಿದ್ದೆ, ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವನಾಗಿ ಸೇರಿಸಲು ನಾನು ಈ ಮೂಲಕ ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ಈ ಒಪ್ಪಿಗೆ ಫಾರ್ಮ್ಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ಈ ಡಾಕ್ಯುಮೆಂಟ್ನಲ್ಲಿ ನೀಡಲಾದ ಮಾಹಿತಿಯನ್ನು ನನಗೆ ಸ್ಪಷ್ಟವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ಸ್ಪಷ್ಟವಾಗಿ ನನಗೆ ಅರ್ಥವಾಗಿದೆ ಎಂದು ನಾನು ದೃ est ೀಕರಿಸುತ್ತೇನೆ. ಈ ಒಪ್ಪಿಗೆ ದಾಖಲೆಯ ನಕಲನ್ನು ನನಗೆ ನೀಡಲಾಗುವುದು.

ಭಾಗವಹಿಸುವವರ ಮೊದಲಕ್ಷರಗಳು:

ಭಾಗವಹಿಸುವವರ ಹೆಸರು ಮತ್ತು ಸಹಿ / ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ.

_____ _____ _____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ.

ಸಾಕ್ಷಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

_____ _____ _____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ.

ತನಿಖಾಧಿಕಾರಿ ಅಥವಾ ಅವರ ಪ್ರತಿನಿಧಿಯ ಒಪ್ಪಿಗೆ ಪಡೆಯುವ ಹೆಸರು ಮತ್ತು ಸಹಿ:

_____ _____ _____

ಹೆಸರು ಸಹಿ ದಿನಾಂಕ.

ANNEXURE III

INFORMATION SHEET

PARENT/ GUARDIAN INFORMATION SHEET (10-17 years)

Study title : A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS

Study site: R.L Jalappa Hospital and research centre, Tamaka, Kolar.

Aim: **To study the pattern and severity of various cutaneous manifestations over periorificial areas in adolescents girls**.

Skin disorders are common among adolescent girls. The psychological impact these skin disorder can have on an adolescent girls is highly variable and unpredictable. The Cutaneous manifestations over periorificial areas may present as acne, dermatitis, atopic eczema, herpes infection, molluscum contagiosum, papulosquamous disorders, pigmentary disorders, appendageal disorder, urticaria/ dermographism , keloid, papular urticaria, scabies, dermatophytosis , contact dermatitis and verrucae.

Periorificial cutaneous manifestations can be diagnosed by clinical examination and biopsy in doubtful cases.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree for your daughter/ward to participate in this study we will collect information (as per proforma) from you and your daughter. Relevant blood investigations will be carried out if required. This information collected will be used for dissertation and publication only.

All information collected from you regarding your daughter/ward will be kept confidential and will not be disclosed to any outsider. Your daughter's/ward's identity will not be revealed. The expenses required for the investigations will be funded by the study investigator. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care your daughter will get will not change if you don't wish to participate.

You are required to sign/ provide thumb impression only if you voluntarily agree for your daughter to participate in this study.

For any further clarification you can contact the study investigator:

Dr. Pavithra.T.R

Mobile no: 9600093777

E-mail id: pavithratr21@gmail.com

<u>ಪೋಷಕ / ಗಾರ್ಡಿಯನ್ ಮಾಹಿತಿ ಹಾಳೆ (10-17 ವರ್ಷಗಳು)</u>

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಹದಿಹರೆಯದ ಬಾಲಕಿಯರ ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳ ಮೇಲೆ ಕಟಾನಿಯಸ್ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್‌ಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನ.

ಅಧ್ಯಯನ ಸೈಟ್: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ , ಕೋಲಾರ.

ಗುರಿ: ಹದಿಹರೆಯದ ಬಾಲಕಿಯರಲ್ಲಿ ಬಾಹ್ಯ ಪ್ರದೇಶಗಳ ಮೇಲೆ ವಿವಿಧ ಕಟುವಾದ ಅಭಿವ್ಯಕ್ತಿಗಳ ಮಾದರಿ ಮತ್ತು ತೀವ್ರತೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡುವುದು.

ಹದಿಹರೆಯದ ಹುಡುಗಿಯರಲ್ಲಿ ಚರ್ಮದ ಕಾಯಿಲೆಗಳು ಸಾಮಾನ್ಯವಾಗಿದೆ. ಈ ಚರ್ಮದ ಅಸ್ವಸ್ಥತೆಯು ಹದಿಹರೆಯದ ಹುಡುಗಿಯರ ಮೇಲೆ ಉಂಟುಮಾಡುವ ಮಾನಸಿಕ ಪರಿಣಾಮವು ಹೆಚ್ಚು ವ್ಯತ್ಯಾಸಗೊಳ್ಳುತ್ತದೆ ಮತ್ತು ಅನಿರೀಕ್ಷಿತವಾಗಿದೆ. ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳಲ್ಲಿನ ಕ್ಯುಟೇನಿಯಸ್ ಅಭಿವ್ಯಕ್ತಿಗಳು ಮೊಡವೆ, ಡರ್ಮಟೈಟಿಸ್, ಅಟೊಪಿಕ್ ಎಸ್ಜಿಮಾ, ಹರ್ಪಿಸ್ ಸೋಂಕು, ಮೃದ್ವಂಗಿ ಕಾಂಟ್ಯಾಜಿಯೊಸಮ್, ಪಾಪುಲೋಸ್ಕ್ವಾಮಸ್ ಅಸ್ವಸ್ಥತೆಗಳು, ವರ್ಣದ್ರವ್ಯದ ಅಸ್ವಸ್ಥತೆಗಳು, ಅಪೆಂಡೇಜಿಯಲ್ ಡಿಸಾರ್ಡರ್, ಉರ್ಟೇರಿಯಾ / ಡರ್ಮೋಗ್ರಾಫಿಸಮ್, ಕೆಲಾಯ್ಡ್, ಪಾಪ್ಯುಲರ್ ಉರ್ಟೇರಿಯಾ, ಸ್ಕ್ಯಾಟೈಟಿಸ್, ಡರ್ಮಟೊಫೈಟಿಸ್‌ಅನುಮಾನಾಸ್ಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ಬಯಾಪ್ಸಿ ಮೂಲಕ ಪೆರಿಯೊರಿಫಿಕಲ್ ಕಟಾನಿಯಸ್ ಅಭಿವ್ಯಕ್ತಿಗಳನ್ನು ಕಂಡುಹಿಡಿಯಬಹುದು.

ದಯವಿಟ್ಟು ಈ ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನಿಮ್ಮ ಮಗಳು / ವಾರ್ಡ್ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಮತ್ತು ನಿಮ್ಮ ಮಗಳಿಂದ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ (ಪ್ರೊಫಾರ್ಮಾದ ಪ್ರಕಾರ). ಅಗತ್ಯವಿದ್ದರೆ ಸಂಬಂಧಿತ ರಕ್ತ ತನಿಖೆ ನಡೆಸಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮ ಮಗಳು / ವಾರ್ಡ್‌ಗೆ ಸಂಬಂಧಿಸಿದಂತೆ ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮಗಳ / ವಾರ್ಡ್‌ನ ಗುರುತು ಬಹಿರಂಗಗೊಳ್ಳುವುದಿಲ್ಲ. ತನಿಖೆಗೆ ಅಗತ್ಯವಾದ ಖರ್ಚುಗಳನ್ನು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಗಳು ನೀಡುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯ

ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನಿಮ್ಮ ಮಗಳಿಗೆ ಸಿಗುವ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮಗಳು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆಗೆ ಸಹಿ / ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

ಡಾ.ಪವಿತ್ರ.ಟಿ.ಆರ್

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 9600093777

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ASSENT INFORMATION SHEET (12-17 years)

Study Title : A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS

Study site: R.L Jalappa Hospital and research centre, Tamaka, Kolar.

Aim: To study the pattern and severity of various cutaneous manifestations over periorificial areas in adolescents girls.

Skin disorders are common among adolescent girls. The psychological impact these skin disorder can have on an adolescent girls is highly variable and unpredictable. The Cutaneous manifestations over periorificial areas may present as acne, dermatitis, atopic eczema, herpes infection, molluscum contagiosum, papulosquamous disorders, pigmentary disorders, appendageal disorder, urticaria/ dermographism , keloid, papular urticaria, scabies, dermatophytosis , contact dermatitis and verrucae.

Periorificial cutaneous manifestations can be diagnosed by clinical examination and biopsy in doubtful cases.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study we will collect information (as per proforma) from you. Relevant blood investigations will be carried out if required. This information collected will be used for dissertation and publication only.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. The expenses required for the investigations will be funded by the study investigator. This study has been reviewed by the Institutional Ethics Committee and you are free

to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care that you get will not change even if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

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<u>ಅಸೆಂಟ್ ಮಾಹಿತಿ ಹಾಳೆ (12-17 ವರ್ಷಗಳು)</u>

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಹದಿಹರೆಯದ ಬಾಲಕಿಯರ ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳ ಮೇಲೆ ಕಟಾನಿಯಸ್ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್‌ಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನ.

<u>ಅಧ್ಯಯನ ಸೈಟ್</u>: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ , ಕೋಲಾರ.

<u>ಗುರಿ:</u> ಹದಿಹರೆಯದ ಹುಡುಗಿಯರಲ್ಲಿ ಬಾಹ್ಯ ಪ್ರದೇಶಗಳ ಮೇಲೆ ವಿವಿಧ ಕಟುವಾದ ಅಭಿವ್ಯಕ್ತಿಗಳ ಮಾದರಿ ಮತ್ತು ತೀವ್ರತೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡುವುದು.

ಹದಿಹರೆಯದ ಹುಡುಗಿಯರಲ್ಲಿ ಚರ್ಮದ ಕಾಯಿಲೆಗಳು ಸಾಮಾನ್ಯವಾಗಿದೆ. ಈ ಚರ್ಮದ ಅಸ್ವಸ್ಥತೆಯು ಹದಿಹರೆಯದ ಹುಡುಗಿಯರ ಮೇಲೆ ಉಂಟುಮಾಡುವ ಮಾನಸಿಕ ಪರಿಣಾಮವು ಹೆಚ್ಚು ವ್ಯತ್ಯಾಸಗೊಳ್ಳುತ್ತದೆ ಮತ್ತು ಅನಿರೀಕ್ಷಿತವಾಗಿದೆ. ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳಲ್ಲಿನ ಕ್ಯುಟೇನಿಯಸ್ ಅಭಿವ್ಯಕ್ತಿಗಳು ಮೊಡವೆ, ಡರ್ಮಟೈಟಿಸ್, ಅಟೊಪಿಕ್ ಎಸ್ಜಿಮಾ, ಹರ್ಪಿಸ್ ಸೋಂಕು, ಮೃದ್ವಂಗಿ ಕಾಂಟ್ಯಾಜಿಯೊಸಮ್, ಪಾಪುಲೋಸ್ಕ್ವಾಮಸ್ ಅಸ್ವಸ್ಥತೆಗಳು, ವರ್ಣದ್ರವ್ಯದ ಅಸ್ವಸ್ಥತೆಗಳು, ಅಪೆಂಡೇಜಿಯಲ್ ಡಿಸಾರ್ಡರ್, ಉರ್ಟೇರಿಯಾ / ಡರ್ಮೋಗ್ರಾಫಿಸಮ್, ಕೆಲಾಯ್ಡ್, ಪಾಪ್ಯುಲರ್ ಉರ್ಟೇರಿಯಾ, ಸ್ಕ್ಯಾಟೈಟಿಸ್, ಡರ್ಮಟೊಫೈಟಿಸ್ ಅನುಮಾನಾಸ್ಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ಬಯಾಪ್ಸಿ ಮೂಲಕ ಪೆರಿಯೊರಿಫಿಕಲ್ ಕಟಾನಿಯಸ್ ಅಭಿವ್ಯಕ್ತಿಗಳನ್ನು ಕಂಡುಹಿಡಿಯಬಹುದು.

ದಯವಿಟ್ಟು ಈ ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ (ಪ್ರೊಫಾರ್ಮಾದ ಪ್ರಕಾರ). ಅಗತ್ಯವಿದ್ದರೆ ಸಂಬಂಧಿತ ರಕ್ತ ತನಿಖೆ ನಡೆಸಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತು ಬಹಿರಂಗಗೊಳ್ಳುವುದಿಲ್ಲ. ತನಿಖೆಗೆ ಅಗತ್ಯವಾದ ಖರ್ಚುಗಳನ್ನು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಗಳು ನೀಡುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು

ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಇಚ್ಛಿದಿದ್ದರೂ ಸಹ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿ ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ ಸಹಿ / ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

ಡಾ.ಪವಿತ್ರ.ಟಿ.ಆರ್
ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 9600093777
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PATIENT INFORMATION SHEET (18-19 years)

Study title : A CLINICO EPIDEMIOLOGICAL STUDY OF CUTANEOUS MANIFESTATIONS OVER THE PERIORIFICIAL AREAS IN ADOLESCENT GIRLS

Study site: R.L Jalappa Hospital and research centre, Tamaka, Kolar.

Aim: **To study the pattern and severity of various cutaneous manifestations over periorificial areas in adolescents girls**.

Skin disorders are common among adolescent girls. The psychological impact these skin disorder can have on an adolescent girls is highly variable and unpredictable. The Cutaneous manifestations over periorificial areas may present as acne, dermatitis, atopic eczema, herpes infection, molluscum contagiosum, papulosquamous disorders, pigmentary disorders, appendageal disorder, urticaria/ dermographism , keloid, papular urticaria, scabies, dermatophytosis , contact dermatitis and verrucae.

Periorificial cutaneous manifestations can be diagnosed by clinical examination and biopsy in doubtful cases.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study we will collect information (as per proforma) from you. Relevant blood investigations will be carried out if required. This information collected will be used for dissertation and publication only.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. The expenses required for the investigations will be funded by the study investigator. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no

compulsion to agree to this study. The care that you get will not change even if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

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ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ (18-19 ವರ್ಷಗಳು)

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಹದಿಹರೆಯದ ಬಾಲಕಿಯರ ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳ ಮೇಲೆ ಕಟಾನಿಯಸ್ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್‌ಗಳ ಕ್ಲಿನಿಕೊ ಎಪಿಡೆಮಿಯೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನ

ಅಧ್ಯಯನ ಸೈಟ್: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ , ಕೋಲಾರ.

ಗುರಿ: ಹದಿಹರೆಯದ ಹುಡುಗಿಯರಲ್ಲಿ ಬಾಹ್ಯ ಪ್ರದೇಶಗಳ ಮೇಲೆ ವಿವಿಧ ಕಟುವಾದ ಅಭಿವ್ಯಕ್ತಿಗಳ ಮಾದರಿ ಮತ್ತು ತೀವ್ರತೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡುವುದು.

ಹದಿಹರೆಯದ ಹುಡುಗಿಯರಲ್ಲಿ ಚರ್ಮದ ಕಾಯಿಲೆಗಳು ಸಾಮಾನ್ಯವಾಗಿದೆ. ಈ ಚರ್ಮದ ಅಸ್ವಸ್ಥತೆಯು ಹದಿಹರೆಯದ ಹುಡುಗಿಯರ ಮೇಲೆ ಉಂಟುಮಾಡುವ ಮಾನಸಿಕ ಪರಿಣಾಮವು ಹೆಚ್ಚು ವ್ಯತ್ಯಾಸಗೊಳ್ಳುತ್ತದೆ ಮತ್ತು ಅನಿರೀಕ್ಷಿತವಾಗಿದೆ. ಪೆರಿಯೊರಿಫಿಕಲ್ ಪ್ರದೇಶಗಳಲ್ಲಿನ ಕ್ಯುಟೇನಿಯಸ್ ಅಭಿವ್ಯಕ್ತಿಗಳು ಮೊಡವೆ, ಡರ್ಮಟೈಟಿಸ್, ಅಟೊಪಿಕ್ ಎಸ್ಜಿಮಾ, ಹರ್ಪಿಸ್ ಸೋಂಕು, ಮೃದ್ವಂಗಿ ಕಾಂಟ್ಯಾಜಿಯೊಸಮ್, ಪಾಪುಲೋಸ್ಕ್ವಾಮಸ್ ಅಸ್ವಸ್ಥತೆಗಳು, ವರ್ಣದ್ರವ್ಯದ ಅಸ್ವಸ್ಥತೆಗಳು, ಅಪೆಂಡೇಜಿಯಲ್ ಡಿಸಾರ್ಡರ್, ಉರ್ಟೇರಿಯಾ / ಡರ್ಮೋಗ್ರಾಫಿಸಮ್, ಕೆಲಾಯ್ಡ್, ಪಾಪ್ಯುಲರ್ ಉರ್ಟೇರಿಯಾ, ಸ್ಕ್ಯಾಟೈಟಿಸ್, ಡರ್ಮಟೊಫೈಟಿಸ್ ಅನುಮಾನಾಸ್ಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ಬಯಾಪ್ಸಿ ಮೂಲಕ ಪೆರಿಯೊರಿಫಿಕಲ್ ಕಟಾನಿಯಸ್ ಅಭಿವ್ಯಕ್ತಿಗಳನ್ನು ಕಂಡುಹಿಡಿಯಬಹುದು.

ದಯವಿಟ್ಟು ಈ ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ (ಪ್ರೊಫಾರ್ಮಾದ ಪ್ರಕಾರ). ಅಗತ್ಯವಿದ್ದರೆ ಸಂಬಂಧಿತ ರಕ್ತ ತನಿಖೆ ನಡೆಸಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತು ಬಹಿರಂಗಗೊಳ್ಳುವುದಿಲ್ಲ. ತನಿಖೆಗೆ ಅಗತ್ಯವಾದ ಖರ್ಚುಗಳನ್ನು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಗಳು ನೀಡುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಇಚ್ಛಿಸದಿದ್ದರೂ ಸಹ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿ

ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ ಸಹಿ / ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

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ANNEXURE IV

KEY TO MASTERCHART

HEADINGS:

1. DIAG – Diagnosis
2. SYM – Symptoms
3. LM – Lesion morphology
4. SV – Seasonal variation
5. FH – Family history
6. MOD – Moderate
7. SEV – Severe
8. COS – Cosmetics
9. ST – Steroid

DIAGNOSIS:

1. PA – Pityriasis Alba
2. PIH – Post Inflammatory Hyperpigmentation
3. PMLE – Polymorphic Light eruption
4. POcD – Periocular dermatitis
5. POcM – Periocular melanosis
6. POD – Perioral dermatitis
7. POM – Perioral melanosis
8. PTS – Post traumatic scar
9. SCAB – Scabies
10. SD – Seborrheic dermatitis
11. TC – Tinea cruris
12. TF – Tinea Facei
13. VIT – Vitiligo
14. XP – Xanthelasma palpebrarum
15. AV – Acne Vulgaris
16. DPN – Dermatosis papulosa nigra

17. AC – Angular chelitis
18. AS – Acne scar
19. CD – Contact Dermatitis
20. FOL – Folliculitis
21. FRE – Freckles
22. HL – Herpes Labialis
23. IMP – Impetigo
24. KLD – Keloid
25. LPP – Lichen planus pigmentosus
26. MC – Molluscum contagiosum

SYMPTOMS :

1. A- Asymptomatic
2. B- Burning
3. I- Itching
4. P- Pain
5. R- Redness

LESION MORPHOLOGY:

1. PA – Papule
2. PL – Plaque
3. PU – Pustule
4. C – Comedone
5. EX – Excoriation
6. F – Fissure
7. M – Macule
8. N – Nodule
9. PAT – Patch
10. S – Scar
11. Hyper – Hyperpigmentation
12. E – Erosion
13. SC – Scales

14. V – Vesicle
15. CR – Crusting

SEASONAL VARIATION:

1. SUM- Summer
2. WIN- Winter

SITE:

1. POC – Periocular
2. PN – Perinasal
3. PA – Periaural
4. PO – Perioral
5. PAN – Perianal