"A CLINICO EPIDEMIOLOGICAL STUDY OF VARIOUS DERMATOSES IN ADOLESCENT GIRLS"

By

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Under the Guidance of

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"A CLINICO EPIDEMIOLOGICAL STUDY OF VARIOUS DERMATOSES IN ADOLESCENT GIRLS"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. T. S. RAJASHEKAR** , Professor and Head of the department, Department of Dermatology, Venereology and Leprosy, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award "**M.D. IN DERMATOLOGY, VENEREOLOGY AND LEPROSY**", the examination to be held in May 2017 by SDUAHER. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

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LIST OF ABBREVIATIONS USED

PCOS: Polycystic ovarian disease.

5-FU: 5-fluorouracil.

TE: Telogen effluvium.

AA: Alopecia areata.

AGA: Androgenetic alopecia.

FDA: Food and Drug association.

TTM: Trichotillomania.

IH: Idiopathic hirsuitism.

PH: Primary hyperhidrosis.

KOH: Potassium hydroxide.

DLSO: Distal lateral subungual onychomycosis

PSO: Proximal subungual onychomycosis.

WSO: White superficial onychomycosis.

HSV: Herpes simplex virus.

UVB: Ultraviolet B.

AD: Atopic dermatitis.

ICD: Irritant contact dermatitis.

ACD: Allergic contact dermatitis.

SD: Seborrheic dermatitis.

PMLE: Polymorphic light eruptions.

LP: Lichen planus.

PUVA: Psoralens plus ultraviolet A.

POHM: Peri ocular hypermelanosis.

PIH: Post inflammatory hyperpigmentation.

STI: Sexually transmitted infection.

ABSTRACT

BACKGROUND:

Adolescence is associated with myriad of hormonal changes in the body which may result in skin disorders. Adolescent self-esteem can be easily threatened when a highly visible skin disorder becomes a focus for unwelcome peer attention.

Skin diseases are an important reason for consultation especially in young girls because they are more conscious of their body complexion, physical well-being and outlook.

Since there is paucity of dermato-epidemiological studies exclusively in girls of this age group, we aim to heighten the clinical awareness on various dermatoses in adolescent girls.

AIMS AND OBJECTIVES:

-To study the pattern and severity of different skin lesions in adolescent girls among general population.

-To differentiate the pattern and severity of skin lesions in adolescent girls of both rural and urban population.

-To study the demographic profile of each dermatoses in them.

MATERIALS AND METHODS:

It was a descriptive study done from January 2015 to July 2016 for a period of one and half years in 231 female patients who attended the outpatient department of Dermatology, Venereology and Leprosy in R.L.Jalapppa hospital, Tamaka, Kolar.

A detailed history of the patient was taken including name, age, nature and duration of illness, predisposing factors like drug intake, topical application of medicines and cosmetics etc. Photographs were taken concealing the individual's identity, for documentation.

Various investigations were carried out whenever required. Informed consent was taken for all patients.

RESULTS:

Out of 231 patients, most common dermatoses were appendageal disorders (n=70,30.3%) followed by infections (n=52,22.51%), eczemas (n=43,18.6%), pigmentary disorders (n=23,9.96%), papulosquamous disorders (n=15,6.49%) and miscellaneous dermatoses (n=28,12.12%). The most common age group being affected is the late adolescent group (39.39%) followed by the middle (33.33%) and early adolescent age group (27.27%). Dermatological diseases were commonly observed in urban population (62.77%) than rural population (37.23%).

CONCLUSION:

The findings of this study highlight the most predominant skin diseases,to be acne, infections and eczema which may incur significant morbidity in the affected individuals Apart from having general health importance, these disorders if not properly managed, can lead to significant cosmetic disfigurement thereby leading to psychological distress in the young adolescent minds. Hence, there is a need to monitor the epidemiology of common skin problems in children and adolescents so that relevant preventive measures can be planned and implemented effectively

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INTRODUCTION:

The World Health Organisation defines adolescence as the age between 10 and 19 years. Adolescence is a critical phase of life when many young people experience difficulties associated with transition from a structured family setting to uncertainties of adult world. During this period, a myriad of hormonal changes occurs in the body to which the skin reacts in various ways. Adolescent self-esteem can be easily threatened when a highly visible skin disorder becomes a focus for unwelcome peer attention[1].

Adolescents are susceptible to a variety of dermatoses, many of which occur due to physiological changes encountered during puberty. They are more prone to developing skin diseases than adults because of excessive exposure to climatic and social conditions. The school environment also makes them more vulnerable to cross-transmission of communicable skin diseases because of their excessive involvement in activities that involve higher interpersonal contact.[1]

Skin disorders are an important reason for consultation, especially in adolescent girls, as they are more conscious of their body complexion, physical well-being and outlook.[2] Some of the physiological differences between male and female skin can be attributed to different levels of sex and stress hormones.[3] It has been pointed out that, for a girl, the pressures of coping with a maturing skin are particularly acute. Her skin should be free from grease, acne and moreover, should be odourless. Puberty makes this ideal image virtually impossible to achieve[4].

The impact of dermatological conditions on the quality of life of adults is well documented, relatively little is known about this relationship in adolescents. This could be

because adolescence is a stage of development that has only recently begun to receive empirical attention. But it is hypothesized that the psychological impact of skin disorders would be greater among adolescents than adults due to the critical role the teenage years play in the development of an individual's self-identity, a portion of which is determined by the self-perception of one's physical attractiveness.[5]

The impact of skin diseases can be enormous because of the visibility of skin diseases and the psychological vulnerability of the young adolescent minds.[1]

An attractive appearance is essential for feminine gender and the search for beauty can cause special adjustment problems especially in adolescent girls. Psychologically they can suffer from negative body image, lowered self-esteem and achievement conflict.[6]

The burden of skin disease in absolute terms is said to be the product of its morbidity multiplied by its prevalence.[1] This burden is virtually unknown in adolescents, due to the paucity of dermato epidemiological studies exclusively in this age group.

Epidemiological data are essential to determine the size and importance of a health problem and define priorities with a view to offering solutions. Hence, with this study, we aim to heighten the clinical awareness on various dermatoses in adolescent girls.

OBJECTIVES

OBJECTIVES:

- To study the pattern and severity of different skin lesions in adolescent girls among general population.
- To differentiate the pattern and severity of skin lesions in adolescent girls of both rural and urban population.
- To study the demographic profile of each dermatosis in them.

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

The World Health Organisation defines adolescence as the period of life between the age 10 and 19 years.[1] Adolescence is a difficult period for most people. The physiological changes which occur in the skin during puberty and adolescence have several effects which may result in sufficient distress to cause the individual to seek medical advice.[4]

The period of adolescence is associated with gross somatic and psychological changes in the body. The hormonal changes around the time of puberty lead to growth and development of sebaceous gland, increased sebum production, development of apocrine glands, growth of axillary and pubic hair, appearance of hair in male pattern, thinning of scalp hair, increased distribution of terminal hair in androgen sensitive area and acne. These disorders occur with increased frequency and severity at this stage of life, combined with other dermatosis which can result from constitutional and exogenous factors such as eczema and infections make these patients more vulnerable to psychological upset. Increase consciousness of youth of their body and beauty further aggravates their anxiety.[7]

Diseases affecting adolescents commonly include the following:

1. Appendageal disorders – acne, alopecia areata, telogen effluvium, diffuse hair loss, hyperhidrosis.
2. Infections – pityriasis versicolor, scabies, dermatophytosis, viral warts, molluscum contagiosum, Pityriasis rosea.
3. Eczemas – seborrheic dermatitis, contact dermatitis, atopic dermatitis, dyshidrotic eczema, nonspecific eczemas, polymorphic light eruptions.
4. Pigmentary disorders – vitiligo, periorbital melanosis, fixed drug eruption.

5. Papulosquamous disorders – keratosis pilaris, psoriasis vulgaris, lichen planus.
6. Miscellaneous –phrynoderma, keloids and hypertrophic scars, urticaria.[1,8]

Here we review the common adolescent dermatoses with their salient features.

1)APPENDAGEAL DISORDERS:

Acne:

Introduction: Acne vulgaris is one of the most common skin diseases affecting adolescents, having a prevalence rate of 70–87%.[9] Severe diseases can leave permanent scarring and there by lead to psychological consequences.[9]

Pathogenesis: Clinically, adolescent acne consists of a combination of non-inflammatory (open and closed comedones) and inflammatory (papules, pustules, and nodules) lesions.[9]

Acne is a disease of the pilosebaceous unit with a complex pathophysiology.

Four main pathogenic factors are known to lead to the development of acne:[10]

1) Follicular epidermal hyperproliferation.

2) Excess sebum production.

(3) Inflammation

(4) Presence and activity of Propionibacterium acnes.

As the puberty sets in, there is increased sebum production as a result of androgen-mediated stimulation of the sebaceous gland. Follicular hyperkeratinization and increased keratinocyte adhesion subsequently result in follicular plugging and comedone formation.

These events combine to create an environment favourable for colonization by the gram-positive bacteria, Propionibacterium acnes (P. acnes).[10]

Role of androgens: Androgen receptors are expressed in the basal layer of sebaceous glands and in the outer root sheath keratinocytes of the hair follicle. Hence excess androgen production is implicated in both exaggerated sebum production as well as follicular hyperkeratinisation.[10]

Role of P. acnes: A normal anaerobic resident of the skin surface, colonizes the occluded pilosebaceous follicles and breaks down sebum to free fatty acids and peptides eliciting an inflammatory response leading to formation of papules, pustules, and nodules.[10]

Recent molecular genetic studies: 1) Complete sequencing of the P. acnes genome showed that the bacterium produces lipases that breakdown sebum, heat-shock proteins (lead to cytokine production and inflammation), and enzymes to produce porphyrins that induce neutrophil chemokine production and accelerated oxidation of squalene in sebum for comedogenesis.[10]

2) It has also shown that the bacteria may express genes for polysaccharides and lipoglycan which may provide a protective barrier (biofilm) against host defences.[10]

Relationship of acne with dietary and lifestyle factors: It is hypothesized that for girls, greater amounts of milk consumption, regardless of fat content, was associated with a higher prevalence of acne and for boys only skim milk (not whole or low-fat milk) had a positive association. On the basis of these results, it is hypothesized that non-fat portion of

milk contains hormones and bioactive molecules, such as androgens, progesterone, and insulin growth factor-1 (IGF-1), that can have an acne stimulating effect.[10]

Dietary glycaemic load: High-glycaemic-load (HGL) diets, such as Western diets, can elevate blood insulin and IGF-1 levels which stimulate adrenal and gonadal androgen production, leading to increased sebum production and acne.[10]

Stress: Psychological stress is also thought to exacerbate acne and acne itself can cause significant psychological distress. The mechanism by which stress negatively impacts on acne is still unknown but could be because of increased hormone levels or inflammation-inducing neuropeptide production.[10]

Management:

Prior to initiating treatment in a female patient, a hyper androgenic state must be considered and ruled out through history, physical examination, and laboratory evaluation. If found normal, then the patient is started on appropriate topical or oral therapies depending on the grade and severity of acne.

Table 1: Topical agents used for acne vulgaris[11]

DRUG	DOSE	SIDE EFFECTS
1)Retinoids		
-Tretinoin	Once bed-time. 0.025-0.1%	Irritation
-Adapalene	Once bed-time.	Minimal irritation
-Tazarotene	Once bed-time.	
2)Antimicrobials		

-Benzoyl peroxide	2.5-10% once /twice daily	Irritation
-Clindamycin, -Erythromycin	once /twice daily	Propensity to resistance
-Combination with zinc, retinoids or other antibiotics	once /twice daily	
3)Other topicals		
10-20% Azelic acid, sulfur, 5% dapsone	once /twice daily	

Table 2: Oral therapy for acne vulgaris[11]

DRUG	**DOSE**	**SIDE EFFECTS**
1)Oral antibiotics		
-Tetracycline	250–500 mg once or twice daily	Gastro intestinal intolerance.
-Doxycycline	50–100 mg once or twice daily	Phototoxicity.
-Minocycline	50–100 mg once or twice daily	Hyperpigmentation of teeth, oral mucosa, and skin;
-Trimethoprim–sulfamethoxazole	One dose (160 mg trimethoprim, 800 mg sulfamethoxazole) b.d.	Toxic epidermal necrolysis and allergic eruptions.
-Erythromycin	250–500 mg b.d. or q.i.d.	Gastro intestinal intolerance.
2)Oral retinoids		
-Isotretinoin	0.5–1.0 mg/kg/day in divided doses	Teratogenicity, mucosal dryness, hyperlipidemia

Table 3: Treatment algorithm:[10]

	Comedonal	Mild papular/pustular	Moderate papular/pustular	Moderate nodular	Severe nodular/cystic
First-line therapies	Topical retinoid	Topical retinoid ± BPO or topical antibiotic/BPO	Topical retinoid + oral antibiotic + BPO or topical antibiotic/BPO	Topical retinoid + oral antibiotic + BPO or topical antibiotic/BPO	Oral isotretinoin
Alternatives	Salicylic acid	Sulfur/sodium sulfacetamide		Oral isotretinoin	Oral antibiotic + topical retinoid + BPO or topical antibiotic/BPO
	BPO Azelaic acid	Azelaic acid			
Alternatives for female patients			Hormonal therapy + topical retinoid ± BPO or topical antibiotic/BPO	Hormonal therapy + topical retinoid ± BPO or topical antibiotic/BPO; oral isotretinoin if refractory	Hormonal therapy + topical retinoid ± BPO or topical antibiotic/BPO

Hormonal therapy for acne vulgaris: Hormonal therapies offer a valuable adjuvant to standard therapy. They act by blocking the androgen receptor and/or decreasing circulating androgens which leads to decreased sebum production. They include spironolactone, other antiandrogens, and oral contraceptives.[12]

The main goal of these therapies is to oppose the effects of androgens on the sebaceous gland. Indications of hormonal therapy in acne: Women with signs of hyperandrogenism and recalcitrant acne.[12]

Table 4: Hormonal therapy for acne vulgaris:[12]

Drug	Mechanism of Action	Standard Dose	Side Effects
Spironolactone	Androgen receptor blocker	25 to 200 mg daily	Menstrual irregularities, breast tenderness, hyperkalemia, hypotension, birth defects
Cyproteroneacetate*	Androgen receptor blocker	50 to 100 mg daily on days 5 to 14 of menstrual cycle or 2 mg combined with 35 g of ethinylestradiol	Breast tenderness, headache, nausea, and breakthrough bleeding, hepatotoxicity, birth defects
Flutamide	Androgen receptor blocker	250 to 500 mg daily	Hepatotoxicity, breast tenderness, gastrointestinal upset, hot flashes, and decreased libido, birth defects
Oral contraceptives	Ovarian androgen blocker	Depends on specific pill	Menstrual irregularities, breast tenderness, gastrointestinal upset, weight gain (see text for contraindications)
Glucocorticoids	Adrenal androgen production blocker	2.5 to 5 mg of prednisone at bedtime	Adrenal suppression (higher risk with dexamethasone)

Chemical peels: Chemical peels are used in the treatment of mild to moderately severe acne vulgaris as adjuncts to topical and oral preparation because of their ability to improve skin's appearance, texture and pore size.

Most commonly used superficial chemical peels like salicylic acid (20-30%) and glycolic acid (20-70%) cause improvements in both inflammatory and non-inflammatory acne. They also increase the penetration of other topical agents used.[13,14]

Lasers and light devices: include photodynamic therapy (PDT), light-emitting diode (LED) therapy and a combination of pneumatic energy and light for the treatment of acne. They mainly act as adjuvant therapies.[15]

Newer modalities:

Resveratrol containing gels: Resveratrol is a natural compound produced by some spermatophytes, such as grapes which inhibits keratinocytic hyperproliferation process.[16]

Vaccines for acne targeting P. acnes: Genomic data has revealed many gene encoded virulence factors, including sialidase, that are involved in adherence to sebocytes, degrading host tissue and inducing inflammation. Adhesion process of bacteria occurs at the early stage of infection and is essential for its colonization, and in turn, colonization may be required for subsequent development of symptoms of diseases. Thus, vaccination targeting sialidase of P. acnes may prove to be an efficient modality for the prevention of early infection of P. acnes.[17]

Picolinic acid 10 % gel: It is an intermediate metabolite of the amino acid tryptophan which has antiviral, antibacterial, and immunomodulatory properties and seems to play a

key role in zinc transport. Picolinic acid gel 10% applied twice daily for 12 weeks has been shown to be a safe and effective treatment for inflammatory and noninflammatory lesions.[18]

Zinc: Zinc acts via inhibition of polymorphonuclear cell chemotaxis, inhibition of growth of P. acnes, decrease in TNF-α production and the inhibition of Toll-like receptor 2 (TLR2) surface expression by keratinocytes. It is used in a dose of 30–150 mg of elemental zinc daily for 3 months for the treatment of inflammatory acne vulgaris.[18]

Insulin sensitizing agents: Polycystic ovary syndrome (PCOS) may present with acne as a marker of hyperandrogenism. Hyperinsulinemia seems to be an important factor in many cases of PCOS. Hence antidiabetic like metformin and thiazolidinediones lower insulin levels, improve ovarian function and plasma androgens, menstrual and metabolic abnormalities. Hence it is reported to be effective in treating hirsutism and acne.[18]

Complications of acne:

1) Post inflammatory hyperpigmentation.
2) Acne scars: Scarring caused by acne is associated with substantial physical and psychological distress, particularly in adolescents.[19]

Table 5: Types of acne scars:[20]

Acne Scars Subtype	Clinical Features
Icepick	Icepick scars are narrow (<2 mm), deep, sharply marginated epithelial tracts that extend vertically to the deep dermis or subcutaneous tissue.
Rolling	Rolling scars occur from dermal tethering of otherwise relatively normal-appearing skin and are usually wider than 4 to 5 mm. Abnormal fibrous anchoring of the dermis to the subcutis leads to superficial shadowing and a rolling or undulating appearance to the overlying skin.
Boxcar Shallow <3 mm diameter >3 mm diameter	Boxcar scars are round to oval depressions with sharply demarcated vertical edges, similar to varicella scars. They are clinically wider at the surface than icepick scars and do not taper to a point at the base.
Deep <3 mm diameter >3 mm diameter	They may be shallow (0.1–0.5 mm) or deep (≥0.5 mm) and are most often 1.5 to 4.0 mm in diameter.

Treatment of acne scars:

Atrophic scars: Chemical peels like glycolic acid, salicylic acid, jessner's solution, trichloroacetic acid(TCA), TCA CROSS (chemical reconstruction of skin scars), dermabrasion/microdermabrasion, lasers, tissue augmentation techniques, subcision.[19,20]

Hypertrophic scars: Silicone gels, intralesional therapies which include intralesional steroids, 5-FU, bleomycin, imiquimod, pulsed dye lasers**,** surgical excision.[19,20]

Hair loss in adolescents:

Introduction: Hair is of limited functional benefit but has a significant impact on self-esteem and quality of life, particularly in our society that emphasizes physical appearance. It may be upsetting to patients, particularly adolescents who are experiencing physical, emotional, and psychological transitions. The most common forms of alopecia in adolescence are telogen effluvium, androgenetic alopecia (AGA), and alopecia areata.[21]

In a patient of hair loss, history regarding hair care practices, such as brushing, styling, and chemical treatments should be asked as these practices are common in adolescent girls.[21]

Telogen effluvium:

Telogen effluvium describes excessive loss of telogen hairs due to abnormal hair cycling.[22]

Etiopathogenesis: Normal hair cycle consists of a growing phase (anagen) lasting 3– 5 years, followed by a transition phase (catagen) and a resting phase (telogen) lasting 3 months, during which hair is shed from the follicle. Telogen hair is replaced by a new anagen hair, restarting the cycle. Losing up to 100 hairs per day is considered as a normal consequence of hair cycling. Acute events that trigger an abrupt change of numerous hair

follicles from anagen to telogen result in increased shedding of telogen hairs 2–3 months later. Common trigger factors include:[22]

(1) Acute telogen effluvium: Acute, severe illness (especially with high fever), Major trauma or surgery.

(2) Chronic telogen effluvium (Shedding of telogen hairs more than 6 months): Iron deficiency anaemia, thyroid disease (hypothyroidism or hyperthyroidism), malnutrition or malabsorption, Chronic illness (e.g. SLE), zinc deficiency, syphilis.

(3) Medication-related (may occur with medication initiation or withdrawal): Oral contraceptives, isotretinoin, terbinafine, anticonvulsants, lithium, cimetidine.[22]

Emotional stress, menstruation and malnutrition appear to be the most common triggering factors in adolescent girls.[22]

Clinical features: Patients complain of losing more hair than normal, often saying the hair is shed in handfuls. Examination of the scalp is normal and shows a diffuse loss of hair rather than any particular region of the scalp with less hair density. The diagnosis is aided by documenting shedding of more than 100 hairs per day or a pull test with greater than 25% telogen hairs.[22]

Evaluation: History about changes 2–3 months prior to onset of hair loss should be elicited in detail. If a particular inciting agent is noted, then patient should be reassured that hair growth will return to normal in 3–6 months and further intervention is not needed.[22]

If a trigger is not identified or if the duration of the hair loss suggests chronic telogen effluvium, laboratory testing like complete blood cell count (CBC), thyroid profile should be done. Additional studies like iron studies [ferritin, total iron binding capacity (TIBC),

serum iron, transferrin saturation] can be done to fully evaluate iron deficiency.[25] Treatment should be aimed at the underlying disorder.[23]

2) Androgenetic alopecia (AGA):

AGA is an androgen-dependent, hereditary disorder which produces a typical pattern of progressive hair loss in affected men and women.[24]

Mechanism of AGA: It is based on dihydrotestosterone, the conversion product of 5-alpha reductase and testosterone, which acts on genetically susceptible hair follicles. Effects include shortening the anagen phase and miniaturization (transformation of large follicles into small ones). This results in finer and shorter hair that does not cover the scalp well.[24]

Although pattern hair loss is seen in both sexes, men often view this process as expected, whereas women are more likely to feel upset and confused by the diagnosis. It is the most common form of alopecia in adults, but it often begins in adolescence.[24]

Clinical features: Women present with decreased frontal hair density, with preservation of the frontal hairline. The central part may be widened, especially toward the anterior scalp, a pattern resembling a Christmas tree.[24]

The diagnosis of AGA is most often clinical. Patients may give history of decreased hair density, decreased thickness of their pony tail and that their hair does not grow as long as before. In a case of adolescent girl with AGA, additional history and examination should be sought to evaluate for potential androgen excess, which may be exogenous or endogenous.[24]

Along with early onset or presence of severe acne, hirsutism, truncal obesity, and acanthosis nigricans, FPHL can be a sign of polycystic ovary syndrome (PCOS).[25] Diagnosing this disorder is important as it is associated with long-term health effects such as insulin resistance and infertility which are important especially in an adolescent girl.[26]

Laboratory diagnosis: Laboratory studies are recommended for all women with suspected FPHL. They include complete blood count, thyroid profile, free and total testosterone, and dehydroepiandrosterone sulfate (DHEA-S), Prolactin and 17-hydroxyprogesterone levels.[24]

Treatment: AGA is a progressive disorder, and treatment is aimed at halting the progression of hair loss and promote regrowth of hair. Two percent minoxidil is Food and Drug Administration (FDA)-approved for use in women atleast for a period of 12months. Both 2% and 5% minoxidil are well tolerated and effective in women, but the 5% solution is more likely to cause reversible facial hypertrichosis.[27] Progression of hair loss will recur once treatment is discontinued.

Spironolactone, an aldosterone antagonist, 50-200 mg/day can also be given in androgenetic alopecia affecting women.[24]

3) Alopecia areata (AA):

Introduction: Alopecia areata is an immune-mediated form of non-scarring hair loss that often presents in childhood or adolescence.[21]

Etiopathogenesis: It is largely undetermined but it is hypothesized that, in genetically susceptible individuals, loss of immune privilege of the hair follicle leads to T-cell-mediated inflammation against anagen hair follicles.[21]

Clinical features: It presents abruptly as round, smooth patches of hair loss that may be isolated or multifocal with broken, short hairs and tapered proximal ends ('exclamation-mark' hairs) at the periphery. Other presentations of AA include the ophiasis pattern: a band of hair loss affecting the lower periphery of the scalp, alopecia totalis (loss of all scalp hair), and alopecia universalis (loss of all scalp and body hair). It can also involve the eyebrows and eyelashes. Nail changes include regular uniform pitting, longitudinal striations and leukonychia.[21]

Alopecia areata has an unpredictable course. Spontaneous improvement is common but about 10% can go into chronic form. Poor prognostic factors include early onset of the disease, severe extent at first presentation, positive family history, presence of nail changes and association with other diseases like thyroid disease and atopic dermatitis.[21]

Treatment: Topical: Potent topical corticosteroids, contact sensitizers such as squaric acid dibutyl ester (SADBE) or diphencyprone (DCP) are carefully applied to the scalp to produce a low-grade allergic contact dermatitis that allows hair to regrow.[21]

Intralesional: corticosteroids like triamcinolone acetonide at a concentration of 5mg/ml can be used per treatment, repeated at 4–6-week interval.[21]

Systemic therapy: oral corticosteroids, steroid sparing drugs can also be used.[21]

4) Trichotillomania:

It is defined as an irresistible urge to pull one's own hair. It arises primarily in children and adolescents, and is a relatively common cause of childhood alopecia.[28]

Scalp is the most common site of pulling, where the pattern of hair loss is often bizarre, with irregularly shaped angular or linear borders, the side of the scalp correlating directly with handedness of the patient. It may also involve eyebrows and eyelashes.[28]

Diagnostic criteria for TTM includes:[29]

A) Recurrent pulling out of one's hair, resulting in noticeable hair loss.

B) An increasing sense of tension immediately before pulling out the hair or when attempting to resist the behaviour.

C) Pleasure, gratification, or relief when pulling out the hair.

D) The disturbance is not better accounted for by another mental disorder and is not caused by a general medical condition (e.g., a dermatologic condition).

E) The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Management is often difficult and referral to psychiatrist is advised. Pharmacotherapy can be used as adjunctive therapy. Non pharmacological treatments in the form of behavioural and/or supportive family and professional counselling, should be considered first-line therapy for children with TTM.[28]

Patients of alopecia should be provided reassurance and emotional support and help them to develop positive coping strategies for their condition. In addition, hair prosthesis like wigs and hair pieces should be considered for any patient with extensive hair loss which can help adolescents appear more like their peers and improve self-esteem.[28]

Premature graying of hair:

Introduction: Canities, or hair graying, is a process of chronological aging and occurs irrespective of gender or race, but the age of onset varies. Hair is said to gray prematurely only if graying occurs before the age of 20 years in Whites and before 25 years in Asians.[30]

Hair length, colour, and style play an important role in people's physical appearance and self-perception. Hence premature hair graying adversely effects the appearance, self-

esteem, and socio-cultural acceptance of the affected individual. It is most commonly viewed as a sign of old age and loss of youthful appearance which renders the affected individuals to develop social stigma, discrimination, and marriage difficulties which is especially in girls.[30]

Pathogenesis: It may reflect a genetically regulated early exhaustion of the melanocyte reservoir's regenerative capacity or due to defects in cell activation/migration triggered by environmental factors, inflammation or emotional stress.[30]

Etiology: genetic, auto immunity, copper deficiency, drugs like chloroquine, impairment of antioxidant mechanism secondary to drugs, stress, smoking.

Differential diagnosis: 1) Various types of oculocutaneous albinism.

2) Silver hair syndromes: Griscelli syndrome, Elejalde Syndrome, Chediak-Higashi syndrome.
3) Metabolic syndromes like phenylketonuria, histidinemia.
4) Localised whitening of the hair seen in vitiligo, piebaldism.[30]

Treatment: No effective therapy is yet available. Few of them include:

1) Para-aminobenzoic acid (PABA), 100mg t.i.d for 2-4 months.
2) Calcium pantothenate.
3) Prostaglandin agonist, latanoprost as they are the most potent stimulators of melanocyte growth and melanogenesis.
4) Camouflage techniques like hair colourants form the mainstay of therapy.[30]

Hirsutism:

Introduction: Hirsutism is defined as the presence of terminal coarse hairs in females in a male-like distribution.[31]

Etiology: Occurs as results of increased androgen levels in females from increased production of androgens (testosterone) either by the adrenals or due to an ovarian disease. The ovarian causes include polycystic ovarian syndrome (PCOS) and ovarian tumors. Adrenal causes include Cushing's syndrome, androgen-producing tumors, and congenital adrenal hyperplasia (CAH), most commonly due to 21-hydroxylase deficiency. Androgenic drugs are also an important cause of hirsutism.[31]

Idiopathic hirsutism (IH): who have normal androgen levels and ovarian function, occurs in about 20% of patients. Onset of IH occurs shortly after puberty with slow progression. PCOS and IH constitute about 90% of the hirsutism in women.[31]

Pathogenesis: Hirsutism is attributed either to increased production or increased sensitivity of the hair follicles to circulating androgen (testosterone). IH with normal androgen levels is postulated to result from exaggerated peripheral 5-alpha reductase activity, androgen receptor polymorphisms, or altered androgen metabolism.[31]

Clinical features: Patients usually present with increased growth of terminal hair at sides of the face, upper lip, chin, upper back, shoulders, sternum, and upper abdomen.[31]

Management: Evaluation of a patient of hirsutism should include detailed history about the pattern and sites of involvement, menstrual irregularities, increased libido, intake of any drugs and investigations like hormonal profile.[31]

Treatment includes oral contraceptives, androgen receptor blockers, gonadotropin releasing hormone agonists, 5-alpha reductase inhibitors. Biological modifiers of hair follicular growth like Eflornithine hydrochloride is known to inhibit the enzyme ornithine decarboxylase involved in the proliferation of hair matrix cells.[31]

Lasers for permanent hair reduction have emerged as the preferred choice in recent times, which work on the principle of selective photothermolysis targeting the melanin.[31]

Disorders of the sweat glands:

Hyperhidrosis: Primary hyperhidrosis (PH) is one of the chronic diseases characterized by excessive sweating which may cause mood changes associated with anxiety, depression and irritability.[32,33]

Hyperhidrosis occurs in both children and adults, with the average age of onset of primary hyperhidrosis being 14–25 years.[33,34] There may be positive family history in patients with disease onset before the age of 20.[35]

It can be mild, moderate or severe. Palmar and plantar regions are the most commonly affected areas, followed by the axillary and craniofacial zones.[32]

PH causes embarrassment, discomfort and even serious social, occupational and psychological problems, there by affecting the quality of life of patients leading to mental disorders that can last a lifetime.[32]

Pathogenesis: Emotional stress hypothesis: Sweat glands of the palms and soles are activated by emotional stimuli and it is even speculated that the hypothalamic sweat centre that controls the palms and soles (and axillae in some patients) is distinct from the rest of

the “hypothalamic sweat centres” and is under the exclusive control of the cortex without inputs from the thermosensitive elements.[36]

Classification: 1) Primary/Physiologic causes: Without any apparent cause as in cases of increased environmental temperature**,** exercise**,** severe pain**,** anxiety, ingestion of spicy foods, alcohol, obesity, pregnancy.

2) Secondary: Secondary to other underlying disorders.

-Generalised: infections, neoplasm, endrocrine disorders, neurological diseases, drugs and substance abuse.

-Regional: Compensatory: spinal cord lesions, neuropathy.

-Focal: Frey syndrome, Chorda tympani syndrome, gustatory sweating.[32]

Clinical features: The criteria for a diagnosis of PH are: focal, visible, excessive sweating of at least six months’ duration without apparent cause with at least two of the following characteristics:[37]

1. Bilateral and relatively symmetrical involvement.

2. Impairment of daily activities.

3. Frequency of at least one episode per week.

4. Age of onset less than 25 years.

5. Positive family history.

6. Cessation of sweating during sleep.

Diagnosis: The diagnosis of primary focal hyperhidrosis is clinical based on the above mentioned criteria.

Tests used to quantify sweat production are not routinely used in clinical practice. Minor's starch iodine test or the quinizarin test can be used to map areas of excessive sweating, which is more useful if botulinum toxin injections are planned.[32]

Table 6: Treatment of hyperhidrosis.[36]

Treatment	Treatment type	Common sites of use	Side effects
Topical therapy	Aluminum salts (±4% salicylic acid) Topical anticholinergics: glycopyrrolate (topical or with iontophoresis) Other (rarely used): tannic acid, formaldehyde, glutaraldehyde	Axilla, palms, soles	Burning, itching, erythema, irritation Skin staining may be seen with aldehydes and tannic acid
Oral anticholinergics	Glycopyrrolate Propantheline bromide Oxybutynin	Axilla, palms, soles, face	Dry mouth, blurred vision, tachycardia, urinary retention, constipation
Other systemic treatments	Calcium-channel blockers (diltiazem) Clonidine Alpha-adrenoceptor antagonists Benzodiazepines	Axilla, palms, soles, face	Treatment dependent
Iontophoresis	Tap water iontophoresis, with or without anticholinergic drugs (glycopyrrolate)	Palms, soles	Stinging, burning, occasional reports of blister formation
Botulinum toxin	Botulinum toxin A (Botox®, Dysport®) Botulinum toxin B (Myobloc®)	Axilla, palms, soles	Muscle weakness Pain at injection site
Surgery	Curettage Liposuction Thoracic sympathectomy (via either "open" or "minimally invasive" approach)	Axilla, palms	All treatment modalities: wound infection, scar Sympathectomy: compensatory hyperhidrosis in surrounding areas, hemothorax, pneumothorax, atelectasis, subcutaneous emphysema, Horner's syndrome

Miliaria:

Introduction: Miliaria is the commonest sweat gland disorder seen in the tropics. It refers to a group of disorders in which there is obstruction to the egress of sweat. Different types of miliaria occur as a result of obstruction at different levels of the intra epidermal sweat duct.

Miliaria crystallina: level of stratum corneum.

Miliaria rubra: stratum granulosum and below

Miliaria profunda: dermoepidermal junction.[38]

Epidemiology: Miliaria rubra (prickly heat) is the most common sweat retention disorder among the other types often affecting the neonates but can also occur in children and adolescents who are exposed to hot and humid environments and who wear tight and impermeable clothing. A study conducted at Nigeria showed that miliaria occurred more in adolescent girls, many of whom were noticed to be wearing numerous layers of nylon vests/underwear underneath their school uniforms, which can generate a significant amount of heat.[1]

Clinical features: Miliaria crystallina: Asymptomatic, discrete or confluent, transparent, thin walled vesicles, 1–2 mm in diameter appear often in crops, resembling "dew drops" appear on an uninflamed skin, rupture spontaneously leaving behind a superficial branny desquamation.[38]

Miliaria rubra: Appear as small, discrete nonfollicular erythematous macules or papules that may contain a minute vesicle sometimes forming "sheets of erythema" often accompanied by a paroxysmal stinging sensation mainly involving trunk and neck especially under the clothing.[38]

Miliaria profunda: Asymptomatic pale or flesh coloured, 1–3 mm sized papules that resemble gooseflesh which are transient and subside in less than an hour once the stimulus for sweating has been removed.[38]

Treatment: The most effective remedial measure for miliaria is to place the patient in a cool environment and avoid excessive exposure to heat, humidity, use of tight or occlusive clothing. Emollients like anhydrous lanolin or hydrophobic ointment are sometimes helpful

in relieving poral occlusion and in facilitating the egress of sweat. Calamine lotion can be given for soothening the irritated skin. Isotretinoin can be given in cases of recalcitrant miliaria profunda.[38]

INFECTIONS:

Fungal infections:

Introduction: Fungal skin infections are broadly classified into superficial and deep infections.[39,40]

Superficial infections are limited to the epidermis, hair, nails and the mucous membrane; the most common of them being dermatophytosis and tinea versicolor.[39,40]

Adolescents have more risk of contracting a superficial mycotic infection, due to youths' high participation in sporting activities, close mingling with each other and use of swimming pools.[39,40]

Tinea capitis is uncommon in this age group, because of the higher content of fungistatic fatty acids in the sebum. In contrast, they may have high frequencies of pityriasis versicolor infection, probably because during puberty the increasing levels of sexual hormones modulate distribution of grease and sebaceous gland secretions, which are the key elements in the metabolism of the lipophilic yeasts of the Malassezia.[39,40]

The presence of these lesions may negatively affect the development of teenagers' personalities and self-esteem.[39,40]

Common superficial fungal infections will be reviewed here.

Tinea corporis and Tinea cruris:

Tinea corporis and tinea cruris are superficial dermatophyte infections, commonly known as 'ringworm.' Tinea corporis includes all superficial dermatophyte infections of the glabrous skin, excluding the scalp, beard, face, hands, feet, and groin. Tinea cruris includes infections of the genitalia, pubic area, perineal skin, and perianal skin.[41]

Etiology: Tinea corporis and tinea cruris may be caused by any of the dermatophytes making up the genera Trichophyton, Microsporum, and Epidermophyton, Trichophyton rubrum being the most common organism causing T. corporis and Trichophyton rubrum, Mentagrophytes sp or Epidermophyton floccosum more commonly causing T. cruris.[41]

It is more common in summer months, and in children or adolescent girls who are obese, perspire more or who wear tight fitting under garments.[41]

Clinical features: Present as pruritic annular scaly erythematous plaque with a raised edge and central clearing. The clearance in the centre of the lesion may be the manifestation of an immune response of the host to the infecting organism.[41]

Majocchi's granuloma: Viable hyphae invade the hair shaft and extend into the dermis, may be because of trauma caused by shaving leading to formation of inflammatory papules and pustules with erythema and peri folliculitis.[41]

Diagnosis: Scrapings from the lesion subjected to 10-20% KOH mount shows fungal hyphae. Culture of the scales on Sabouraud's agar media incubated at room temperature will show the causative organism in 2-3 weeks.[41]

Treatment: Topical: Azoles (oxiconazole, miconazole, clotrimazole, econazole, and ketoconazole); the allylamines (naftifine and terbinafine); benzylamine derivatives (butenaftine); and hydroxypyridones (ciclopirox olamine).[41]

Systematic: Commonly used drugs are terbinafine, griseofulvin, itraconazole**,** fluconazole.[41]

Tinea pedis:

This is a common condition in children and athletes commonly known as athlete's foot. Infection is usually in between toes where moist skin favours the growth of dermatophytes, producing itchy, macerated lesions with fissuring often with a foul odour. Common risk factors are use of community shower stalls at schools, swimming pools, sports clubs, sharing slippers, wearing tight shoes and non-absorbent socks for long time. Most common organisms are the same as in tinea cruris.[41]

Tinea rubrum affect relatively dry area of foot like heels, soles, and sides producing slightly erythematous plaques covered with fine silver scales.[41]

Differential diagnosis includes contact and atopic dermatitis, psoriasis, and candidal infection between the toes.[41]

Treatment: Topical antifungals may be used in mild cases but most patients require oral antifungals.[41]

Tinea incognito:

The term tinea incognito was originally described in patients with a dermatophytic infection that had an atypical clinical presentation caused by previous treatment with steroids.[42,43]

Over-the-counter access to steroids and other immune suppressants in some countries, as well as the increase in medications containing steroids, makes tinea incognito more likely,

and therefore, the diagnosis is frequently missed or delayed. Such self-medications are more common in adolescents owing to the skin lightening effect of topical steroids.[42,43]

Clinically, they differ from classical lesions by having a less raised margin and are less scaly. They tend to be pustular, pruritic, extensive, and erythematous mimicking other skin diseases.[42,43]

The clinical history is fundamental, because the clinical appearance may be confusing. The definitive diagnosis must be attained by direct examination with potassium hydroxide, which demonstrates fungal structures. The species may also be identified by culture.[42,43]

It usually requires systemic treatment with oral antifungal agents. Terbinafine, itraconazole, and fluconazole have been shown to be superior to treatment with griseofulvin, because they accumulate in the skin. Therapy is generally indicated for 2 weeks.[42,43]

Pityriasis Versicolor:

This superficial fungal infection is caused by different genus Malassezia and most commonly by the species Malassezia furfur.[44]

It gives rise to typical fine scaly macular lesions mostly affecting the trunk, back and arms but can also involve other areas, especially in children where face involvement is common. It is more frequent in hot and humid climates. It presents differently according to the colour of skin of the individual affected hence the name versicolor. In light coloured individuals, lesions are hyperchromic or brownish while in dark coloured skin the macules are hypochromic.[44]

Lesions are usually asymptomatic and only reason patients seek help is for cosmetic concerns.[44]

Bedside tests: Woods lamp examination shows pale yellow fluorescence. KOH preparation reveals short hyphae and grape like clusters of spores ("spaghetti and meatball" appearance).[44]

Treatment: Topical antifungals like selenium sulfide 2.5% shampoo or lotion, zinc pyrithione shampoo 1% are preferred in children. However, oral antifungals like single-dose ketoconazole (400 mg orally) or fluconazole (400 mg orally) can be given in older adolescents.[44]

Pityrosporum folliculitis:

Pityrosporum folliculitis involves follicular occlusion followed by an overgrowth of the yeast Malassezia that thrives in a sebaceous environment. It is commonly found in adolescents probably because of the increased activity of their sebaceous glands.[45]

Clinically, it presents as 1 to 2 mm pruritic, monomorphic, pink papules and pustules on the chest, back, upper arms, and less frequently on the face, mimicking acne vulgaris.[45]

A KOH examination on scrapings of the monomorphic pustules reveals spores and budding yeast forms.[45]

Antibiotics commonly used to treat acne may suppress normal bacterial flora and allow overgrowth of Malassezia. This may explain some cases of what appears to be persistent acne that shows no improvement and actually worsens with oral antibiotic treatment and usually responds well to oral antifungal medications.[45]

Onychomycosis:

Introduction: It denotes nail infection from dermatophytes, non dermatophytic moulds, or yeasts while tinea unguium refers specifically to the infection of the nail plate by dermatophytes.[46]

Epidemiology: The reported prevalence of onychomycosis ranges from 0.2% to 2.6% among children approximately 1/30th of adults.[47,48]

Onychomycosis is rare in childhood probably because of protective factors typical of that age, including rapid nail growth, small contact area of childrens' nails (which means infrequent trauma and minor possibility of fungal colonization), lower incidence of tinea pedis in young age and infrequent exposure to infectious agents in community places.[46]

Etiology: a) Onychomycosis in children is usually due to dermatophytes, and mostly to T. rubrum and T. interdigitale, as in adults.

b) Candidal onychomycosis is rare in children and occurs usually in immune compromised individuals.[46]

Predisposing factors: Are more common in children accounting for 53% as opposed to only 5-10% in adults. Development of onychomycosis in children possibly needs strong predisposing factors such as positive family history, immune suppression and contact with fungi in the environment such as to counteract the protective ones.[46]

Clinical features: Dermatophyte onychomycosis in children presents with 3 clinical varieties seen in adults, usually limited to one or two digits.

a) Distal lateral subungual onychomycosis(DLSO): more common in the toe nails than finger nails as the plantar skin may be a reservoir of fungi.[46]

b) Proximal subungual onychomycosis (PSO): Occurs in both immunocompromised and healthy patients as opposed to PSO being more in immune compromised adults. It presents as a whitish discoloration of the proximal nail plate that extends distally. Nail invasion starts from the proximal nail fold and reaches the nail matrix.[46]

c)White superficial onychomycosis (WSO): Affects several toenails and often presents as deep WSO, characterized by a deeper nail invasion and a larger surface of involvement than the 'classical' WSO. Fungi easily invade the thin nail plate of children, and this anatomic reason explains the particular feature of WSO in young age.[49]

Treatment: Optimal management of onychomycosis in children includes confirming the clinical diagnosis with potassium hydroxide and culture, and looking for associated tinea pedis and other predisposing factors, such as onychomycosis/tinea pedis in family members.[49]

Commonly used oral medications include itraconazole, fluconazole and terbinafine.[50]

Viral infections:

Herpes simplex infection:

HSV infections are distributed worldwide. Humans remain the sole reservoir for transmission of these viruses to other humans. The virus is transmitted to susceptible hosts by close personal contact.[39,51,52]

The incidence of HSV-1 infection is influenced largely by geographic location, socioeconomic status and age. HSV-2 is usually common after the age of onset of sexual activity. [39,51,52]

Varicella zoster infection (chicken pox):

This is highly contagious infection which spreads by direct contact and droplet infection caused by varicella zoster virus. [39,51,52]

Incubation period: 10-23 days. [39,51,52]

C/F: Disease starts with prodromal flu-like symptoms, fever, sore throat, and headaches.

Rash first appears on trunk and spreads laterally. The mildly pruritic rash comes in crops starts as erythematous macules, which evolve in vesicles (dew-drop on a petal), and then into pustules which later rupture and become encrusted before completely healing. Usually there is no scarring or pigmentary change, though it may occur if pruritus is more severe than usual. [39,51,52]

Varicella usually runs a benign course but complications include secondary infections, necrotizing fasciitis, meningitis, encephalitis, transverse myelitis.[39,51,52]

Management: Supportive and symptomatic treatment is all that is needed in cases of an uncomplicated varicella infection. [39,51,52]

Anti-viral treatment: Oral acyclovir, valcyclovir, famciclocvir begun within 24 hours of onset of symptoms reduces the number of lesions, the time to cessation of new lesion formation, and the duration of rash, fever and constitutional symptoms. [39,51,52]

Antibiotic treatment will be indicated in secondary bacterial infections. [39,51,52]

Varicella vaccine is considered effective in up to 85% of cases. For susceptible immunocompetent older children, two doses of 0.5 ml is administered subcutaneously 4-8 weeks apart. [39,51,52]

Molluscum contagiosum:

Molluscum contagiosum is a common, benign, self-limiting viral infection of the skin. It generally affects children and sexually active adults which mainly occurs following autoinoculation or contact with affected people. [39,51,52]

The incubation period is 2-6 months. It is more common in young children and in children who swim or bathe together, and also in children with atopic dermatitis. [39,51,52]

Mollusca present as multiple dome shaped pearly or flesh coloured papules with a central depression (umbilication), which usually appear on the trunk and flexural areas. They vary in size from 1 mm to 10 mm, with growth occurring over several weeks. [39,51,52]

As the condition is benign and typically resolves spontaneously, treatment is usually not necessary. Treatment modalities include cryotherapy with liquid nitrogen, imiquimod, excision and curettage and electrodessication. [39,51,52]

Verruca vulgaris:

Cutaneous viral warts are discrete benign epithelial proliferations caused by the human papilloma virus. Prevalence increases during childhood, peaks in adolescence, and declines thereafter. [39,51,52]

Clinical features and types: Common warts: Begin as smooth flesh coloured papules that enlarge and develop a characteristic hyperkeratotic surface of grossly thickened keratin. Koebnerisation can be seen.[39,51,52]

Other morphological variants include plantar warts, plane warts, mosaic warts, filiform warts.[39,51,52]

Treatment: Topical therapy with keratolytic agents like salicylic acid 10-25%, cryotherapy with liquid nitrogen are commonly used treatment modalities. Intralesional bleomycin, topical immunotherapy, photodynamic therapy, and pulsed dye laser treatment are best confined to resistant cases. [39,51,52]

Pityriasis rosea:

Pityriasis rosea is an acute benign self-limited papulosquamous disorder with characteristic skin rash and clinical course mainly involving children and young adults.[39,51,52]

Etiology: The exact etiology of this condition is unknown, but some studies have demonstrated the presence of HHV-7 in lesional skin and in mononuclear cell, plasma, and peripheral blood, suggesting their role in the pathogenesis of pityriasis rosea. [39,51,52]

Clinical features: Skin lesions are preceded by upper respiratory tract infection in some cases. Clinically appears as a single isolated oval lesion defined as "herald patch" (in 50% patients) usually on the trunk, upper arm, neck or thigh with surrounding fine white scale "collar sign", usually seen 1-2 weeks prior to appearance of other lesions. [39,51,52]

The secondary lesions reveal generalized erythematous patches, papules with fine white scale bilaterally and symmetrically discrete to the line of skin cleavage, in a "Christmas

tree pattern" on the upper arm, neck, back, trunk and upper thigh. The white scales reveal a "collarette of scale" surrounding the lesion, characteristic of pityriasis rosea. [39,51,52]

Spontaneous recovery usually occurs within 6-12 weeks but may be prolonged over a period of 6 months. There may be residual post-inflammatory hypo or hyperpigmentation.[39,51,52]

Treatment: Patients and families should be reassured of the self-limited natural course of this condition. The treatment is supportive with topical or systemic antipruritic medications. Optional treatments include oral erythromycin, topical corticosteroid and UVB. [39,51,52]

Pyodermas:

Bacterial skin infections are broadly termed pyodermas.

Primary pyodermas: Which arise on the normal skin.

Secondary pyodermas: Which may not have a characteristic morphology and originate on a pre-existing skin condition.[39,53]

Impetigo:

Introduction: A common skin infection more commonly young children rather than adolescence. Typical lesions can be bullous or non-bullous, as well as have a mixed presentation occurring within the same area. [39,53]

Staphylococcus aureus is the most commonly implicated etiological factor. [39,53]

Types: a) Bullous impetigo: Most common form, starts as innocuous reddish papules which coalesce and form small bullae which easily rupture forming erosions covered with the typical honey coloured thick crust. [39,53]

b) Non bullous impetigo: This less common form presents with expanding honey-coloured crusts leaving a raw erosive area. [39,53]

c) Secondary infection of minor cuts, insect bites and excessive scratching of other pruritic lesions especially in diabetic or immunocompromised children can result in impetigenous lesions, usually of the non-bullous type. [39,53]

Folliculitis: Folliculitis is a bacterial infection of the hair follicle which can be superficial or deep. [39,53]

Superficial folliculitis: Can be infective (caused by Staphylococcus aureus) or non-infective (caused by contact with mineral, occlusive dressings and use of coal tar preparations). It is characterised by dome-shaped pustules and papules surrounding the hair follicles. [39,53]

Deep folliculitis: The infection reaches to the base of follicle resulting in painful papules and pustules often heals with scarring. [39,53]

Furuncle (boil): Infection of the hair follicle and perifollicular area leading to perifollicular inflammation and suppuration usually caused by Staphylococcus aureus. Primary source of infection is usually nasal carriage. [39,53]

Hyperhidrosis commonly predisposes to furunculosis and hence maintaining good hygiene of intertriginous areas is necessary to prevent autoinoculation and recurrence. [39,53]

Pitted keratolysis: Infection of palms and soles by Corynebacterium bacteria produces proteolytic enzymes which leads to digestion of keratin and formation of pits. [39,53]

Clinically characterised by multiple, tiny, small, shallow crateriform lesions involving the palms and soles which coalesce to form irregular erosions. It is associated with hyperhidrosis. [39,53]

Treatment of pyodermas: Topical antibiotics like mupirocin, bacitracin or fusidic acid to be applied twice daily in case of small lesions. Extensive lesions can be treated with oral antibiotics. [39,53]

INFESTATIONS:

Pediculosis:

Introduction: The three lice species that infest humans are Pediculus humanus capitis—head louse, Phthirus pubis—crab or pubic louse, and Pediculus humanus corpus—body louse all of them being obligate human parasites. Most common of them affecting children and adolescents is pediculosis capitis.[54,55]

Pediculosis Capitis:

Infestation with pediculosis humanus capitis is very common all over the world usually affecting children between ages of 3 to 12 years with a higher preponderance for girls and a lower predilection for African Americans. [54,55]

Head to head contact is the main source of transmission but can also occur through sharing combs, hats, scarves, hair bands etc. or sleeping on the bedding of the infested person. Outbreaks in preschools and elementary schools are common. [54,55]

Head lice feed on the host's blood and can cause significant itching which leads to frequent scratching in turn giving rise to secondary infections like impetigo. [54,55]

Diagnosis is best established by visualizing the live lice which at times could be very difficult as they tend to hide in areas where the hair is thickest as in the nape of the neck. Use of a louse comb aids in diagnosis. [54,55]

Prevention of spread is the most effective way of controlling the infection. Education not to share personal items like combs, hairbrush, hats can be effective in limiting spread. [54,55]

Treatment: Application of 1% Permethrin or 0.5% Malathion is effective. 1% Permethrin is very effective in form of a crème rinse when left in the hair for 10 minutes and rinsed. The residue it leaves behind continues to kill the nymphs emerging from eggs. A second application is suggested in 7-10 days. [54,55]

All clothes, hairbrushes, combs and linens used 2 days prior to treatment should be washed in hot water. [54,55]

Scabies:

Caused by an arthropod Sarcoptes scabiei var hominis, this mite can not only transmit by skin-to-skin contact but also by contact with contaminated material like bedding or clothing where it can survive for up to 36 hours.[56,57]

Scabies affects people of all social classes and ages, but it is more commonly seen in children and adolescents especially girls. Children are more at risk due to their propensity of being in close contact with each other. [56,57]

After an incubation period of 3-6 weeks, the female mite burrows in the epidermis where it lays eggs. The larva emerges within 2-3 days and matures in an adult mite within 15 days increasing the population of mite thus the risk of transmission to others. [56,57]

Clinical features: Itchy lesions commonly involving the finger webs, along the sides of fingers, flexor aspects of wrist, genitalia, axilla and buttocks. [56,57]

The pruritis is due to a hypersensitivity reaction to the mite. Itching is worse during night when the skin is warmer. [56,57]

The initial lesion is a small erythematous papule, which can become a vesicle or pustule. Typical lesions are small linear brownish lesions called burrows. Scratching may cause excoriations and breaks in skin leading to secondary infections like impetigo. [56,57]

Diagnosis is confirmed by clinical examination and by finding typical burrow lesions in between finger webs. Attempt should be made to extract the mite, or eggs from these burrows by gently scraping and examining under light microscope. Other techniques include epiluminescence microscopy and dermatoscopy.[56,57]

Treatment: Topical agents: Permethrin 5% cream to be applied to the whole body below the level of the chin and washed off after 8-12 hours, with reapplication recommended after 2 weeks. Other modalities include lindane (gamma benzene hexachloride) 1% lotion or cream, benzyl benzoate 10% and 25% lotion or emulsion, precipitated sulphur 2–10% ointment. Oral therapy includes ivermectin 0.2mg/kg single dose is sufficient. [56,57]

ECZEMAS:

Atopic dermatitis (AD):

Introduction: It is a chronic inflammatory skin disease which typically affects children during the first years of life, with a prevalence around 10-30%.[58,59]Recent studies have shown that in 10-20% of children with AD, the disease persists through adolescence.[60]

The persistence of AD in adolescence is correlated with psychological distress such as anxiety; moreover, adolescents affected by AD might have relationship problems with their peers. AD can lead to significant stress and psychological problems which can cause worsening of the patients' quality of life (QoL).[58]

Etiopathogenesis: Etiological factors which have been implicated are hereditary factors, environmental factors, immune dysregulation, infective factors and psychological factors. [58,59]

Hereditary factors: A family history of "atopy" can be obtained in approximately 43%–73% of cases. Patients without a family history of "atopy" present clinically in a similar way but less severe and with better chance of spontaneous remission. [58,59]

Filaggrin (responsible to form cross-links between the corneocytes to form the stratum corneum) gene mutation leading to primary epithelial defect is considered to be main mechanism. This finding establishes a key role for impaired skin barrier function in the development of AD. [58,59]

Environmental factors: Atopic dermatitis is supposed to be more common in urban than rural areas which is probably because of industrialization and a changed lifestyle. Pollution plays a significant role not only in the precipitation of allergic rhinitis or bronchial asthma but also in AD. [58,59]

AD is known to aggravate during winter. However, heat and exercise-induced sweating can increase the pruritus of AD. Cholinergic hyper responsiveness may thus be responsible for influencing the course of AD. [58,59]

Inhaled allergens elicit or aggravate eczematous skin lesions and contribute to the flare-ups of AD in a subgroup of patients. Reduction of exposure to some common allergens such as house dust mites is associated with significant improvement of AD. [58,59]

Immunological mechanisms: Defects in the innate immunity are known to play a role in the development and severity of AD. Some of them include defects in the pattern recognition receptors (toll like receptors), reduced migration and function of the effector cells like langerhans' cells, neutrophils, mast cells into the skin.[61]

Infective factors: The reduction in antimicrobial peptides, diminished recruitment of innate immune cells to the skin, epithelial barrier disruption, and Toll like receptor-2 defects are just some of the explanations for AD patients' susceptibility to pathogens such as Staphylococcus aureus, herpes simplex virus. [58,59]

Psychological factors: Stress is an important factor in the precipitation of flares in patients with AD. [58,59]

Gender differences: A study has shown that girls have a higher skin pH and a lower hydration of the stratum corneum than boys which might cause female patients to have a higher risk of developing eczema. Female AD patients have worsening of the skin condition during the pre-menstrual period, menstruations and pregnancy, suggesting that an association exists between hormonal levels and AD lesions. It may also be due to switching over to TH2 response during pregnancy which may lead to worsening of eczema.[60]

Nutritional factors: Zinc and magnesium deficiency may be associated with AD.[62]

Clinical features: Infantile AD: lesions are exudative and mainly localized in the head, face and in the extensor surfaces of limbs. In older children, lesions are mainly concentrated

on the flexural surfaces of the limbs, the popliteal and antecubital folds, back of hands and feet. The skin is commonly dry with lichenification and intense itch. The lips are frequently dry brittle, chapped and develop fissures. The disease is characterised by phases of remissions and exacerbations. [58,59]

Clinical features typical of adolescence are represented by eyelid dermatitis, and the palmar and plantar dermatitis; the eczematous lesions are often localized to the neck, and are often associated with infection by Malassezia. [58,59]

Treatment: General measures: Avoid the use of clothing that may act as an irritant (e.g., wool or synthetics). Cotton clothing is preferable. Advice regarding adequate clothing (which may reduce exposure to irritants and trauma from scratching).[63]

Bathing: Bathe once daily for several minutes in warm water. After bathing, pat dry and immediately apply topical medication or emollient or both. [63]

Emollients: Emollients are effective as first-line agents in the management of AD and may be steroid sparing. Ointments and creams provide better barrier function than lotions.[63]

Topical corticosteroids: First-line therapy for the management of disease exacerbations.[63]

Calcineurin inhibitors: Topical tacrolimus (0.03% ointment) and topical pimecrolimus 1% have been approved for use in the treatment of AD in children. [63]

Systemic therapy: Systemic corticosteroids: in severe disease, anti-histamines for symptomatic relief of pruritis, oral antibiotics active against Staphylococcus aureus and Streptococcus pyogenes are indicated for patients with evidence of secondary bacterial infection. [63]

Contact dermatitis:

Introduction: Contact dermatitis is an eczematous eruption secondary to external agents, and can be broadly divided into irritant substances that have a direct toxic effect on the skin (irritant contact dermatitis, ICD) and allergic chemicals where immune mediated delayed hypersensitivity reactions occur (allergic contact dermatitis, ACD).[64]

1)Allergic contact dermatitis:

Allergic contact dermatitis (ACD) was thought to be rare occurrence in children due to the assumption that children were less exposed to contact allergens and that the immune system in children was less susceptible to contact allergens, but in the last few decades, incidence of ACD in children and adolescents is increasing. This increase may be a result of more frequent exposure to allergens at a younger age, new trends in body piercing, use of cosmetic products, and participation in sports and hobbies.[65]

ACD acquired in childhood has important repercussions for patients, hence may affect decisions regarding future occupation in adulthood.[65]

Epidemiology: Sensitivity to contact allergens increases with age, through childhood and adolescence, as a result of increased environmental exposure to contact allergens. In the adolescent age group, females have significantly higher rates of allergic contact dermatitis on the face, which is explained by increased exposures to nickel in piercings and to preservative and fragrance chemicals in cosmetic products.[65]

Clinical features: ACD presents as pruritic papules, erosions or eczematous plaque which may extend beyond the borders of the region exposed to the allergen. Anatomical regions involved like the scalp, face, lips, neck, trunk, may provide clues to the allergen.[66]

Diagnosis: Patch testing remains the gold standard and a valuable testing tool in the diagnosis of ACD in children.[66]

But there is no Food and Drug Administration-approved commercially available patch test tool designated for patients less than 18 years of age.[66]

Table 7: Common allergens in children and adolescents:[67]

Allergen	Description	Source	Frequent distribution
Nickel	Metal	Jewellery, buckles and snaps, eyeglasses, orthodontics, cell phones, keys, coins	Face/eyelids, earlobes, neck, wrists
Neomycin	Topical antibiotic	Antibiotic ointments, combinations with corticosteroids	Face/eyelids
'Balsam of Peru'/fragrance mix	Fragrance	Perfumes and cosmetics, flavoring agent, toothpaste, lozenges and naturally cross-reacts with tomato and cinnamon	Eyelids/face, neck, mouth/lips
Thimerosal	Preservative	Vaccinations, creams, lotions, mascara	Body/torso
Cobalt	Metal	Jewellery, buttons/snaps, ceramics, cement, vitamin B12	Earlobes, neckline, umbilical area, hands
Chromate	Metal	Tanned leather, matches, cement, paints, green felt	Hands, umbilical area
Thiuram	Rubber accelerant	Elastic waistbands, socks, swimsuits, shoes (soles or insoles), gloves, pesticides	Waistline, feet, hands
Lanolin	Emollient	Emollients, rust-preventive waxes, soaps, lip balms	Hands, any body area with emollient use
Formaldehyde/formaldehyde-releasing products	Preservative	Shampoos, lotions, cosmetics, newsprints, cosmetics, clothing – wrinkle-resistant	Face, ears, hands, trunk
Paraphenylenediamine	Oxidative chemical	Hair dye, printer inks, black henna tattoos	Hairline, ears, hands, tattoo sites

Treatment:

1) Emollients: restore the barrier function and relieve pruritis
2) Topical corticosteroids: form the first line therapy.
3) Topical calcineurin inhibitors
4) Systemic therapy: systemic corticosteroids in severe cases and oral anti-histaminics for symptomatic relief.[66]

2)Irritant contact dermatitis: ICD is the most common form of occupational dermatitis. It includes 3 types based on the irritant and its exposure pattern.[68]

-Subjective irritancy: Idiosyncratic stinging which occurs within minutes of contact, usually on the face, in the absence of visible changes often secondary to cosmetic or sunscreen constituents. [68]

-Acute irritant contact dermatitis: Occurs secondary to single overwhelming exposure or a few brief exposures to strong irritants or caustic agents. e.g. concentrated acids, strong alkali, organic and inorganic salts. [68]

-Chronic (cumulative) irritant contact dermatitis: Occurs following repetitive exposure to weaker irritants such as detergents, soaps, weak acids, and alkalis, powders, and dusts. It is due to stepwise progression of damage to the barrier function of the skin and presents with dry, scaly fissuring, and eczematous lesions on the fingers and hands. [68]

Management of ICD involves treatment of the active lesions with topical corticosteroids, emollients and prevention by using personal protective measures. [68]

3)Contact urticaria: It is an immediate reaction from exposure to a substance and is mediated by either irritant or immunological mechanisms; it can resemble ICD but the onset is immediate and short lived. [68]

Dyshidrotic eczema:

Introduction: It is also known as dyshidrotic dermatitis or pompholyx, and is characterized by pruritic, tense, deep-seated vesicles mainly on the palms and lateral surfaces of the fingers.[69]

Etiology: The exact etiology is unknown. Most cases are idiopathic. Predisposing factors include atopy, contact allergens, contact irritants, dermatophyte infection, allergy to

ingested metal (nickel and cobalt), hyperhidrosis, prolonged use of protective gloves, intravenous immunoglobulin, psychological stress, and smoking. [69]

Clinical features: Clinically, it presents with a sudden onset of symmetrical, pruritic, deep-seated "tapioca-like" vesicles that affect primarily the palms and lateral surfaces of the fingers, hands being more common than the feet. In the chronic phase, scaling, desquamation, fissuring, and, sometimes, lichenification may be seen. [69]

Management: Hydration of the skin helps to improve the dryness and pruritus and restore the disturbed skin's barrier function. A moisturizer or emollient should be applied as soon as possible after hand-washing to prevent evaporation of water and to keep the skin soft. Excessive use of soaps and detergents should be avoided. [69]

Ultrapotent topical corticosteroids are the mainstay of therapy applied once or twice daily and topical calcineurin inhibitors may be prescribed in the maintenance phase of treatment. [69]

Refractory cases: Systemic corticosteroids, immunosuppressives, psoralen plus ultraviolet A (PUVA), UVA1 and UVB. Intradermal botulinum toxin A can be used in patients with concomitant hyperhidrosis. Anti histaminics: to relieve pruritis. [69]

Seborrheic dermatitis (SD):

Introduction: It is a common inflammatory disorder of the skin, characterized by erythema covered with greasy-looking scales commonly involving areas rich in sebaceous glands namely, the scalp, face, chest, back and flexural areas.[70]

Epidemiology: SD occurs most commonly in infants within the first 3 months of life, in adolescents and young adults, with the incidence increasing again in patients older than 50 years of age.[71,72] SD is being recognised to have a substantial negative effect on the patient's quality of life (QoL).[73]

Etiology: SD is a multifactorial skin disease that is predisposed by endogenous and exogenous factors.[74]

a) Hormonal influence: Mainly by androgens. The age prevalence of SD coincides with the period of life when sebaceous glands are most active; moreover, SD lesions are located in sebaceous gland-rich body areas. [74]
b) Malassezia infection is thought to play an etiological role: By-products may cause inflammation by inducing cytokine production by keratinocytes or through involvement of langerhans cells and T-lymphocyte activation. [74]

Clinical features: Erythema and scaling distributed on the scalp, nasolabial folds, eyebrows, post auricular areas, and the sternum. The distribution of lesions is generally symmetrical. Dandruff can be considered a mild form of SD, with scalp scaling and/or mild to marked erythema of the nasolabial fold during times of stress. [74]

Treatment: Topical: Antifungals (ketoconazole 2% shampoo once or twice a week, ciclopirox 1% cream), corticosteroids (low-mid potency, either alone or in combination with antifungals), zinc pyrithrone 1% shampoo, selenium sulphide 2.5% shampoo, coal tar shampoos, metronidazole cream 0.75%, topical calcineurin inhibitors.[75]

Phototherapy: NBUVB given 3 times/week for 2 months or till clearing is shown to be effective. The direct inhibitive effect of UVA and UVB light on Malassezia yeasts cultured from the skin has been experimentally confirmed. [75]

Systemic therapy: Oral antifungals have been used in selected cases of extensive SD resistant to topical therapies. [75]

Polymorphic light eruptions:

Introduction: It is the most common endogenous photodermatosis which affects men and women of all ages but more common in women in their second and third decade and adolescents due to their increased tendency for outdoor activities may be more at risk. Fair-skinned individuals with Fitzpatrick skin types I-IV are more susceptible.[76]

Etiopathogenesis: The cause of PMLE is unknown, although an immunologic basis has been suggested. There appears to be a delayed type hypersensitivity (DTH) response to undefined endogenous, cutaneous photo induced antigens. Most commonly occurs in the action spectrum of 290-365 and rarely visible light.[76]

Clinical features: PMLE manifests as pruritus, erythema, macules, papules, or vesicles on sun-exposed skin arising 1-2 days after exposure and resolving spontaneously over the next 7-10 days. It is most common with initial sun exposures during spring or early summer. With subsequent exposures, sensitivity reduces leading to tolerance which is termed hardening. The lesions are polymorphic, but individual patients tend to develop the same type each year.[76]

Treatment: It consists of avoidance of sun exposure, use of protective clothing, and regular application of broad-spectrum sunscreens.[76]

Mid potent topical corticosteroids in mild symptomatic disease. Short course of oral corticosteroids in severe cases.[76]

Pityriasis alba:

Pityriasis alba is quite a common pigmentary disorder commonly occurring in children but can also persist into adulthood.[77]

The exact aetiology is unknown but various factors like infection, nutritional factors, atopy and dryness of the skin have been implicated. It is considered as a mild form of atopic dermatitis with post inflammatory pigment dispersion.[77]

Clinical features: They present as well defined or ill-defined, hypopigmented macules with fine superficial scaling. Uncommon variants include pigmenting P.alba and generalised P.alba.[77]

Differential diagnosis: Vitiligo, nevus depigmentosus, leprosy, pityriasis versicolor and post inflammatory hypopigmentation.[78]

Treatment: Most important intervention is reassurance that this self-limited and non-permanent hypopigmentation will eventually resolve. Other forms of treatment include moisturisers and sunscreen, mid-potency topical corticosteroids and topical calcineurin inhibitors.[79]

<u>PAPULOSQUAMOUS DIORDERS:</u>

Psoriasis:

Introduction: It is a chronic life-altering skin disorder with possible systemic comorbidities. It affects a considerable proportion of patients in childhood and adolescence

and its management in them poses some challenge due to the lack of officially approved therapies and standardized methodology.[80]

Epidemiology: Affects 2%–4% of the world's population.[80]The prevalence of the disease in childhood and adolescence ranges between 0.5% and 2%.[81,82,83]

Etiopathogenesis: Psoriasis is a chronic T-cell-mediated inflammatory disease characterized by keratinocyte hyperproliferation, vascular endothelial proliferation, and inflammatory cell infiltration of the dermis and the epidermis. Its pathogenesis is based on a complicated interplay of genetic and environmental factors.[80]

The juvenile population is more influenced by intrinsic and extrinsic environmental factors in the precipitation and/or the exacerbation of psoriasis than in adults. Streptococcal infections from group A β-hemolytic streptococcus (pharyngitis or perianal dermatitis) frequently trigger the appearance of guttate psoriasis in children, while stressful events induce new psoriatic lesions more commonly in this age group due to their emotional immaturity.[80]

Differences in the clinical features between the juvenile and adult population:

Plaque-type psoriasis is the most common form of the disease in the juveniles, but the lesions are usually smaller, thinner, and less scaly than those seen in adults. The scalp, face and extensors are the most affected areas.[80,84]

Guttate disease generally more often presents during childhood and adolescence than in adulthood and is characterized by small nummular scaly lesions located on the trunk, abdomen, and back. Prior to the onset of this condition in children, a preceding streptococcal pharyngitis or perianal infection is common.[80,84]

Erythrodermic, pustular, and inverse psoriasis may also occur in adolescents, but less frequently.[80,84]

Nail psoriasis can be presented either in the background of plaque psoriasis or psoriatic arthritis or as isolated nail disease. They include nail pits, discoloration, onycholysis, subungual hyperkeratosis, onychodystrophy, and splinter hemorrhages.[80,84]

The frequency of remissions has also been found to be greater in pediatric-onset psoriasis than in adult-onset disease.[80,84]

Treatment: The management of psoriasis in adolescence is an intriguing and complicated task because of the lack of standardized guidelines. Even relatively mild forms of the disease could have a major impact on the quality of life of children and adolescents, and hence impair their psychological development.[80,85]

Patients and their families should be given social and psychological support along with appropriate education about the nature of the disease, to achieve treatment compliance. It must be clarified that psoriasis does not have a permanent cure, and therefore the main goal of treatments is to establish disease control and prolong the periods between flares.[80,84]

Topical therapy: Includes emollients and moisturisers, corticosteroids, calcineurin inhibitors, vitamin D_3 analogues.[80,85]

Phototherapy: General indications for phototherapy in adolescence are the presence of disseminated guttate lesions or thin plaques, lesions refractory to combination topical therapy, and difficult-to-treat palmoplantar psoriasis. Cumulative dosing of UV treatments, which has been linked in some cases with the possible long-term risk of carcinogenesis should be considered especially when treating children and adolescence. Narrow band UVB

is considered the most efficacious and safe type of phototherapy for children in moderate to severe psoriasis. UVA plus psoralen must be prescribed only to children older than 12 years.[80,85]

Systemic therapy: Classic antipsoriatic drugs like acitretin, methotrexate, or ciclosporine are also used in children.

Biologics: Etanercept is the only biologic agent officially approved for the treatment of childhood plaque psoriasis. Until now, biologic agents have been considered second- or even third-line agents for recalcitrant juvenile psoriasis, mainly because of possible unknown long-term safety issues (possible increased risk of lymphoma in the pediatric population).[80,85]

Lichen planus:

Lichen planus is a common disorder, usually found in all age groups.[39]

Etiopathogenesis: Activation of the cell-mediated immune response destined towards keratinocyte apoptosis is the prime event in the pathogenesis of LP.[39]

Clinical features: The characteristic findings of classical LP are small flat-topped pruritic polyglonal purplish papules distributed mainly over the flexural surfaces of the extremities. Lesions are faintly erythematous to begin with and later turn violaceous over a few weeks. Papules may develop at the site of trauma (Koebner's phenomenon).[39]

The other variants of lichen planus are bullous LP, actinic LP, annular LP, atrophic LP, hypertrophic LP, linear LP, ulcerative LP, LP pemphigoid, LP lupus erythematosus and lichen planopilaris. Bullous LP is usually seen in age group of 13-17years.[39]

Linear LP is the most common variant in pediatric patients.[39]

Oral mucous membranes may be commonly involved which characteristically shows lacy reticulate pattern defined as “Wickham’s striae”. The buccal mucosa, lip and palate are the most commonly affected areas.[39]

Nail involvement is uncommon in pediatric patients with lichen planus, but may present with pitting, lusterless nail with thinning nail plate, longitudinal ridging and twenty-nail-dystrophy.[39]

Differential diagnosis: Includes lichen striatus, lichen nitidus, lichen simplex chronicus, lichenoid drug eruption and papular granuloma annulare.[39]

Treatment: Moderate to potent topical corticosteroids and antihistamines. Other treatments include topical tacrolimus, oral dapsone, PUVA or UVB light therapy, oral retinoid, cyclosporine.[39]

Keratosis Pilaris:

It is an autosomal dominantly inherited disorder characterized by minute, gooseflesh-like, horny plugs at the follicular orifices, distributed on the posterolateral aspect of the thighs, upper arms, buttocks and legs.[39]

Usually associated with icthyosis and atopy.[39]

The disease generally manifests at the age of 2–3 years, remains until the age of 20 and then subsides gradually.[39]

Treatment consists of topical application of mild keratolytics like 3% salicylic acid ointment or 0.1% tretinoin.[39]

DISORDERS OF PIGMENTATION:

Vitiligo:

Introduction: Vitiligo is defined as an acquired cutaneous achromia characterized by milky white macules of various sizes and shapes that tends to enlarge peripherally in course of time.[86,87]

Epidemiology: Vitiligo occurs worldwide with an overall prevalence of 1%. Its incidence ranges from 0.1 to > 8.8% in different countries.[88,89,90,91] The highest incidence of the condition has been recorded in Indians, followed by Mexico and Japan. The difference in its incidence may be due to a higher reporting of vitiligo in a population, because of the apparent colour contrast and stigma attached to the condition forcing them to seek early consultation.[86,87]

Vitiligo usually presents itself in childhood, adolescence or young adults, approximately half to one third of them develop this condition by 20 years of age. Childhood vitiligo differs from the adults by showing a higher incidence in females, segmental vitiligo being more common and less frequent association with other systemic autoimmune and endocrine disorders.[91]

Studies have shown a greater incidence of the disease in females but it could also be attributable to greater aesthetic concern and social consequences among them.[92]

Childhood and adolescent vitiligo is often associated with a marked psychosocial and long lasting effect on the self-esteem of the affected children and their parents, hence an adequate treatment is very essential.[92]

Etiopathogenesis: The cause of vitiligo still remains unknown, although it is clear that several different pathophysiologic processes may be involved.[93]

The autoimmune hypothesis is best supported because of the numerous genetic association and genetic linkage studies, in combination with humoral and cellular immune aberrancies.[93]

Other theories: Neurohumoral, cytotoxic, and oxidative stress theories with moderate evidence.[93]

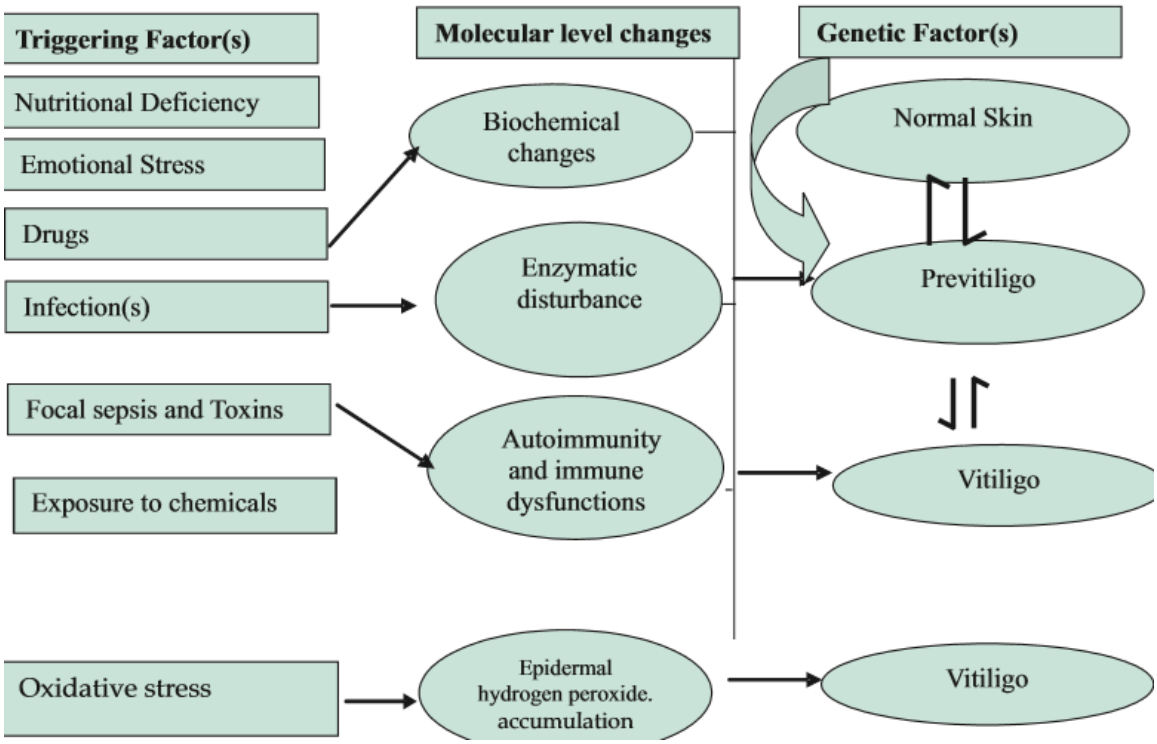


Figure 1: Etiopathogenesis of vitiligo.[86]

Clinical features: Vitiligo is characterized by the appearance of asymptomatic or mildly pruritic patchy discoloration in the form of typical chalky-white or milky macule(s) which are round or oval in shape, often with scalloped margins, varying in size from a few millimetres to several centimeters.[86]

Vitiligo is a slow and progressive disease and may have remissions and exacerbations correlating with triggering events.[86]

Although any part of the skin and / or mucous membranes is can develop vitiligo, the disease has a predilection for normal hyper pigmented regions such as the face, groin, axillae, areolae and genitalia and other areas like the ankles, elbows, knees, which are subjected to repeated trauma / friction, an outcome of koebner's phenomenon.[86]

Lip-tip syndrome, a variant of vitiligo is characterized by depigmentation of the terminal phalanges and the lips.[86]

Table 8: Classification of vitiligo.[86]

1) Segmental	**Features**
Vitiligo zosteriformis	Macules distributed along a dermatome or a near dermatome or lines of body cleavage
1) Non segmental	
-Vitiligo areata	1-2 macules
-Vitiligo vulgaris	Generalized macules involving extensive body areas
-Vitiligo acrofacialis	Macules affecting the face and tips of the hands and feet
-Mucosal vitiligo	An exclusive involvement of mucous membranes

Treatment: (1) Therapies to repigment small surface area (<20%): topical corticosteroids, calcineurin inhibitors, excimer laser therapy.

(2) Therapies to stabilize vitiligo: NBUVB, PUVA, short course oral corticosteroids, pseudocatalase

(3) Other therapies in vitiligo: Depigmentation for severe disease (>70%) in psychologically stable late adolescents, cosmetic camouflage, surgical interventions including mini-grafting, suction-blister grafting, melanocyte transfer therapy.[94]

Periorbital hypermelanosis (POHM):

Introduction: It is characterised by bilateral darkening of the eyelid and orbital skin due to excessive melanin deposits that contrasts with the adjoining facial skin. Hyperchromia of the orbital region can be subdivided into primary and secondary types.[95]

Primary hypermelanosis is also called idiopathic cutaneous hyperchromia at the orbital region (ICHOR) which is characterised by bilateral darkening of the eyelid and orbital skin that is not secondary to systemic diseases.[95]

The secondary type is hyperchromia secondary to environmental factors such as excessive exposure to the sun, endogenous or use of exogenous estrogens, post inflammatory hyperpigmentation, excessive subcutaneous vascularisation and hyper transparency of the skin.[95]

It is of great aesthetic concern for patients because dark circles interfere with the face appearance, giving the patient a tired, sad look which often causes difficulties in acceptance by the society and may impact quality of life.[95]

Force of gravity acting on an area with little subcutaneous tissue and little support causes the skin to move downward, thereby becoming stretched and thinned and allowing the underlying vessels and orbicular musculature to become more apparent. Excessive

exposure to ultraviolet radiation causes skin thinning and increases melanin content, thus favouring the darkening of this region.[95]

The degree of hyperchromia appears to vary with some factors such as fatigue, menstrual cycle, bouts of flu and other situations of physical and emotional stress which is more common in adolescent girls. This indicates that a dynamic factor may be present.[95]

Treatment: Includes avoidance of aggravating factors, routine use of broad spectrum sunscreens, mild depigmenting agents. Lactic acid peels can also be done in severe cases.[95]

Post inflammatory hyperpigmentation (PIH):

Introduction: Post-inflammatory hyperpigmentation (PIH) of the skin is a pigmentary disorder that is seen in all skin types but mostly common in dark skin (Fitzpatrick types IV to VI), resulting in lesions that can persist for months or years which can be psychologically devastating to few patients.[96]

PIH can be caused by endogenous inflammatory skin disorders (acne, infections, eczemas) or iatrogenic sources (lasers) and may be transient or long lasting.[96]

Clinically PIH presents as irregular, darkly pigmented macules and patches at sites of previous injury or inflammation, histopathologically characterized by increased melanocytic activity and dermal melanophages. This condition tends to be worse in patients having preceding inflammatory disease, such as lichen planus and lupus erythematosus which has disrupted the basal layer of the epidermis.[96]

Treatment: It is often difficult, requiring prolonged courses of therapy and excellent patient compliance. Available methods of treatment for post inflammatory

hyperpigmentation include hydroquinone 3% or 4% twice daily, azelaic acid 20% cream twice daily, salicylic or glycolic acid peels, retinoids, and laser therapy.[96]

MISCELLANEOUS:

Urticaria:

Introduction: Urticaria (or 'hives' or 'nettle rash') consists of blancheable, erythematous, oedematous papules or 'wheals' which vary in size from 1 mm to many centimetres, are usually intensely itchy and transient lasting only a few hours. They occur as a result of vasoactive mediators, predominantly histamine, released from mast cells.[97]

Urticaria is a common condition with an estimated lifetime incidence of approximately 15%, with females being affected more often than males. Both children and adults may develop urticaria, with the peak age of onset in adults being between 20 and 40 years.[97]

Urticaria is defined as 'acute' if it lasts for less than 6 weeks and 'chronic' if it lasts for more than 6 weeks. Most urticarial reactions are acute and self-limiting; but patients referred to allergy clinics usually have chronic urticaria or episodic urticaria. The causative factor is easily identified in acute urticaria whereas it is difficult in chronic urticaria.[97]

Pathogenesis: Urticaria results from the release of histamine, bradykinin, leukotriene C4, prostaglandin D2, and other vasoactive substances from mast cells and basophils in the dermis. These substances cause extravasation of fluid into the dermis, leading to urticarial lesions. The intense pruritus of urticaria is a result of histamine released into the dermis. Histamine is the ligand for 2 membrane-bound receptors, the H1 and H2 receptors, which are present on many cell types. The activation of the H1 histamine receptors on endothelial

and smooth muscle cells leads to increased capillary permeability. The activation of the H2 histamine receptors leads to vasodilation.[98,99,100]

Etiology: Acute urticaria usually occurs secondary to the following causes:

1) Drugs: Act through allergic or non-allergic mechanisms. Penicillins, cephalosporins, sulfonamides and tetracyclines are some examples of drug - induced urticaria.
2) Infections: Hepatitis B viral infection, streptococcal throat infection, and Campylobacter jejuni.
3) Infestations: Gastro-intestinal parasites like ankylostoma, strongyloides, echinococcus and Toxacara canis are few examples.
4) Inhalants: Include grass, pollen, mould, spores, animal danders, and house dust.
5) Ingestion: Fish, milk, peanut, beans, potato, rice, carrot and drumstick are responsible for acute urticaria mediated through IgE dependant mechanism.
6) Arthropod bites: Bees or wasp sting can cause acute urticarial or even anaphylaxis.[99]

Chronic urticaria: Can be further divided into 3 subgroups:[98,100]

1. Chronic urticaria with potential provoking factors: Include drugs, food, infections collagen and endocrine disorders, dermatological disorders etc.
2. Autoimmune urticaria: Approximately 50% of adults and children with chronic urticaria have histamine-releasing autoantibodies directed against the alpha subunit of the IgE receptor on mast cells causing mast cell degranulation via activation of the classical complement pathway. They may be demonstrable by the autologous serum skin test (ASST).

3. Chronic idiopathic urticaria. (CIU): Most common form seen in about 50% of patients and includes urticaria in which all the known provocating factors and autoimmunity have been excluded. It is a diagnosis of exclusion.[98,100]

Evaluation of a patient with urticaria should include thorough history and investigations to rule out the triggering factors. [98,100]

Investigations include complete blood count, ESR, urine routine and microscopy, stool for cysts and ova, thyroid profile, autologous serum skin test.[98,100]

Treatment: General measures: Avoidance of identifiable causes, frequent tepid showers and application of 1% menthol or calamine in aqueous cream/lotion can be prescribed as cooling agents.[98,100]

Treatment of underlying disorders and symptomatic therapy which includes H-1 antihistaminics, mast cell stabilizers, leukotriene antagonists.[98,100]

Immunotherapy and intravenous immunoglobulins can be given in cases of autoimmune urticaria.[98,100]

Keloids and hypertrophic scars:

Introduction: Keloids and hypertrophic scars are the result of abnormal healing responses and dermal tissue proliferation.[101]

A keloid is an abnormal proliferation of scar tissue that forms at the site of cutaneous injury (e.g., on the site of a surgical incision or trauma) which does not show a tendency to regress,

and grows beyond the original margins of the scar. In contrast, hypertrophic scars are common after thermal injuries and other injuries that involve the deep dermis; are characterized by erythematous, pruritic, raised fibrous lesions that typically do not expand beyond the boundaries of the initial injury; and may undergo partial spontaneous resolution.[101]

Adolescents and young adults have increasingly acquired practices like body piercings over recent years. Piercing different body sites has been practiced globally by various cultures for centuries. As the popularity of piercings has grown, so has the list of complications. Associated health risks include infections, pain, bleeding, hematoma formation, cyst formation, allergic reactions, hypertrophic scarring, and keloid formation.[101]

Clinical features: Keloids vary in size from a few millimeters in diameter to several centimeters or larger and may be soft and doughy or firm. Areas particularly prone to keloidal scarring include ears, cheeks, shoulders, chest, upper arms, and upper back because of the increased skin tension in these areas.[102]

Pathophysiology: 1) Abberant fibroblastic activity

2) Role of growth factors: Mainly transforming growth factor β (TGF- β) released from platelets at the site of injury, which is a potent stimulator for collagen synthesis.

3) Immunological mechanisms: Altered cellular immune response is noted in keloids.[102]

Treatment: Pressure garments, radiation therapy with superficial X-rays, electron beam therapy, interstitial radiotherapy, excision, intralesional injections with triamcinolone-40mg/ml every 3-4weeks, 5-Fluorouracil- 50mg/ml every 4-6weeks, bleomycin, interferon

alpha, cryotherapy, silicon gel dressings, lasers like carbon dioxide laser, erbium-YAG laser, pulsed dye laser.[103]

Papular urticaria:

Papular urticaria is a characterised by recurrent pruritic papules or vesicles with varying degrees of edema resulting from a hypersensitivity reaction to biting, stinging insects. The severity of the eruption and pruritis are related to the host response to the salivary or contact proteins.[104]

Children seem to be predisposed to papular urticaria; possibly a reflection of immune mechanisms and/or behaviors that facilitate the encounters with the offending insects.[104]

Etiopathogenesis: Hypersensitivity reaction to bites of arthropods, including mosquitoes, flies, gnats, mites, ticks, and caterpillars.[104]

Immune complex deposition and complement activation is suggested in the pathogenesis.[104]

Clinical features: Characterised by pruritic erythematous papules ranging from 3 to 10 mm typically grouped in clusters on exposed area accompanied by excoriations and sometimes complicated by secondary infection distributed particularly over the extensor surfaces of extremities and constricting areas such as the tops of socks and around waistbands. Papules might also have a more diffuse, generalized distribution involving the torso, neck, and face. Perennial or seasonal exacerbations are common and are presumed to be associated with re-exposure to the offending arthropod.[104]

Treatment: Symptomatic treatment remains the cornerstone of management. Washing the involved skin promptly with soap and water often removes both the ecto parasite and the offending toxic or allergenic substance. Cool compresses provide symptomatic relief.[104]

Antihistamines are given for reducing the severe pruritis often accompanying papular urticaria. Topical corticosteroids, especially potent (class 1) steroids can be given. Secondary bacterial infection can be treated with local or systemic antibiotics.[104]

Prurigo:

Prurigo nodularis is a highly pruritic condition characterized by multiple hyperkeratotic nodules erupting on the extensor surfaces of the limbs secondary to itching or rubbing.[105]

Pathophysiology: Chronic mechanical trauma to the skin causes thickening of the skin proportionate to the trauma. Repetitive rubbing, scratching, and touching (induced by a foreign body or self-induced) results in plaque or nodular lichenification, hyperkeratosis and pigmentary changes. Lesional skin biopsies showed significantly decreased intraepidermal nerve fibre density, suggesting the presence of a subclinical small fibre neuropathy in prurigo nodularis.[105]

Th2 cytokines related to STAT6 activation together with some unknown stimuli that activate STAT3 play a principal role in the pathogenesis of prurigo nodularis.[106]

Clinical features: They present as nodules or papules are 3-20 mm in diameter; they are discrete, scaly, generally symmetric, hyper pigmented or purpuric, on the extensor surfaces of the arms, the legs, and sometimes the trunk.[105]

Management: Emollients, steroids mainly high potent topical, intralesional and oral therapies, thalidomide, gabapentine and PUVA therapy have been tried.[105,107]

Sexually transmitted infections:

Introduction: There is an increased risk of adolescent girls developing sexually transmitted infections(STIs) for both biologic and social reasons. Biologically, they have increased cervical ectopy, which increases susceptibility to infection with STI's. Socially, they often lack enough strength and skills to refuse sexual activity or to demand safer sex, rendering them particularly vulnerable to STI's.[108]

Epidemiology: Although the traditional and cultural norms of India oppose premarital sex, some studies indicate a growing trend towards premarital sexual activities among adolescents. An important aspect of neglected adolescent girls is that they are married early and have their first child in their teens.[109] Majority of Indian women marry as adolescents, even though the legal age for marriage is 18. A study has shown that 30% of girls aged 15-19 are currently married or in union, compared to only 5% of boys of the same age.[110]

In India, young women with low socio-economic status are seven times more likely to give birth before age 18 than young women with higher socio-economic status. Hence they are extremely susceptible for acquiring a sexually transmitted infection.[110]

A study done in rural Tamil Nadu showed that each year nearly 1.3 million women die of reproductive health problems that are largely preventable and 1 out of 20 teenagers contract a sexually transmitted disease, some of which causing lifelong disabilities such as infertility or death.[111]

Some of the common sexually transmitted infections in India are:

Vulvo vaginal candidiasis (VVC):

VVC usually is caused by Candida albicans, but occasionally is caused by other Candida species or yeasts.[112]

Clinically they present with pruritus, vaginal soreness, dyspareunia, external dysuria, and abnormal vaginal discharge. In case of recurrent VVC, patients experience four or more episodes of vaginitis per year.[112]

Signs include vulvar edema, fissures, excoriations, or thick, curdy vaginal discharge.[112]

Diagnosis: 10% KOH or gram stain of vaginal discharge demonstrates yeasts, hyphae, or pseudohyphae. Cultures can be done to isolate the species.[112]

Treatment: Single dose of tablet fluconazole 150mg is usually sufficient. Topical antifungals like 1%-2% clotrimazole cream, 2%-4% miconazole cream are effective in uncomplicated cases.[112]

In cases of recurrent VVC, to maintain clinical and mycological control, longer duration of initial therapy is recommended. i.e. 7–14 days of topical therapy or a 100-mg, 150-mg, or 200-mg oral dose of fluconazole every third day for a total of 3 doses [day 1, 4, and 7]. Oral fluconazole (i.e., 100-mg, 150-mg, or 200-mg dose) weekly for 6 months is instituted as a maintenance therapy.[112]

Genital herpes simplex infections:

Genital herpes is a chronic, life-long viral infection. Two types of HSV have been identified as causing genital herpes: HSV-1 and HSV-2, common among them being HSV-2.[112]

Patients are often asymptomatic but shed virus intermittently in the genital tract. Very few patients present with the classical signs of multiple painful vesicular lesions over the genital area. As a result, the majority of genital herpes infections are transmitted by persons being unaware of it.[112]

Both virologic tests like cell culture and PCR, and type-specific serological tests are required to make a diagnosis but are not available in routine laboratories.[112]

Treatment: Primary episode: Acyclovir 200 mg 5 times per day, Famciclovir 250 mg t.i.d, Valaciclovir 500 mg b.d for 5 days.[112]

Recurrent episode: Episodic treatment Acyclovir 400 mg orally three times a day for 5 days, Famciclovir 500 mg once, followed by 250 mg twice daily for 2 days. Suppressive therapy: Acyclovir 400 mg orally twice a day, Valacyclovir 1 g orally once a day for 6-12 months and then reassess disease activity.[112]

Human papilloma virus infection (HPV): Most HPV infections are asymptomatic, unrecognized, or subclinical. Oncogenic, or high-risk HPV types (e.g., HPV types 16 and 18), are the cause of cervical cancers. Non-oncogenic, or low-risk HPV types (e.g., HPV types 6 and 11), are the cause of genital warts and recurrent respiratory papillomatosis.[112]

In the absence of lesions, treatment is not recommended for subclinical genital HPV infection whether it is diagnosed by colposcopy, acetic acid application, or by tests for HPV DNA.[112]

Genital warts can be treated with topical podophyllotoxin b.d. for 3 days a week for 4 to 6 courses. Topical 5% imiquimod 3 times per week for up to 16 weeks. Cryotherapy, curettage and cauterization, excision can also be done.[112]

Genital contact allergy:

The genital area is exposed to various allergens and irritants due to hygienic and sexual practices. Irritants cause more intense reactions on vulval epithelium than nongenital skin due to higher transepidermal water loss, capacitance, and blood flow in vulva. Usually, low-grade erythema of vulva is not readily apparent because of pigmentation of skin of vulva and the patient may only complain of burning and stinging of vulva, but examination may not reveal dermatitis.[113]

Genital allergy can occur as a result of non-sexual and sexual related conditions. Non-sexual causes include topical medications, feminine hygiene sprays, bubble baths, scented soaps, douches and sexual causes include seminal fluid, spermicides, latex.[113]

Genital allergy should be considered as a possible diagnosis in all patients with genital soreness or irritation for which no infection or dermatosis can be identified and in whom symptoms remain unchanged or worsen with treatment. Diagnosis should be made by thorough history and patch testing.[113]

Avoidance of potential sensitizer is the optimal approach to management.[113]

Adolescent attitudes and practices:

Body piercing:

Body piercing is a well-known practice of body ornamentation since centuries mainly connected with cultural norms and a sign of social status but has recently gained popularity among the adolescents due to fashionable modifications and as a way to express their individuality and make a personal statement.[114]

As the popularity of piercings has grown, so has the list of complications. The associated risks include infections, pain, bleeding, hematoma formation, cyst formation, allergic reactions, hypertrophic scarring, and keloid formation. Staphylococcus aureus is the most commonly reported causative organism in piercing-related infections; it causes varied type of conditions ranging from local site infections, such as impetigo and cellulitis, to more extensive infections including osteomyelitis, toxic shock syndrome, and bacteraemia.[114]

Life-threatening bacterial infections can also be associated with body piercing which include septic arthritis, acute glomerulonephritis, and endocarditis.[114]

Site-specific risks include auricular chondritis following ear piercing most commonly by pseudomonas aeruginosa.[114]

Most important infection-related concern secondary to body piercing is the potential risk of transmission of viral hepatitis and human immunodeficiency virus infection.[114]

Artificial tanning:

Artificial tanning is popular with adolescent girls who undergo such procedures to obtain a dusky complexion which is assumed to be more appealing.[115]

Artificial ultraviolet radiation (UVR) exposure can lead acute health effects such as erythema, sunburn, skin dryness, pruritus, nausea, disease exacerbation in case of systemic lupus erythematosis, and disease induction in cases like polymorphous light eruption.

Long-term health effects include skin aging, effects on the eye such as cataract formation, and most dangerously carcinogenesis.[115]

MATERIALS AND METHODS

MATERIALS AND METHODS:

Source of data: All girls aged between 10 and 19 years attending the out-patient department of dermatology, venereology and leprosy in R L Jalappa Hospital and Research centre, Kolar.

WHO defines the period of adolescence between 10 and 19 years. It is divided into early adolescence (10-13), middle adolescence (14-16), late adolescence (17-19).[1]

The patients were categorised into those from urban or rural background depending on the Census of India report, 2011.

The educational status of the patients was assessed based on the report 'National education for all – 2014: review for India'.[116]

Method of collection of data:

Inclusion criteria:

- All girls aged between 10 and 19 years with any dermatological complaint/disorder developing during adolescent period.

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 as well as adulthood.

Study design:

It is an observational study which was done from January 2015 to July 2016 for a period of one and half years. Sample size was estimated based on the anticipated frequency of the

occurrence of dermatoses among adolescent girls as 50%. Based on the above hypothesis, a minimum sample size of 200 was arrived at. However, 231 adolescent girls were included in the study.

A detailed history of the patient was taken including name, age, nature and duration of illness, predisposing factors like drug intake, topical application of medicines and cosmetics etc. Photographs were taken for documentation of the lesions. Informed consent for inclusion into the study as well as for taking photographs, was taken for all the patients.

A thorough cutaneous examination was done, recording the clinical pattern of the lesions, including any involvement of mucosa and genital areas.

To assess the severity of the disorders, various standardised scoring systems available for certain disorders were used as described below.

1) **Scoring system for acne vulgaris:**[117]

Grade 1: Comedones, occasional papules.

Grade 2: Papules, comedones, few pustules.

Grade 3: Predominant pustules, nodules, abscesses.

Grade 4: Mainly cysts, abscesses, widespread scarring.

2) **Scoring system for hyperhidrosis: hyperhidrosis disease severity score (HDSS)**[117]

Score 1: My sweating is never noticeable and never interferes with my daily activities.

Score 2: My sweating is tolerable but sometimes interferes with my daily activities.

Score 3: My sweating is barely tolerable and frequently interferes with my daily activities.

Score 4: My sweating is intolerable and always interferes with my daily activities.

3) **Scoring system for alopecia areata: National Alopecia Areata Foundation working committee has devised "Severity of Alopecia Tool score" (SALT score).**[117]

Scalp is divided into 4 areas, namely, Vertex - 40% (0.4) of scalp surface area; right profile of scalp - 18% (0.18) of scalp surface area; left profile of scalp - 18% (0.18) of scalp surface area; Posterior aspect of scalp - 24% (0.24) of scalp surface area. Percentage of hair loss in any of these areas is percentage hair loss multiplied by percent surface area of the scalp in that area. SALT score is the sum of percentage of hair loss in all above mentioned areas.

4) **Scoring system for atopic dermatitis: SCORing Atopic Dermatitis (SCORAD)**[117]

The SCORAD Index is a composite score based on 3 sub scores:

A = The extent score based on body surface area calculated using the 'Rule of 9'.

B = Intensity score based on 6 clinical findings in atopic dermatitis, namely erythema, edema or papulations, oozing or crusting, excoriation, lichenification, dryness, graded on a scale of 0 – 3 (0-absent, 1- mild, 2- moderate, 3- severe).

C = The score for pruritus and sleep loss graded on a visual analog scale of 0 to 10. The severity is based on the average extent for the last 3 days or nights.

Final formula for calculation of SCORAD is as follows: SCORAD = A/5 + 7(B/2) + C.

5) **Scoring system for dyshidrotic eczema: Dyshidrotic eczema area and severity index (DASI).**[117]

Based on the severity grade of single items –

V: Number of vesicles per square centimetre,

E: Erythema

S: Desquamation.

I: Itch

A: Extension of the affected area

DASI = (pV + pE + pS + pI) × pA.

6) **Scoring system for vitiligo: Vitiligo disease activity score (VIDA)**[117]

The VIDA is a six-point scale for assessing vitiligo activity. Scoring is based on the individual's own opinion of the present disease activity over time. Active vitiligo involves either expansion of existing lesions or appearance of new lesions.

Grading is as follows: VIDA Score

+4: Activity of 6 weeks or less duration.

+3: Activity of 6 weeks to 3 months.

+2: Activity of 3 - 6 months.

+1: Activity of 6 - 12 months.

0: Stable for 1 year or more.

-1: Stable with spontaneous repigmentation since 1 year or more.

7) Scoring system for psoriasis: PASI (Psoriasis area severity index).[117]

Four sites of affection, the head (h), upper limb (u), trunk (t) and lower limbs (l), are separately scored by using three parameters, erythema, induration and desquamation, each of which is graded on a severity scale of 0 to 4, where 0 = nil, 1 = mild, 2 = moderate, 3 = severe and 4 = very severe. The area wise percentage involvement of the involved sites is calculated as: 1 = less than 10% area; 2 = 1029%; 3 = 30-49%; 4 = 50-69%; 5 = 70-89%; and 6 = more than 90%.

The final formula for PASI score is: PASI = 0.1 (Eh + Ih + Dh) Ah + 0.2 (Eu + Iu + Du) Au + 0.3 (Et + It+ Dt) At + 0.4 (El + Il + Dl) Al.

8) Scoring system for urticaria: UAS (urticaria activity score):[117]

The UAS consisted of the sum of the wheal number score and the itch severity score.

The wheal numbers are graded from 0 to 3 as follows: 0 - less than 10 small wheals (diameter, < 3 cm); 1- 10 to 50 small wheals or less than 10 large wheals (diameter, > 3 cm); 2 - greater than 50 small wheals or 10 to 50 large wheals; and 3 almost the whole

body is covered. The severity of the itching is graded from 0 to 3 (0, none; 1, mild; 2, moderate; and 3, severe).

Various investigations were carried out whenever required. Informed consent was taken for all patients.

Socio economic status was classified using the following classification.

Table 9: Revised modified BG Prasad socioeconomic classification scale, January 2014.[118]

SOCIO ECONOMIC STATUS CLASS	PER CAPITA MONTHLY INCOME
UPPER CLASS – I	>5357 and above
UPPER MIDDLE – II	2652-5356
MIDDLE – III	1570-2651
LOWER MIDDLE – IV	812-1569
LOWER – V	<811

OBSERVATION AND RESULTS

RESULTS:

A total of 231 adolescent girls were examined in this study.

The socio-demographic characteristics of the study population is depicted in the following table.

Table 10: Age distribution of the study population.

Age group	Number of patients	%
Early adolescence	63	27.27
Mid adolescence	77	33.33
Late adolescence	91	39.39
Total	231	100

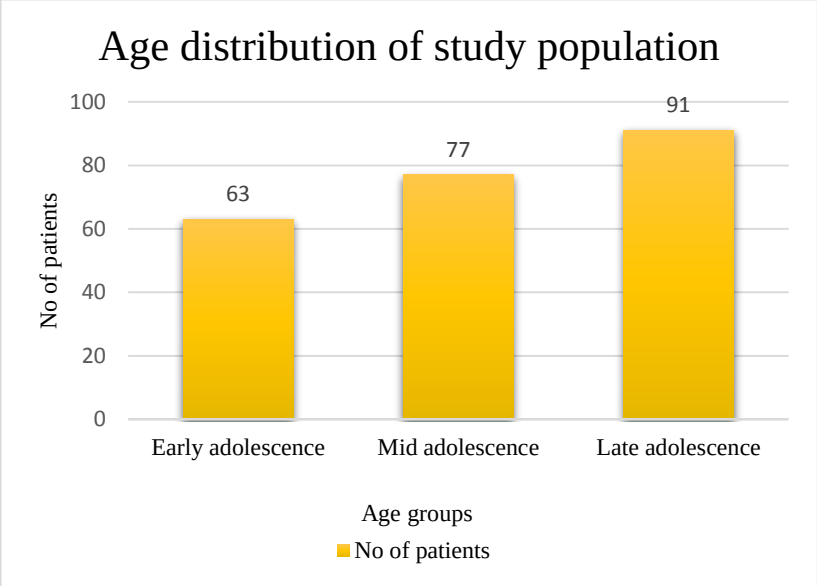


Figure 2: Age distribution of the study population.

Table 11: Educational status of the study population.

Educational status	No of patients	%
Primary	48	20.78
Secondary	65	28.14
Higher secondary	50	21.65
Undergraduate	43	18.61
Illiterate	25	10.82

Table 12: Socio economic status of the study population.

Socioeconomic status	No of patients	%
Class I	5	2.2
Class II	75	32.5
Class III	91	39.4
Class IV	51	22.1
Class V	9	3.9

Out of 231 patients, 5 belonged to the socio economic status class I, 75 belonged to class II, 91 belonged to class III, 51 belonged to class IV and 9 patients belonged to class V according to the modified BG Prasad's classification.

Table 13: Distribution of the study population according to urban/rural background.

Geographic distribution	No of patients	%
Urban (n = 144)	145	62.77
Rural (n = 87)	86	37.23

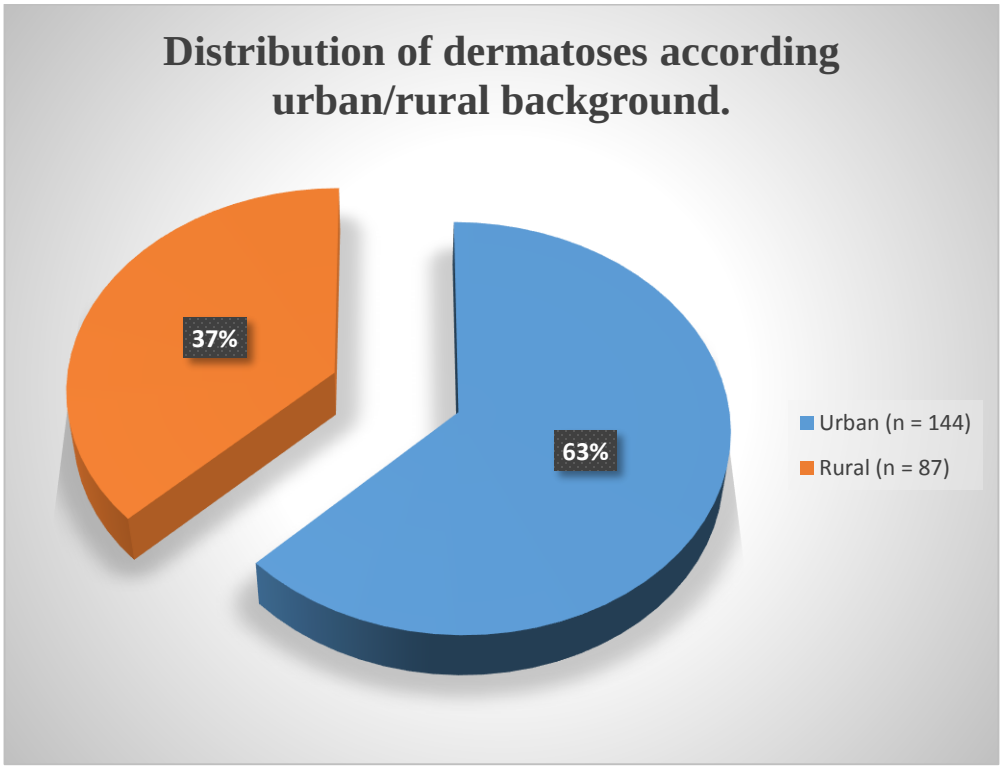


Figure 3: Distribution of the study population according to urban/rural background.

Table 14: Incidence of various dermatoses seen in the study population:

Types	No of patients	%
Appendageal disorders	70	30.30
Infections	52	22.51
Eczemas	43	18.61
Pigmentary disorders	23	9.96
Papulosquamous disorders	15	6.49
Miscellaneous	28	12.12
Total	231	100

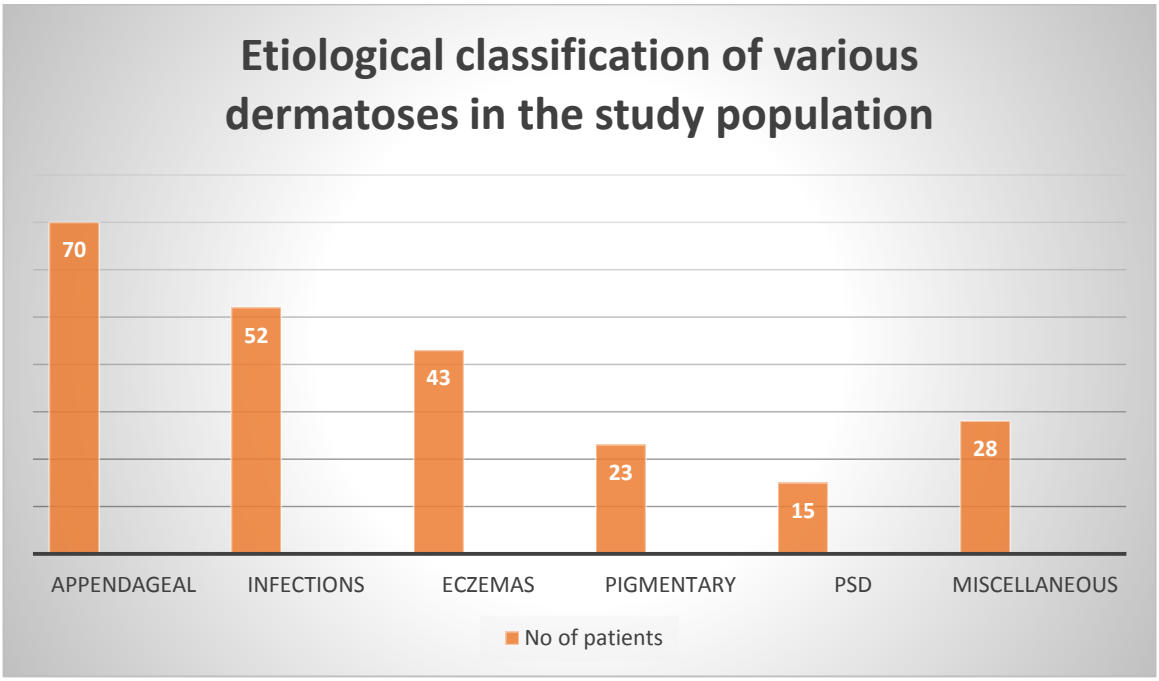


Figure 4: Incidence of various dermatoses seen in the study population.

Out of 231 patients, most common dermatoses were appendageal disorders seen in 70 patients (30.3%) followed by infections seen in 52 patients (22.51%), eczemas in 43 patients (18.6%), pigmentary disorders in 23 patients (9.96%), papulosquamous disorders in 15 patients (6.49%) and miscellaneous dermatoses in 28 patients (12.12%).

Table 15: Incidence of different dermatoses seen in the study population:

Diagnosis	**No of cases**	**%**
Appendageal disorders	**70**	**30.30**
Acne vulgaris	40	17.32
Acneiform eruptions	9	3.90
Alopecia areata	4	1.73
Palmar hyperhidrosis	4	1.73
Telogen effluvium	5	2.16
Premature graying of hair	2	0.87
Hirsutism	3	1.30
Trichotillomania	1	0.43
Miliaria rubra	2	0.87
Eczemas	**43**	**18.61**
Allergic contact dermatitis	8	3.46
Atopic dermatitis	11	4.76
Dyshidrotic eczema	4	1.73
Irritant contact dermatitis	1	0.43
P. alba	5	2.16
PMLE	10	4.33
Seborrheic dermatitis	4	1.73
Infections	**52**	**22.51**
Folliculitis	1	0.43
Furuncle	2	0.87
HSV	2	0.87
Impetigo	1	0.43
Molluscum contagiosum	2	0.87
Tinea	10	4.33
Verruca vulgaris	6	2.60
Onychomycosis	3	1.30
Pediculosis capitis	6	2.60
P.rosea	1	0.43
P. versicolour	5	2.16
Scabies	8	3.46
Varicella zoster	2	0.87
Genital candidiasis	2	0.87
Genital warts	1	0.43

Papulosquamous disorders	15	6.49
Psoriasis vulgaris	8	3.46
Lichen planus	4	1.73
Keratosis pilaris	3	1.30
Pigmentary disorders	**23**	**9.96**
Vitiligo	11	4.76
Periorbital melanosis	1	0.43
Periocular hypermelanosis	2	0.87
Postinflammatory hyperpigmentation	9	3.90
Miscellaneous	**28**	**12.12**
Insect bite reaction	6	2.60
Keloid	5	2.16
Phrynoderma	2	0.87
Prurigo simplex	5	2.16
Urticaria	10	4.33
Total	**231**	**100**

Table 16: Age group wise distribution of dermatoses.

Disorders	Early adolescence	Mid adolescence	Late adolescence	Total
Appendageal disorders	8	26	36	70
Infections	15	17	20	52
Eczemas	20	13	10	43
Papulosquamous disorders	5	3	7	15
Pigmentary disorders	6	10	7	23
Miscellaneous	9	8	11	28
Total	63	77	91	231

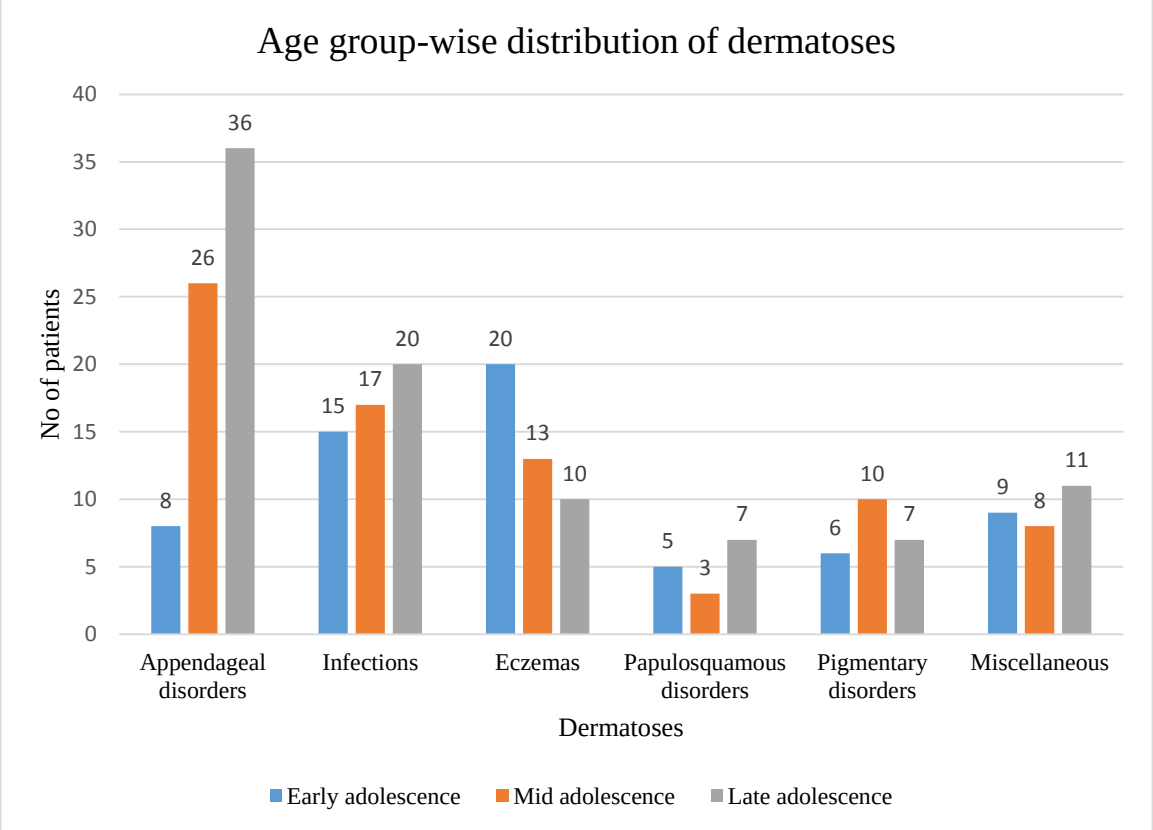


***Figure 5: Age group wise distribution of dermatoses*.**

Appendageal disorders, infections and miscellaneous dermatoses showed an increasing trend with age where as eczemas were common in the early adolescent age group.

Table 17: Distribution of dermatoses based on educational status:

Disorders	Primary	Secondary	Higher secondary	Undergraduate	Illiterate	Total
Appendageal disorders	5	22	20	22	1	70
Infections	13	12	11	4	12	52
Eczemas	17	14	6	5	1	43
Papulosquamous disorders	3	4	1	3	4	15
Pigmentary disorders	4	4	9	5	1	23
Miscellaneous	6	9	3	4	6	28
Total	48	65	50	43	25	231

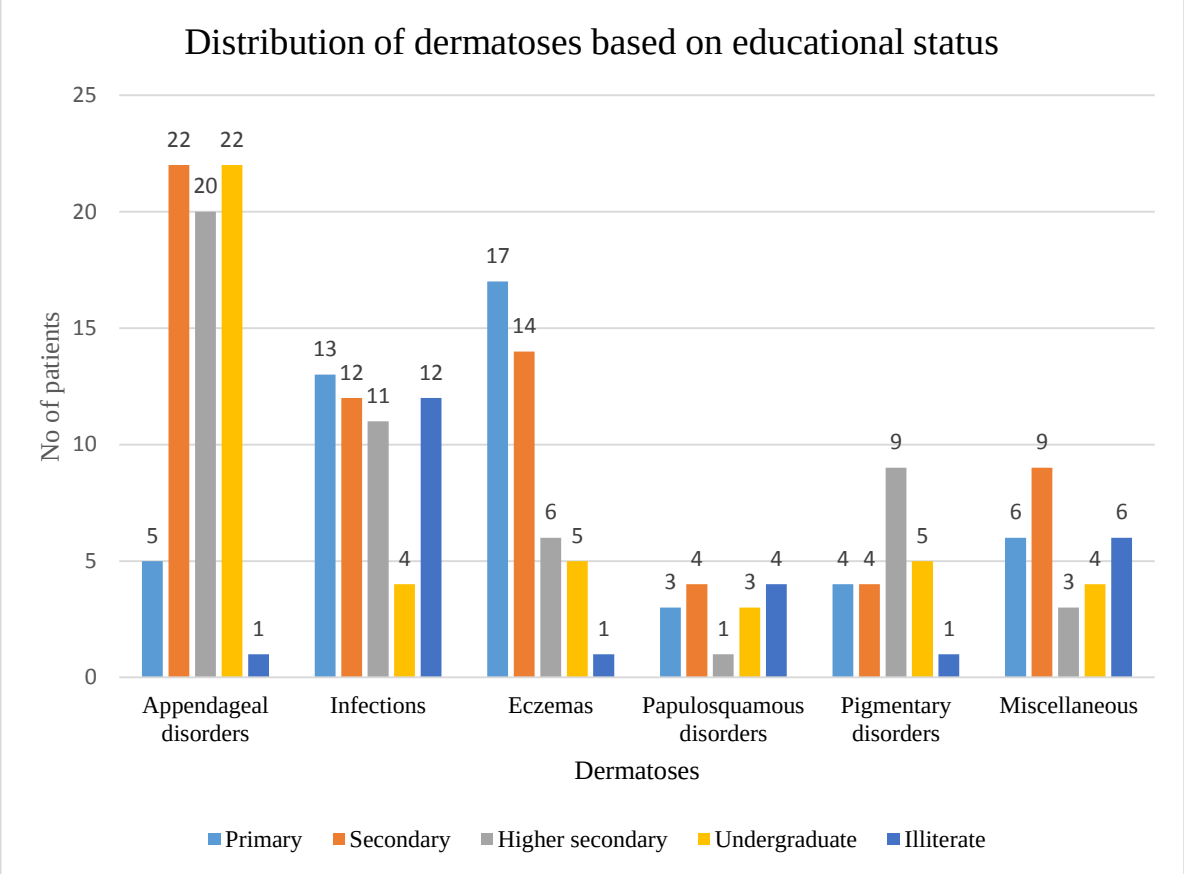


Figure 6: Distribution of dermatoses based on educational status.

Appendageal disorders showed an increasing tendency in the secondary, higher secondary and undergraduate students as compared to the primary students. Infections showed a higher incidence among the primary school children and among those who did not have any educational background as compared to students of other classes. It was interesting to note that all the 3 cases of STI's were in seen in girls with no educational background. Eczemas showed a higher incidence in the primary school children and then declined. Pigmentary, papulosquamous and miscellaneous diseases more or less involved similar numbers among girls of all educational status.

Table 18: Distribution of dermatoses according to the urban/rural background of the study population.

Disorders	Urban	Rural	Total
Appendageal disorders	51	19	70
Infections	20	32	52
Eczemas	28	15	43
Papulo squamous disorders	8	7	15
Pigmentary disorders	22	1	23
Miscellaneous	16	12	28
Total	145	86	231

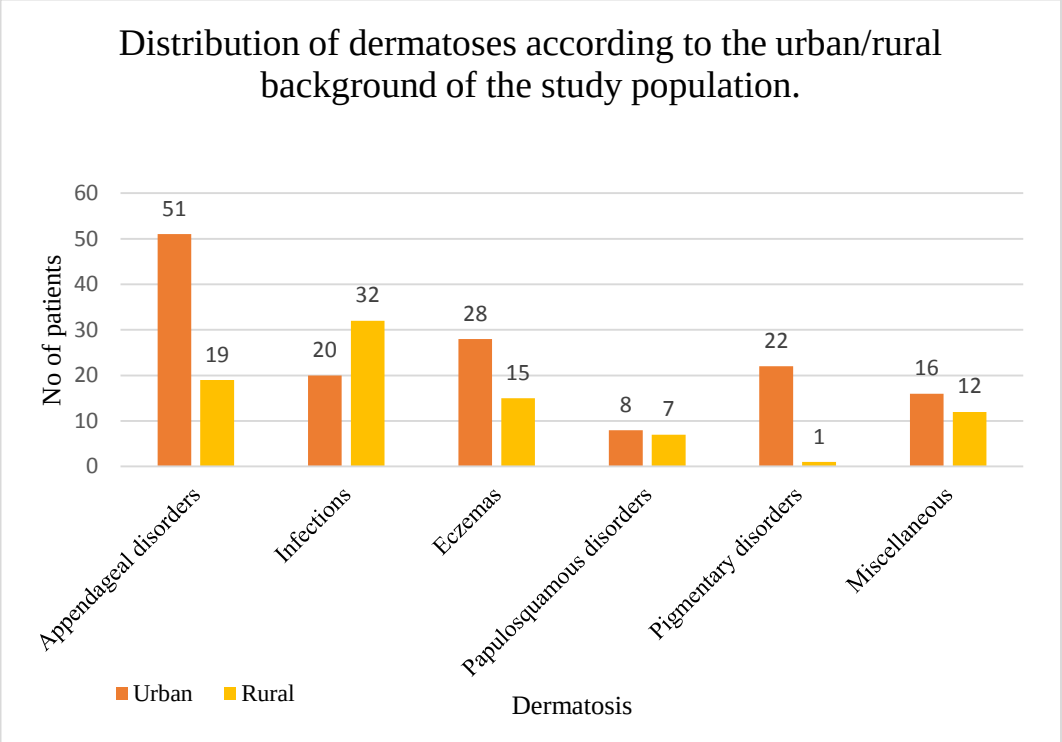


Figure 7: Distribution of dermatoses according to the urban/rural background of the study population.

Table 19: Distribution of dermatoses according to the socio economic status in urban and rural areas.

SES CLASS	DERMATOSES						
	APP	ECZ	INF	MIS	PIGM	PSD	Grand Total
R	**19**	**15**	**32**	**12**	**1**	**8**	**87**
II	9	8	4	1	1		23
III	8	6	6	5		4	29
IV	2	1	18	5		3	29
V			4	1		1	6
U	**51**	**28**	**20**	**16**	**22**	**7**	**144**
I	3	1				1	5
II	24	10	1	3	12	2	52
III	21	12	8	9	8	4	62
IV	3	5	11	2	1		22
V				2	1		3
Grand Total	**70**	**43**	**52**	**28**	**23**	**15**	**231**

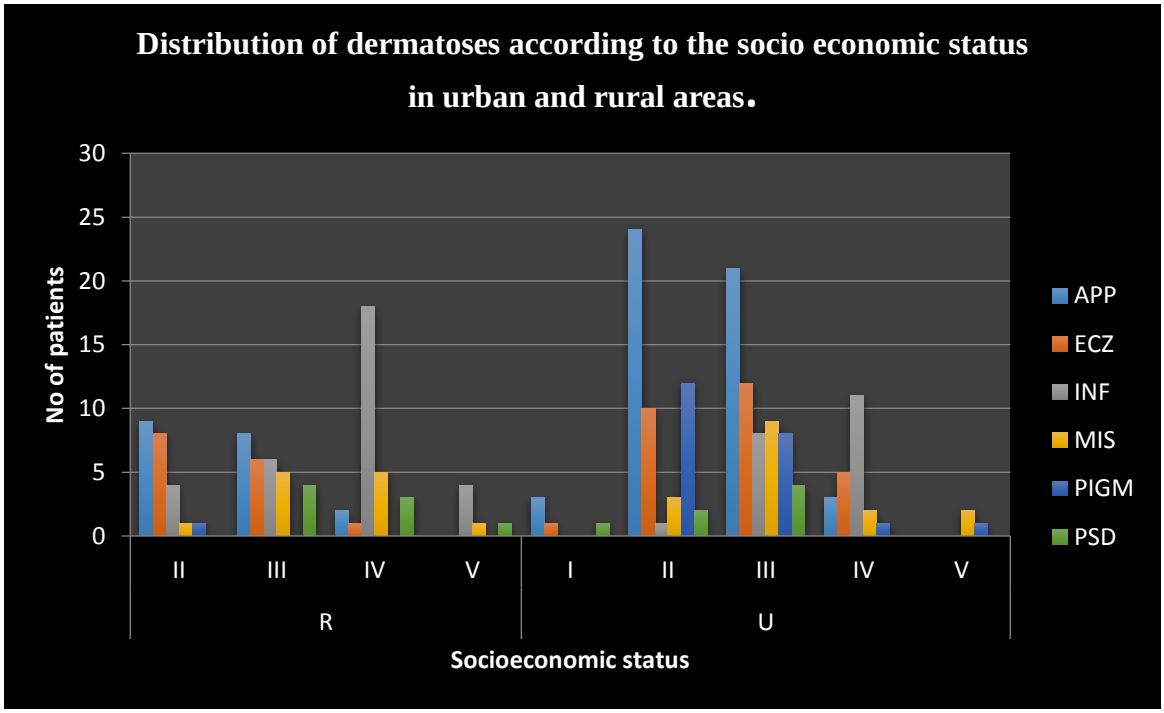


Figure 8: Distribution of dermatoses according to the socio economic status in urban and rural areas.

All the dermatoses were more common in the urban than rural population, mailnly in patients belonging to SES class II and class III. However infections were more commonly seen in the rural population and lower SES (class IV) in both urban and rural areas.

Types and patterns of different groups of dermatoses and their distribution according to age group and place of locality of the study population.

Table 20: Appendageal disorders:

	Urban			Rural			
	Early adolescence	Mid adolescence	Late adolescence	Early adolescence	Mid adolescence	Late adolescence	
Pilosebaceous	4	14	15	3	4	9	49
Hair	0	6	8	0	0	1	15
Sweat glands	1	2	1	0	0	2	6
Total	5	22	24	3	4	12	70

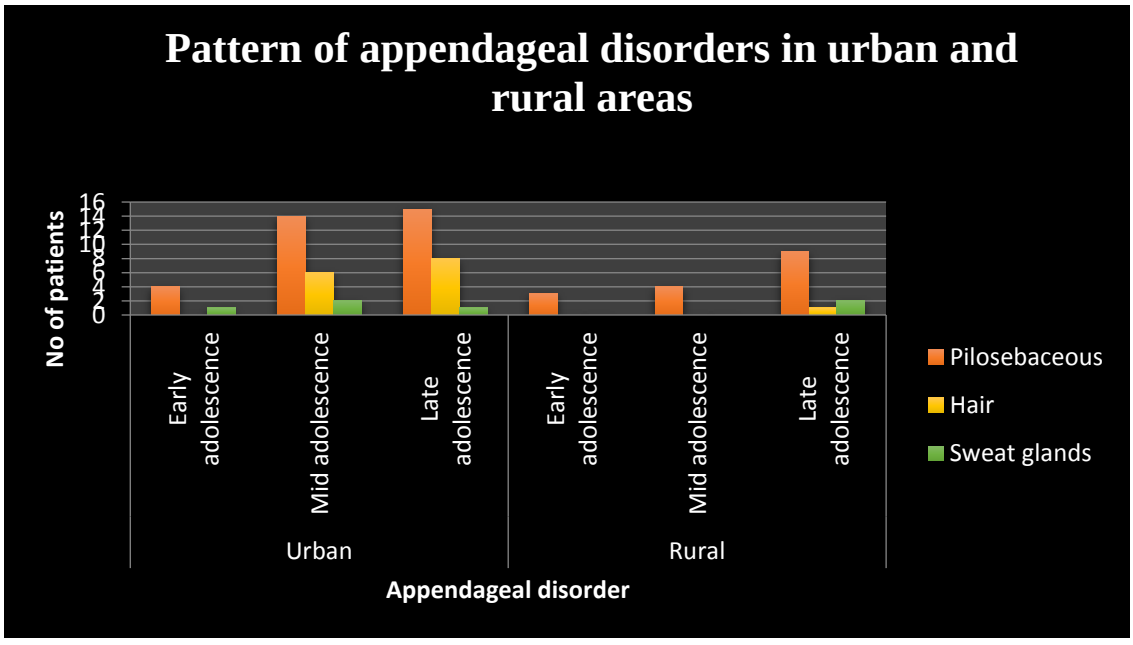


Figure 9: Pattern of appendageal disorders.

Pilosebaceous disorders and hair disorders were the common appendageal disorders observed, both of which showing higher incidence in urban areas and an increasing tendency with age. Sweat gland disorders were also more common in urban than rural population but were more or less equally distributed among all age groups.

Table 21: Types and patterns of infections.

	Urban			Rural			
	Early adolescence	Mid adolescence	Late adolescence	Early adolescence	Mid adolescence	Late adolescence	
Bacterial	1	0	1	1	1	0	4
Fungal	1	3	2	2	3	7	18
Viral	2	3	2	2	2	2	13
Parasitic	3	0	1	3	5	2	14
STI	0	0	1	0	0	2	3
Total	7	6	6	8	11	11	52

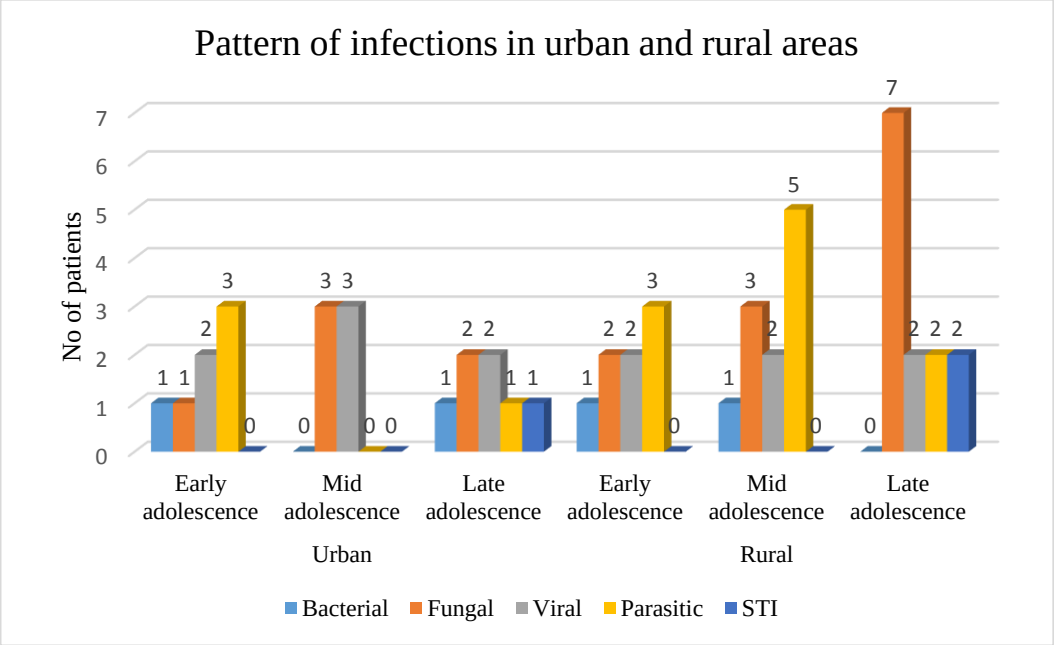


Figure 10: Types and patterns of infections.

Fungal infections were the most common infections in this study. Superficial dermatophyte infections (4.33%) were the commonest followed by pityriasis versicolor (2.16%) and onychomycosis (1.3%).

Parasitic infections were the next common group and comprised of scabies (3.46%) and pediculosis capitis (2.6%).

Among the viral infections, warts were the commonest (2.6 %), followed by molluscum contagiosusm (0.87%), herpes simplex (0.87%), varicella zoster infection (0.87%), and pityriasis rosea (0.43%).

Bacterial infection constituted the least common type accounting for only 4 cases overall and comprised of furuncles, folliculitis and impetigo.

Sexually transmitted infections accounted for 3 cases and comprised of genital candidiasis and genital warts**.**

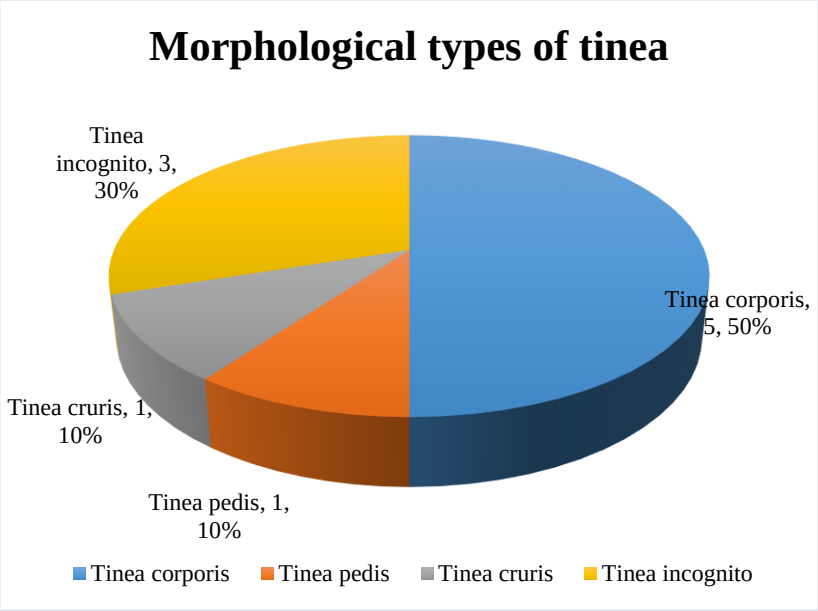


***Figure 11: Different types of tinea infections seen in the study population*.**

Table 22: Types and patterns of eczemas.

	Urban			Rural			
	Early adolescence	Mid adolescence	Late adolescence	Early adolescence	Mid adolescence	Late adolescence	
Endogenous	12	2	1	6	3	0	24
Exogenous	1	5	7	1	3	2	19
Total	13	7	8	7	6	2	43

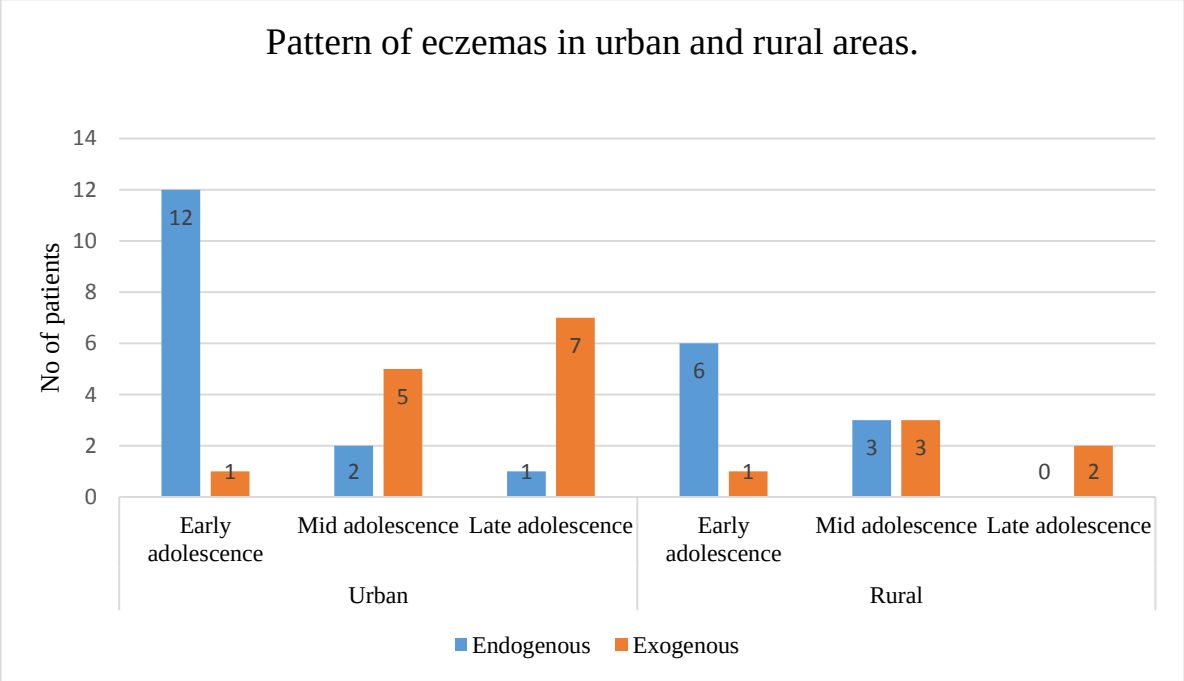


Figure 12: Types and patterns of eczemas.

Endogenous eczemas were the commonest type of eczemas (55.8%) and comprised of atopic dermatitis, pityriasis alba, dyshidrotic eczema and seborrhoiec dermatitis.

Atopic dermatitis was the commonest endogenous eczema and majority of them belonged to the urban areas and higher socio-economic status.

Exogenous eczema constituted 44.1 % of all the eczemas and commonest of them being contact dermatitis and photo eczema.

Among contact eczema, allergic contact eczemas were the commonest constituting 88.9 % followed by irritant contact dermatitis (11.1%). Out of 8 patients with ACD, 5 of them tested positive for nickel sulfate, 1 for paraphenylenediamine, and 2 were patch test negative, but gave clear history of use of new detergent soap since a couple of weeks leading to the diagnosis of ACD.

Table 23: Types and patterns of pigmentary dermatoses.

	Urban			**Rural**			
	Early adolescence	Mid adolescence	Late adolescence	Early adolescence	Mid adolescence	Late adolescence	
Hypopigmentary	6	5	0	0	0	0	11
Hyperpigmentary	0	4	7	0	1	0	12
Total	6	9	7	0	1	0	23

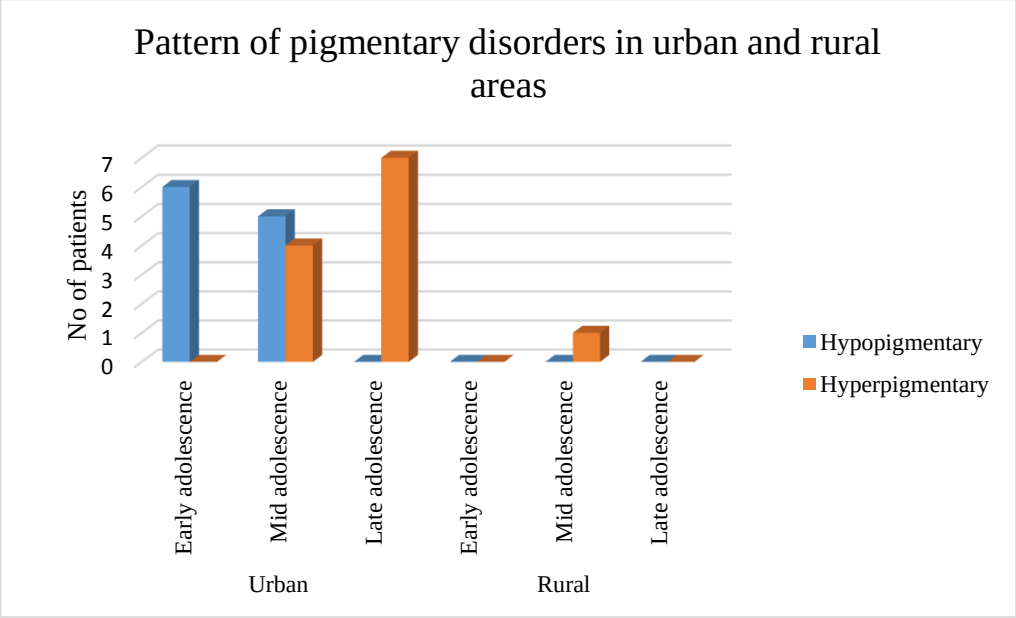


Figure 13: Types and patterns of pigmentary dermatoses.

Hyper pigmentary disorders comprising of PIH, peri orbital and peri ocular hypermelanosis constituted the majority of the pigmentary disorders (52.1%) followed by hypopigmentary conditions. PIH was commonly seen secondary to acne and was mainly seen over the face.

Vitiligo was the only hypopigmentary condition seen in this study. Generalised vitiligo was the most common type and lower limbs was the commonest site affected followed by upper limbs and the face.

Papulo squamous disorders:

Table 24: Different types of psoriasis seen in the study population.

Variants of psoriasis	No of patients	%
Chronic plaque psoriasis	6	75
Scalp psoriasis	1	12.5
Palmoplantar psoriasis	1	12.5

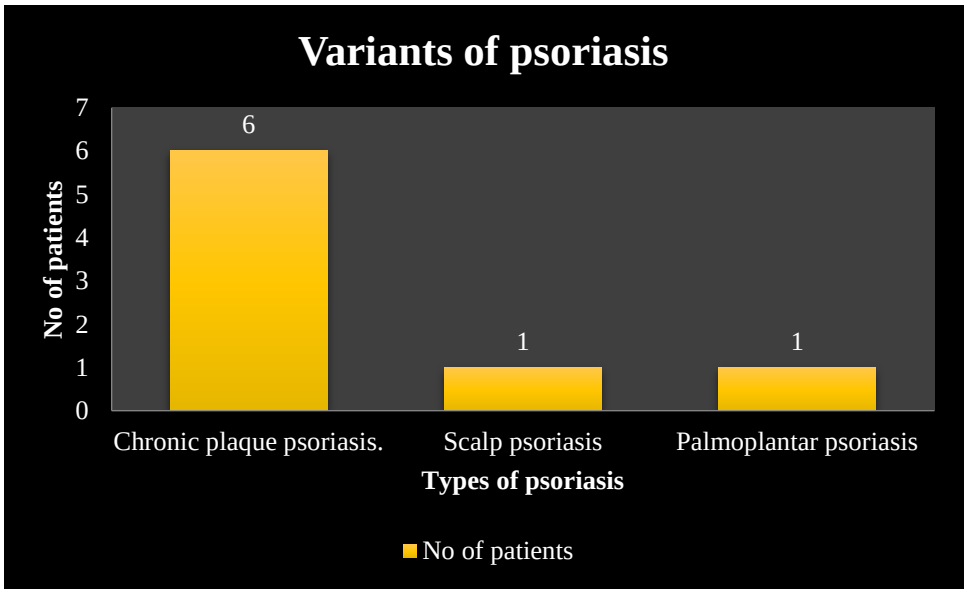


Figure 14: Different types of psoriasis seen in the study population **:**

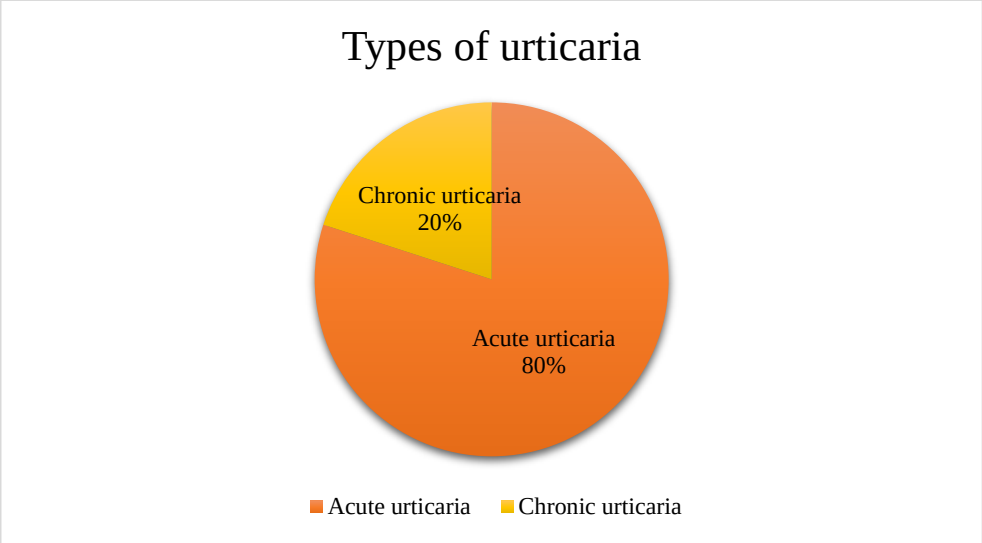


Figure 15: Types and patterns of urticaria.

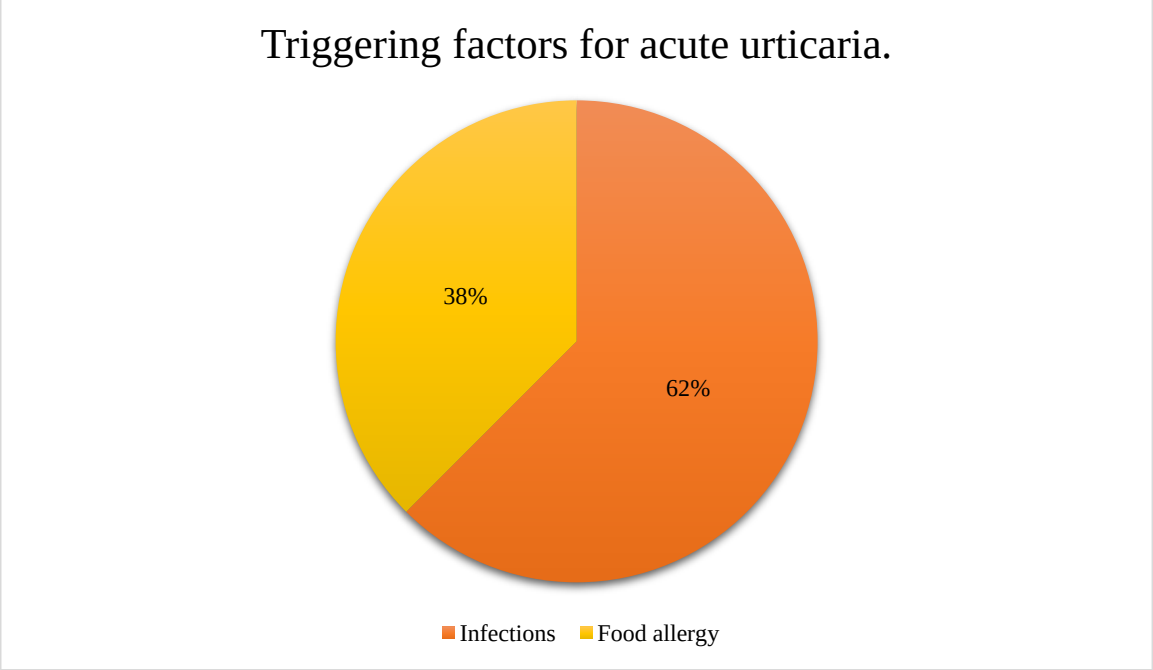


Figure 16: Triggering factors for acute urticaria.

Comparison of severity of dermatoses between urban and rural population using standard scoring systems:

Table 25: Grading of acne vulagris.

Acne vulgaris grading	Urban	Rural
I	10	0
II	7	9
III	8	4
IV	1	1

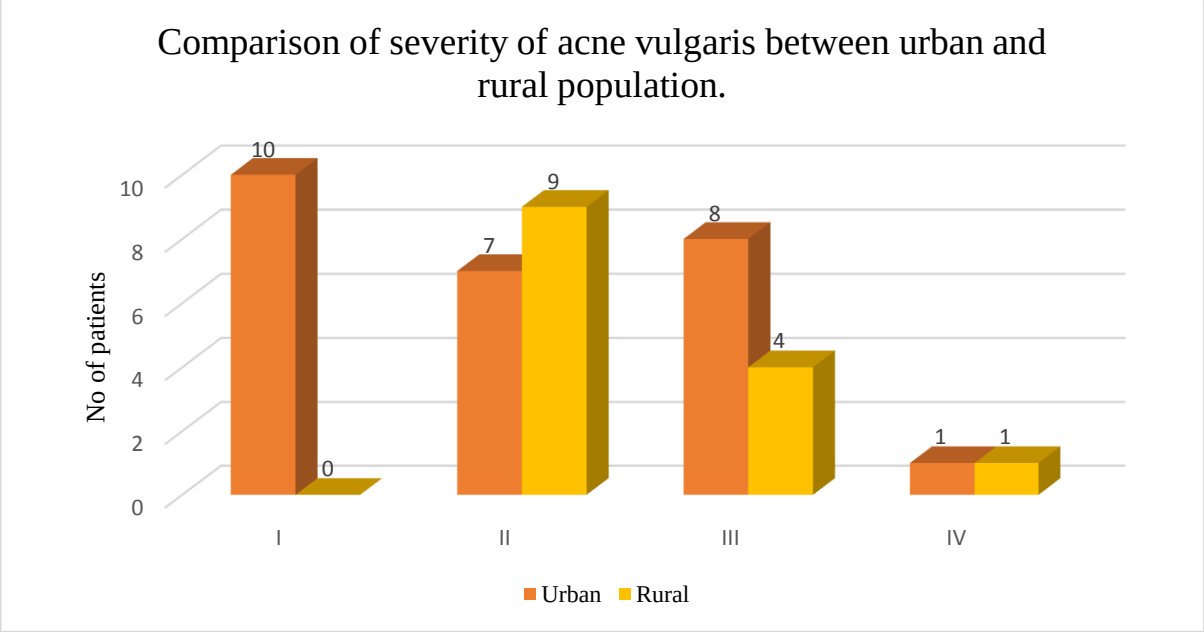


Figure 17: Grading of acne vulagris.

Table 26: Severity of alopecia areata.

	Urban	Rural
Mean SALT score	8.6	5.4
***P* = not significant**		

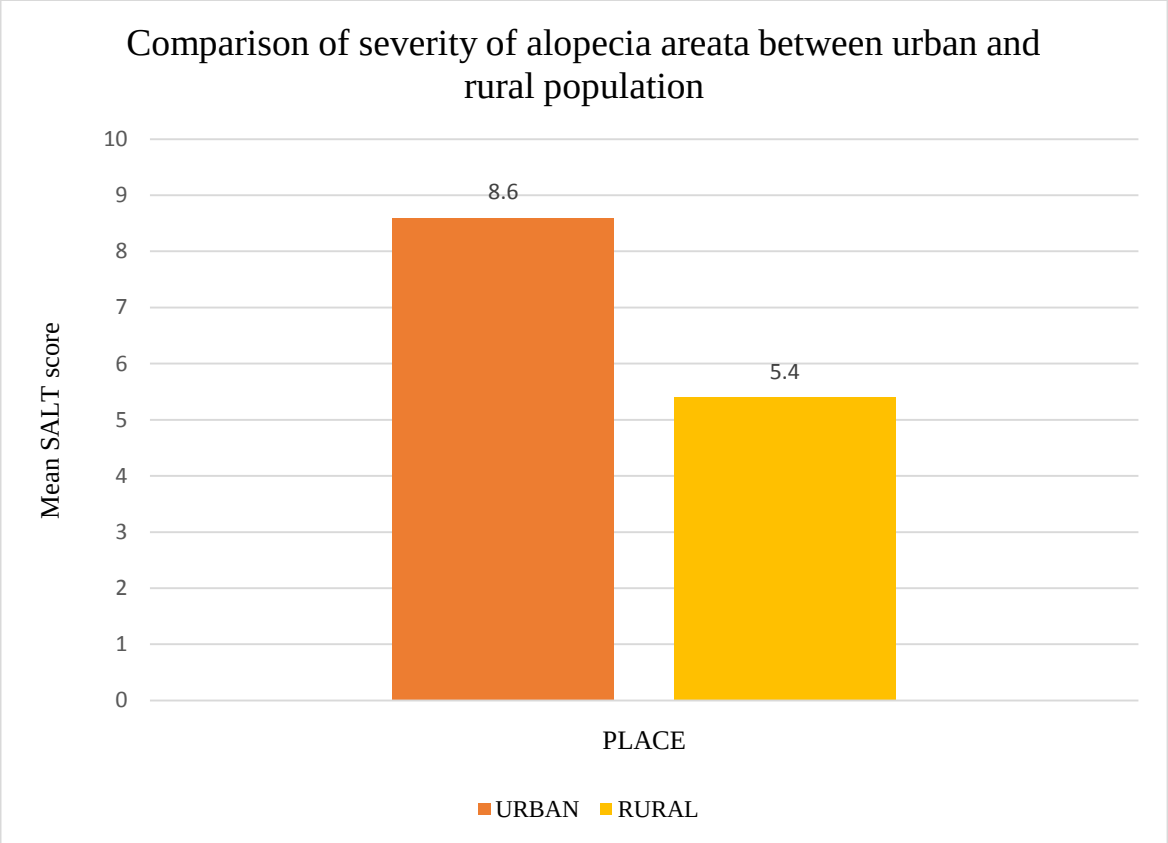


Figure 18: Severity of alopecia areata.

Table 27: Severity of hyperhidrosis

	Urban	Rural
Mean HDSS score	2.75	0
***P* = not significant**		

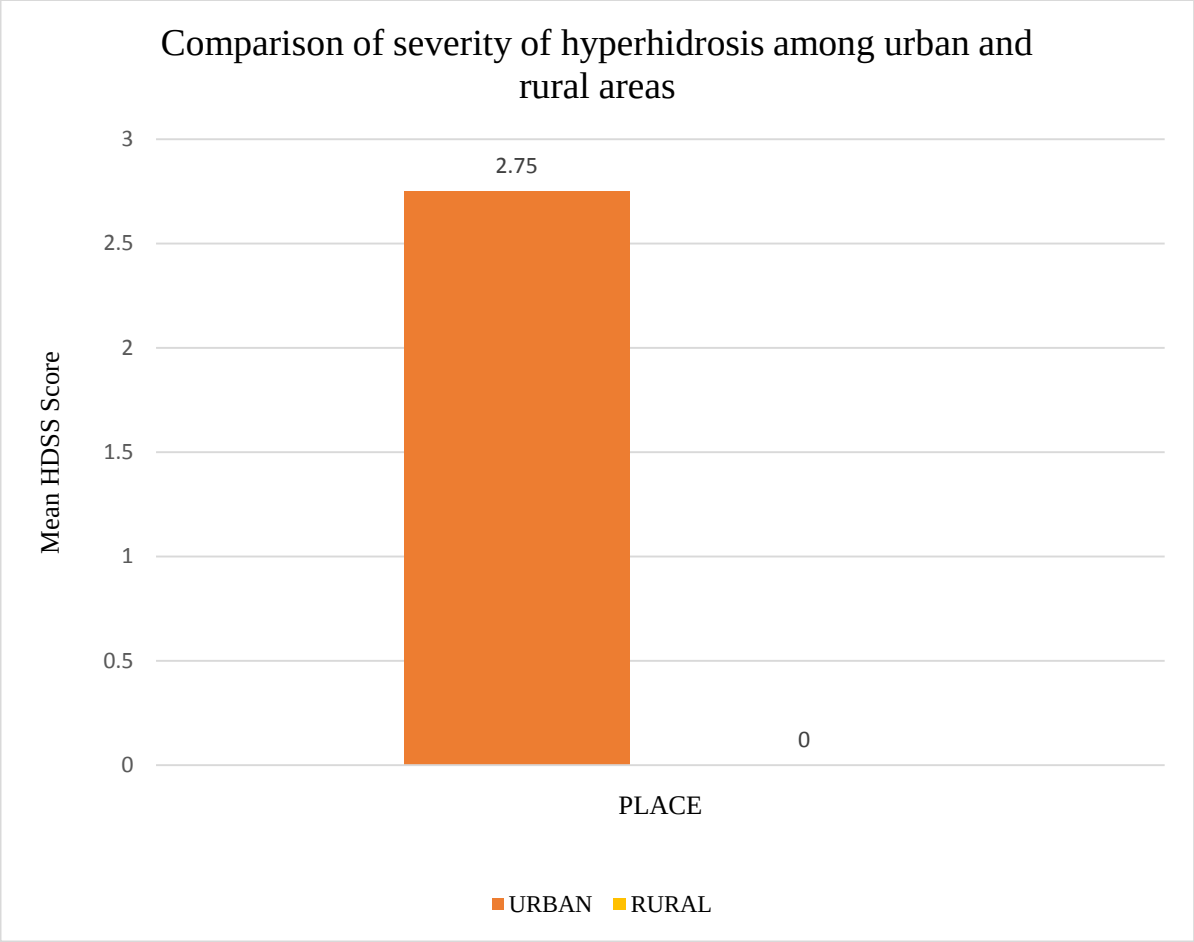


Figure 19: Severity of hyperhidrosis.

Table 28: Severity of atopic dermatitis.

	Urban	Rural
Mean SCORAD score	14.9	10.9
***P* = 0.015 (significant)**		

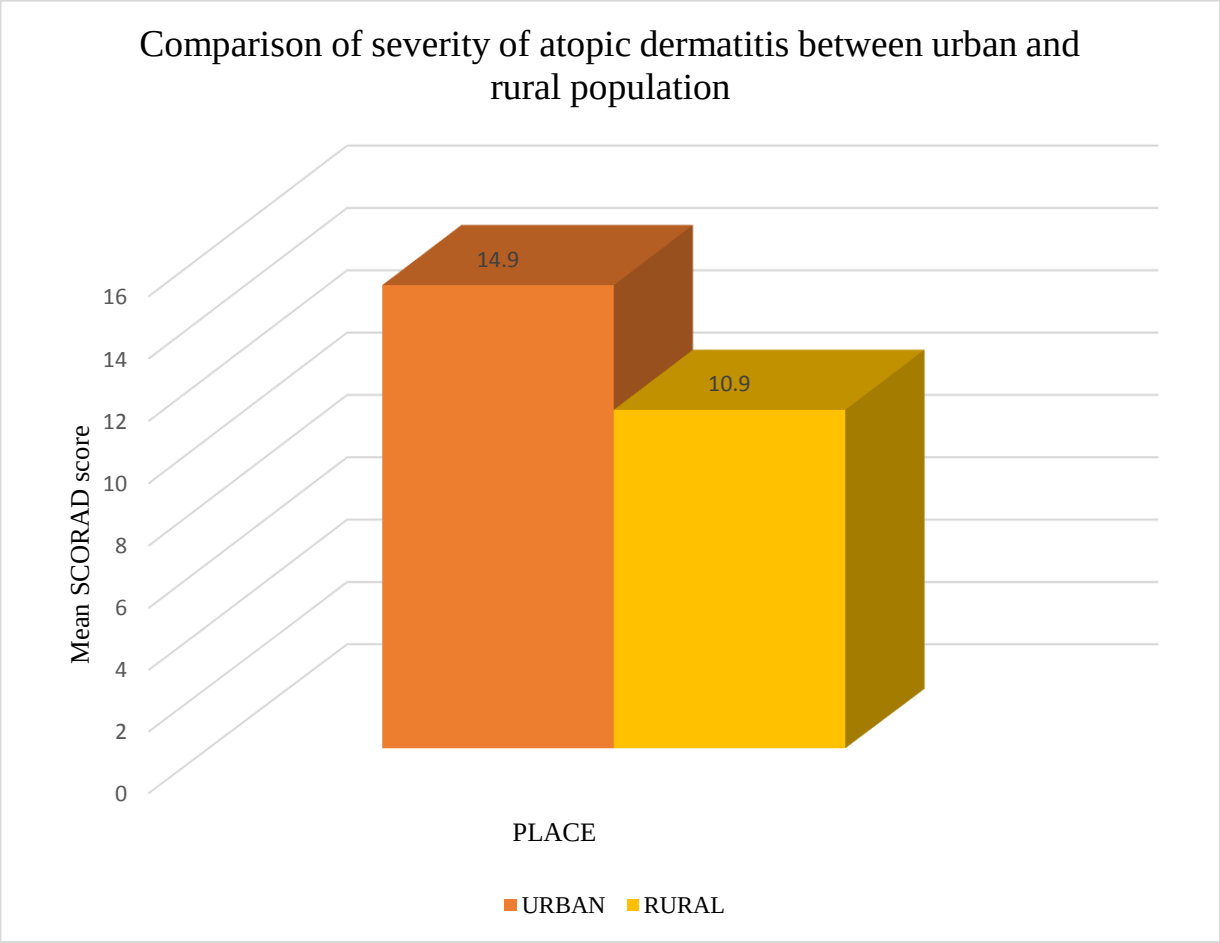


Figure 20: Severity of atopic dermatitis.

Table 29: Severity of dyshidotic eczema.

	Urban	Rural
Mean DASI score	14	16
***P* = not significant**		

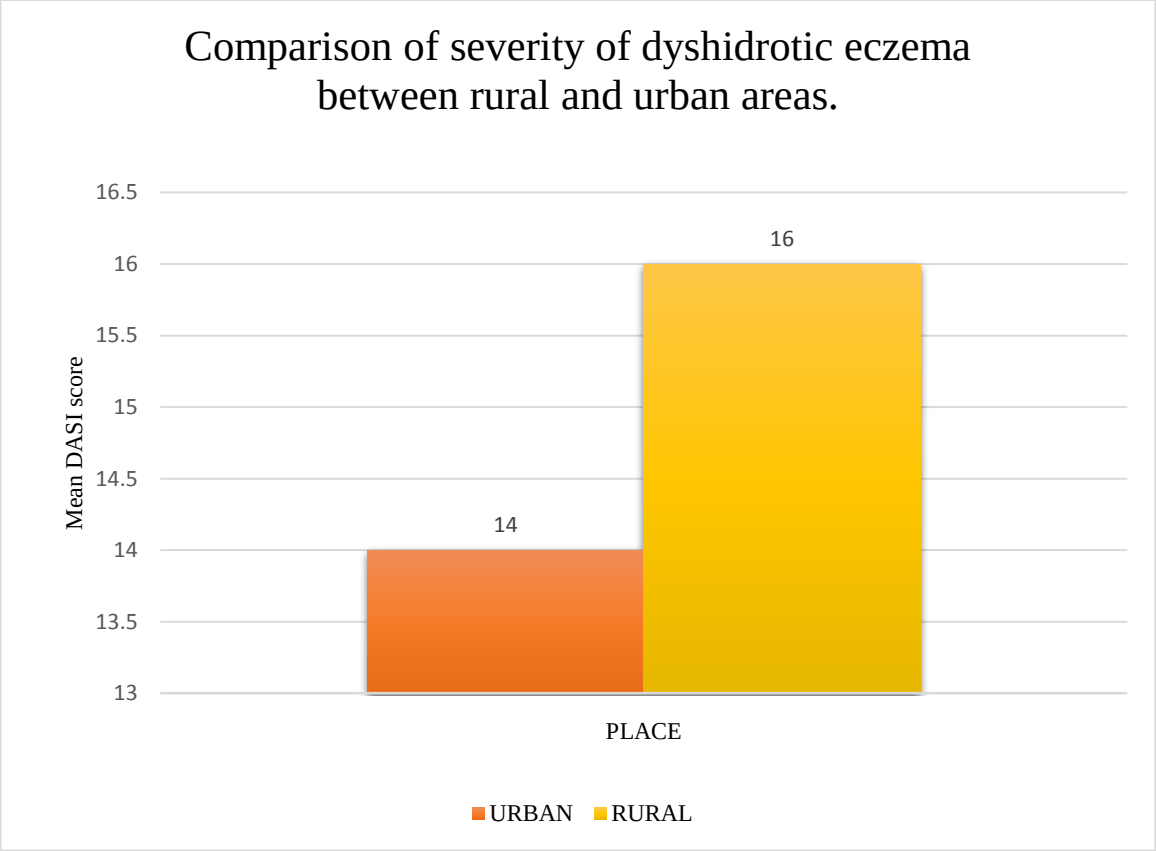


***Figure 21: Severity of dyshidotic eczema*.**

Table 30: Severity of vitiligo.

VIDA scores	Urban	Rural
0	1	0
1+	1	0
2+	3	0
3+	5	0
4+	1	0

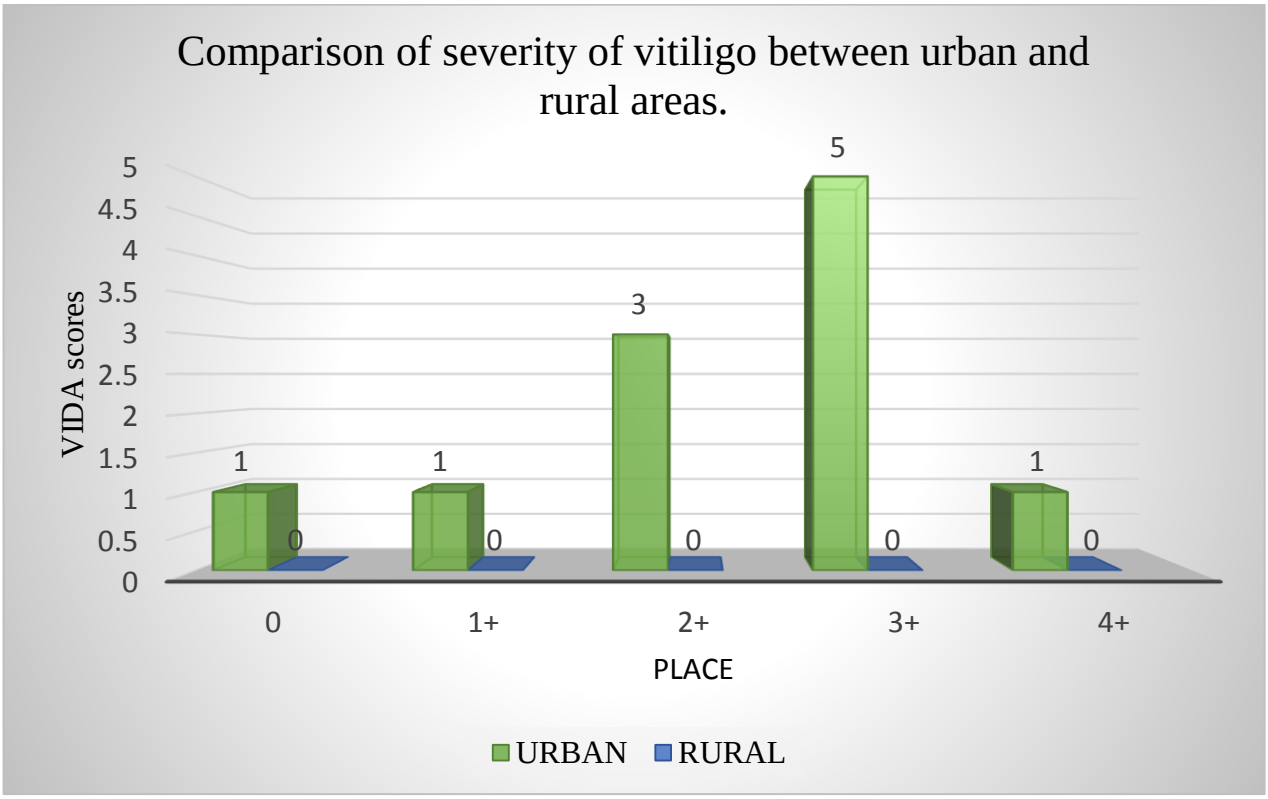


Figure 22: Severity of vitiligo.

Table 31: Severity of psoriasis:

	Urban	Rural
Mean PASI score	**7.24**	**8.8**
***P* = 0.64 (not significant)**		

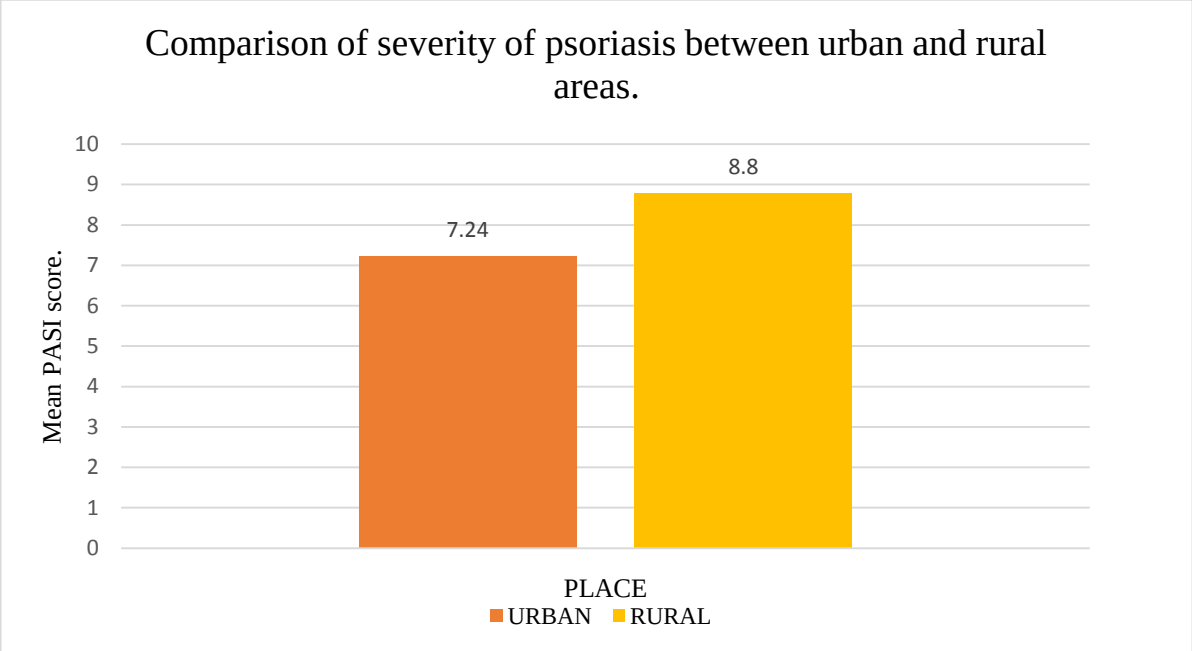


Figure 23: Severity of psoriasis:

Table 32: Severity of urticaria.

	Urban	Rural
Mean UAS score	5	4.75
***P* <0.05 (significant)**		

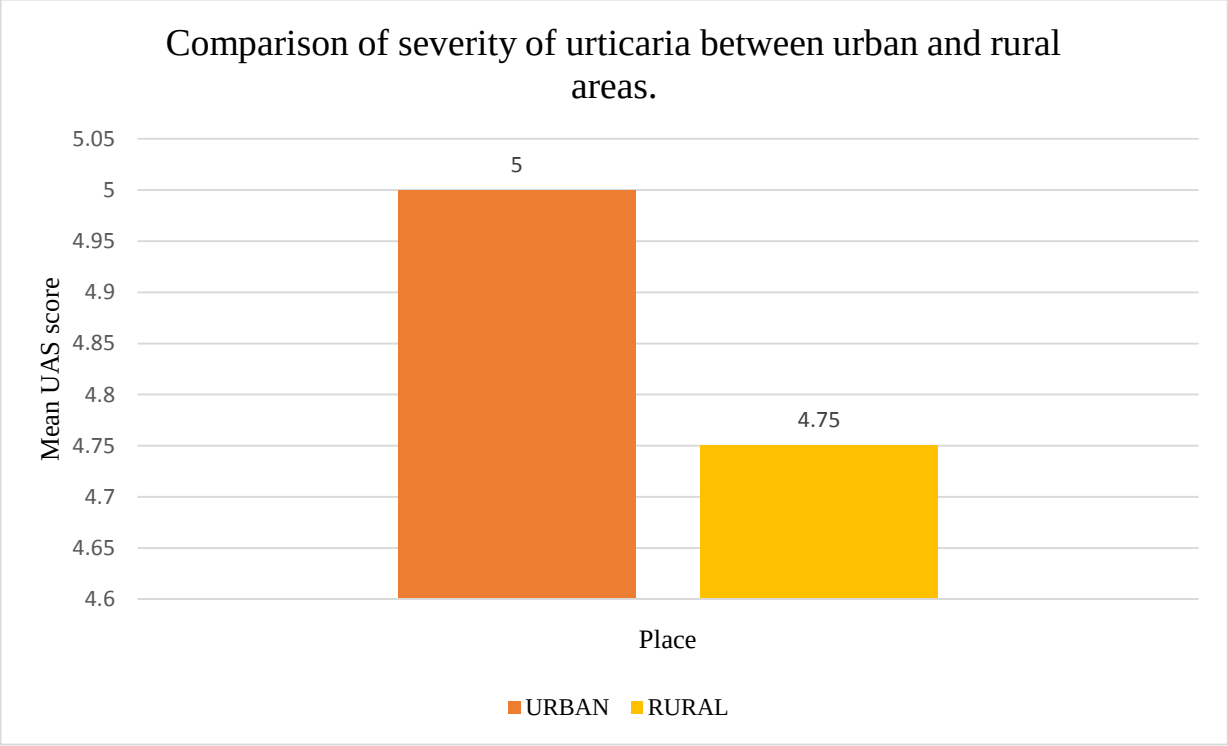


Figure 24: Severity of urticaria.

PHOTOGRAPHS

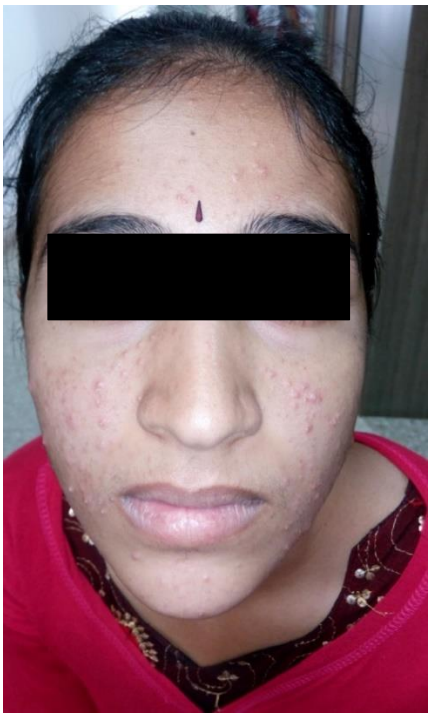


FIG 25: ACNE VULGARIS III

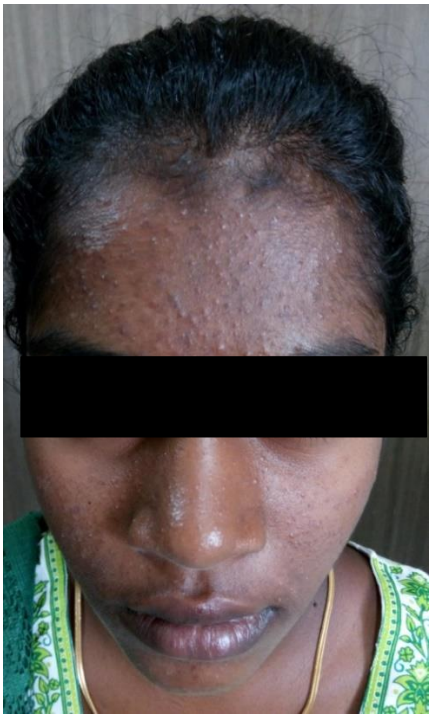


FIG 26: ACNEIFORM ERUPTIONS

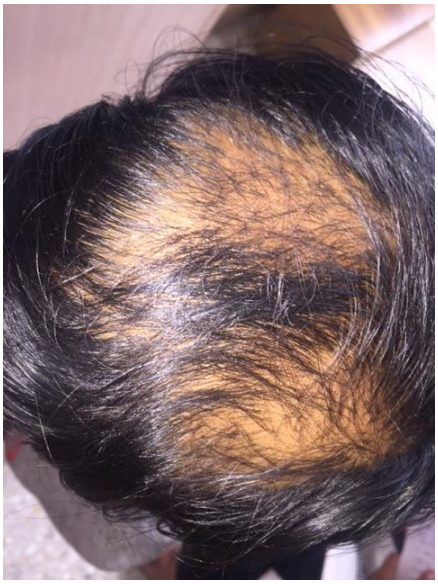


FIG 27: TRICHOTILLOMANIA

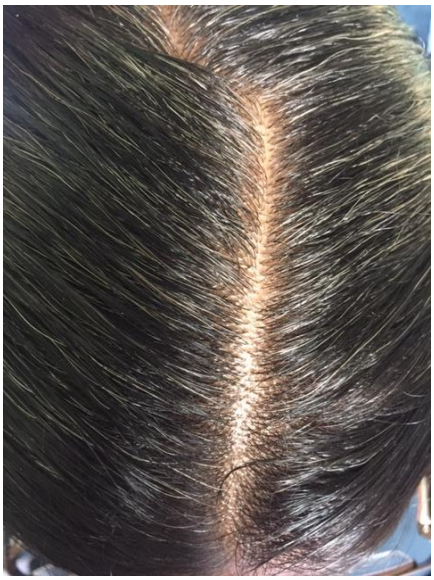


FIG 28: PREMATURE GRAYING OF HAIR

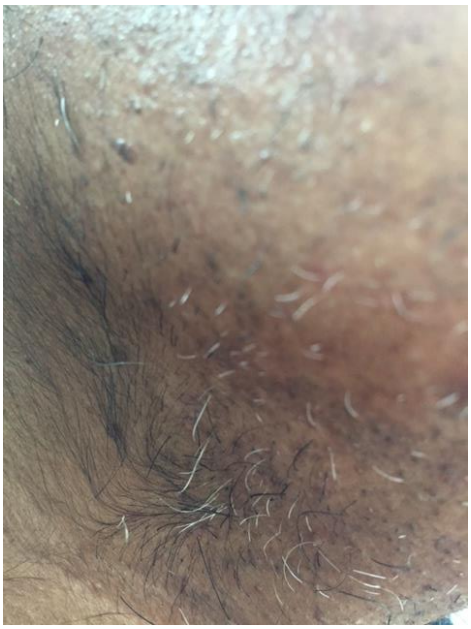


FIG 29: HIRSUTISM

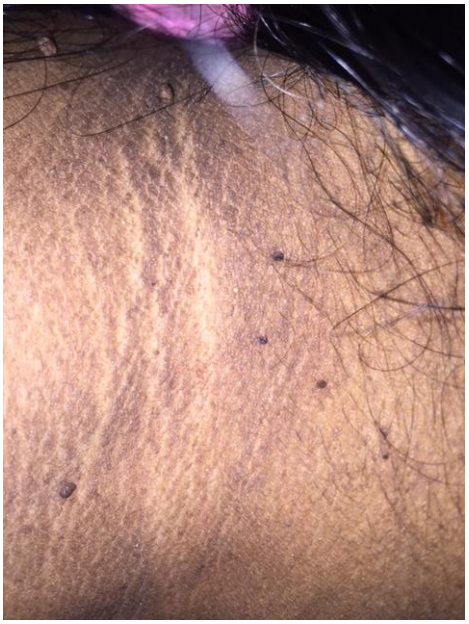


FIG 30: ACANTHOSIS NIGRICANS

FIG 31: PALMAR HYPERHIDROSIS

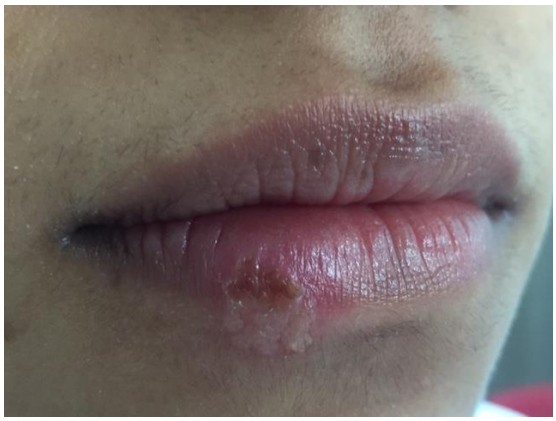


FIG 32: HERPES LABIALIS

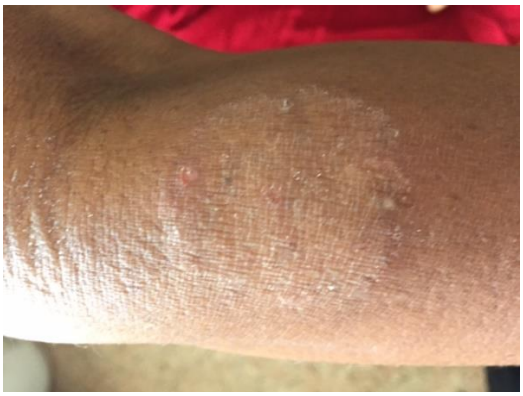


FIG 33: TINEA CORPORIS

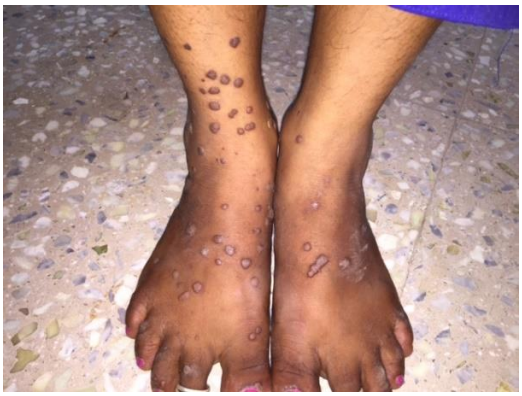


FIG 34: VERRUCA VULGARIS

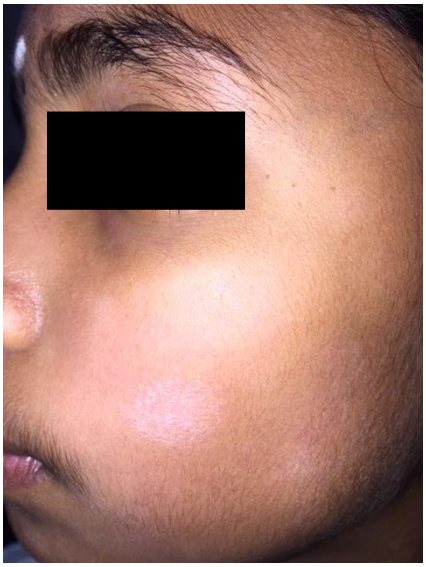


FIG 35: PITYRIASIS ALBA

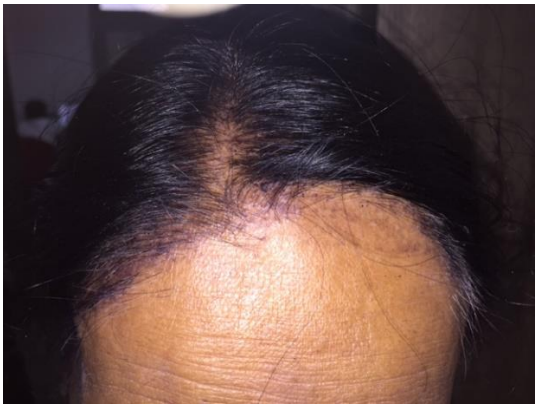


FIG 36: ALLERGIC CD TO HAIR DYE

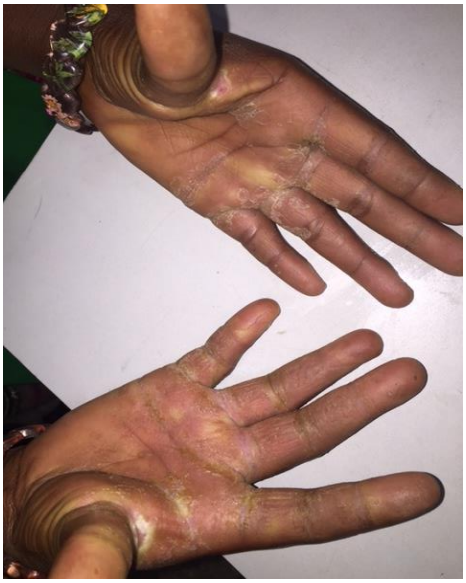


FIG 37: ALLERGIC CONTACT DERMATITIS TO DETERGENTS

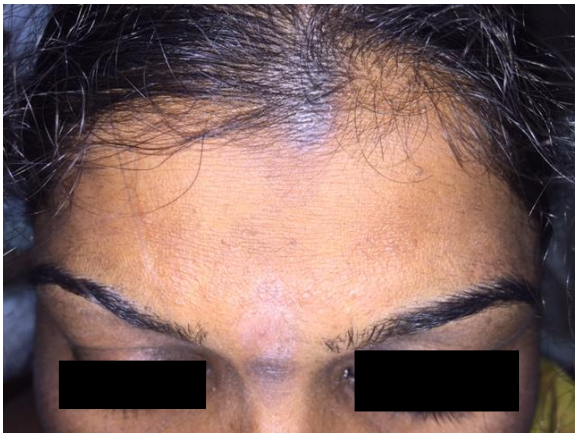


FIG 38: ALLERGIC CONTACT DERMATITIS TO KUMKUM

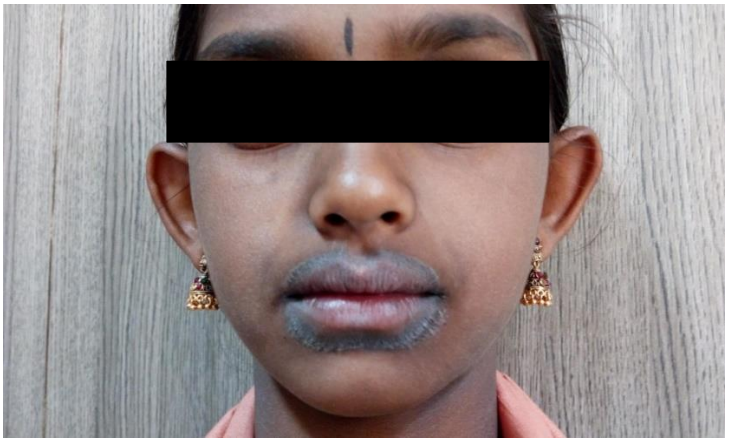


FIG 39: IRRITANT CONTACT DERMATITIS (LIP LICK DERMATITIS)

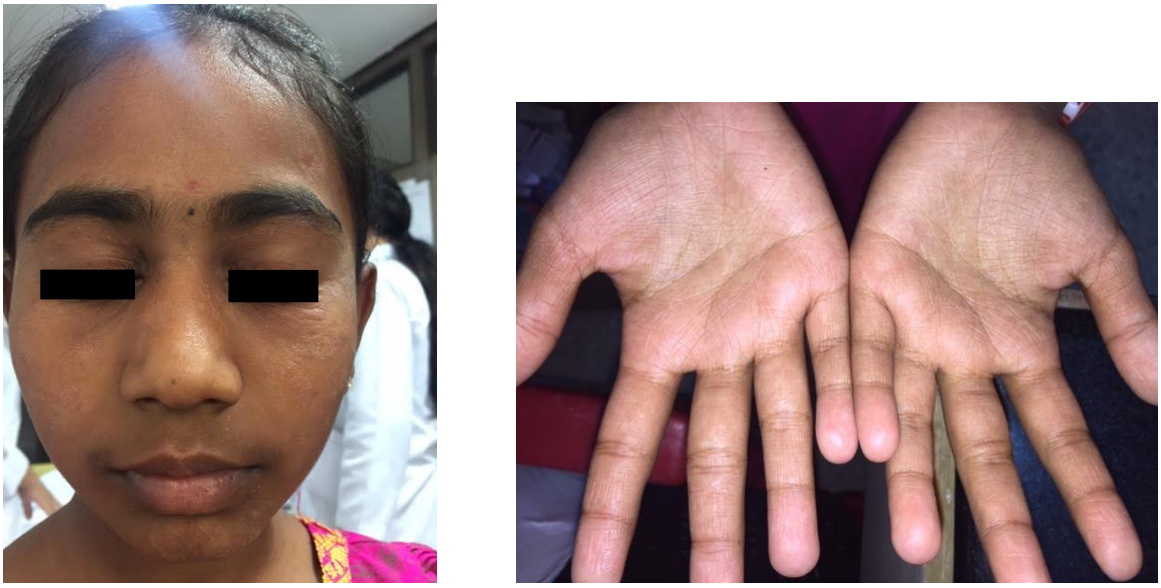


FIG 40: PATIENT OF ATOPIC DERMATITIS WITH LESIONS ON THE FACE AND HYPERLINEARITY OF THE PALMS.

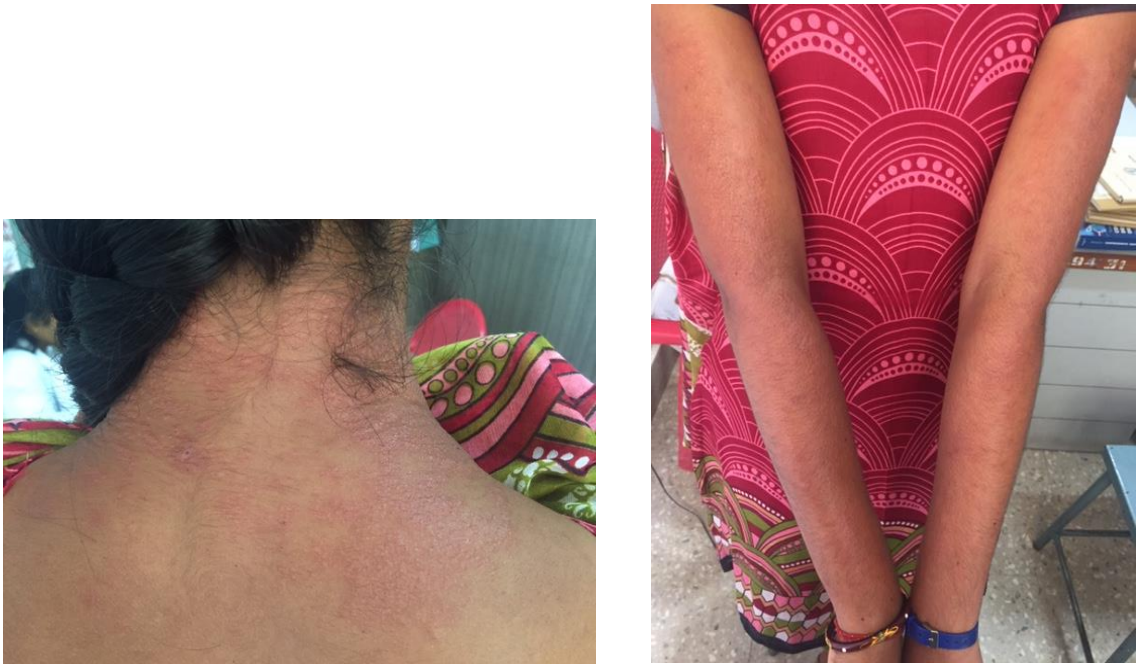


FIG 41: A PATIENT WITH POLYMORPHIC LIGHT ERUPTIONS INVOLVING THE BACK OF NECK AND EXTENSORS OF FOREARMS.

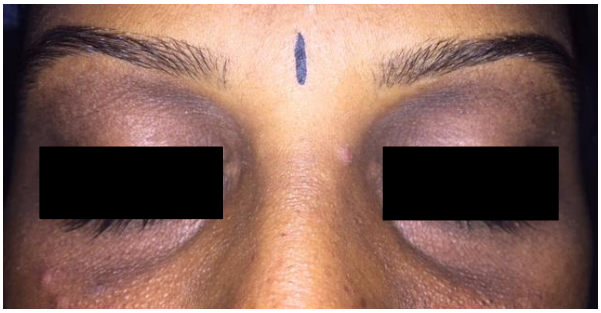


FIG 42: PERI OCULAR HYPERMELANOSIS

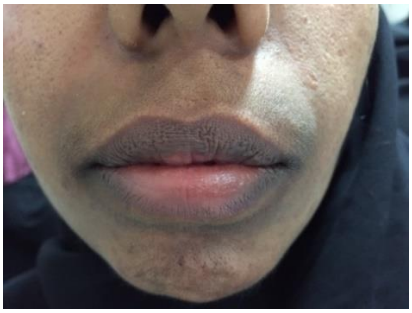


FIG 43: PERI ORBITAL MELANOSIS

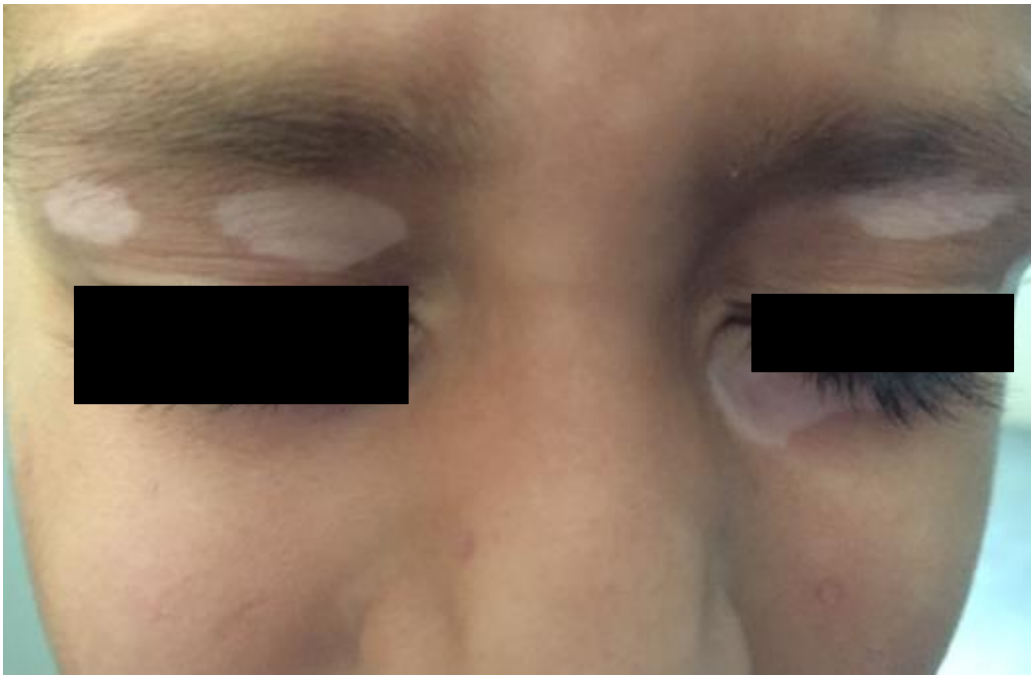


FIG 44: VITILIGO INVOLVING THE PERIORBITAL AREAS.

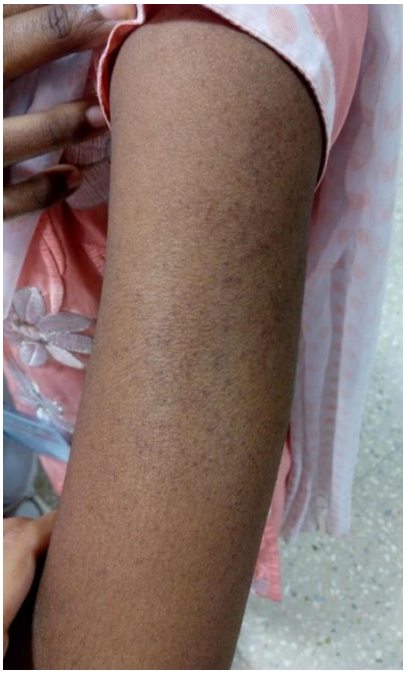


FIG 45: KERATOSIS PILARIS

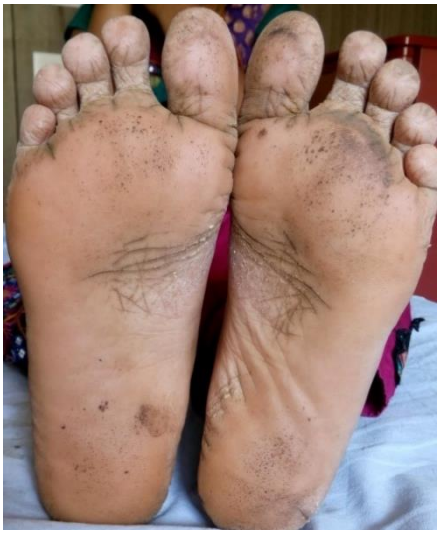
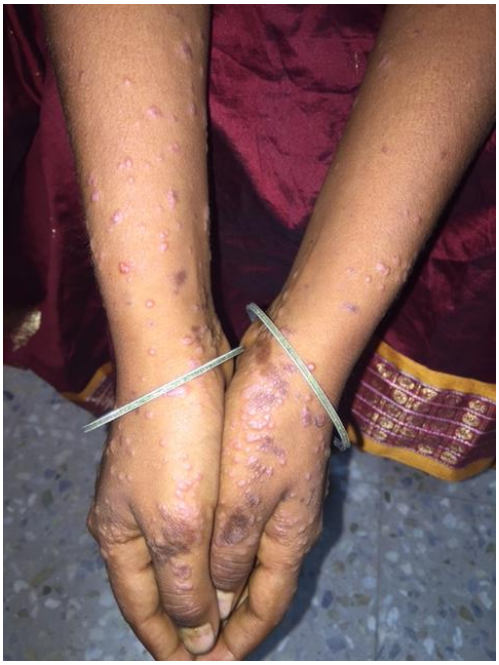


FIG 46: PLANTAR PSORIASIS



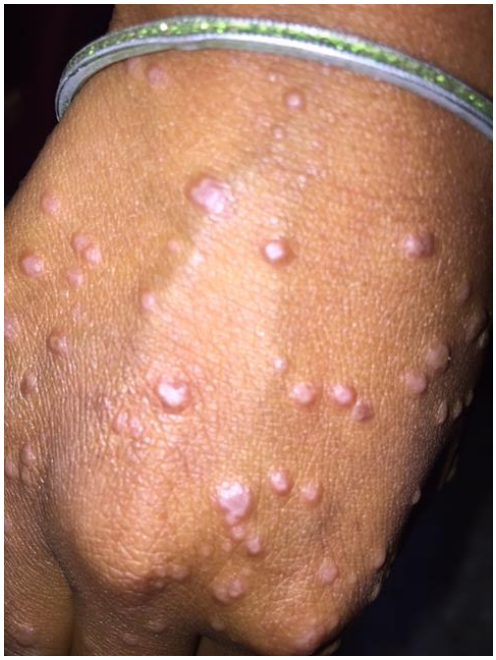
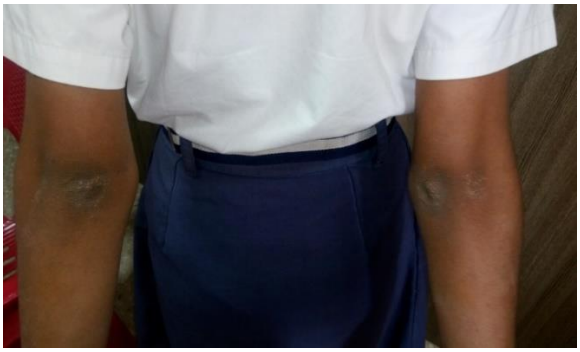


FIG 47: PURPLISH FLAT TOPPED PAPULES OF LICHEN PLANUS.



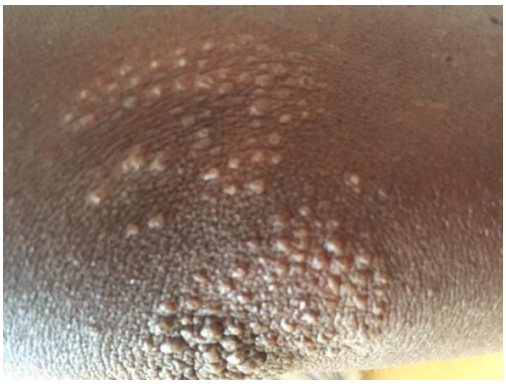


FIG 48: PHRYNODERMA

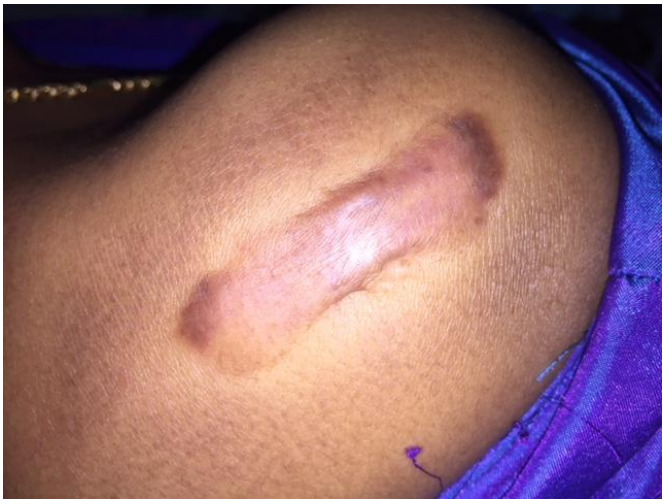


FIG 49: KELOID OVER THE SHOULDER AREA.

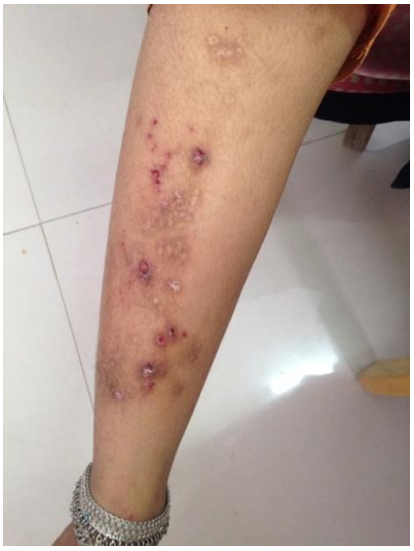


FIG 50: PRURIGO SIMPLEX.

DISCUSSION

DISCUSSION:

In our study, 43 specific dermatologic disorders were seen in 231 patients which were classified into six categories (based on etiology) which included appendageal disorders (n=70, 30.3%), infections (n=52, 22.51%), eczemas (n=43, 18.6%), pigmentary disorders (n=23, 9.96%), papulosquamous disorders (n=15, 6.49%) and miscellaneous dermatoses (n=28, 12.12%).

Age distribution of dermatosis:

The most common age group being affected is the late adolescent group (39.39%) followed by the middle (33.33%) and early adolescent age group (27.27%), which was comparable to a study conducted by Fung and Lo.[8]

Distribution of dermatoses according to the place of living:

Majority of our patients were from urban background (62.77%) as compared to rural background (37.23%), which could be attributed to better access to health care facilities and increased awareness among urban population. However other studies have shown higher incidence of dermatoses in rural areas as compared to urban areas.[119]

Socio economic status: Majority of the patients in our study belonged to the class III, class IV, class V socio economic constituting about 65% of patients followed by class II (32.5%) and only a minority of patients belonged to higher SES (2.2%). Our results were similar to data published in other studies in India.[120,121] However, there is sparse data based on the modified B.G.Prasad criteria employed in our study. We believe the new system of SES classification is more objective and therefore can be easily reproducible.

An analysis of the different dermatosis with respect to socio economic status showed an increasing trend of infections among patients of lower SES. A similar trend has been reported in other studies as well.[120,121] We observed a higher incidence of fungal infections and parasitic infestations. Other studies have also shown a predominance of parasitic infection followed by either bacterial or fungal infections.[120,122]

All the three cases of sexually transmitted diseases belonged to lower socio economic status (class IV). UNICEF in 2011 reported that in India, young women with low socio-economic status are seven times more likely to give birth before early and have higher chances of acquiring a sexually transmitted disease than young women with higher socio-economic status, which is also seen in our study.[110]

Appendageal disorders:

Appendageal disorders were the most common conditions seen in the study (30.3%) predominant among them being acne vulgaris which accounted for 17.3%. Studies conducted elsewhere have shown varying trends of incidence of appendageal disorders, including incidence of acne vulgaris,[1,123] with one study showing a similar incidence compared with our study.[7]

Face was the commonest site affected accounting for 75% of the cases followed by trunk (25%) similar to other studies.[1,123]

Grade II and grade III acne (mild-moderate type) were the most common type of acne seen in our study where as nodulo cystic acne was seen only 2 patients. This is consistent with data reported in other studies.[1]

The peak incidence of acne in our study was in the late adolescent group. Hormonal changes at puberty have been shown to affect the incidence of acne which explains the increased frequency among late teens. Similar results have been shown in other studies.[7,8]

All patients with acneiform eruptions (n=9) had a history of application of over the counter creams such as topical corticosteroids owing to their skin lightening effect. A study conducted in India, on misuse of topical corticosteroids showed that majority of them had facial acne and more than half of them belonged to the adolescent and youth age group and interestingly 78.89% were females.[43]

Other appendageal disorders seen in our study were hair loss, hirsuitism, premature graying of hair and sweat gland abnormalities such as palmar hyperhidrosis and milaria rubra similar to other studies.[1,7]

The main causes of hair loss were telogen effluvium, alopecia areata and trichotillomania. The importance of stress in the pathogenesis of these disorders is well proved. The presence of stress related events in adolescents and their immaturity to cope up with it could be contributory to the development of these disorders.

The mean SALT score in our study was 7. (8.6 and 5.4 in urban and rural population respectively) which represents a milder form of the disease. Our results were similar to findings published by Yang et al who reported a mean SALT score of 6.9 at the first visit.[124]

Hirsutism was seen in 3 patients. Most of them had over the moustache area, chin and mandible consistent with findings reported in other studies.[7,123] Additionally, one patient in our study had associated acne, acanthosis nigricans and menstrual irregularities as well.

The mean HDSS score in our study was 2.75. All the patients were from urban background. The mean HDSS score observed in our study was similar to a study in which majority of the patients (65%) had HDSS score of 3.[125] However, another study showed that majority of the patients (55.2%) had the HDSS score of 4, which represents the most severe form of the disease and 35% had a score of 3.[126]

Miliaria could be attributed to the hot and humid environment in and around the town and also due to excessive sports activities common in this age group.

Infections:

Infections constituted the second most important group of disorders in this study accounting for 22.5% similar to other studies.[1,127] However, other studies have shown infections to be the predominant skin diseases accounting for 37.7% to 54.5% of the population studied.[119,128]

Fungal infections were the most common infections seen (n=18, 34.6%) followed by parasitic infestations (n=14, 26.9%), viral infections (n=13, 25%), bacterial infections (n=4, 7.6%) and sexually transmitted infections (n=3, 5.7%) which is similar to other studies conducted in India.[128] However, Nadia et al. reported viral infections as the commonest infection (15.7%), followed by fungal (13.5%) and bacterial (10.8%) infections.[129]

It is interesting to note that infections were significantly more common in patients of rural background as compared to urban (37.2% vs 13.8% respectively; P <0.0001). This could possibly be explained by the lower socio economic strata resulting in overcrowding and poor living standards, poor hygiene and lack of awareness. Our results were similar to a study conducted in Turkey who concluded that improvement in socioeconomic conditions

and education level is essential to decrease the prevalence of communicable skin diseases.[130]

Superficial dermatophyte infections were the most common fungal infections followed Pityriasis versicolor and onychomycosis. Tinea corporis was the most common dermatophyte infection. Our results are similar to findings reported by another study.[8] A study conducted in India on misuse of topical corticosteroids showed that tinea incognito was one of the common disorders seen perhaps due to its anti-inflammatory effects leading to temporary relief in the symptoms.[43]Onychomycosis affecting the toe nail was more common and all of them had DLSO similar to a study conducted in Korea.[131] Parasitic infestations which includes scabies and pediculosis capitis were a close second.

Warts were the most common viral infections followed by molluscum contagiosum, herpes simplex and varicella zoster infection. A similar pattern of spectrum of viral infections were reported by a study in south India as well.[132]

Sexually transmitted infections were seen in 3 patients and included mainly genital candidiasis and genital warts. However, a study conducted by Bunnel et al. in adolescent females showed Chlamydia trachomatis and Nesseria gonorrhoeae to be more common.[108] All 3 of them were married at the age of 18, belonged to lower socioeconomic status (class IV) and did not have any educational background.

Eczemas:

Eczemas were the third most common condition seen in our study (n=43, 18.6 %). Endogenous eczema comprising atopic dermatitis, dyshidrotic eczema, pityriasis alba, seborrheic dermatitis constituted majority of the eczemas seen (n=24, 55.8%) followed by

exogenous eczemas (n=19, 44.1%) comprising contact dermatitis and photodermatitis similar to another study.[8]

Atopic dermatitis itself accounted for almost half of the endogenous eczemas (n=11, 45.8%). Results of our study correlate with findings reported elsewhere.[8,130] However, few studies have reported low incidence of atopic dermatitis constituting a mere 0.01% of all the dermatoses.[119]

It was interesting to note that atopic dermatitis was commonest in early adolescent group and significantly less common in the later age groups. This supports the general finding in most studies that atopic eczema usually begins in childhood and tends to clear spontaneously in adulthood. It was also more common in the urban population with a higher socio economic strata.

The mean SCORAD score in our study was 12.9 (14.9 and 10.9 in urban and rural population respectively). Our results are similar to findings published in another study. in which a mean SCORAD score of 13.9 at the first visit was reported.[133]

Dyshidrotic eczema was seen in 1.73% (n = 4) of patients with a mean DASI score of 15, (14 and 16 in urban and rural population respectively) which is similar to another study in which a mean DASI score of 16 at the first visit was reported.[134]

Polymorphic light eruption was the commonest exogenous eczema, probably attributed to increased sun exposure especially during peak hours due to higher participation in outdoor activities among adolescents.

Contact dermatitis was the next most common exogenous eczema seen in this study. It showed an increasing trend with age which supports the general belief that sensitivity to

contact allergens increases with age in childhood and adolescence because of increased environmental exposure to contact allergens. Results of our study are similar to other studies conducted in children and adolescence which also showed that it is more common in girls.[135,136,137] Nickel sulphate was the most common allergen causing contact dermatitis in our study, consistent with data reported elsewhere.[138,139,140]

Pigmentary disorders:

Hyper pigmentary disorders comprising post inflammatory hyperpigmentation, peri orbital and peri ocular melanosis were common (n=12, 52.1%) than the hypo pigmentary conditions like vitiligo (n=11, 47.9%) which is similar to other study.[1] However, other studies conducted in adolescents found that vitiligo was the commonest pigmentary disorder in adolescents.[141,142]

Vitiligo vulgaris was the commonest type of vitiligo seen in this study similar to other studies.[141,142] However, a study done in south India showed that segmental variety is more common. Our study showed an increased prevalence in the early adolescence.[143]

All the patients with vitiligo were from the urban background. Among them, majority had a VIDA score of 2+ and 3+ (n=8, 72.7%) consistent with findings reported by another study.[144] Vitiligo is associated with immense stigma in the society especially where there is lack of awareness about the disease and this could be a possible explanation why there were no patients of vitiligo from the rural areas.

Hyper pigmentary disorders showed a higher rate in late adolescence and urban areas which can be attributed to their increased awareness concerning any pigmentary discrepancy on their body.

Papulo squamous disorders(PSD):

They constituted 6.49 % of the different types of skin diseases seen in this study. Most common among them being psoriasis (3.46%) followed by lichen planus (1.7 %) and keratosis pilaris (1.3%).

Chronic plaque psoriasis was the most common type of psoriasis (75%) seen in this study similar to a study conducted at a psoriasis clinic in north India (60.6%).[145] Exclusive scalp and palmo-plantar involvement was seen in one patient each. However, we did not get a single patient with guttate psoriasis during the study period, even though it is one of the most common variants of psoriasis being described in childhood and adolescents. Nail involvement in the form of pitting, ridging and discoloration was seen only in 3 patients (37.5%) which is similar to 31% in another study.[145]

The mean PASI score in our study was 8.02 (7.24 and 8.8 in urban and rural areas respectively) which is similar to another study in which majority of the adolescents had PASI scores ranging from 5-10.[146]

The most common age group being affected was the early adolescent age group similar to a study conducted at a tertiary psoriasis clinic in Korea where the mean age of onset was 10.5 ± 4.3.[146]

Positive family history was noted only in 2 patients (25%), where as in another study it was noted in 32.4 %[146] even though it is said that childhood psoriasis has a higher incidence of positive family history as compared to the adult psoriasis. The low familial incidence in our study may be explained by the ignorance of family members about the existence of the

disease, or the actual absence of the disease at the time of examining the patient, which may subsequently appear.

Lichen planus was the next most common keratinising disorder seen in 4 patients (26.6%). Upper and lower limbs were the commonest sites involved. There were no patients with nail or mucosal involvement in our study. Studies done elsewhere have reported similar findings.[147]

Keratosis pilaris was seen in 3 patients (1.3%) and involved outer aspect of the arms which is the commonest location for this disease. A similar study in Saudi Arabia reported a 2.2% incidence of keratosis pilaris in a similar patient population as compared to our study.[148]

Miscellaneous conditions:

Miscellaneous conditions accounted for 12.12 %, most common of them being urticaria (4.3%), insect bite reactions (2.6%), keloids (2.16%), prurigo simplex (2.16%) and phrynoderma (0.87%).

Urticaria was the predominant condition among the miscellaneous group (4.3%), among which acute urticaria constituted 80% (n=8) and chronic idiopathic urticaria constituted 20% (n=2) similar to another study which showed the incidence of acute urticaria in their study to be 68.5%.[149]

The most common triggering factor for acute urticaria in our study was infections seen in 62.5% (n=5), all of them giving a history of preceding upper respiratory infection and food allergy seen in 37.5% (n=3), in accordance to a study conducted by Liu et al who reported infections to be the most common triggering factor for acute urticaria in children.[150]

The mean UAS score in our study was 4.87 (5 and 4.75 in urban and rural population respectively) similar to another study in which the mean UAS score was found to be 5.3.[151]

CONCLUSION

CONCLUSION:

The findings of this study highlight the most predominant skin diseases, which were acne, infections and eczema which may incur significant morbidity in the affected individuals and also use considerable resources in the community

Apart from having general health importance, these disorders if not properly managed, can lead to significant cosmetic disfigurement thereby leading to disfiguring sequel and psychological distress in the young adolescent minds.

Hence, there is a need to monitor the epidemiology of common skin problems in children and adolescents so that relevant preventive measures can be planned and implemented effectively.

Thus, creating awareness among this subset of population is of utmost importance as small changes in the perception of need for medical help can have a great impact on the delivery of health care.

SUMMARY

SUMMARY:

The salient findings of the present study are summarised below:

- Out of 231 patients, most common dermatoses were appendageal disorders (n=70,30.3%) followed by infections (n=52,22.51%), eczemas (n=43,18.6%), pigmentary disorders (n=23,9.96%), papulosquamous disorders (n=15,6.49%) and miscellaneous dermatoses (n=28,12.12%).
- The most common age group being affected is the late adolescent group (39.39%) followed by the middle (33.33%) and early adolescent age group (27.27%).
- Dermatological diseases were commonly observed in urban population (62.77%) than rural population (37.23%).
- Out of 231 patients, 5 belonged to the socio economic status class I, 75 belonged to class II, 91 belonged to class III, 51 belonged to class IV and 9 patients belonged to class V.
- Appendageal disorders were the most common conditions seen in the study (30.3%) predominant among them being acne vulgaris which accounted for 17.3%. Face was the most important site affected and majority of the patients had grade II and Grade III acne (mild to modeare type).
- Other appendageal disorders seen in this study were acneiform eruptions, hair loss (secondary to telogen effluvium, alopecia areata and trichotillomania), hirsuitism, premature graying of hair and sweat gland abnormalities like palmar hyperhidrosis and milaria rubra.
- Infections constituted the second commonest type of disorders seen. Fungal infections were the commonest infections seen (36.7%) followed by parasitic

infestations (28.5%), viral infections (26.5%) and bacterial infections (8.1%). The predominance of infections may also represent the inadequacies in the primary health care facilities.

- Infections were more common in the rural areas and lower socio economic strata probably attributed to overcrowding and living standards, poor hygiene and lack of awareness. The predominance of infections may also represent the inadequacies in the primary health care facilities.
- Eczemas were the third most common condition seen in our study (18.6 %). Endogenous eczema comprising atopic eczema, dyshidrotic eczema, pityriasis alba, seborrheic dermatitis constituted majority of the eczemas seen (55.8%) followed by exogenous eczemas (44.1%) comprising contact eczema and photo eczema.
- Pigmentary disorders were the next most common disorders in this study. Hyper pigmentary disorders comprising post inflammatory hyperpigmentation, peri orbital and peri ocular melanosis were common (52.1%) than the hypo pigmentary conditions like vitiligo (47.9%).
- Papulo squamous disorders formed a minor percentage (6.49 %) of the different types of skin diseases seen in this study. Most common among them being psoriasis (3.46%) followed by lichen planus (1.7 %) and keratosis pilaris (1.3%).
- Miscellaneous conditions accounted for 12.12 %, most common of them being urticaria (4.3%), insect bite reactions (2.6%), keloids (2.16%), prurigo simplex (2.16%) and phrynoderma (0.87%).

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ANNEXURES

PROFORMA

SL. No: **Date:**

Name:

Age:

Occupation:

Address:

OP/ IP No:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

Onset:

Duration:

Progression:

Associated symptoms:

Aggravating/relieving factors:

Seasonal variation:

H/o photosensitivity:

H/o topical application of medicines/cosmetics. Details if any.

PAST HISTORY:

PERSONAL HISTORY:

FAMILY HISTORY:

<u>ON EXAMINATION:</u>

GENERAL PHYSICAL EXAMINATION:

CUTANEOUS EXAMINATION:

- Morphology of the skin lesions.
- Site of distribution.

SYSTEMIC EXAMINATION:

-CVS

-RS

-P/A

-CNS

INVESTIGATIONS:

PROVISIONAL DIAGNOSIS:

PATIENT INFORMATION SHEET

Study title: A Clinico-epidemiological study of various dermatoses in adolescent girls

Study site: R.L Jalappa hospital, Tamaka, Kolar.

Aim: To study the pattern, severity and demographic profile of various dermatoses in adolescent girls.

Adolescence is associated with myriad of hormonal changes in the body which may result in skin disorders. They may also result from exogenous factors. The most common dermatoses in this age group are acne, infections, dermatitis, pigmentary disorders, appendageal disorders, papulosquamous disorders, sexually transmitted infections etc.

This study will help to know the pattern, severity and demographic profile of various dermatoses in adolescent girls. 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, required information will be collected from you (as per the proforma). Relevant investigations will be carried out if required. Photographs will be taken to document the morphology of the lesions at the time of presentation. 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 above 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 you 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 to participate in this study.

For any further clarification you can contact the study investigator:

Dr. Soumya Soragavi

Mobile no: 9591154574

E-mail id: soumyasorgavi09@gmail.com

CONSENT FORM

Study title: **A CLINICO-EPIDEMIOLOGICAL STUDY OF VARIOUS DERMATOSES IN ADOLESCENT GIRLS.**

Chief researcher/ PG guide's name: **Dr. RAJASHEKAR T S**

Principal investigator: **Dr. SOUMYA SORAGAVI**

Name of the subject:

Age:

Address:

a. I have been informed in my own vernacular language the purpose of the study, the necessity of relevant investigations to be carried out and photographs to be taken.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

e. I confirm that _____ (chief researcher/ name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that I may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant's signature

Signature of the witness: Date:

I have explained to _____ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/ Guide signature Date:

KEY TO MASTER CHART:

OP: Outpatient number.

EDU: EDUCATIONAL STATUS.

PRI: Primary.

SEC: Secondary.

H.SEC: Higher secondary.

UG: Under graduate.

ILL: Illiterate.

PLACE:

U: Urban.

R: Rural

SES: **SOCIO ECONOMIC STATUS**

SOSL: SITE OF SKIN LESION

UL / LL: Upper limb / Lower limb

F&T: Fingers and toes.

GEN: Generalised.

LAB MAJ: Labia Majora.

RA: Retroauricular.

PE: Photoexposed.

ABD: Abdominal.

FWS: Finger web spaces.

DIAGNOSIS:

AA: Alopecia areata.

AD: Atoipc dermatitis.

ACD: Allergic contact dermatitis.

AE: Acneiform eruptions.

AV: Acne vulgaris.

DE: Dyshidrotic eczema.

FO: Folliculitis.

FU: Furunculosis.

GC: Genital Candidiasis.

GW: Genital Warts.

HIR: Hirsutism.

HS: Herpes simplex.

IBR: Insect bite reaction.

ICD: Irritant contact dermatitis.

IMP: Impetigo.

KEL: Keloid.

KP: Keratosis pilaris.

LP: Lichen planus.

MC: Molluscum contagiosum

MR: Miliaria rubra

ONY: onychomycosis.

P. ALBA: Pityriasis alba.

PC: Pediculosis capitis.

PGH: Premature graying of hair.

PH: Palmar hyperhidrosis.

PHRY: Phrynoderma.

PIH: Post inflammatory hyperpigmentation.

PIV: Pityriasis versicolor

PMLE: Polymorphic light eruptions.

POHM: Periorbital hypermelanosis.

POM: Periorbital melanosis.

PPP: Palmo plantar psoriasis.

PR: Pityriasis rosea.

PS: Prurigo simplex.

PV: Psoriasis vulgaris.

SCAB: Scabies.

SD: Seborrheic dermatitis.

SP: Scalp psoriasis.

TC: Tinea corporis.

TCR: Tinea cruris.

TE: Telogen effluvium

TI: Tinea incognito.

TP: Tinea pedis.

TTM: Trichotillomania.

URT: Urticaria.

VIT: Vitiligo.

VV: Verruca vulgaris.

VZ: Varicella zoster.

GROUPS:

APP: Appendageal disorder.

INF: Infection.

ECZ: Eczema.

PSD: Papulosquamous disorder.

PIGM: Pigmentary disorder.

MIS: Miscellaneous.

SUBGROUPS:

EXO ECZ: Exogenous eczema.

ENDO ECZ: Endogenous eczema.

PILO SEB: Pilosebaceous

BACT: Bacterial.

STI: Sexually transmitted infection.

VIR: Viral

FUNG: Fungal

PARA: Parasitic

SG: Sweat gland disorder

HYPER.P: Hyper pigmentary

HYPO.P: Hypo Pigmentary

AU: Acute urticarial.

CU: Chronic urticarial.

SEVERITY SCORES:

SALT: Severity of Alopecia Tool.

SCORAD: Scoring for Atopic dermatitis.

DASI: Dyshidrotic eczema area severity index.

HDSS: Hyperhidrosis disease severity score.

PASI: Psoriasis area severity index.

UAS: Urticaria activity score.

VIDA: Vitiligo disease activity score.

NA: Not applicable.

MASTER CHART:

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
1	100939	11	PRI	U	III	LEGS	IBR	MIS	NA	NA
2	100872	12	PRI	U	II	FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 13.4
3	100910	18	SEC	R	III	FACE	ACD	ECZ	EXO ECZ	NA
4	101227	17	H.SEC	U	II	FACE	AV-I	APP	PILO SEB	AV-I
5	100843	18	ILL	R	IV	TRUNK	MR	APP	SG	NA
6	101217	17	H.SEC	U	IV	LEG	FU	INF	BACT	NA
7	100850	18	UG	U	III	WRIST	ACD	ECZ	EXO ECZ	NA
8	100850	12	ILL	R	IV	UL,LL	PV	PSD	NA	PASI 8.4
9	100930	14	SEC	U	II	FACE	AV-I	APP	PILO SEB	AV-I
10	100935	11	PRI	U	III	UL,LL	PV	PSD	NA	PASI 7.2
11	101229	19	ILL	U	III	PALMS	ICD	ECZ	EXO ECZ	NA
12	101949	14	SEC	U	III	UL,LL	LP	PSD	NA	NA
13	101957	13	SEC	U	II	FACE	AV-I	APP	PILO SEB	AV-I
14	102083	17	ILL	R	IV	UL,LL	LP	PSD	NA	NA
15	102081	18	UG	R	III	SCALP	AA	APP	HAIR	SALT 5.4
16	102148	13	SEC	R	II	FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 12.8
17	102137	11	PRI	U	II	FACE	VIT	PIGM	HYPO.P	VIDA 3+
18	102129	12	PRI	U	III	PALMS	DE	ECZ	ENDO ECZ	DASI 14
19	107417	10	PRI	U	III	LEGS	IBR	MIS	NA	NA
20	107543	16	H.SEC	U	II	ARMS	VIT	PIGM	HYPO.P	VIDA 2+
21	107416	18	UG	U	II	FACE,RA	SD	ECZ	ENDO ECZ	NA
22	912946	17	H.SEC	U	II	FACE	AV-III	APP	PILO SEB	AV-III
23	79153	13	SEC	R	III	LEGS	IBR	MIS	NA	NA
24	79889	12	PRI	U	III	FACE	AV-III	APP	PILO SEB	AV-III
25	79884	12	PRI	R	IV	UL,LL	IBR	MIS	NA	NA
26	79884	11	PRI	U	II	FACE	P.ALBA	ECZ	ENDO ECZ	NA
27	71465	13	SEC	R	III	LEGS	IBR	MIS	NA	NA
28	87093	14	SEC	U	II	FACE	AV-I	APP	PILO SEB	AV-I
29	73231	15	SEC	R	III	SCALP	PC	INF	PARASITIC	NA
30	99511	13	SEC	U	III	F&T	VIT	PIGM	HYPO.P	VIDA 3+
31	99507	17	H.SEC	U	III	TRUNK	AV-III	APP	PILO SEB	AV-III
32	99466	12	PRI	U	II	UL,LL	PV	PSD	NA	PASI 10.8

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
33	99483	18	UG	U	III	UL,LL	PV	PSD	NA	PASI 10
34	99509	19	UG	U	II	FACE	AV-I	APP	PILO SEB	AV-I
35	99460	12	PRI	U	II	ARMS	VIT	PIG M	HYPO.P	VIDA 3+
36	99471	11	PRI	U	III	FACE	MC	INF	VIRAL	NA
37	99324	12	PRI	U	II	BACK	AV-II	APP	PILO SEB	AV-II
38	99506	12	PRI	R	III	FWS	SCAB	INF	PARASITIC	NA
39	99514	12	PRI	U	II	GEN	AD	ECZ	ENDO ECZ	SCORAD 16.3
40	99332	11	PRI	R	I V	GEN	SCAB	INF	PARASITIC	NA
41	99333	16	H.SEC	R	II	EARLOBE	ACD	ECZ	EXO ECZ	NA
42	99527	15	SEC	U	I	FACE	AV-I	APP	PILO SEB	AV-I
43	99327	10	PRI	U	II	PALMS	PH	APP	SG	HDSS 2
44	101998	17	H.SEC	U	II	FACE	PIH	PIG M	HYPER.P	NA
45	101949	19	UG	R	II	FACE	AV-III	APP	PILO SEB	AV-III
46	101957	15	SEC	U	III	FACE	AE	APP	PILO SEB	NA
47	101951	12	PRI	R	III	UL,LL	IBR	MIS	NA	NA
48	102057	14	SEC	U	II	FACE	AV-I	APP	PILO SEB	AV-I
49	100172	16	H.SEC	U	II	ARMS	VIT	PIG M	HYPO.P	VIDA 2+
50	100184	15	SEC	R	I V	GEN	SCAB	INF	PARASITIC	NA
51	100181	16	H.SEC	R	II	BACK	AV-II	APP	PILO SEB	AV-II
52	100476	14	SEC	U	II	SCALP	AA	APP	HAIR	SALT 15.2
53	80336	16	H.SEC	U	III	SCALP	TE	APP	HAIR	NA
54	95932	15	SEC	U	II	FACE	PIH	PIG M	HYPER.P	NA
55	100903	14	SEC	R	III	PALMS	DE	ECZ	ENDO ECZ	DASI 16
56	100853	16	H.SEC	U	II	SCALP	AA	APP	HAIR	SALT 3.6
57	100904	15	SEC	U	III	FACE	AV-I	APP	PILO SEB	AV-I
58	100873	14	SEC	R	I V	SCALP	PC	INF	PARASITIC	NA
59	100906	16	H.SEC	U	II	FACE	ACD	ECZ	EXO ECZ	NA
60	100851	17	H.SEC	U	II	SCALP	PGH	APP	HAIR	NA
61	101778	16	H.SEC	U	III	BACK	AE	APP	PILO SEB	NA
62	100920	18	UG	U	III	TRUNK	AV-III	APP	PILO SEB	AV-III
63	100944	19	UG	R	II	LEGS	PS	MIS	NA	NA
64	100866	11	PRI	U	II	FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 14
65	101998	12	PRI	R	II	FACE	P.ALBA	ECZ	ENDO ECZ	NA
66	101951	19	UG	U	III	FACE	AV-I	APP	PILO SEB	AV-I

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
67	102083	17	H.SEC	R	III	FN	ONY	INF	FUNGAL	NA
68	102126	16	H.SEC	U	I	FACE	AV-III	APP	PILO SEB	AV-III
69	102077	15	SEC	R	III	LEGS	PS	MIS	NA	NA
70	101996	15	SEC	U	II	FACE	AE	APP	PILO SEB	NA
71	81729	17	ILL	R	IV	LEGS	PS	MIS	NA	NA
72	104182	16	H.SEC	U	II	FACE	PIH	PIGM	HYPER.P	NA
73	104188	16	H.SEC	U	II	BACK	AV-II	APP	PILO SEB	AV-II
74	81019	15	SEC	U	II	FACE	AV-I	APP	PILO SEB	AV-I
75	89777	18	UG	U	III	UL,LL	PS	MIS	NA	NA
76	85544	19	UG	U	I	SCALP	PGH	APP	HAIR	NA
77	85604	18	UG	U	II	FACE	AV-III	APP	PILO SEB	AV-III
78	85650	11	PRI	U	III	ELBOWS	PHRY	MIS	NA	NA
79	85654	17	H.SEC	R	II	BACK	AV-II	APP	PILO SEB	AV-II
80	85581	15	SEC	U	II	EARLOBE	KEL	MIS	NA	NA
81	86322	13	SEC	R	III	UL,LL	PS	MIS	NA	NA
82	86807	17	H.SEC	U	II	EARLOBE	KEL	MIS	NA	NA
83	87006	12	PRI	R	II	FACE,BACK	AV-II	APP	PILO SEB	AV-II
84	234540	12	PRI	U	II	FACE	P.ALBA	ECZ	ENDO ECZ	NA
85	90442	11	PRI	R	II	FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 8.9
86	90553	12	PRI	U	IV	SCALP	PC	INF	PARASITIC	NA
87	95705	17	H.SEC	R	II	FACE,CHEST	AV-II	APP	PILO SEB	AV-II
88	95730	15	SEC	U	III	FACE	AE	APP	PILO SEB	NA
89	95738	18	UG	U	III	EARLOBE	ACD	ECZ	EXO ECZ	NA
90	95745	19	UG	U	II	FACE	AV-III	APP	PILO SEB	AV-III
91	96096	15	SEC	U	I	GEN	LP	PSD	NA	NA
92	96143	17	H.SEC	U	II	FACE	ACD	ECZ	EXO ECZ	NA
93	96147	11	PRI	U	II	PALMS	DE	ECZ	ENDO ECZ	DASI 12
94	96492	12	PRI	U	III	BACK	PR	INF	VIRAL	NA
95	96579	13	SEC	U	III	FACE	P.ALBA	ECZ	ENDO ECZ	NA
96	96485	15	SEC	U	II	SCALP	AA	APP	HAIR	SALT 7.2
97	96643	17	H.SEC	R	III	ARMS	KP	PSD	NA	NA
98	96583	18	UG	R	II	TRUNK	VZ	INF	VIRAL	NA
99	96479	19	UG	U	II	EARLOBE	KEL	MIS	NA	NA
100	96766	13	SEC	U	I	FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 12.4
101	84986	15	SEC	U	II	PALMS	DE	ECZ	ENDO ECZ	DASI 16

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
102	106332	12	PRI	U	IV	SCALP	PC	INF	PARASITIC	NA
103	92967	15	SEC	U	III	TRUNK,FACE	VZ	INF	VIRAL	NA
104	102060	16	H.SEC	U	III	CHEST	KEL	MIS	NA	NA
105	101251	14	SEC	R	IV	FACE,RA	SD	ECZ	ENDO ECZ	NA
106	100843	18	UG	U	III	FACE	AV-III	APP	PILO SEB	AV-III
107	100664	19	ILL	R	IV	LAB MAJ	GW	INF	STI	NA
108	100478	13	SEC	R	II	FACE	P.ALBA	ECZ	ENDO ECZ	NA
109	100489	18	UG	U	II	FACE	AV-II	APP	PILO SEB	AV-II
110	100485	14	SEC	U	III	PE	PMLE	ECZ	EXO ECZ	NA
111	31400	13	SEC	U	II	FACE	AV-II	APP	PILO SEB	AV-II
112	111027	12	PRI	U	III	ELBOWS	PHRY	MIS	NA	NA
113	32376	17	H.SEC	R	III	ABD	TC	INF	FUNGAL	NA
114	146995	16	ILL	R	V	ABD	TC	INF	FUNGAL	NA
115	127189	15	SEC	U	IV	FACE,RA	SD	ECZ	ENDO ECZ	NA
116	121720	16	H.SEC	R	II	LEG	FU	INF	BACT	NA
117	121727	17	H.SEC	U	III	EARLOBE	KEL	MIS	NA	NA
118	121709	15	SEC	U	IV	PE	PMLE	ECZ	EXO ECZ	NA
119	86783	13	SEC	R	II	BACK	AV-II	APP	PILO SEB	AV-II
120	115745	19	UG	U	III	FACE	AV-IV	APP	PILO SEB	AV-IV
121	116687	18	UG	U	III	HANDS	VV	INF	VIRAL	NA
122	47104	13	SEC	U	IV	FACE	PMLE	ECZ	EXO ECZ	NA
123	111855	12	PRI	R	IV	FACE	IMP	INF	BACT	NA
124	83326	11	PRI	U	II	FACE	FO	INF	BACT	NA
125	106356	18	ILL	U	IV	VAG	GC	INF	STI	NA
126	106342	15	SEC	U	III	FACE	AE	APP	PILO SEB	NA
127	151805	16	H.SEC	U	IV	HANDS	VV	INF	VIRAL	NA
128	151467	15	SEC	U	II	BACK	AV-II	APP	PILO SEB	AV-II
129	152545	17	H.SEC	R	II	FACE	AV-II	APP	PILO SEB	AV-II
130	86783	18	UG	U	III	PE	PMLE	ECZ	EXO ECZ	NA
131	151560	16	H.SEC	R	IV	FACE	AE	APP	PILO SEB	NA
132	151214	15	SEC	U	IV	BACK	PIV	INF	FUNGAL	NA
133	63983	16	H.SEC	R	III	FACE,RA	SD	ECZ	ENDO ECZ	NA

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
134	51690	12	PRI	U	III	BACK	PIV	INF	FUNGAL	NA
135	64231	14	SEC	U	IV	PE	PMLE	ECZ	EXO ECZ	NA
136	64713	11	PRI	R	II	FACE	AV-II	APP	PILO SEB	AV-II
137	66394	18	UG	U	III	PE	PMLE	ECZ	EXO ECZ	NA
138	25267	16	H.SEC	R	IV	ABD	TC	INF	FUNGAL	NA
139	74357	17	H.SEC	U	III	FACE	AE	APP	PILO SEB	NA
140	74359	15	SEC	R	IV	SCALP	PC	INF	PARASITIC	NA
141	83054	16	H.SEC	U	III	BACK	PIV	INF	FUNGAL	NA
142	28294	15	SEC	U	IV	NECK	PIV	INF	FUNGAL	NA
143	100218	17	H.SEC	U	IV	FEET	VV	INF	VIRAL	NA
144	70458	14	SEC	R	III	FACE	AV-II	APP	PILO SEB	AV-II
145	70473	18	ILL	U	IV	ARMS	KP	PSD	NA	NA
146	136375	17	H.SEC	U	IV	UP CHEST	PIV	INF	FUNGAL	NA
147	155957	16	H.SEC	R	III	FACE	VV	INF	VIRAL	NA
148	155977	15	SEC	R	II	FACE	AV-II	APP	PILO SEB	AV-II
149	155971	18	UG	R	III	ARMS	KP	PSD	NA	NA
150	159694	11	PRI	U	IV	SCALP	PC	INF	PARASITIC	NA
151	161936	14	SEC	R	II	HANDS	VV	INF	VIRAL	NA
152	165028	15	SEC	R	III	FWS	SCAB	INF	PARASITIC	NA
153	174824	17	PRI	R	III	PE	PMLE	ECZ	EXO ECZ	NA
154	8999	16	H.SEC	R	II	FACE	POM	PIGM	HYPER.P	NA
155	71645	12	PRI	U	II	SCALP	SP	PSD	NA	PASI 1
156	178654	11	PRI	R	III	PE	PMLE	ECZ	EXO ECZ	NA
157	155060	12	PRI	U	IV	UL,LL	VIT	PIGM	HYPO.P	VIDA 3+
158	183369	12	PRI	R	IV	FEET	TP	INF	FUNGAL	NA
159	189984	13	SEC	U	III	F&T	VIT	PIGM	HYPO.P	VIDA 1+
160	193105	11	PRI	R	II	FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 12.8
161	210048	13	SEC	R	IV	HANDS	TI	INF	FUNGAL	NA
162	292241	13	SEC	R	IV	FACE	HS	INF	VIRAL	NA
163	291161	11	PRI	U	III	GEN	AD	ECZ	ENDO ECZ	SCORAD 15.2
164	192979	15	SEC	U	III	SCALP	TE	APP	HAIR	NA

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
165	289688	16	H.SEC	U	III	FACE,LL	VIT	PIGM	HYPO.P	VIDA 2+
166	178686	17	H.SEC	U	IV	SCALP	TTM	APP	HAIR	NA
167	213638	14	ILL	U	V	UL,LL	VIT	PIGM	HYPO.P	VIDA 4+
168	295774	11	PRI	R	IV	HANDS	MC	INF	VIRAL	NA
169	195317	10	PRI	U	II	LIP	VIT	PIGM	HYPO.P	VIDA 0
170	155759	17	H.SEC	U	II	FACE	AV-II	APP	PILO SEB	AV-II
171	232662	18	UG	R	III	FACE	AV-IV	APP	PILO SEB	AV-IV
172	289727	19	UG	U	II	FACE	AV-II	APP	PILO SEB	AV-II
173	290131	18	UG	R	III	TRUNK	AV-III	APP	PILO SEB	AV-III
174	222133	11	PRI	R	II	FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 9.1
175	274494	19	UG	U	III	TN	ONY	INF	FUNGAL	NA
176	238418	18	UG	U	III	FACE,CHEST	HIR	APP	HAIR	NA
177	106666	16	H.SEC	U	IV	PE	PMLE	ECZ	EXO ECZ	NA
178	289913	14	SEC	U	III	FACE,LL	VIT	PIGM	HYPO.P	VIDA 3+
179	208547	10	PRI	R	IV	GEN	SCAB	INF	PARASITIC	NA
180	290766	19	UG	U	II	FACE	PIH	PIGM	HYPER.P	NA
181	289596	18	UG	R	II	TN	ONY	INF	FUNGAL	NA
182	194631	17	H.SEC	U	III	FACE	AE	APP	PILO SEB	NA
183	49068	14	SEC	U	III	FACE	HS	INF	VIRAL	NA
184	208082	14	SEC	R	II	FACE	ACD	ECZ	EXO ECZ	NA
185	238290	19	ILL	R	V	GEN	SCAB	INF	PARASITIC	NA
186	278650	18	UG	U	II	FACE	POHM	PIGM	HYPER.P	NA
187	284691	16	H.SEC	U	III	FACE	PIH	PIGM	HYPER.P	NA
188	295328	18	ILL	R	IV	GROINS	TCR	INF	FUNGAL	NA
189	208550	19	ILL	U	IV	GEN	URT	MIS	AU	UAS 5
190	296470	17	PRI	U	III	PE	PMLE	ECZ	EXO ECZ	NA
191	164240	19	UG	R	III	FACE	AV-III	APP	PILO SEB	AV-III
192	208573	17	H.SEC	U	III	FACE	POHM	PIGM	HYPER.P	NA
193	291733	18	UG	U	II	FACE	PIH	PIGM	HYPER.P	NA
194	289791	19	ILL	R	IV	FWS,GENI	SCAB	INF	PARASITIC	NA
195	290092	18	ILL	U	V	GEN	URT	MIS	AU	UAS 6

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
196	294635	16	H.SEC	R	IV	ABD	TC	INF	FUNGAL	NA
197	200954	14	SEC	R	III	UL,LL	LP	PSD	NA	NA
198	228682	10	PRI	U	III	FACE, FLEXURES	AD	ECZ	ENDO ECZ	SCORAD 13.4
199	198024	18	UG	U	II	SCALP	TE	APP	HAIR	NA
200	961282	19	UG	U	II	ARMS	PIH	PIGM	HYPER.P	NA
201	282620	19	ILL	R	V	FEET	VV	INF	VIRAL	NA
202	273273	17	H.SEC	U	III	SCALP	TE	APP	HAIR	NA
203	221986	19	ILL	R	IV	THIGH	TC	INF	FUNGAL	NA
204	272140	19	UG	R	III	TRUNK	AV-III	APP	PILO SEB	AV-III
205	274453	18	UG	U	III	FACE	PIH	PIGM	HYPER.P	NA
206	290826	16	H.SEC	U	III	FACE	PIH	PIGM	HYPER.P	NA
207	289795	19	UG	R	III	FACE	AE	APP	PILO SEB	NA
208	289797	10	PRI	U	III	GEN	AD	ECZ	ENDO ECZ	SCORAD 19.6
209	297388	19	UG	U	III	GEN	URT	MIS	AU	UAS 5
210	205575	18	UG	U	III	FACE,CHEST	HIR	APP	HAIR	NA
211	228715	19	UG	U	III	SCALP	TE	APP	HAIR	NA
212	210656	17	H.SEC	R	III	TRUNK	MR	APP	SG	NA
213	234460	19	UG	U	III	PALMS	PH	APP	SG	HDSS 3
214	83098	19	ILL	R	IV	VAG	GC	INF	STI	NA
215	178572	17	ILL	R	V	ABD,LL	TC	INF	FUNGAL	NA
216	216083	16	H.SEC	R	III	WRIST	ACD	ECZ	EXO ECZ	NA
217	289484	14	SEC	U	IV	GEN	URT	MIS	CU	UAS 4
218	270172	18	ILL	R	IV	ABD	TC	INF	FUNGAL	NA
219	167818	15	SEC	U	III	GEN	URT	MIS	AU	UAS 5
220	112719	15	SEC	U	IV	FACE,CHEST	HIR	APP	HAIR	NA
221	289440	16	ILL	R	V	GEN	URT	MIS	AU	UAS 5
222	288811	13	SEC	U	III	UL,LL	PV	PSD	NA	PASI 7.2
223	289498	17	ILL	R	IV	GEN	URT	MIS	AU	UAS 4
224	288844	18	UG	R	III	P&S	PPP	PSD	NA	PASI 1.8
225	288623	17	ILL	R	IV	GEN	URT	MIS	AU	UAS 4
226	252142	15	SEC	U	III	PALMS	PH	APP	SG	HDSS 3
227	277328	14	SEC	R	IV	GEN	URT	MIS	CU	UAS 4

SL NO	OP/IP	AGE(YRS)	EDU	PLACE	SES	SOSL	DIAGNOSIS	GROUP	SUBGROUPS	SEVERITY
228	287237	18	ILL	U	IV	GEN	SCAB	INF	PARASITIC	NA
229	286937	15	SEC	U	IV	PALMS	PH	APP	SG	HDSS 3
230	276542	15	SEC	U	V	GEN	URT	MIS	AU	UAS 5
231	276438	17	ILL	R	V	ABD,UL,LL	PV	PSD	NA	PASI 16.2