A CLINICAL SPECTRUM OF SCALP DERMATOSES IN ADULTS PRESENTING TO A TERTIARY REFERRAL CARE CENTRE

BY

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Dissertation Submitted to

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Doctor of Medicine (M.D)

in

DERMATOLOGY, VENEREOLOGY AND LEPROSY

UNDER THE GUIDANCE OF

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April 2014

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I hereby declare that this dissertation entitled **"A CLINICAL SPECTRUM OF SCALP DERMATOSES IN ADULTS PRESENTING TO A TERTIARY REFERRAL CARE CENTRE"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. RAJENDRA OKADE, M.D,DNB. Professor, Dept of DERMATOLOGY, VENEREOLOGY, LEPROSY,** Sri Devaraj Urs Medical College, Kolar.

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LIST OF ABBREVIATIONS

LPP	:	Lichen planopilaris
SCAP	:	Syringocystadenoma papilliferm
HLA	:	Human leukocyte antigen
IL	:	Interleukin
TNF-α	:	Tumour necrosis factor-α
NF-κB	:	Nuclear factor kappa-light-chain-enhancer of activated B cells
TGF	:	Transforming growth factor
LPC	:	Liquor picis carbonis
LCD	:	Liquor carbonis detergens
HIV	:	Human immunodeficiency virus
AIDS	:	Acquired immunodeficiency syndrome
α-MSH	:	α- melanocyte stimulating hormone
M. furfur	:	Malassezia furfur
M. globosa	:	Malassezia globosa
M.restricta	:	Malassezia restricta
Dsg	:	Desmoglein
PV	:	Pemphigus vulgaris
AGA	:	Androgenetic alopecia
LE	:	Lupus erythematosus
DLE	:	Discoid lupus erythematosus
SLE	:	Systemic lupus erythematosus
PRP	:	Pityriasis rubra pilaris
HIV-EF	:	HIV-related eosinophilic folliculitis
IRIS	:	Immune reconstitution inflammatory syndrome

ABSTRACT

BACKGROUND: Hair and scalp disorders generally are not associated with significant physical morbidity but the psychological impact of visible scalp problems may be very high. The scalp is unique among skin areas in humans, with high follicular density and a high rate of sebum production. The skin of the scalp has several unique features that aid in its critical role of protecting the head. The follicular density is much higher, creating a dark, warm and moist environment. This provides thermal insulation, but also creates an environment conducive to the superficial mycotic infections that play a role in dandruff, seborrheic dermatitis, and tinea capitis, and for parasitic infestations such as pediculosis capitis. As there is a paucity of studies on scalp dermatoses in the Indian and the Western literature, a clinical spectrum of these scalp dermatoses can unravel the common clinical manifestations in our population.

OBJECTIVES: To study the spectrum of scalp dermatoses in adults.

To study the various clinical patterns of scalp dermatoses in adults

MATERIAL AND METHODS: The study was undertaken from January 2012 to June 2013. All adult patients reporting to the Department Of Dermatology, Sri R.L. Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar were evaluated for entry into the study and patients having scalp lesions were enrolled. A detailed history of all such patients was taken including general status of the patient, systemic diseases, medications used, precipitating factors such as sunlight, alcohol, smoking, drugs and trauma. Complete clinical and a thorough scalp examination was performed. During the clinical

examination, the following elements were analysed such as morphology of the lesion, anatomical location and colour changes.

RESULTS AND DISCUSSION: Scalp dermatoses are common in adult population, with prevalence of 2.85%. Majority of the patients with scalp dermatoses belonged to the age group of 41-50 years (30.9%), followed by 3rd decade (29.8%).Males (60.8%) were affected more than females (39.2%), with a male: female ratio 1.55:1. Scalp was the initial site of involvement in 73.1% of the cases studied. Lesions exclusively over the scalp were seen in 47.3% of adults. Multiple regions of the scalp were affected in 69.6% of the patients, with parietal area being involved in 57.3% of cases. Inflammatory conditions (72.5%) predominated in our study. The most common dermatosis was psoriasis which constituted 33.3% of cases, followed by seborrhoeic dermatitis(18.7%), pityriasis sicca (11.6%) and vitiligo (9.9%).

KEY WORDS: scalp dermatoses, scalp psoriasis, seborrhoeic dermatitis

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“ What is most difficult of all? It is what appears most simple: To see with your eyes what lies in front of your eyes.”

- Goethe

INTRODUCTION

Although hair and scalp disorders generally are not associated with significant physical morbidity, the psychological impact of visible scalp problems may be very high.[1]

In human societies, hair and scalp now play an important role in appearance and sexual signaling; original functional roles of protection and heat conservation are secondary, and changes in the appearance of skin and hair affect self-esteem and confidence in social settings. It should also be recognized that scalp changes in some cases may be a sign of a more substantial medical problem, thus making the differential diagnosis critically important.

Many of the common scalp conditions share similar clinical manifestations of pruritus, scaling, inflammation and hair loss, complicating diagnosis, but a correct diagnosis is essential to initiate a proper treatment.[1]

The scalp also participates in many systemic disorders and frequently is the chief site of involvement. Similarly, many generalized cutaneous disorders exhibit their most typical manifestations in the scalp.

Hyperkeratosis (scaling), pruritus, alopecia, and inflammatory signs (erythema, purulence) are common symptoms of scalp disorders. Scaling and pruritus are extremely common complaints. In a survey of 735 adults in the United States, 39% reported having experienced some flaking, and almost 50% complained of scalp itch.[1]

As there is a paucity of studies on scalp dermatoses in the Indian and the Western literature, a clinical study of these scalp dermatoses can unravel the common clinical manifestations in our population.

AIMS OF THE STUDY

- To study the spectrum of scalp dermatoses in adults.
- To study the various clinical patterns of scalp dermatoses in adults.

REVIEW OF LITERATURE

"Skin, skin is a wonderful thing,

Keeps the outside out and the inside in."

HISTORICAL ASPECTS

Unna and Sabouraud were among the first to describe seborrhoeic dermatitis, favouring an aetiological role for bacteria, yeasts, or both.[2]

Raymond Sabouraud identified a dandruff-causing organism in 1904 and called it "Pityrosporum malassez", honoring Malassez, but at the species level as opposed to the genus level.[3]

Pityriasis amiantacea was first described by Alibert in 1832, and was called 'la porrigineamiantacée' because its scales resembled the grayish substance surrounding the feathers of juvenile and molting birds and had the appearance of asbestos (amiante).[4,5]

Psoriatic alopecia was first described by Shuster in 1972.[6]

Aplasia cutis congenita, a rare congenital disorder characterized by localized absence of skin and dermal appendages was first described by Cordon in 1767.[7]

Nevus sebaceous was first identified in 1895 by Jadassohn [8]and later the term was used to describe a congenital hamartoma on the scalp or face showing an excess of sebaceous glands, abortive hair follicles and ectopic apocrine glands. Mehregan and Pinkus preferred the term 'organoid neavus'.[9]

Eosinophilic pustular folliculitis is a recurrent pruriginous condition which was first described in 1984 by Lucky and coworkers.[10] The first clear description of lupus erythematosus was given by Biett and it was reported by his student Cazenave under

the term erythema centrifugum in 1833. In 1851 Cazenave renamed erythema centrifugum as lupus erythematosus and gave a classic description of discoid lupus erythematosus. In 1872, Kaposi subdivided lupus into the discoid and systemic forms and introduced the concept of systemic disease with a potentially fatal outcome.[11]

The description of an acute inflammatory disease of the scalp with production of purulent material is attributed to Celsus around 30 BCE in Rome, leading to the name kerion celsi. During the 19th century, tinea capitis was a very serious public health problem that reached epidemic proportions; it was apparently introduced to the American continent by the Europeans.[12]

Lichen planopilaris (LPP) also known as lichen follicularis, is a cutaneous disorder selectively involving hair follicles which was initially described by Pringle in 1895.[13] The North American Hair Research Society (NAHRS) placed LPP in the lymphocytic group of disorders in their classification.[14]

Ancell first described multiple cylindromas of the head and abdomen in 1824.[15] Spiegler and Brooke described the clinicopathological features of cylindroma and named it Brooke–Spiegler syndrome, an inherited disease affecting the folliculosebaceous apocrine unit.[16,17]

Lynch et al described a case of atypical necrobiosis lipoidica as arcuate plaques that appeared on the face and scalp.[18]

Syringocystadenoma papilliferm (SCAP), an uncommon benign tumor of disputed histogenesis with a predilection for the scalp and forehead was first described by John Stokes in 1917 under the term nevus syringoadenomatous papilliferum.[19]

The scalp is unique among skin areas in humans, with high follicular density and a high rate of sebum production. The skin of the scalp has several unique features that aid in its critical role of protecting the head.

First, the follicular density is much higher, creating a dark, warm and moist environment. This provides thermal insulation, but also creates an environment conducive to the superficial mycotic infections that play a role in dandruff, seborrheic dermatitis, and tinea capitis, and for parasitic infestations such as pediculosis capitis.[20]

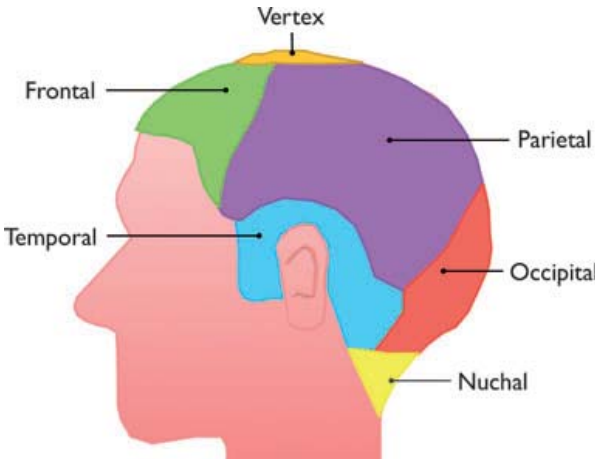
Second, in adults there is a high rate of sebum production, which along with desquamated skin cells can provide a food source for microorganisms.

Finally, the scalp skin is subjected to brushing and contact with fingers, combs, hats, other styling implements that can cause frictional injury and may introduce microorganisms, thereby increasing the likelihood of infections and infestations.

Scalp changes may also be seen in inflammatory conditions such as psoriasis.

The similarities in clinical signs and symptoms of many scalp conditions can complicate accurate diagnosis.

Scalp can be divided into the following anatomic regions as shown in the figure below .



AETIOLOGY:

Scalp disorders can be classified as:[21]

GENETIC/CONGENITAL	:	Epidermal naevi
		Sebaceous naevi
		Aplasia cutis congenita
INFLAMMATORY	:	Psoriasis
		Seborrhoeic dermatitis and Dandruff
		Pityriasis amiantacea
		Pemphigus vulgaris
		Lichen planopilaris
		Lupus erythematosus
		Pityriasis rubra pilaris
INFECTIVE		
Bacterial	:	Folliculitis
		Furuncle
Fungal	:	Tinea capitis
Viral	:	Herpes Zoster
		Varicella
SCLEROSING	:	Morphea
		Scleroderma
		Lichen sclerosus
GRANULOMATOUS	:	Sarcoidosis
		Necrobiosis lipoidica

NEOPLASTIC

Benign	:	Cylindroma
		Syringocystadenoma papilliform
Malignant		
Primary	:	Basal cell carcinoma
		Squamous cell carcinoma
Secondary	:	Renal cell carcinoma
		Breast carcinoma
MISCELLANEOUS	:	Vitiligo
		Trauma
		Radiodermatitis
		Post operative

1. GENETIC/CONGENITAL DISORDERS

SEBACEOUS NAEVUS:

Synonym: Naevus sebaceus of Jadassohn

Organoid nevus

Sebaceous naevi are epidermal hamartomas comprising predominantly of sebaceous glands. Sebaceous naevi and verrucous epidermal naevi as variants of the same disorder is supported by the occurrence of both types in the same individual and in the epidermal naevus syndrome.[22,23]

They are usually sporadic, probably reflecting lethal genes rescued by mosaicism. Because sebaceous naevi have a tendency to develop tumours, various

tumour genes have been implicated in their aetiology. [24] These nevi have a predisposition to neoplastic change.

Epidemiology:

Sebaceous naevi occur in about 0.3% of all neonates with equal incidence by sex.[24]

Clinical features:

Naevus sebaceous presents as circumscribed, hairless, yellow, waxy, and verrucous plaque with a velvety surface. It can be flat at birth, becoming plaque-like under the hormonal influences of puberty. It most commonly occurs singly, but may be multiple and extensive, like verrucous epidermal naevi. Most occur on the head and neck, favouring the scalp, the areas around the ears, the temples, forehead and the central part of the face. They can extend on to the oral mucosa.

A variety of appendageal tumours, sometimes multiple, may develop within sebaceous naevi. The most commonly reported are syringocystadenoma papilliferum and trichoblastoma;[25] less common tumours include nodular hidradenoma,[26] apocrine cystadenoma,[27]syringoma, infundibuloma, trichoadenoma,[28] trichilemmoma and pilomatricoma.[29] Locally invasive and malignant tumours include keratoacanthoma ,[30] proliferating trichilemmal cyst ,[31] basal cell ,[32] sebaceous, apocrine ,[33] eccrine [34] and squamous carcinomas[35] and malignant melanoma.[30]

APLASIA CUTIS CONGENITA:

Synonym : **Congenital Absence of skin.**

It is hypothesized to be due to a basic developmental defect which may be aggravated or complicated by factors in the intrauterine environment such as

nutritional, chemical and toxic insults.[36] An autosomal pattern of inheritance is noted in several familial reports.[37,38]

Clinical features:

About 80% of all lesions are located on the scalp. The parietal hair whorl is the commonest site; this predilection may be explained on the basis of scalp tension which is maximum in the dorsal midline due to rapid brain growth in the tenth to eighteenth weeks of gestation.[39,40] It is usually single and of variable diameter; the lesions may present as superficial or deep ulcers, bullae or scars.

Scalp lesions may show defects of the underlying skull and dura as well.

2. INFLAMMATORY DISORDERS

PSORIASIS:

Psoriasis is a common, chronic, inflammatory and proliferative condition of the skin, in which both genetic and environmental influences have a critical role.

Epidemiology:

Psoriasis of the scalp is a frequently occurring condition affecting approximately 2% of the Western population.[41] According to one survey, [42] the scalp is the most commonly affected part of the body with involvement occurring in 79% of psoriasis patients. The scalp is one of the first sites affected in 25% of patients with psoriasis and the frequency of involvement increases with duration.[43, 44] Scalp psoriasis also represents a social impediment in nearly 50% of patients, with pruritus and scaling of lesions being the most commonly identified symptoms.[45] Psoriasis observed on the scalp could be an early indication of psoriatic arthritis, as anywhere from 6 to 39% of those with psoriasis develop inflammation of the joints.[46]

Aetiopathogenesis:

The riddle of psoriasis remains unsolved despite intensive basic and clinical investigations. Various factors like genetic, environmental and immunologic play a key role in the pathogenesis of psoriasis.

Role of genetic factors:

In a North Indian study, 9.8% of the children had a positive family history; the figure was as high as 28% in another study from Kuwait.[47] If only one parent has psoriasis, then the risk for the child developing psoriasis is 16%.[48] It increases to 50% if both parents have psoriasis.[49] Twin pair analysis has revealed 72% concordance among monozygotic twins compared to 22% concordance among dizygotic twins.[50] Due to genomic imprinting, men are more likely than women to transmit psoriasis to the offspring.[51]

Psoriasis has been associated with many (human leukocyte antigen) HLA haplotypes. By using linkage analysis and genome-wide association studies, at least nine candidate loci have been identified: 6p (PSORS1), 17q25 (PSORS2), 4q34 (PSORS3), 1q21 (PSORS4), 3q21 (PSORS5), 19p13 (PSORS6), 1p32 (PSORS7), 16q (PSORS8) and 4q31 (PSORS9).[52]

Role of environmental factors:

Several environmental factors such as physical trauma, psychological stress, drugs [53]and infections, may trigger the disease in a genetically predisposed individual.

Drugs can cause drug triggered psoriasis (i.e., induction of psoriatic lesions on clinically uninvolved skin in patients with psoriasis) as well as drug induced psoriasis (i.e.precipitation of the disease in genetically predisposed individuals). Although a plethora of drugs have been implicated in provoking psoriasis, the strongest evidence is for lithium, beta-blockers, anti-malarials, non-steroidal anti-inflammatory drugs and

tetracyclines. In addition, angiotensin-converting enzyme inhibitors, interferons, digoxin, clonidine, carbamazepine, valproic acid, calcium-channel blockers, granulocyte-colony stimulating factor, potassium iodide, ampicillin, penicillin, progesterone, morphine and acetazolamide have been reported to exacerbate psoriasis.[54,55,56,57]

Immunologic factors:

Activated T cells are believed to be the primary modulators in the pathogenesis of psoriasis.[58,59,60] Disordered cellular immunity involving inflammatory cytokines such as IL-1, IL-6, tumour necrosis factor-α [TNF-α] and proinflammatory transcription factors (NF-κB signal transduction and transcription and AP-1) have all been implicated.[61,62]

Naïve T-cells can differentiate into any of the four types of inflammatory cells (viz. Th1, Th2, Th17 or T regulatory cells) depending on the presence of TNF-α, TGF-β and IL-6. [63] In the presence of TGF-β and IL-6, naive T-cells transform into Th17 cells which enter the circulation and extravasate through the endothelium to the sites of inflammation in skin where they produce the Th1-Th2-Th17 imbalance.[64] The role of the IL-23/Th17 pathway has been intensely researched in recent years. IL-23, a heterodimer composed of p19 and p40 subunits, is produced by dendritic cells and macrophages.[65,66] It causes activation of Th17 cells to produce IL-17 and IL-22 which in turn results in an increase in the pro-inflammatory cytokines like S-100, A7, β-defensins and lipocalin.[67,68] Increased levels of IL-17 also promote keratinocytes to produce CXC-chemokines and CCL-20, both of which attract neutrophils to the site of inflammation.[69] Increased IL-22 levels lead to epidermal acanthosis and abnormal keratinocyte differentiation.

Angiogenic factors produced by epidermal keratinocytes are now recognized as drivers of abnormal dermal vascular proliferation and angiogenesis. Levels of vascular endothelial growth factor are raised in psoriatic plaques.[70]

Clinical features:

Scalp psoriasis is characterized by sharply demarcated erythematosquamous lesions with silvery-white scaling. The whole scalp may be diffusely involved, or multiple discrete plaques of varying size may be seen. Often, very thick plaques develop, especially at the occiput. Pruritus and burning may accompany the lesions and the severity can fluctuate with time.

Hair shafts may appear funneled together, producing the ‘‘tepee” sign.[71]Hair shafts may also be dry and brittle, and, in some cases, the disease process leads to telogen effluvium, causing extensive hair loss.[72] Long lasting psoriatic plaques may cause scarring alopecia.

Similar lesions may appear on other body parts, which can aid in diagnosis.

Histopathology:

The histopathology in psoriasis is characterized by acanthosis with elongated rete ridges, parakeratosis and a mixed inflammatory infiltrate around tortuous and elongated capillaries. Penetration of lymphocytes and polymorphonuclear neutrophils into the epidermis is a characteristic feature.[73]

Differential diagnosis:

In seborrhoeic dermatitis, the lesions are lighter in colour, less well-defined and covered with a dull or branny scale.

Lichen simplex can resemble psoriasis closely, particularly on the scalp. The intensified skin markings, rather ill-defined edge and the marked itching are characteristic.

Treatment:

Scalp psoriasis continues to be a disease that is difficult to treat and causes significant patient morbidity. Topical corticosteroids remain the mainstay of treatment when short-term treatment is required. Long-term therapy utilizing topical corticosteroids carries an associated risk of atrophy and corticosteroid absorption. Therefore, topical corticosteroids should be used intermittently or various steroid-sparing agents, including calcipotriol/calcipotriene, tazarotene, salicylic acid, anthralin, and tar, should be used when long-term administration is required. These medications should be considered in combination with corticosteroids to enhance efficacy while minimizing the risk of toxicity of topical corticosteroids.[74]

Clobetasol propionate 0.05% and betamethasone dipropionate 0.05% are among the most potent topical corticosteroid preparations currently used.

Anthralin 0.1–3% cream has been used for long-term treatment of scalp psoriasis. Anthralin is applied in a thin layer to the psoriatic area once daily, rubbed in well and left on the scalp for 5–10 minutes before washing with a shampoo and rinsing well.

Coal tar is an effective and a cheap treatment modality for scalp psoriasis. Staining and a pungent odor are the problems associated with its use. Topical tar solution [liquor picis carbonis (LPC) or liquor carbonis detergens (LCD)] is widely available and commonly used for scalp psoriasis.

The treatment of scalp psoriasis can be divided into four phases.

First phase: involves descaling using salicylic acid or urea preparations.

Second phase: clearing phase in which topical corticosteroids, vitamin D analogs, tar, dithranol, antifungal treatment, ultraviolet B light therapy or systemic treatment are used.

Third phase: stabilization phase, wherein a steroid sparing vitamin D analog during the week and a super potent topical corticosteroid at weekends are used.

Fourth phase: is maintenance, using a vitamin D analog alone or with a tar shampoo.[75]

Second line treatments for recalcitrant disease include phototherapy and systemic medications like methotrexate, retinoids, cyclosporine and biologics.

First line therapies[75]

Salicylic acid / Urea

Topical corticosteroids (short term use)

Calcipotriol

Dithranol / Anthralin

Coal tar (Shampoo / pomade)

Tazarotene

Combination therapies

Second line therapies (For recalcitrant or severe disease)

Phototherapy

Systemic drugs (Methotrexate, Acitretin, Cyclosporine)

Biologics

SEBORRHOEIC DERMATITIS AND DANDRUFF:

Synonyms : Pityrosporal dermatitis

Dermatitis of the sebaceous areas

Pityriasis sicca

Seborrhoeic dermatitis is a chronic inflammatory skin disorder characterized in immunocompetent adults by erythema, greasy-looking scales over areas rich in

sebaceous glands namely the scalp, face, chest, back and flexural areas.[76,77] Dandruff (pityriasis sicca) represents a mild form of seborrhoeic dermatitis with fine scalp scaling without the visible presence of inflammation.[78] An oily type, pityriasis steatoides, is accompanied by erythema and an accumulation of thick crusts.[78]

Epidemiology:

The reported prevalence of seborrhoeic dermatitis in the adult population ranges from 1 to 5%.[76, 79]

Seborrhoeic dermatitis has two age peaks, one in infancy within the first three months of life and the second around fourth to seventh decade.[2] The adult form of seborrhoeic dermatitis presents first around puberty, correlating with the increase in cutaneous lipids resulting from androgen-driven sebaceous gland development and sebum secretion.[80] At all ages, seborrhoeic dermatitis is more common in males than in females.

Seborrheic dermatitis is found in upto 85% of patients with HIV and AIDS.[2] An increased incidence of seborrhoeic dermatitis is also seen in patients with Parkinson's disease and other neurological disorders like post cerebrovascular accidents, epilepsy, central nervous system disorders, facial nerve palsy, and syringomyelia induced by neuroleptic drugs with extrapyramidal side effects.[81]

Aetiopathogenesis:

Although the aetiology of seborrhoeic dermatitis is not definitely known, there are three principal factors that appear to play a role:

i) Increased sebaceous gland secretion (seborrhoea)

ii) Alteration in colonization and metabolism of cutaneous microflora (*Malassezia spp and others*),

iii) Individual susceptibility and host response.[82]

Seborrhoea:

Although maturation of the sebaceous glands may be a permissive factor for the development of seborrhoeic dermatitis, the role of seborrhoea in the pathogenesis of the condition is debatable. The supporting evidences for the role of seborrhoea are the sites of predilection – face, ears, scalp, and upper trunk, which are particularly rich in sebaceous follicles[2] and an increased prevalence of seborrhoeic dermatitis among patients with acne vulgaris and rosacea.[83] The increase in the pool of sebum in immobile skin could be the pathogenic factor for the increased incidence of seborrhoeic dermatitis seen in patients with Parkinson's disease and other neurological disorders.[84] Elevated levels of circulating α- melanocyte stimulating hormone (α-MSH) in Parkinson's patients has been documented, supporting the hypothesis that changes in sebum levels are due to endocrine rather than neurotropic factors.[85] Treatment of parkinsonism with levodopa can improve seborrhoeic dermatitis in some patients with associated reduced sebum excretion.[86]

In patients with seborrhoeic dermatitis, qualitative abnormalities in the composition of sebum have not been demonstrated consistently [2] except in a study,[87] where the lipid composition of sebum was characterized by an increased proportion of cholesterol, triglycerides, and paraffin, and a decrease in squalene, free fatty acids, and wax esters.

Alteration in colonization and metabolism of cutaneous microflora

A more causal link seems to exist between seborrhoeic dermatitis and the proliferation of Malassezia species (e.g., *Malassezia furfur, Malassezia ovalis*) found in normal dimorphic human flora.[88,89] The causal relationship is implied because of the ability to isolate Malassezia in patients with seborrhoeic dermatitis and by its therapeutic response to antifungal agents.[90] Yeasts of this genus predominate and are

found in seborrhoeic regions of the body that are rich in sebaceous lipids (e.g., head, trunk, upper back). Yeast of the genus *Malassezia* is also increased in the scaly epidermis of dandruff and seborrhoeic dermatitis. Although it has been suggested that this is secondary to the increase in size of the habitat provided by the scaling, it is generally accepted that the presence of yeast of the genus *Malassezia* causes the condition.[91]

The malassezia species that have been most commonly associated with seborrhoeic dermatitis are *M.globosa* and *M. restricta*, both of which are commensal yeasts that require an exogenous source of lipids.[92] A cell mediated immune response to *M. furfur* has been found in normal individuals also.

Seborrheic dermatitis is also associated with nutritional deficiencies, but there is no firm linkage.[93]

Overgrowth of M. furfur may lead to inflammation, either through introduction of yeast- derived metabolic products into the epidermis or as a result of the presence of yeast cells on the skin surface.[2] The mechanism of production of inflammation is through activation of Langerhans cells and T lypmhocytes by *Malassezia* or its by-products. When M.furfur comes into contact with serum, it can activate complement via the direct and alternative pathways and this may play some part in the role of inflammation.

The role of *Propionibacterium acnes* in seborrhoeic dermatitis is unexplained, though the counts are low in patients with seborrhoeic dermatitis.[2]

Individual susceptibility and host response:

Evidences suggesting altered host immune function or response in those with seborrhoeic dermatitis have been documented. The observation of increase in natural killer cells (NK1+) and CD16+ cells, increase in inflammatory interleukins, and

activation of complement in lesional skin as compared to nonlesional skin in patients with seborrhoeic dermatitis and in skin of healthy controls suggests an augmented inflammatory response in individuals with seborrhoeic dermatitis.[94]

A similar link has been suggested in studies of patients with seborrheic dermatitis associated with acquired immunodeficiency syndrome (AIDS).[95]

Clinical features:

Adult seborrhoeic dermatitis presents most often on the face and/or scalp as ill-defined erythematous patches associated with fine (pityriasiform) scaling, involving one or more sites of predilection. These commonly affected sites include scalp, anterior hairline , eyebrows, glabella region of the forehead, nasal alar creases, mesolabial folds, ears (including the external canals, anterior auricular region, retroauricular region), central chest (sternum area), and genital region.[96]

Morphological variants: There are several morphological variants of seborrhoeic dermatitis, which in adults occur in various combinations and degrees of severity.

Clinical patterns of seborrhoeic dermatitis[83]

Infantile

Scalp (cradle cap)

Trunk (including flexures and napkin area)

Leiner's disease

Non-familial

Familial C5 dysfunction

Adult

Scalp

Dandruff

Inflammatory—may extend onto non-hairy areas (e.g. postauricular)

Face (may include blepharitis and conjunctivitis)

Trunk

Petaloid

Pityriasiform

Flexural

Eczematous plaques

Follicular

Generalized (may be erythroderma)

Other types of seborrhoeic dermatitis on the scalp include arcuate, polycyclic, or petaloid patches, and psoriasiform, exudative, or crusted plaques.[78]

The scalp scaling associated with seborrhoeic dermatitis and dandruff is often bothersome as flakes, which are shed from the scalp and are often visibly apparent on darker clothing. In some cases, seborrhoeic dermatitis may produce thicker, more confluent areas of involvement, sometimes with oval, discoid plaques (medallion lesions).[76] In extreme cases the entire scalp is covered by a greasy, dirty crust with an offensive odor.[78]

Differential diagnosis[93]

A number of disorders are similar to seborrhoeic dermatitis. Scalp psoriasis may be difficult to distinguish from seborrhoeic dermatitis. Psoriasis of the scalp presents as sharply demarcated scaly plaques, usually extending beyond the hairline.

Other signs of psoriasis, such as nail pitting or distal onycholysis, also may facilitate distinction.[97]

Seborrhoeic dermatitis also may resemble atopic dermatitis, tinea capitis, and, rarely, cutaneous lymphoma or Langerhans cell histiocytosis.

Atopic dermatitis in adults characteristically appears in antecubital and popliteal fossae. The distinction is a clinical one because elevated immunoglobulin E (IgE) levels associated with atopic dermatitis are a nonspecific finding.

Tinea capitis can be diagnosed by demonstrating the presence of hyphae on 10% potassium hydroxide examination.

Concomitant disorders (e.g., psoriasis, scabetic eczema, superficial fungal infection) may complicate seborrhoeic dermatitis, especially in patients with AIDS. Skin biopsies may effectively distinguish seborrhoeic dermatitis from similar disorders. Seborrhoeic dermatitis have neutrophils in the scale crust at the margins of follicular ostia. Yeast cells sometimes are visible within keratinocytes on special stains.

Histopathology:

The histopathology is not diagnostic, but generally shows features of both psoriasis and chronic dermatitis. There is parakeratosis, mild to moderate acanthosis, with slight spongiosis. Spongiosis is the major feature which distinguishes it from psoriasis. The dermis shows a mild, chronic inflammatory infiltrate.

The histopathology of seborrhoeic dermatitis in patients with AIDS tends to show follicular involvement, and more plasma cells. *Malassezia* yeasts are prominent in the skin of AIDS patients with seborrhoeic dermatitis compared with seborrhoeic dermatitis patients without AIDS.[98]

Treatment [99-102]

Effective therapies for seborrhoeic dermatitis include anti-inflammatory (immunomodulatory) agents, keratolytic agents, antifungals, and alternative medications.

Therapies for Treating Seborrhoeic dermatitis

Anti-inflammatory (immunomodulatory) agents	**Therapy Usage**
Steroid shampoo	
Fluocinolone	Two times per week
Topical steroids	
Fluocinolone	Daily
Betamethasone valerate lotion	Daily
Keratolytics	
Salicylic acid shampoo	Two times per week
Tar shampoo	Three times per week
Zinc pyrithione shampoo (also has antifungal properties)	Two times per week
Antifungals	
Ketoconazole shampoo	Three times per week
Selenium sulfide shampoo	Two times per week
Alternative medication	
Tea tree oil shampoo	Daily

PITYRIASIS AMIANTIACEA:

Pityriasis amiantacea is an inflammatory scaling reaction of the scalp of unknown aetiology. It represents a particular reaction pattern of the scalp to various inflammatory scalp diseases.

Epidemiology:

Pityriasis amiantacea may occur at any age from 5 years upto 63 years [39,40] with females more affected than males.[103,104]

Aetiopathogenesis:

Pityriasis amiantacea is not an aetiologically coherent entity. It represents a reaction pattern of the scalp skin to various causes such as:[105]

Non-infective causes

- Psoriasis
- Seborrhoeic dermatitis
- Lichen simplex chronicus
- Atopic dermatitis
- Lichen planus

Infective causes

- Superficial fungal infections
- Pyogenic infections

Previous studies which have addressed the potential relationship between pityriasis amiantacea and psoriasis have concluded that psoriasis is the most common cause and pityriasis amiantacea might be the first clinical manifestation of it.[103,105] One Scandinavian follow-up study reported that psoriasis occurred in 15% of patients with pityriasis amiantacea.[106]

Clinical features:

Clinically thick, sticky, masses of adherent asbestosis-like or silvery scales, overlapping like the tiles on a roof, adhere to the scalp and are attached in layers to the shafts of the hairs, which they surround. The underlying scalp is usually inflamed and moist and adjacent areas may show features of associated dermatoses like psoriasis, seborrhoeic dermatitis, or lichen simplex chronicus. The disease may be confined to discrete areas of the scalp, or may be very extensive.

Pityriasis amiantacea may result in nonscarring or scarring alopecia.[107]

Histopathology:

The most consistent findings are spongiosis, parakeratosis, migration of lymphocytes into the epidermis and a variable degree of acanthosis. The essential features responsible for the asbestos like scaling are diffuse hyperkeratosis and parakeratosis together with follicular keratosis, which surrounds each hair with a sheath of horn.[104]

Treatment

Therapy consists of identification and treatment of the underlying dermatoses predisposing to this condition. Generous applications of an oil-based product are needed to remove the scales. Tar or salicylic acid ointments are commonly used. The preparation is left on the scalp for several hours and then washed out. The scales may be gently removed using a metal-toothed comb. Once the scaling is controlled, an antidandruff or tar-based shampoo may help to maintain remission, but most patients will need retreatment with an ointment from time to time. [108]

Oil of Cade ointment, which contains 6% oil of Cade, 2 % precipitated sulfur, and 2% salicylic acid in emulsifying ointment base, is an alternative.

The use of 2% Cetavlon (cetrimide and chlorhexidine hydrochloride) or Betadine hair washes helps control and prevent bacterial infection. Potent topical corticosteroid lotions are beneficial in some cases, but are not effective in removing thick scales.

PEMPHIGUS VULGARIS:

The word Pemphigus is derived from the Greek word *pemphix* meaning blister or bubble. It describes a group of chronic bullous diseases, originally named by Wichmann in 1791.[109]

Epidemiology:

Pemphigus vulgaris affects all races and both sexes equally.[110]It is a disease of middle age, which affects children rarely; but patients are younger at presentation in India than in Western countries.[111]

Pemphigus vulgaris has a worldwide prevalence of 0.1- 0.5 per 1,00,000 population, which can be as high as 3.2 per 1,00,000 in certain races.[112]

Pemphigus vulgaris accounts for around 70% of all cases of pemphigus and may be the commonest autoimmune blistering disease in Eastern countries, such as India, Malaysia, China and the Middle East.[111,112]

Aetiopathogenesis:

A genetic predisposition to develop pemphigus has been documented, which, in the presence of certain environmental triggers, results in acantholysis. First degree relatives of patients with pemphigus vulgaris are more susceptible to the development of autoimmune diseases than are controls[113,114] and have a higher incidence of circulating antidesmoglein antibodies.[115] Higher incidence and early age of onset for pemphigus seen in Indian population have been attributed to higher frequency of DSG3*TCCCC haplotype in Indian population. It has been observed that two related

haplotypes, DSG3*TCCTC and DSG3*TCCCC, are involved in the pathogenesis of PV in patients with British and North Indian descent, respectively.[116]

Although acantholysis as the basic pathomechanism in pemphigus is well accepted, the mechanism by which disruption of adhesion between keratinocytes occurs is not fully elucidated. The possible mechanisms are:

- Binding of autoantibodies to their antigens can disrupt adhesion of the bound antigens by stearic hindrance.
- Secondly, the antigen-antibody complex induces production of plasminogen activator, which in turn leads to the production of active plasmin culminating in cell dissociation.
- Thirdly, the autoantibodies may lead to reorganization of the keratinocyte cytoskeleton, leading to cellular shrinkage and separation of keratinocytes.[117]

In PV, autoantibodies are primarily directed against desmosomal cadherins, desmoglein (Dsg) 3 and Dsg 1. Approximately 85% of the pemphigus patients develop antibodies against keratinocyte acetylcholine receptors (AChR) - AChR α9 and Pemphaxin.[118] PV-IgG autoantibodies weakens intercellular adhesions between keratinocytes via inactivation of the cholinergic receptor-mediated physiologic control of cadherin (Dsg) expression and/or function and causes acantholysis.

Although the autoantibodies have been shown to be pathogenic, the role of the cellular immune system is unclear. One of the earliest pathogenic events in PV is the activation of protein kinase, including the protein kinase R (PKR) -like endoplasmic reticulum kinase (PERK). Decreased expression of PERK in vivo has been shown to reduce the effects of PV serum on the cell cycle and keratinocyte viability, two key events in PV pathophysiology.[119]

In pemphigus, cytokines IL-4 and IL-10, markers of Th2 driven immunity, are overexpressed, whereas IL-2 and IFN-γ, markers of Th1 driven immunity, are significantly suppressed. The underexpression of Th1 cytokines has been explained by suppressive effect of IL-4 and IL-10.[120]

Pemphigus occurs in patients with disorders characterized by immunological disturbances such as thymoma, myasthenia gravis, rheumatoid arthritis, pernicious anaemia, Hashimoto's thyroiditis, scleroderma and Sjogren's syndrome.[110] Pemphigus may also develop in patients with lupus erythematosus, bullous pemphigoid, lymphoproliferative diseases such as Castlemann's tumours.[121]

Clinical features:

The disease has a predilection for the scalp, face, axillae, groins and pressure points. On occasion, the pemphigus lesions may be confined to the scalp only, presenting as crusted lesions commonly misdiagnosed as pyoderma. Lesions may remain localized to the scalp for several months prior to the appearance of generalized involvement.

Histopathology:

The earliest histological feature seen in pemphigus vulgaris is intercellular oedema with loss of intercellular attachments in the basal layer. Suprabasal splitting of the epidermis leads to blister formation, with the basal layer still remaining adherent to the basement membrane giving the appearance of row of the tomb stones.[122,123] Within the blister cavity, the acantholytic keratinocytes are seen singularly or in clusters. Blistering is preceded by eosinophilic spongiosis in some cases. The superficial dermis has a mild, superficial, mixed inflammatory infiltrate which includes some eosinophils.[124]

IMMUNOFLUORESCENCE

The diagnosis of pemphigus is confirmed by direct immunofluorescence which shows IgG deposition on the surface of keratinocytes throughout the epidermis. IgG1 and IgG4 are the most common subclasses; C3, IgM and IgA are present less frequently. Circulating pemphigus autoantibodies are detected by indirect immunofluorescence in over 80% of patients. The use of more than one substrate improves sensitivity; oesophageal substrate being preferable for the detection of antibodies to desmoglein-3.[71]

Treatment:

Pemphigus vulgaris, initially even though limited in extent, should be generally treated at its onset, because it will ultimately generalize and the prognosis without therapy is poor. In addition, it is easier to control the disease at an early stage than at a later widespread stage, where the mortality is high.

The systemic administration of glucocorticoids, usually prednisolone, is the mainstay of therapy for pemphigus. Prednisolone (1.0 to 1.5 mg/kg) with an adjuvant is the preferred treatment to control the disease in many patients.[125]

Potent topical or intralesional steroids may reduce the requirement for oral steroids. Potassium permanganate and topical antiseptics may help reduce the risk of cutaneous infection.

Adjuvant therapy is meant to shorten the duration of the course of the disease and its use must be tailored to the individual patient. [126] Azathioprine is widely used in a dose of 2.5 mg/kg/day.[125]Azathioprine is the most effective steroid-sparing agent, followed by cyclophosphamide (pulse therapy) and mycophenolate mofetil

(2g/day).[127]Azathioprine may be an effective monotherapy in mild cases, although the therapeutic effect is delayed for 3 to 5 weeks.

Cyclophosphamide, an alkylating agent, in a single dose of 1-3 mg/kg/ day has been reported to be effective both as first line adjuvant in patients with pemphigus and in the treatment of those whose disease has previously failed to respond to azathioprine.[125]

Rituximab, an anti-CD-20 monoclonal antibody has produced remission in some patients with treatment-resistant disease by depletion of B-lymphocytes .[127,128]

Removal of circulating antibodies by plasmapheresis or by immune adsorption[129] has induced remission, but concomitant immunosuppression with steroids or cyclophosphamide is needed to prevent a rebound increase in the synthesis of antibody.

LICHEN PLANOPILARIS (LPP):

SYNONYMS: Frontal fibrosing alopecia

Follicular lichen planus.

Lichen planus is an idiopathic inflammatory disease that may affect the skin, hair and nails. Lichen planopilaris is a chronic scarring alopecia characterized by follicular hyperkeratosis, perifollicular erythema, and loss of follicular orifices. Three variants of cicatricial alopecia resulting from lichen planus are recognized: Lichen planopilaris, Graham-Little syndrome and frontal fibrosing alopecia. In each of these conditions the scalp may be affected alone or in conjunction with lichen planus elsewhere.

Epidemiology:

Lichen planus occurs throughout the world, although there are no data on its frequency in different ethnic groups. Lichen planus occurs at any age, but in over 80%

of cases the onset is between 30 and 70 years [130] Significant involvement of the scalp is relatively infrequent—only 10 of 807 patients in one series[130]—but the incidence is probably higher than such figures suggest, because of the exclusion of those patients in whom alopecia, classified as pseudopelade, was the only manifestation of the disease. Scalp involvement occurs in over 40% of patients with either of two unusual variants of lichen planus: the bullous or erosive form and LPP.

Clinical features:

Scalp lesions may show violaceous papules, erythema and scaling. These papules are replaced quickly by follicular plugs and scarring which are eventually shed from the scarred area, thus remaining white, smooth and atrophic. Follicular orifices are absent within the area of alopecia. The triad of follicular lichen planus of the skin (lichen planus spinulosus) and/or scalp, multifocal cicatricial alopecia of the scalp, non-scarring alopecia of the axillary and pubic areas is termed as Graham-Little-Picarrdi-Lassueur syndrome (Graham Little Feldman syndrome).

Another clinical variant of Lichen planopilaris is frontal fibrosing alopecia.[131] This condition superficially resembles androgenetic alopecia with frontal recession, but on close inspection there is loss of follicular orifices, perifollicular erythema and hyperkeratosis at the marginal hairline. It typically occurs in postmenopausal women, although it can occur earlier. In contrast with AGA, the frontal hairline recedes in a straight line rather than bitemporally.

Treatment:

Potent topical steroids such as clobetasol propionate ointment twice daily can be used to inhibit the process and relieve the associated symptoms such as itching and pain.[132] Hydroxychloroquine and acitretin have been tried with variable success. Ciclosporin is very effective for cutaneous lichen planus, and has been reported as

useful in Graham-Little Syndrome[133], as is thalidomide.[134] In some cases, a short course of systemic treatment with corticosteroids is desirable. The natural history of frontal fibrosing alopecia is slow progression over many years. There is no effective treatment.

DISCOID LUPUS ERYTHEMATOSUS (DLE):

Discoid lupus erythematosus may occur on its own or associated with SLE. If the initial DLE is confined to the head and neck, the risk of SLE is 1–2%, whereas if the lesions are generalized the risk is 22%.[135]

Only DLE regularly produces cicatricial alopecia. Inflammation of the infundibular region of the hair follicle that contains the stem cells is thought to be the basis of the scarring alopecia that occurs in DLE.

Epidemiology:

DLE occurs most commonly in women and is about three times more common in African Americans than in white people. The incidence is approximately 1 in 2000. Familial cases occur in approximately 10%. The peak age of onset is around 40 years.[135]

Scarring alopecia occurs in 20% of men and 50% of women affected with DLE, and the scalp is the only area affected in a significant number of patients.

Clinical features:

The common sites of distribution includes sun-exposed areas such as scalp, face, ear, but occasionally is much more extensive, involving large areas of skin. Patches on the scalp are often itchy. Areas of erythema and scaling with follicular plugging extend irregularly across the scalp and produce scarring. The depressed scars, hair loss, and pigmentary changes are often extremely disfiguring, particularly in darker-skinned people.

Squamous cell carcinoma has been reported in chronic cicatricial LE of the scalp.[136]

Histopathology:

The histopathology of DLE shows hyperkeratosis with follicular plugging, a perivascular and periadnexal lymphoid infiltrate, which may be sparse, moderate or heavy, and the essential feature of focal basal layer vacuolar degeneration. This may be associated with colloid body formation, pigmentary incontinence, papillary dermal oedema, thickening of the basement-membrane zone and exocytosis of lymphocytes into the epidermis and follicular epithelium.[135.] Scarring occurs in DLE and manifests as homogenized collagen fibres running parallel to the surface, a loss of appendages and lone arrector pili muscles.

Antinuclear antibody (ANA) is positive in approximately 35% of patients with DLE. Anti-Ro antibodies are found in 10%.

Treatment:

Potent topical corticosteroid appears to be effective in the treatment of DLE. Hydroxychloroquine (200-400 mg/day) produces a remission within 3 months in majority of the cases. Chloroquine, acitretin, dapsone, thalidomide or a combination of these medications may be useful in refractory cases. Early effective treatment may lead to total clearing of the skin lesions, but failure of treatment results in permanent scarring.

PITYRIASIS RUBRA PILARIS:

Pityriasis rubra pilaris (PRP) is a keratinisation disorder, characterised by follicular keratosis and palmoplantar keratoderma. It affects males and females equally.

Clinical features:

The classical adult onset type starts most often on the head, neck, or upper trunk. Typical lesions are erythematous perifollicular papules, with a central keratotic plug. Lesions occur singly, but may coalesce into groups. The scalp shows diffuse bran-like scaling. A biopsy of the skin is essential, to differentiate PRP from similar conditions, like eczema and psoriasis.

Treatment:

The use of emollients to restore the disrupted skin barrier is important in the treatment of PRP. Topical and systemic steroids are ineffective. Retinoids, like acitretin, are the most effective treatment for PRP. About 80% of cases resolve spontaneously within three years.[137]

3. INFECTIVE DISORDERS

FOLLICULITIS:

Folliculitis is the inflammation of one or more hair follicles which is characterized by erythema and pustules located around a hair follicle on the scalp. Superficial and deep forms are the chief subdivisions.

Epidemiology:

Superficial folliculitis is more common and can occur at any age.[138] It tends to affect adults more frequently than children. The exact incidence is unknown.

Aetiopathogenesis:

It is ordinarily initiated by coagulase- positive staphylococci. Under unusual circumstances, however, when the host resistance is impaired, other organisms, such as coliform gram-negative organisms and possibly even the normal skin micrococci, may be causative.[139]

Persistent bacterial folliculitis can be caused by diabetes, occlusive headgears, and shaving.[140]

Superficial folliculitis has also been termed *follicular* or *Bockhart impetigo.* It is a subacute or chronic folliculitis, in which the inflammatory changes are confined to the ostium or extend only slightly below it, and which heals without scar formation.[140]

Clinical features:

Folliculitis can be pruritic or painful. The lesions develop into 1-5 mm yellow-grey papules or pustules, with surrounding erythema, confined to the follicular ostia. They can be grouped or discrete and usually occur on the scalp, face, buttocks, and extremities. There are usually no systemic symptoms.

Treatment:

Gram stain, culture and sensitivity of lesional exudate confirm the diagnosis and guide treatment. If the infection of the follicle is deeper and involves more follicles it usually needs incision and drainage.[138] Soap and water cleansing and local application of topical antibiotics are effective measures. Antiseptics, including chlorhexidine, triclosan, and povidone-iodine, can be used as creams or lotions, soap substitutes, and bath additives. Systemic antibiotics are indicated in case of severe or refractory folliculitis.[140] Resistant lesions respond to topical mupirocin or fusidic acid.

FOLLICULITIS DECALVANS:

Folliculitis decalvans is a heterogeneous group of syndromes, in which clinically evident chronic folliculitis leads to progressive scarring on the scalp.

Epidemiology:

The exact cause is unknown. It occurs in both sexes, and typically affects women in age group of 30-60 years, and men from adolescence onwards.

Clinical features:

Clinical manifestation is a central patch of scarring alopecia, surrounded by crops of follicular pustules, with destruction of such follicles. *Staphylococcus aureas* may be cultured from the pustules.[141]

Treatment:

Treatment of folliculitis decalvans is difficult. Topical and systemic antibiotics are the mainstay of treatment. They can inhibit extension of the disease, but only for as long as they are administered. [142]

EOSINIOPHILIC PUSTULAR FOLLICULITIS OF THE SCALP:

Eosinophilic pustular folliculitis is a recurrent pruriginous condition characterized by pustular lesions on the scalp and other body areas.

Epidemiology:

It is typically seen in adults, and the Eosinophilic pustular folliculitis associated with immunosuppression, is seen mainly in HIV-seropositive patients.

Clinical features:

HIV-related eosinophilic folliculitis (HIV-EF) is characterized by perifollicular erythematous papules and pustules that cause intense itching. Recently, HIV-EF has been associated with IRIS, especially associated with a rapid increase in CD4 cell blood counts related to HAART.[143,144]

FOLLICULITIS KELOIDALIS NUCHAE:

Folliculitis keloidalis nuchae is a chronic inflammatory process, that affects the hair follicles of the nape of the neck and may extend to the occipital scalp.[145] The cause is unknown. In-growing hair following close shaving, and bacterial folliculitis, has been postulated as possible causes.

Epidemiology:

The disease is 10 times more common in males than females, and predominantly affects males of African ancestry.[146]

Clinical features:

The disease starts as papules and pustules, which may coalesce into plaques, and heal with small, or large, keloids, on the nuchal area and over the scalp.

Treatment:

Early disease, with papules and pustules, may be treated with topical antibiotics,

oral antibiotics, intralesional corticosteroid injections, or destruction of firm papules with cryotherapy. Once lesions have progressed to hypertrophic scars and keloids, surgical excision may be considered.

ACNE NECROTICA VARIOLIFORMIS:

Synonyms : Folliculitis Varioliformis

The term acne necrotica includes a chronic follicular necrotizing process, which evolves into small, round scars, affecting mainly areas close to scalp margins. Acne necrotica miliaris is a rare and chronic form of the disease occurring throughout the scalp without significant scarring. The relationship between the two is still uncertain, but the latter is regarded synonymous with *Propionibacterium acnes* folliculitis of the scalp.[147]

Epidemiology:

It occurs slightly more frequently in men than in women, and is usually seen between the ages of 30 and 50 years, but never before puberty.

Aetiopathogenesis:

The lesion is essentially a folliculitis, but the cause is uncertain. The most widely suggested pathogens have been *Staphylococcus aureus* and *Propionibacterium acnes*, but neither is universally accepted; it is the follicular and perifollicular necrosis, presumably mediated by host response mechanisms, which characterizes the condition.

Emotional disturbance has been a frequent finding in some studies;[148,149] it has been suggested that repeated excoriation of lesions of a primary folliculitis leads to more destructive changes.[148]

Clinical features:

The lesions are persistent brown papulopustules, usually localized to the anterior margins of the scalp and the brow. The nose, cheeks and trunk can also be involved. The top of the papule usually erodes and a crust forms beneath which there is a necrotic zone.[139] When the lesions are numerous, they may ultimately produce disfiguring scarring.

Histopathology:

Early lesions show extensive individual cell necrosis of keratinocytes in the external root sheath and the surrounding epidermis, and marked subepidermal oedema along with lymphocytic folliculitis. Later, the follicles and adjacent epidermis and dermis show confluent necrosis.

Treatment:

Topically clindamycin is found to be effective. Anti-septic shampoos are also advised. Prolonged course of antibiotics such as tetracycline can be given in refractory cases.[150,151]

PROPIONIBACTERIUM ACNES SCALP INFECTION:

It is characterized by pinhead to 3mm, usually dome to flat, white to yellow follicular pustules. Scalp erythema is often noted. The occiput is more commonly involved than the remainder of the scalp. It usually affects males more than the females.

On Grams staining, numerous gram positive pleomorphic rods are noted. Tetracycline in the dose of 1g/day given orally may abort the development of a pustule. Isotretinoin 1-2 mg/kg for 20 weeks has also been proved effective.

EROSIVE PUSTULAR DERMATOSIS OF THE SCALP:

This disease particularly affects the elderly.[152,153] Though the cause is unknown, there are several precipitating factors such as sun damage, local trauma, surgery, cryosurgery, skin grafting and radiation therapy.[154]

Clinical features:

Initially, a small area of scalp becomes red, crusted and irritable; crusting and superficial pustulations overlie a moist, eroded surface. As the condition extends, areas of activity coexist with areas of scarring. Squamous cell carcinoma can develop in the scars.[155]

Differential diagnosis:

Pyogenic and yeast infection are excluded by bacteriological examination and the lack of response to antibacterial or antifungal agents.

Treatment:

Potent topical corticosteroids will suppress the inflammatory changes.

TINEA CAPITIS:

It is a ring worm infection of the scalp in which the essential feature is invasion of the hair shafts by a dermatophtye fungus. Tinea capitis is predominantly an infection of children, although adult cases are seen, particularly with *T. tonsurans* infections. Tinea capitis may also be seen in adults with AIDS.

Trichophyton and Microsporum species account for the majority of disease worldwide; Trichophyton tonsurans is the predominant pathogen in North America, and Microsporum canis is a significant cause of disease in other parts of the world.[156,157]

Tinea capitis caused by T tonsurans can occur at any age from the neonate to the elderly.[158,159] The incidence in adults is increasing and ranges from 2.7% to 11.4% of reported cases.[160,161]

Clinical features:

The manifestation varies from a few dull grey, broken-off hairs with a little scaling, detectable only on careful inspection, to a severe, painful, inflammatory mass covering most of the scalp. Itching is variable. In all types, the cardinal features are partial hair loss with inflammation of some degree. Sporulation inside the hair shaft causes breakage of the hairs near the scalp surface, leading to ‘‘black dot’’ alopecia. In severe cases, swollen boggy scalp lesion, called kerion and regional lymphadenopathy can occur.

Although tinea capitis is a clinical diagnosis, laboratory investigations is performed to confirm the diagnosis. A simple side-room test involves scraping out scales from the lesion onto a glass slide, adding a few drops (10%) of potassium hydroxide (KOH), warming with a Bunsen burner, and looking for hyphae and spores, under a light microscope.

Differential diagnosis:

The differential diagnosis of tinea capitis includes all conditions capable of causing patchy baldness with inflammatory changes of the scalp. Traumatic alopecia from hairdressing procedures and trichotillomania may be confused with T.capitis. Seborrhoeic dermatitis is usually more diffuse than tinea capitis, but in tinea amiantacea the changes are often localized. In this condition, the scaling is adherent to the hair, but breakage of the hair shaft does not normally occur. In psoriasis, hair loss is found only occasionally, and again broken-off hairs are not usually present. In impetigo, which may be secondary to pediculosis of the scalp, loosening of the hair is not normally present. A carbuncle of the scalp is much more acutely painful, and shedding of loosened hairs much less evident than in kerion. Discoid lupus erythematosus, lichen planus and other causes of cicatricial alopecia may sometimes have to be considered.

Treatment:

The mainstay of treatment for tinea capitis is griseofulvin 20 mg/kg orally, daily, for three months.[162] Topical therapy is not indicated, but frequent shampooing may help to remove matted crusts.

4. GRANULOMATOUS

NECROBIOSIS LIPOIDICA:

Necrobiosis lipoidica, a rare degenerative connective tissue disorder occurs in 0.2–0.3% of cases of diabetes mellitus. The oval atrophic plaques classically occur on the shins but may be seen on other parts of the body, including the scalp. The patches are glazed and yellowish, often with conspicuous telangiectases.

Scarring may be dense. The clinical features in the scalp vary from large plaques of cicatricial alopecia to multiple small areas of scarring.[163] An atrophic form affecting predominantly the forehead and the scalp has been described.[164]

5. NEOPLASTIC CONDITIONS

CYLINDROMA:

Synoyms - TURBAN TUMOR

SPIEGLER'S TUMOR

Cylindroma is an uncommon skin appendage tumor that may occur either as solitary or multiple lesions over the scalp. This syndrome is an inherited disease and affects the folliculosebaceous apocrine units. [165,166]

Epidemiology:

This is an uncommon tumour, affecting females more frequently than males. It has been reported to follow radiotherapy epilation of the scalp. The onset is usually in adult life, but maybe in childhood or adolescence.

Clinical features:

The tumours are frequently multiple, smooth, firm, pink to red in colour and often somewhat pedunculated. The commonest site is the scalp and adjacent skin. A great many papules and nodules may obscure the whole scalp, like a turban.[167] Tumours on the scalp may be almost hairless when pedunculated, but the smaller lesions form dermal nodules with little loss of overlying hair.

Histopathology:

The tumours have a rounded outline and are composed of closely set mosaic-like masses ('jigsaw-puzzle'appearance) and columns of cells that are invested by a hyaline basal membrane of variable thickness. Thin bands of stroma separate tumour lobules from one another.

Differential diagnosis:

Trichelemmal cysts are most likely confused with cylindromas. They are usually smoother, firmer, and more mobile than cylidromas.

Treatment:

Surgery is the treatment of choice. Extensive involvement of the scalp may require wide excision and replacement of the whole area by a graft.

SYRINGOCYSTADENOMA PAPILLIFORM (SCAP):

Synonym : Naevus syringocystadenomatosus papilliferus

Syringoadenomatous papilliferum is a rare benign hamartomatous cutaneous adnexal neoplasm with variable clinical appearance and characteristic histology.

They show features of apocrine glands, or occasionally eccrine glands. Syringocystadenoma papilliferum is a frequent component of sebaceous naevi and associated hamartomatous malformations of hair follicles and sebaceous glands are common.

Clinical features:

SCAP has been commonly described in head (on scalp, forehead, cheeks, pinna and eyelids) and neck region. The plaque and linear varieties are usually present at birth, or appear during infancy,[168] and may frequently increase in size and become papillomatous, verrucous or crusted at puberty. The plaque form usually presents as a hairless area in the scalp.

Ulceration or rapid enlargement may indicate malignant transformation, usually to basal cell carcinoma,[169] but occasionally adenocarcinoma.

Histopathology:

The epidermis shows papillomatosis. There are cystic invaginations extending downwards from the papillomatous epidermis, with numerous villous projections extending into the lumen. The lining is glandular epithelium, comprising an outer cuboidal layer and an inner cylindrical layer occasionally demonstrating 'decapitation' secretion.

Differential diagnosis:

The plaque and linear forms on the head and neck may be difficult to distinguish clinically from sebaceous naevus, although syringocystadenoma papilliferum tends to be pinker and more nodular.

Treatment:

Excision is the treatment of choice.

MATERIALS AND METHODS

The study was undertaken from January 2012 to June 2013. All adult patients reporting to the Department Of Dermatology, Sri R.L.Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar were evaluated for entry into the study and patients having scalp lesions were enrolled.

A detailed history of all such patients was taken including general status of the patient, systemic diseases, medications used, precipitating factors such as sunlight, alcohol, smoking, drugs and trauma. Complete clinical and a thorough scalp examination was performed. During the clinical examination, the following elements were analysed such as morphology of the lesion, anatomical location and colour changes.

The clinical diagnosis was established. In relevant cases, necessary investigations were done to establish the definitive diagnosis. The data collected was documented in a prescribed proforma.

CRITERIA FOR SELECTION:

Inclusion criteria:

- All patients aged 18 years and above with scalp lesions.

Exclusion criteria:

- Patients with hair disorders.
- Patients on immunosuppressive medications.

OBSERVATIONS AND RESULTS

A total of 171 cases having scalp lesions fulfilling inclusion criteria attending to dermatology OPD at R. L. Jalappa Hospital and Research centre, Tamaka, Kolar district, Karnataka during the period of January 2012 – June 2013 were enrolled in this clinical study.

Table 1: Prevalence of scalp dermatoses

Total number of patients screened	Patients with scalp dermatoses	Prevalence (%)
6000	171	2.85 %

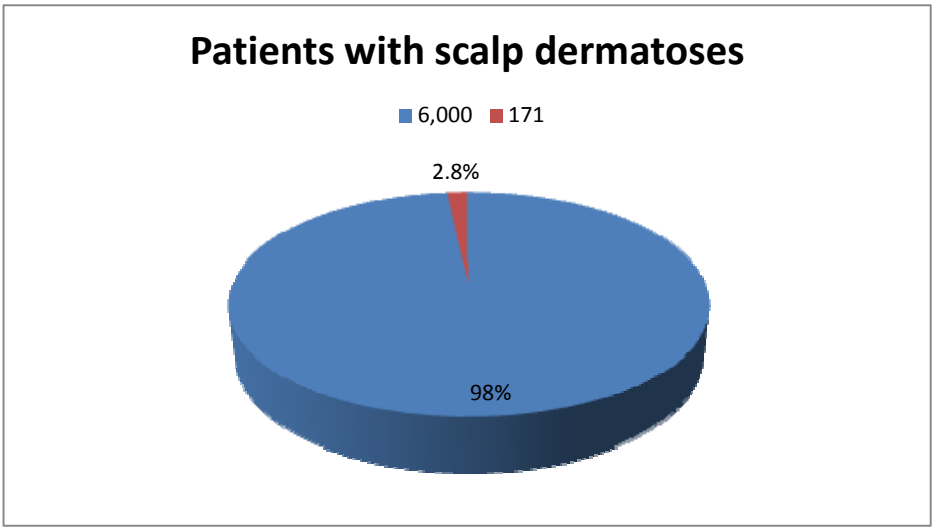


Figure 1. Shows the overall prevalence of scalp dermatoses in adults in the study population.

The overall prevalence of scalp dermatoses in our study population was 2.85%.

AGE DISTRIBUTION:

Table 2: Age group distribution of common scalp dermatoses

Age Group (in yrs)	Psoriasis	Seborrheoic dermatitis	Pityriasis sicca	Vitiligo	Pemphigus vulgaris	Folliculitis	Miscella-neous	Total (%)
18-30	13	10	12	4	3	4	5	**51 (29.8)**
31-40	10	8	6	2	2	1	2	**31 (18.1)**
41-50	23	9	2	7	3	2	7	**53 (30.9)**
51-60	5	5	-	3	1	1	2	**17 (9.9)**
>61	6	-	-	1	-	2	10	**19 (11.1)**
Total no (%)	**57 (33.3)**	**32 (18.7)**	**20 (11.6)**	**17 (9.9)**	**9 (5.2)**	**10 (5.8)**	**26 (15.2)**	**171 (100)**

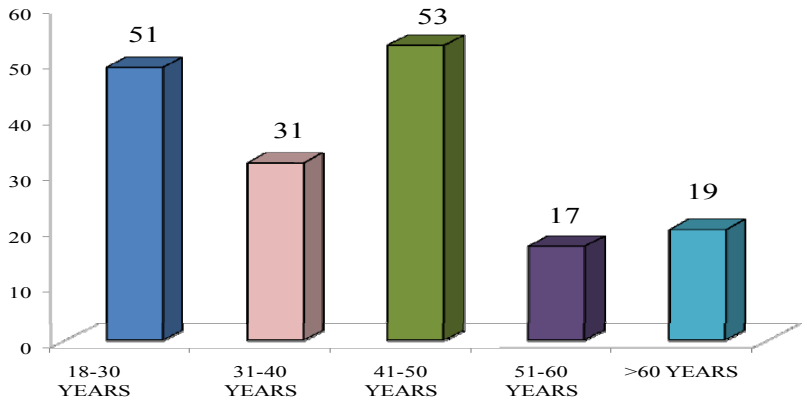


Figure 2. Age group distribution of common scalp dermatoses.

Out of 171 patients, 53 (30.9%) patients belonged to the age group of 41-50 years, followed by 51 (29.8%) in the age group of 18-30 years, 31 (18.1%) patients in the age group of 31-40 years, 17(9.9%) patients in the age group of 51-60 years and 19 (11.1%) patients in the age group of above 61 years.

SEX DISTRIBUTION:

Table 3: Sex wise distribution of patients with scalp dermatoses

Sex	Total (no.)	Percentage (%)
Males	104	60.8%
Females	67	39.2%

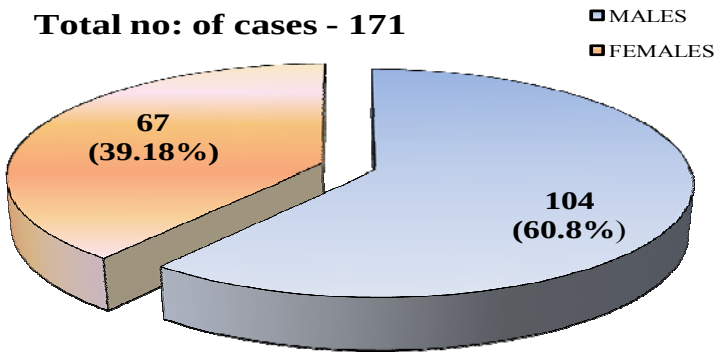


Figure 3. Sex distribution of patients with scalp dermatoses.

Males (60.8%) were affected more than females (39.2%) in our study population.

Table 4: Distribution of various scalp dermatoses according to age and sex

Scalp dermatoses	18-30yrs		31-40yrs		41-50yrs		51-60yrs		>61 yrs		Total (n=171)
	M	F	M	F	M	F	M	F	M	F	
Psoriasis	5	7	8	2	18	5	5	1	6	-	57 (33.3%)
Seborrhoeic dermatitis	8	2	5	3	5	4	3	2	-	-	32 (18.7%)
Pityriasis sicca	3	9	2	4	1	1	-	-	-	-	20 (11.6%)
Vitiligo	-	4	2	-	4	3	1	2	-	1	17 (9.9%)
Folliculitis	3	1	1	-	-	2	-	1	1	1	10 (5.8%)
Pemphigus vulgaris	1	2	2	-	2	1	1	-	-	-	9 (5.2%)
Discoid lupus erythematosus	-	-	1	-	1	1	-	-	1	-	4 (2.3%)
Chronic actinic dermatitis	-	-	-	-	1	-	1	-	1	-	3 (1.7%)
Seborrheoic keratoses	-	-	-	-	-	-	-	-	1	1	2 (1.1%)
Dermoid cyst	-	-	-	-	-	-	-	1	-	1	2 (1.1%)
Pyogenic granuloma	2	-	-	-	-	-	-	-	-	-	2 (1.1%)
Drug reactions	-	-	-	-	1	-	-	-	1	-	2 (1.1%)
Trauma	-	-	-	-	-	1	-	-	-	1	2 (1.1%)
Scarring alopecia	1	-	1	-	-	-	-	-	-	-	2 (1.1%)
Pityriasis amiantiacea	-	-	-	-	-	1	-	-	-	-	1 (0.5%)
Keratinous cyst	-	-	-	-	-	-	-	-	-	1	1 (0.5%)
Bullous pemphigoid	-	-	-	-	-	-	-	-	-	1	1 (0.5%)
Acquired icthyoses	-	-	-	-	-	-	-	-	1	-	1 (0.5%)
X- linked icthyoses	1	-	-	-	-	-	-	-	-	-	1 (0.5%)
Port wine stain	-	-	-	-	-	-	-	-	1	-	1 (0.5%)
Cherry angioma	-	-	-	-	-	1	-	-	-	-	1 (0.5%)

More females presented with scalp dermatoses in the age group of 18-30 years as compared to males, whereas in all other age groups, males were more in number.

PRESENTING SYMPTOMS:

Table 5: Presenting symptoms of scalp dermatoses

Presenting symptoms	Patients (n =171)	Percentage (%)
Itching	123	71.9%
Burning sensation	21	12.2%
Pain	3	1.7%
Discoloration	17	9.9%
No symptoms	7	4.1%

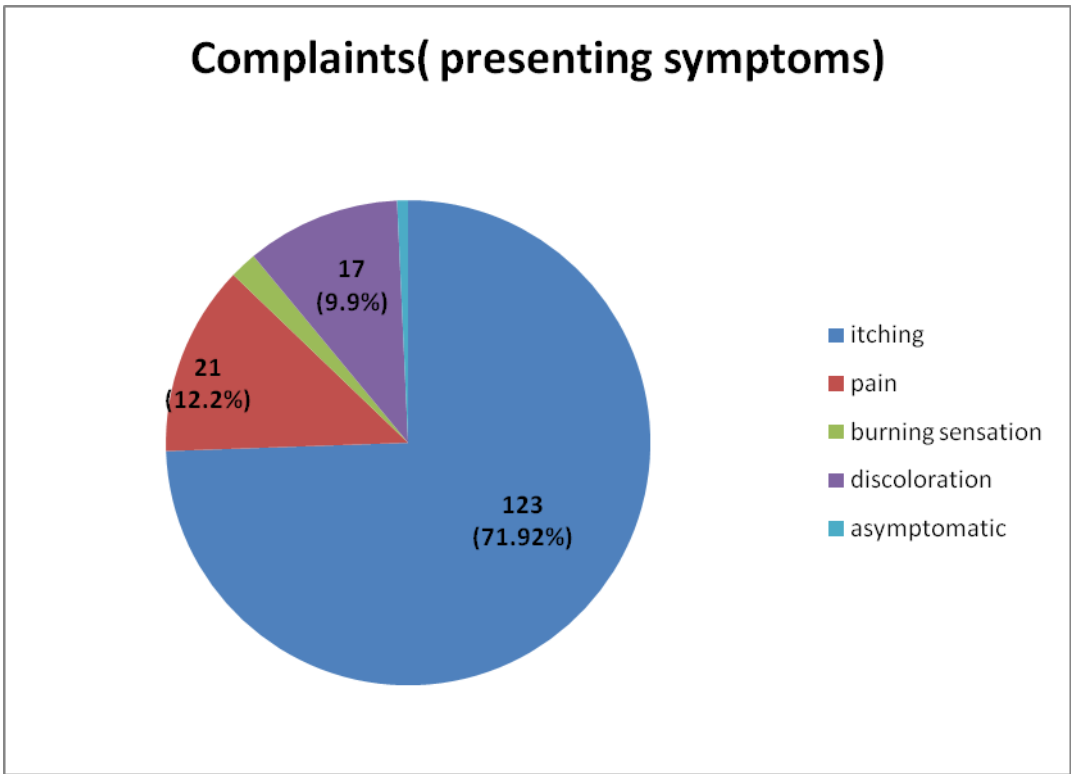


Figure 4. Shows the presenting symptoms of patients with scalp dermatoses.

Of the 171 patients, 123 patients (71.9%) complained of itching of scalp. Burning sensation and discolouration were the other presenting complaints in 21(12.2%) and 17(9.9%) patients respectively.

DURATION OF DISEASES:

Table 6: Distribution of scalp dermatoses according to duration

Duration	Patients (n=171)	Percentage (%)
Since birth	3	1.7 %
<6 weeks	6	3.5%
>6 weeks	162	94.7%

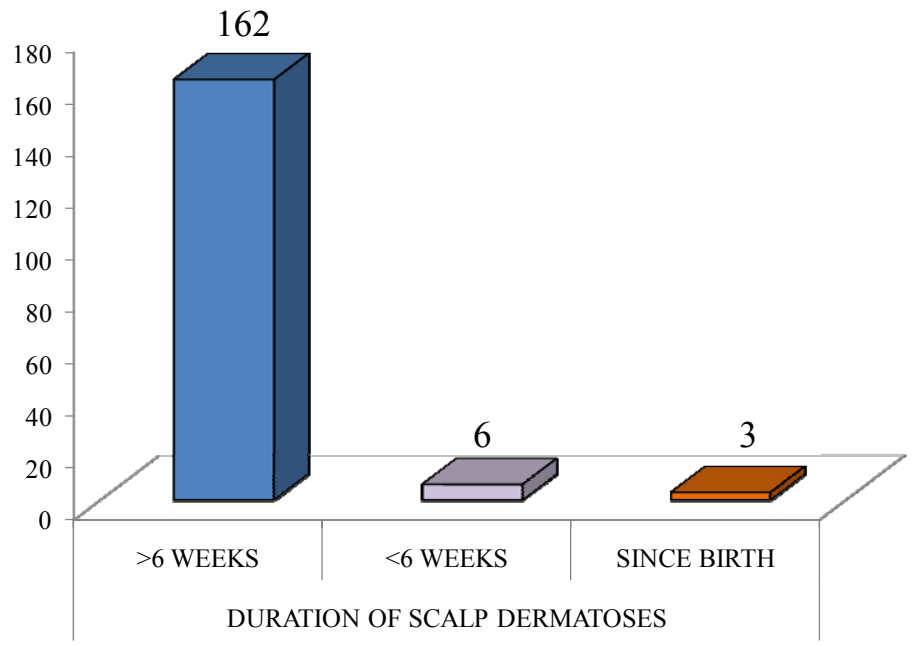


Figure 5. Duration of the diseases.

Majority of the patients (94.7%) had symptoms of more than 6 weeks duration.

ASSOCIATED RISK FACTORS:

Table 7: Associated risk factors with scalp dermatoses

Associated risk factors	Males	Females	Total* (n=61) (%)
Sunlight	3	-	3 (4.9%)
Alcohol	10	-	10 (16.3%)
Smoking	10	-	10 (16.3%)
Stress	3	6	9 (14.7 %)
Season (Winter)	17	5	21 (34.4%)
Trauma	_	4	4 (6.5%)
Drugs	3	-	3 (4.9%)

* Associated risk factors could be identified in only 61 patients.

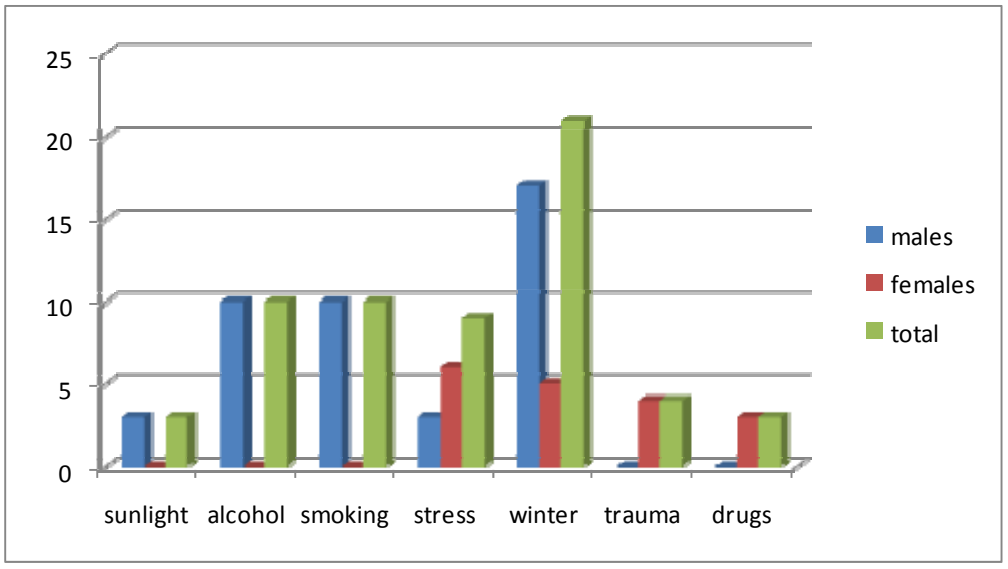


Figure 6. Associated risk factors with scalp dermatoses.

Associated risk factors could be elicited in 61(31.6%) patients with scalp dermatoses. Exacerbation of scalp dermatoses in winter season was complained by 34.4% of patients belonging to both sexes. In males, alcohol consumption and smoking worsened the condition in 16.3% of cases respectively. Emotional stress was the common associated risk factor in females (14.7%).

Table 8: Associated risk factors and various scalp dermatoses

Risk factors	Psoriasis	Seborrhoeic dermatitis	Miscellaneous	Total (n=61) (%)
Sunlight	-	-	3	3 (4.9%)
Alcohol	8	2	-	10 (16.3%)
Smoking	6	2	2	10 (16.3%)
Stress	6	3	-	9 (14.7%)
Seasonal (winter)	20	1	-	21 (34.4%)
Trauma	-	-	4	4 (6.5%)
Drugs	1	-	2	3 (4.9%)

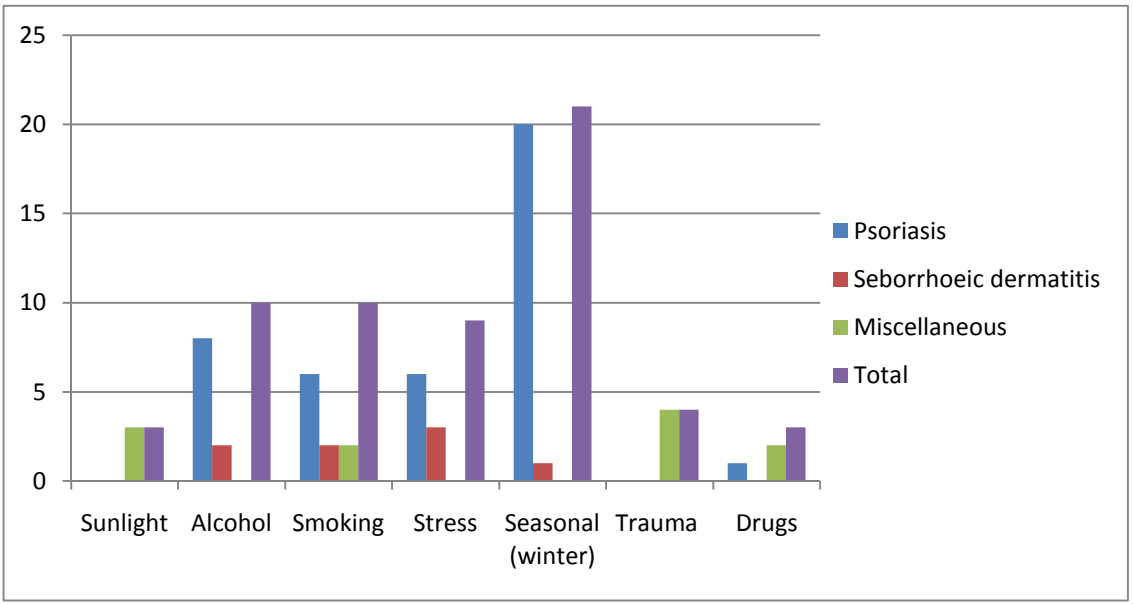


Figure 7. Associated risk factors and various scalp dermatoses.

Table 9: Associated diseases with scalp dermatoses

Associated diseases	Psoriasis	Seborrhoeic dermatitis	Miscellaneous	Total %(n=19)
Diabetes	3 (15.7%)	1 (5.2%)	3 (15.7%)	7 (36.8%)
Hypertension	7 (36.8%)	1 (5.2%)	2 (10.5%)	10 (52.6%)
Ischaemic heart disease	1 (5.2%)	-	1 (5.2%)	2 (10.5%)
Psychiatric illness	-	-	-	-
Immunosuppression	-	-	-	-

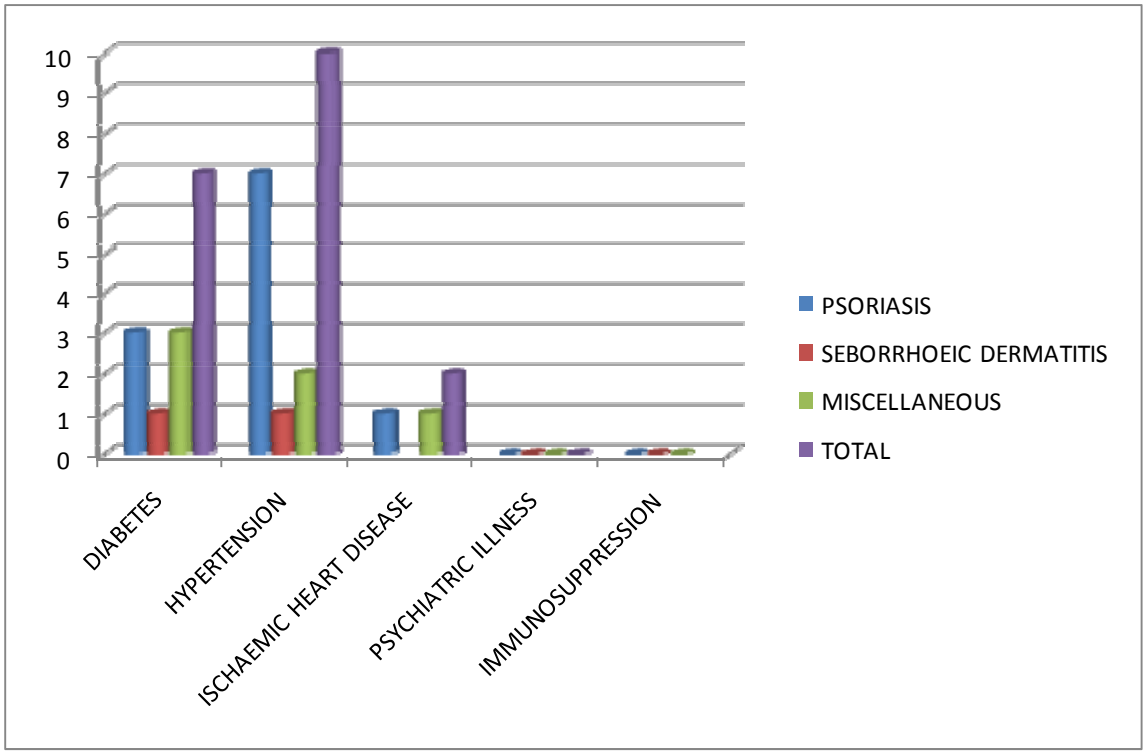


Figure 8. Associated diseases with scalp dermatoses.

In patients with scalp dermatoses, associated co-morbidities were seen in 19 (11.1%) cases.

DISTRIBUTION OF SCALP DERMATOSES/ SITES:

Table 10: Distribution of scalp dermatoses/ sites

Sites on scalp	Total* (n=171)	Percentage (%)
Frontal	68	39.7
Temporal	75	43.8
Parietal	98	57.3
Occipital	15	8.7
Vertex	12	7.0
All	28	16.3

*More than one scalp region involved in many patients

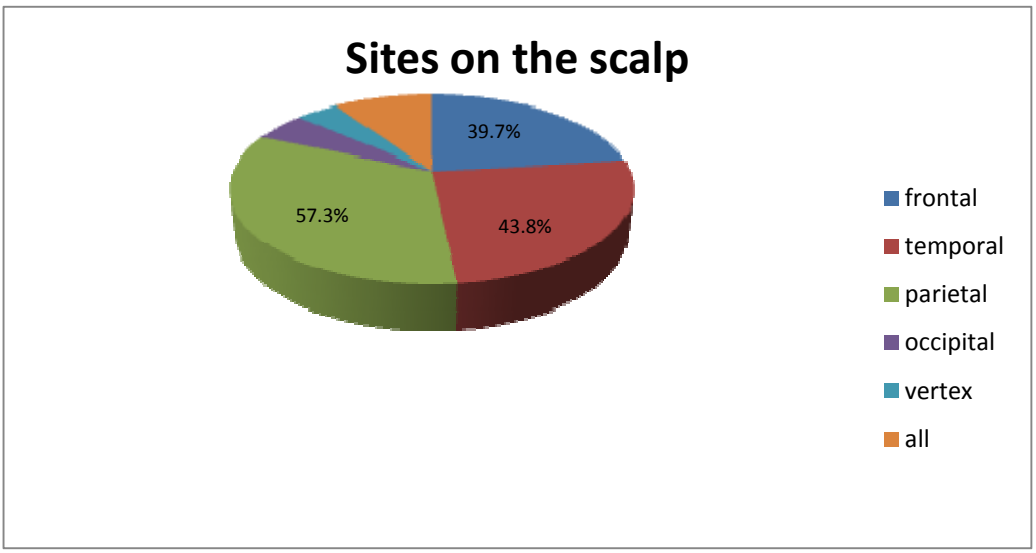


Figure 9. Distribution of scalp dermatoses/ sites.

Parietal area (57.3%) of the scalp was the most common site involved, followed by temporal (43.8%) and frontal regions (39.7%). Multiple sites were involved in 69.5% of cases.

MORPHOLOGY OF LESIONS:

Table 11: Morphology of scalp lesions

Primary lesion	**Patients (n=171)**	**Percentage (%)**
Macule	17	9.9
Papule	4	2.3
Plaque	132	77.1
Pustule	13	7.6
Nodule	4	2.3
Cyst	1	0.5
Secondary lesion		
Scaling	141	82.4
Crusting	12	7.0
Ulcer	5	2.9
Erosion	9	5.2
Atrophy	4	2.3
Margins		
Well-defined	111	64.8
Ill-defined	60	35.0
Colour		
Erythematous	85	49.7
Hyperpigmented	60	35.0
Skin coloured	6	3.5
De pigmentation	20	11.6

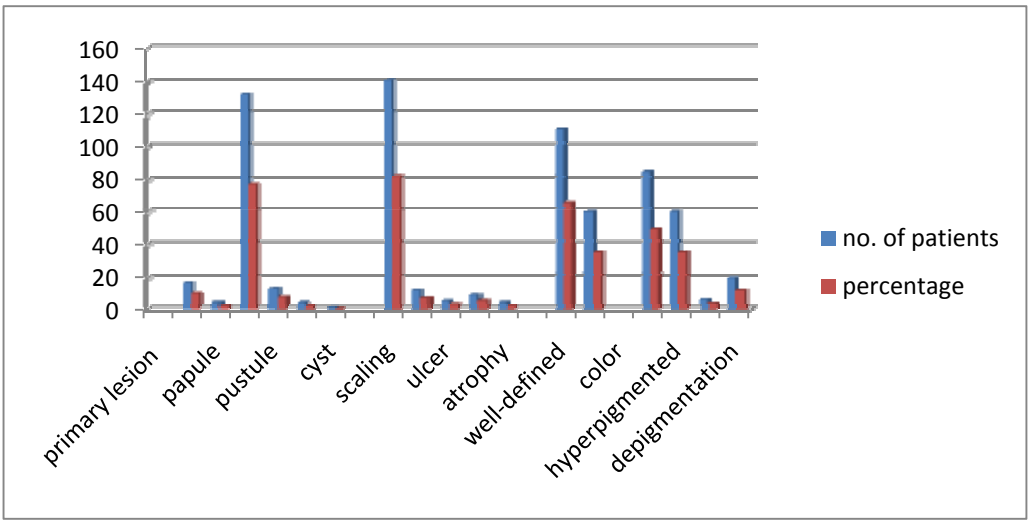


Figure 10. Shows morphology of scalp lesions.

Plaque (77.1%) was the commonest primary lesion seen among patients with scalp dermatoses in this study and scaling (82%) was the commonest secondary change.

AETIOLOGICAL CLASSIFICATION OF SCALP DERMATOSES:

Table 12: Aetiological classification of scalp dermatoses

Aetiology	Total patients (n=171)	Percentage (%)
Genetic / congentital	3	1.7 %
Inflammatory	124	72.5 %
Infective	10	5.8 %
Neoplastic	5	2.9 %
Miscellaneous	29	16.9 %
Total	**171**	**100 %**

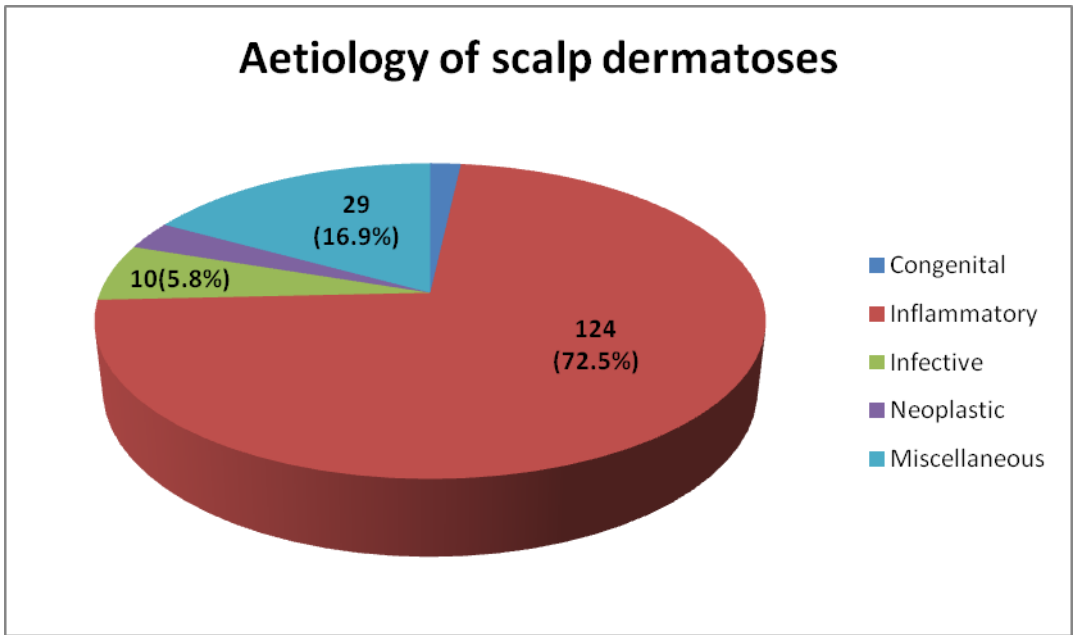


Figure 11. Aetiology of scalp dermatoses.

Inflammatory disorders (72.5%) were the common scalp dermatoses seen in adults.

CLINICAL TYPES OF SCALP DERMATOSES:

Table 13: Clinical types of scalp dermatoses

Types	Total patients (n=171)	Percentage (%)	Prevalence *
Chronic plaque psoriasis	57	33.3 %	1.0%
Seborrhoeic dermatitis	32	18.7%	0.5%
Pityriasis sicca	20	11.7 %	0.3%
Vitiligo	17	9.9 %	0.3%
Folliculitis	10	5.8 %	0.2%
Pemphigus vulgaris	9	5.3 %	0.2%
Discoid lupus erythematosus	4	2.3 %	0.2%
Chronic actinic dermatitis	3	1.8%	0.2%
Seborrheoic keratoses	2	1.2%	0.03%
Dermoid cyst	2	1.2%	0.03%
Pyogenic granuloma	2	1.2%	0.03%
Drug reactions	2	1.2%	0.03%
Trauma	2	1.2%	0.03%
Scarring alopecia	2	1.2%	0.03%
Pityriasis amiantiacea	1	0.5%	0.01%
Keratinous cyst	1	0.5%	0.01%
Bullous pemphigoid	1	0.5%	0.01%
Acquired icthyoses	1	0.5%	0.01%
X- linked icthyoses	1	0.5%	0.01%
Port wine stain	1	0.5%	0.01%
Cherry angioma	1	0.5%	0.01%

- Prevalence calculated based on total number of patients screened (6000)

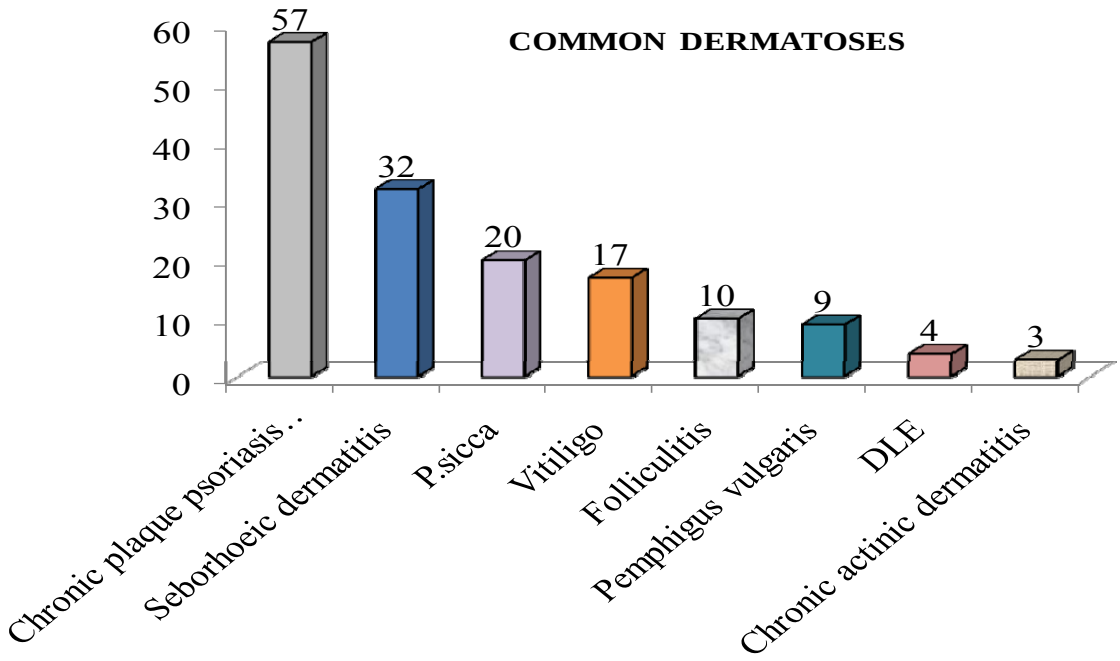


Figure 12. Shows the clinical types of scalp dermatoses.

Out of the 171 cases, psoriasis (33%) was the most common dermatoses followed by seborrheoic dermatitis(18.7%) , pityriasis sicca (11.6%) , vitiligo (9.9%), folliculitis (5.8%) and pemphigus vulgaris(5.2%) in our study.

SCALP PSORIASIS:

Table 14: Demographic and disease characteristics of scalp psoriasis

	Male (n=42)	Female (n=15)	Total (n=57)(%)
Age group			
18-30 yrs	5	7	12 (21.0%)
31-40 yrs	8	2	10 (17.5%)
41-50 yrs	18	5	23 (40.4%)
51-60 yrs	5	1	6 (10.5%)
>61 yrs	6	-	6 (10.5%)
Subtype			
Chronic plaque psoriasis	30	10	40 (70.2%)
Scalp psoriasis	12	4	16 (28.1%)
Pustular psoriasis	1	-	1 (1.8%)
Erythroderma	-	1	1 (1.8%)
Associated risk factors*			
Smoking	6	-	6 (10.5%)
Alcohol	8	-	8 (14.0%)
Stress	3	3	6 (10.5%)
Seasonal (winter)	16	4	20 (35.1%)
Drugs	1	-	1 (1.8%)
Sites **			
Frontal	34	10	44 (77.2%)
Parietal	39	8	47 (82.5%)
Temporal	22	1	23 (40.4%)
Occipital	2	1	3 (5.3%)
Vertex	1	-	1 (1.8%)
All	2	3	5 (8.8%)

* >1 risk factor present

** >1 site involved

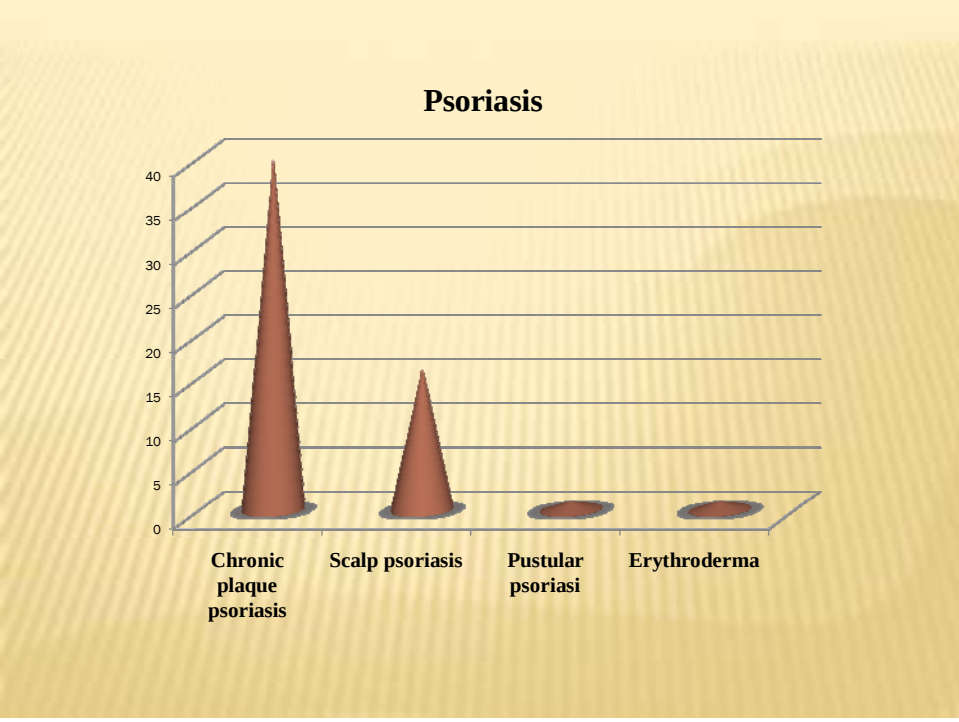


Figure 13. Demographic and disease characteristics of scalp psoriasis.

- The prevalence of psoriasis in our study is 33.33% with a median age of 45 yrs; seen in 42 males (73.6%) and 15 females (16.4%).
- Scalp was the only site of involvement in 16 patients (28%) and the initial site of involvement in 21 patients (36.8%).
- Commonest region of scalp involved was frontal, parietal and temporal areas.
- Scalp involvement was more common in patients with chronic plaque psoriasis.
- Scalp involvement was seen in only one patient of generalized pustular psoriasis.
- It was seen over the bald regions of the scalp in 3 patients above 50 years of age.
- History of exacerbation in winter months was seen in 20 patients (35%).
- Nail changes like pitting and onycholysis were seen in 18 patients.

SEBORRHOEIC DERMATITIS:

Table 15: Demographic and disease characteristics of Seborrhoeic dermatitis

	Males (n = 21)	**Females (n=11)**	**Total (n=32)(%)**
Age group			
18-30 yrs	8	2	10 (31.2%)
31-40 yrs	5	3	8 (25.0%)
41-50 yrs	5	4	9 (28.1%)
51-60 yrs	3	2	5 (15.6%)
>61 yrs	-	-	-
Subtypes			
Pityriasis sicca	6	14	20 (62.5%)
Pityriasis amiantiacea	-	1	13 (3.1%)
Polycyclic	15	10	25 (78.1%)
Petalloid	8	7	15 (46.8%)
Sites *			
Frontal	5	2	7 (21.9%)
Parietal	13	8	21 (65.6%)
Temporal	15	5	20 (62.6%)
Occipital	5	-	5 (15.6%)
Vertex	-	-	-
All	1	1	1(3.1%)

* >1 site

- The prevalence of seborrhoeic dermatitis in our study was 18.7%; seen in 21 males (65.6%) and 11 females (34.4%).
- Diffuse involvement of scalp was seen in majority of cases.
- It was seen in 2 cases of bald scalp in patients above 50 years.
- In 1 case of androgenetic alopecia, it was seen only over the hairy areas of the scalp.
- Scalp was the only site of involvement in 18 (56.2%) cases.

SCALP VITILIGO:

Table 16: Demographic and disease characteristics of vitiligo

	Males (n =7)	**Females (n=10)**	**Total (n=17)(%)**
Age group			
18-30 yrs	-	4	4 (23.5%)
31-40 yrs	2	-	2 (11.7%)
41-50 yrs	4	3	7 (41.2)
51-60 yrs	1	2	3 (17.6%)
>61 yrs	-	1	1 (5.8%)
Subtypes			
Vitiligo vulgaris	6	9	20 (62.5%)
Segmental vitiligo	1	1	2 (11.7%)
Sites *			
Frontal	7	8	15 (88.2%)
Parietal	3	5	8 (47.1%)
Temporal	10	5	15 (88.2%)
Occipital	1	1	2 (11.7%)
Vertex	2	2	4 (23.5%)
All	2	3	5 (29.4%)

** >1 site

- The prevalence of vitiligo in our study was 9.94%; seen in 7 males (41.2%) and 10 females (58.8%).
- Most common age group involved was 41-50 yrs (41.2%).
- Two cases had only scalp involvement.
- Scalp involvement was common in vitiligo vulgaris patients.
- Two cases of scalp involvement was seen in segmental vitiligo.
- Leukotrichia were present in all 17 cases**.**

SCALP FOLLICULITIS:

Table 17: Demographic and disease characteristics of folliculitis

	Males (n =5)	Females (n=5)	Total (n=10)
Age group			
18-30 yrs	3	1	4 (40%)
31-40 yrs	1	-	1 (10%)
41-50 yrs	-	2	2 (20%)
51-60 yrs	-	1	1 (10%)
>61 yrs	1	1	2 (20%)
Sites *			
Frontal	-	-	1 (10%)
Parietal	1	2	3 (30%)
Temporal	3	1	4 (40%)
Occipital	1	1	2 (20%)
Vertex	-	1	1 (10%)
All	-	-	-

- The incidence in our study was 5.8 %.
- It was seen in 10 patients, 5 males (50%) and 5 females (50%) with a median age of 56 years.
- All the five patients had only scalp involvement
- Grams staining of the pustules showed gram positive cocci in clusters in all the cases.

PEMPHIGUS VULGARIS:

Table 18: Demographic and disease characteristics of pemphigus vulgaris

	Males (n =5)	Females (n=4)	Total (n=9)
Age group			
18-30 yrs	1	2	3 (33.3%)
31-40 yrs	2	-	2(22.2%)
41-50 yrs	2	1	3(33.3%)
51-60 yrs	-	1	1 (11.1%)
>61 yrs	-	-	-
Sites			
Frontal	1	1	2 (20.0%)
Parietal	3	2	5 (55.5%)
Temporal	3	2	5 (55.5%)
Occipital	2	1	3 (33.3%)
Vertex	-	1	1 (11.1%)
All	2	-	2 (22.2%)

- The incidence of pemphigus vulgaris in our study was 6.4%; seen in five males (55.6%) and four females (44.4%) with a median age of 37 years.
- It involved all the regions of the scalp.
- Scalp was the initial site of involvement in two cases.

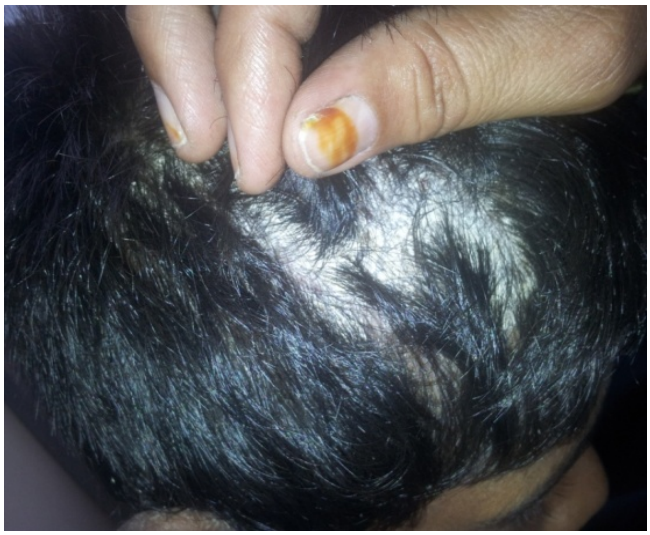
The incidence of Discoid lupus erythematosus was 2.3% in our study. Males and females showed equal incidence. In all the cases it was seen on hairy part of the scalp and associated hair loss was present in 75% of the cases in our study.

The incidence of chronic actinic dermatitis was 3 cases (1.8%) in our study. It was seen only in males. History of exacerbation in summer was seen in all the three patients.

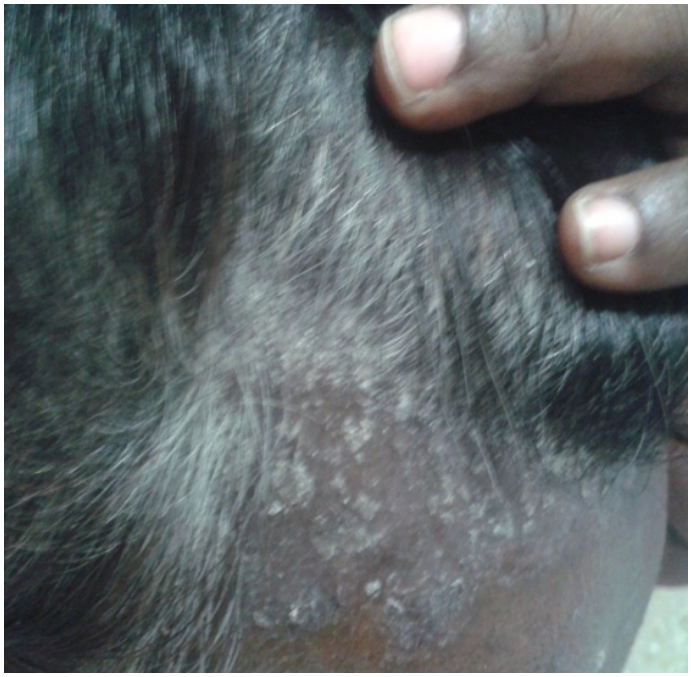
Seborrhoeic keratoses with scalp involvement was seen in (2 cases)1.2% of our study. Both the cases were seen in patients above 61 years.

There were two cases of dermoid cyst in this study with an incidence of 1.2% seen in one male and one female.

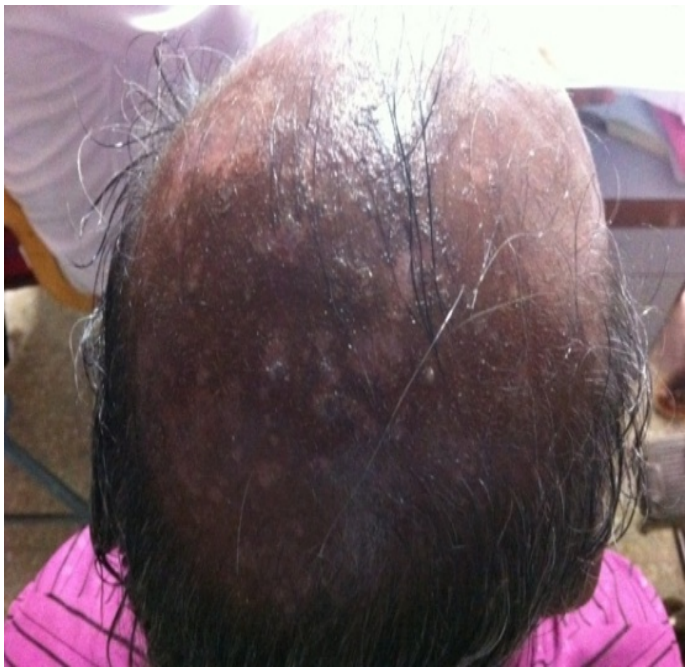
The miscellaneous and rare conditions with scalp dermatoses in our study were pyogenic granuloma (2 cases, 1.16%), Drug reactions (2 cases, 1.16%), Scarring alopecia (2 cases, 1.16%), Pityriasis amiantiacea (1 cases, 0.5%), Cherry angioma (1 case, 0.5%), Keratinous cyst (1 case, 0.5%), Acquired icthyoses (1 case, 0.5%), X-linked icthyoses-(1 case,0.5%), Port- wine stain (1 case, 0.5%) and Bullous pemphigoid (1 case, 0.5%).



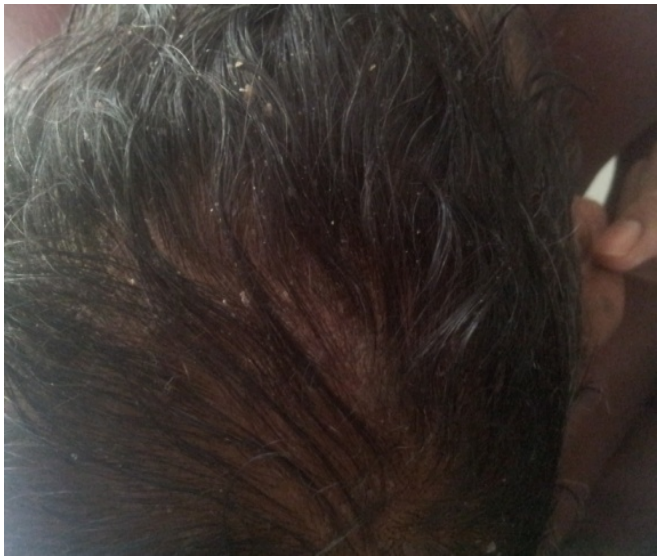
Chronic plaque psoriasis with scalp involvement.



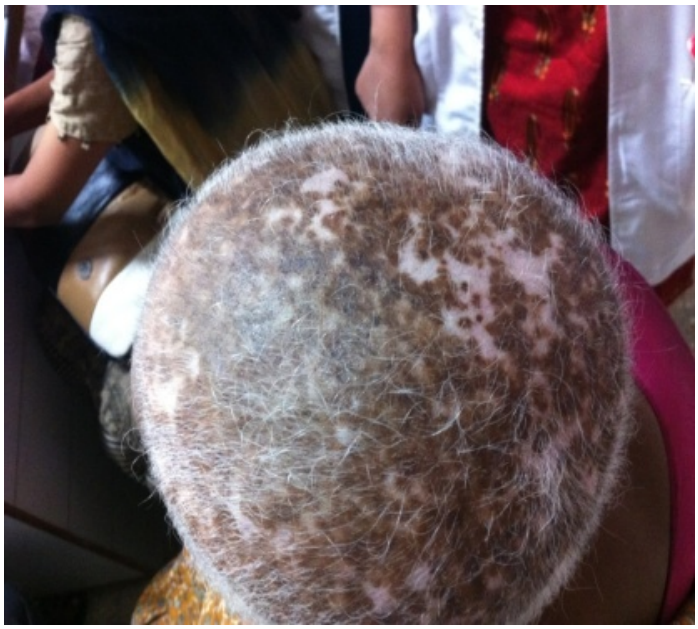
Chronic plaque psoriasis on the scalp extending beyond the hair line.



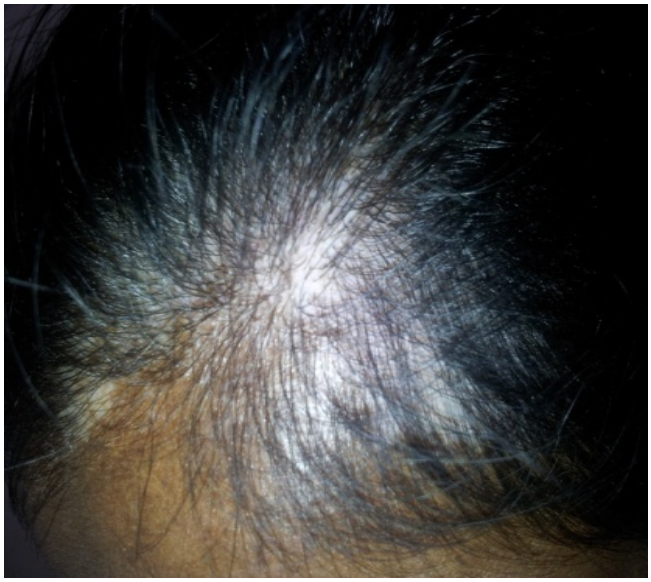
Seborrhoeic dermatitis involving a bald scalp .



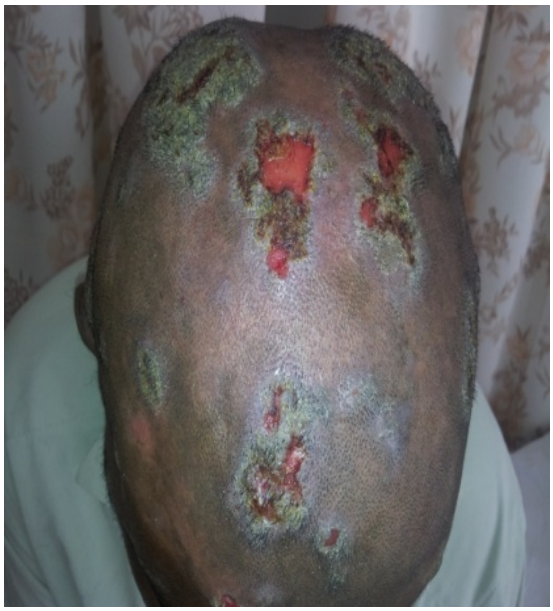
Pityriasis sicca over the scalp.



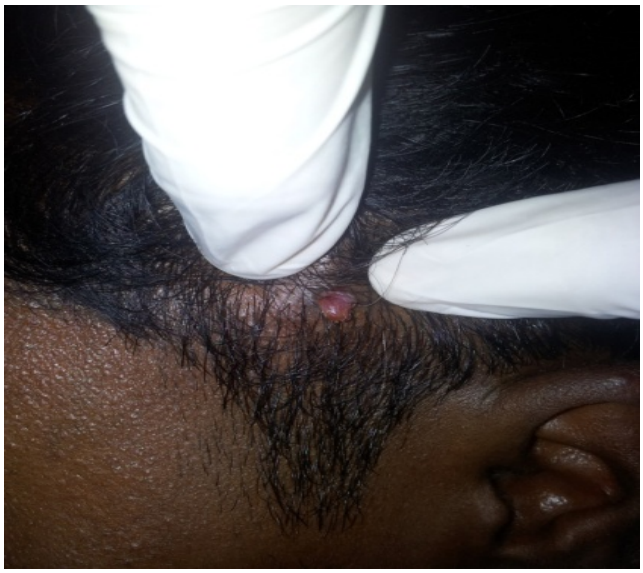
Scalp vitiligo over the frontal and parietal area in a lady.



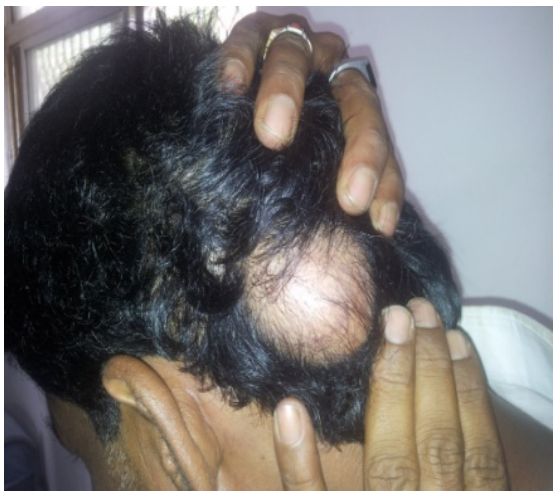
Scalp involvement in a case of vitiligo vulgaris.



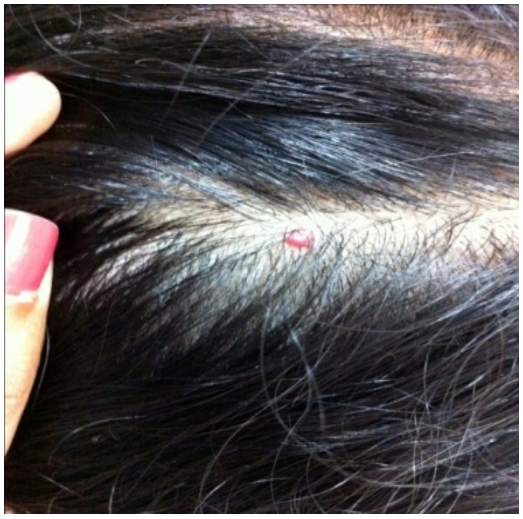
Multiple erosions over the scalp in a case of pemphigus vulgaris.



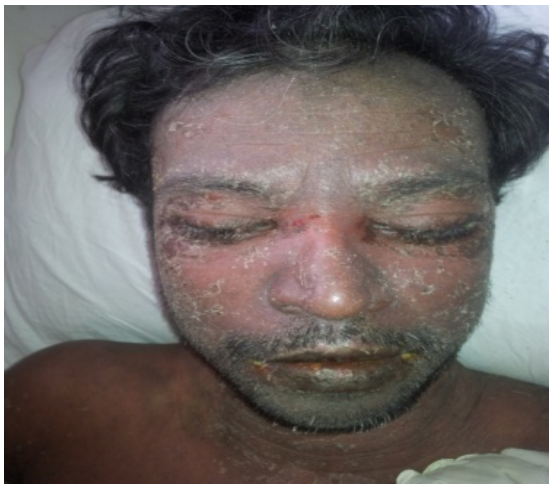
Pyogenic granuloma over the scalp.



Dermoid cyst over the scalp.



Cherry angioma over the scalp



A case of toxic epidermal necrolysis to carbamazepine.

DISCUSSION

Scalp dermatoses are very common in general practice, and result in emotional stress in adult patients due to the associated social stigma. They occur as primary diseases of the scalp, as part of a generalised inflammatory skin disease, or as part of a systemic disease.

Many common scalp conditions have similar symptoms, clinical features and in some cases it may be a sign of a more substantial medical problem, complicating diagnosis; so correct diagnosis is critical to determine proper treatment.[1]

There are no comprehensive studies on the various clinical patterns of scalp dermatoses in the Indian and Western literature.

In our study, a total of 171 adult patients with scalp lesions were enrolled. The overall prevalence of scalp dermatoses in our study of patients representing rural population was around 3%. There is no comparable prevalence rate of scalp dermatoses reported from any other population group.

The most common age group affected with scalp dermatoses in adults in our study was 5th decade (30.9%), followed by 3rd decade (29.8%). This finding is similar to the observations of several previous studies of seborrhoeic dermatitis and psoriasis.[1,20,93]

In our study, scalp dermatoses were more common in males (60.8%) than in females (39.2%), which is in concordance with previous studies.[76,93]

In our study, the predominant symptom complained of by the patients was itching (71.9%). The less reported symptoms were burning sensation (12.3%) and discolouration (9.9%). This is similar to the findings in earlier studies of seborrhoiec dermatitis and psoriasis.[1,20,170]

In a study conducted on a quantitatively representative sample of the French population, scalp itching was reported in only 25% of the population .[171]This discordance could be explained by the genetic and different environmental factors prevalent here.

Majority of our patients (94.7%) had symptoms persisting for more than 6 weeks duration, as inflammatory disorders were more common in our study population. This is consistent with the reported symptom duration in previous studies of inflammatory diseases. [172]

In the present study, the associated risk factors varied with the gender of the patient. Exacerbation of scalp dermatoses in winter season was documented in 21(34.4%) patients of both sexes; alcohol consumption (16.3%) and smoking (16.3%) were the other common associated risk factors in males, whereas in females, stress (9.8%) was the commonest associated risk factor. This is in agreement with the findings of the earlier studies.[93]

Scalp was observed to be the initial site of involvement in 73.1% of cases in our study population. Similar findings were reported in previous published studies.[1,20,93,170] Exclusive scalp involvement was seen in 47.4% of patients.

The most common scalp area involved in our study population was parietal region (57.3 %), followed by temporal (43.8%) and frontal regions (39.7%). Multiple sites were involved in 69.5% of our patients. There are no other comprehensive studies for comparison of common sites involved in scalp dermatoses.

Various morphological types were observed in our study, with plaques (63.7%) and scaling (67.8%) being the predominant primary and secondary types respectively. These findings are consistent with the morphological types seen in previous studies of seborrhoeic dermatitis and psoriasis.[1,20,76,93]

Inflammatory disorders were the common aetiological cause for scalp dermatoses in our study population, which is in agreement with the previous study. [173]

The most common scalp dermatoses observed in our study was psoriasis (33.3%), followed by seborrhoeic dermatitis (18.7%) and pityriasis sicca (11.7%), which is in concordance with the previous published study.[173]

Prevalence of scalp psoriasis in our study population was around 1% which is consistent with the reported prevalence in previous published studies from India. However, the prevalence rate is lower than that reported in Western populations. [77,174,175,176]This variance may be related to different environmental conditions, dietary habits and genetic differences.

In patients with chronic plaque psoriasis, scalp involvement was noticed in 30% of patients. Scalp was the only site to be involved by psoriasis in 28% of patients and it was the initial site to be involved in 66.6% of cases of psoriasis. This is in agreement with the previous published studies from India.[177,178] In our study, scalp involvement was more common in males (73.7%) than in females (26.3%), which is in concordance with study from North India.[174] Majority of our patients with scalp psoriasis were in the age group of 41-50 years and 18-30 years. This is in agreement to the bimodal distribution of psoriasis reported earlier. [174,177]

Chronic plaque type of psoriasis (98.2%) was the commonest morphological phenotype seen in our study, which is in concordance to the findings in other studies.[174,178]

Pruritus was the commonest associated symptom present in 98.2% of cases, which is in agreement to the previous published studies.[177]

Winter exacerbation was reported by % of patients in the present study, which was also observed in previous studies.[174]

Nail changes associated with psoriasis was observed in 31.5% of cases, which is similar to the finding in a previous study.[179] Joint involvement was seen in 3.5% of our cases which is low in comparison to the other studies.[180 ,181] This variance could be due to early presentation of patients with scalp psoriasis.

In our study, the prevalence of seborrhoeic dermatitis was around 1 % of our population. The mild form, dandruff /pityriasis sicca was seen in 11.7% of all patients with scalp dermatoses, whereas seborrhoeic dermatitis was observed in 18.7% of all cases studied. This is in concordance to the observations of previous studies.[20,93]

Pityriasis sicca and seborrhoeic dermatitis were seen in 60% and 31.2% of cases in the age group of 18- 30 years respectively. Seborrhoeic dermatitis occurring in patients above 50 years was seen in 15.6% of our cases. This is in agreement to the observations of previous studies.[1,20,76]

In our study, pityriasis sicca was more frequently seen in females(70%), whereas seborrhoeic dermatitis affected males predominantly (65.6%). Pruritus was the commonest presenting symptom seen in 98.6 % of the cases of pityriasis sicca and seborrhoeic dermatitis in our population.

The prevalence of vitiligo in our study of scalp dermatoses was 0.28 %, which is in concordance to previous studies.[182]

Scalp as the first site of involvement was noticed in 23% of vitiligo cases in our study. Scalp involvement was more common in patients of vitiligo vulgaris in our study, constituting 94.1% of cases. Only one case (5.8%) of scalp involvement was seen in segmental vitiligo. Majority of the patients (41.1%) in this study belonged to the age group of 41-50 years and females (58.8%) were more commonly affected than the males (41.17%).

The incidence of folliculitis occurring on the scalp in our study was 5.8%. Males and females showed equal incidence, with 40% of cases belonging in the age group of 18-30 years in our study.

The prevalence of pemphigus vulgaris in our study was 0.2%. Scalp as the initial site of involvement in pemphigus patients was seen in 22.2% of our pemphigus cases, which is similar to the observation of a previous study. [183]Scalp involvement in pemphigus patients was more common in males (55.5%) in our series.

The incidence of DLE in our study was 2.3%. Scalp was the initial and the only site of involvement in all the cases (2.3%). In all the cases it was seen on hairy part of scalp and associated hair loss was present in 75% of the cases. Males and females showed equal incidence in our study.

Other conditions recorded in our study with scalp dermatoses in adults were Chronic actinic dermatitis(3 cases,1.75%) , Seborrhoeic keratoses (2 cases 1.16%) , Trauma (2 cases, 1.16%), Pyogenic granuloma (2 cases, 1.16%), Dermoid cyst (2 cases, 1.16%), Drug reactions (2 cases, 1.16%), Scarring alopecia (2 cases, 1.16%), Pityriasis amiantiacea (1 cases, 0.5%), Cherry angioma (1 case, 0.5%), Keratinous cyst (1 case, 0.5%), Acquired icthyoses (1 case, 0.5%), X-linked icthyoses-(1 case,0.5%), Port- wine stain (1 case, 0.5%) and Bullous pemphigoid (1 case, 0.5%) which are not reported in any other study.

CONCLUSION

- Majority of the world's population experience scalp related symptoms at some point or the other.
- Though scalp dermatoses do not account for majority of the dermatological problems, the psychological impact is significant affecting the social profile of these patients.
- This study emphasizes the fact that many of the common dermatoses can significantly involve the scalp and can have overlapping symptoms and presentations making the diagnosis difficult.
- A proper management of scalp dermatoses is essential to improve the quality of life of the patients.
- This study gives a **precise clinical insight** into **scalp dermatoses** and thereby helps us manage the patient better.

SUMMARY

A total of 171 patients with scalp lesions who presented to the department of Dermatology at R.L. Jalappa hospital and research centre, attached to Sri Devaraj Urs Medical College, Tamaka, Kolar between January 2012 to June 201 were studied. The findings of our study is summarized below:

- Scalp dermatoses are common in adult population, with prevalence of 2.85%.
- Majority of the patients with scalp dermatoses belonged to the age group of 41-50 years (30.9%), followed by 3rd decade (29.8%).
- Males (60.8%) were affected more than females (39.2%), giving a male: female ratio 1.55:1.
- Majority of the patients (94.7%) presented with symptoms of more than 6 weeks duration.
- Itching was the most common presenting symptom in 71.9% of the cases.
- Associated risk factors with scalp dermatoses was elicited in 31.6% patients, with exacerbation of scalp dermatoses in winter season seen in 34.4% of those patients belonging to both sexes. In males, alcohol consumption and smoking worsened the condition in 16.3% of cases respectively. Emotional stress was the common associated risk factor in females (14.7%).
- Co morbid conditions were seen 11.1% of cases.
- Scalp was the initial site of involvement in 73.1% of the cases studied. Lesions exclusively over the scalp were seen in 47.3% of adults.
- Multiple regions of the scalp were affected in 69.6% of the patients, with parietal area being involved in 57.3% of cases.

- Plaques (77.1%) and scaling (82.4%) were the commonest morphological presentation in our study.
- A total of 21 different types of scalp dermatoses were noted in our study. Inflammatory conditions (72.5%) predominated in our study. The most common dermatosis was psoriasis which constituted 33.3% of cases, followed by seborrhoeic dermatitis(18.7%), pityriasis sicca (11.6%) and vitiligo (9.9%).

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PROFORMA

Name Age Sex

Occupation OP/IP no

Religion Address

HISTORY

Complaints: Flaking Skin lesions

Associated symptoms: Itching Pain

Burning sensation

Onset : Acute Chronic

Progression: Rapid Gradual

Initial site :

Other sites involved: Face Scalp

Trunk Upper limbs

Lower limbs

Seasonal variation: Summer Rainy season

Winter

Precipitating factors: Sunlight Alcohol

Smoking Stress

Treatment history : Topical Improved/ Worsened

Systemic Improved/Worsened

Associated diseases Diabetes Hypertension

Immunosuppresion Psychiatric disorders

Past history

Family history

EXAMINATION

General examination

Pallor Icterus Cyanosis Clubbing

Lymph nodes Oedema

CUTANEOUS EXAMINATION

Primary changes Macule Papule Plaque

Pustule Nodule Cyst

Secondary changes Scaling Crusting Ulcer

Erosion Hyper/hypopigmentation

Distribution (Scalp) Frontal Temporal Vertex

Parietal Occipital

Size of lesion

Shape

Margin Well defined Ill-defined

Color

Hair Hairy scalp Loss of hair

Non-hairy scalp Site

Any change in the color of hair

Involvement of other areas Face Trunk

Upper limbs Lower limbs

Nail changes

DIAGNOSIS

INVESTIGATIONS

TREATMENT

KEY TO MASTER CHART

1. SERIAL NUMBER:
2. IP/OP NO.
3. AGE : A-18-30 YEARS B- 31-40 YEARS C- 41-50 YEARS
   D- 51-60 YEARS E- >61 YEARS.
4. SEX : M- MALE F- FEMALE
5. SYMPTOMS : 1- Itching 2- Pain 5-Asymptomatic
   3- Burning sensation 4- Discoloration
6. Duration : 1- since birth
   2- <6 weeks
   3->6 weeks
7. Initial site : S- Scalp F- Face U- Upper limbs
   T- Trunk L- Lower limbs O-Oral cavity
8. Other sites involved : S- Scalp F- Face U- Upper limbs
   T- Trunk L- Lower limbs. O-Oral cavity A-All
9. Precipitating factors: A- Sunlight B- Alcohol E-Winter G-Drugs
   C- Smoking D- Stress F- Trauma
10. Associated diseases : D- Diabetes H- Hypertension S-Seizures
    I- Immunosuppression P- Psychiatric I-IHD
11. Site on the scalp : A- Frontal C- Temporal
    B- Parietal D- Occipital E- Vertex F-ALL
12. Primary changes: A- Macule B- Papule C- Plaque
    D- Pustule E- Nodule F- Cyst

13. Secondary changes : A- Scaling B- Crusting C- Ulcer

D- Erosion E- Hypopigmentation F-Hyperpigmentation

14. Margins : A- Well defined B- Ill- defined

15. Color : E- Erythematous H- Hyperpigmented

S- skin colored D- Depigmented

16. Hairy scalp : Y- Yes N- No

17. Loss of hair : Y-Yes N-No

18. Non- hairy scalp : : A- Frontal C- Temporal

B- Parietal D- Occipital E- Vertex

19. Color of the hair : B- Black G-Grey

20. Other sites : F- Face U- Upper limbs O- oral cavity

T- Trunk L- Lower limbs

21. Diagnosis :

										PRECIPITATING	TREATMENT			EXAMINATION OF LESION					HAIR					
S	HOSPITAL NO	AGE	SEX	OCCUPATION	SYMPTOMS	DURATION	INITIAL SITE	OTHER SITES INVOLVED		FACTORS	TOPICAL	SYSTEMIC	ASS DISEASES	SITE ON SCALP	PRIMARY CHANGES	SECONDARY CHANGES	MARGINS	COLOR OF LESION	HAIRY SCALP	LOSS OF HA	NON HAIRY SCALP	COLOR OF HAIR	OTHER SITES	DIAGNOSIS
1	725834	A	F	clerk	1	3	SCALP	-		-	-	-	-	F	-	A	B	-	Y	-	B	B	-	P.SICCA
2	726071	A	M	STUDENT	1	3	SCALP	_		-	-	-	-	F	-	A	B	-	Y	-	-	-	-	P.SICCA
3	829768	A	F	HOUSEWIFE	2	3	SCALP	A		-	-	Y	-	B+C	C	B	A	H	Y	-	-	-	F+U+T	PEMPHIGUS VULGARIS
4	839056	A	F	STUDENT	1	3	SCALP	-		-	-	-	-	F	-	A	B	-	Y	-	-	-	-	P.SICCA
5	740480	A	M	clerk	1	3	SCALP	T		-	-	-	-	B+C	C	A	B	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
6	734488	A	M	AGRICULTURIST	2	3	SCALP	-		-	-	-	-	C	E	-	A	H	Y	-	-	-	-	PYOGENIC GRANULOMA
7	730106	B	M	ATTENDER	1	3	U	S+L		-	-	-	-	B+C+D	C	A	A	E	Y	-	-	-	U+T+L	CHRONIC PLAQUE PSORIASIS
8	729479	A	M	TEACHER	1	3	SCALP	-		-	Y	-	-	B+C	C	A	B	E	Y	-	A + E	-	-	SEBORRHOEIC DERMATITIS
9	711222	E	F	HOUSEWIFE	2	2	SCALP	-		F	-	-	D	V	-	C	A	E	Y	Y	-	B	-	TRAUMA
10	725833	B	F	HOUSEWIFE	1	3	SCALP	-		-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
11	796679	C	F	HOUSEWIFE	1	3	U	S+U+T+L		-	Y	-	D	F	C	A	A	E	Y	-	-	-	U+T+LL	CHRONIC PLAQUE PSORIASIS
12	796692	A	M	STUDENT	1	3	UL	S+U+T+L		G	Y	-	-	A+B+C	C	A	A	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
13	786303	C	F	HOUSEWIFE	1	3	UL	S+U+LL		-	-	-	D	A+B+C	C	A	A	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
14	5235656	B	F	HOUSEWIFE	1	3	SCALP	POSTAURICULAR		-	-	-	-	B+C	C	A	B	H	Y	-	-	B	POSTAURICULAR	SEBORRHOEIC DERMATITIS
15	876348	E	M	BUSINESS	1	3	SCALP	-		-	-	-	-	A	C	A , ATROPHY	A	E	Y	Y	-	B	-	DLE
16	878107	A	M	BUSINESS	1	3	SCALP	-		-	-	-	-	A+B+C	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
17	878108	B	M	BUSINESS	1	3	SCALP	B		B	-	-	-	A	C	A	A	E	Y	-	-	-	-	DLE
18	614571	E	F	HOUSEWIFE	5	3	SCALP	-		-	-	-	-	B+C	F	-	A	SKIN COLORED	Y	-	-	B	-	EPIDERMAL CYST
19	763665	C	M	BUSINESS	1+3	3	U	S+T+LL		-	Y	-	H	C+D	D	-	A	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
20	843130	A	M	AGRICULTURIST	1	3	SCALP	-		-	-	-	-	C+D	C	A	B	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
21	542677	B	M	AGRICULTURIST	1	3	SCALP	-		D	-	-	-	A+B+C	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
22	877065	E	M	AGRICULTURIST	4	3	SCALP	T		-	-	-	D	B+C	C	HYPERPIGMENTATION	A	H	N	-	BALD SCALP	G	T	SEBORRHOEIC KERATOSES
23	881445	C	M	AGRICULTURIST	1	3	SCALP	-		E	-	-	H	A+B+C	C	A	A	H	Y	-	-	B	-	SCALP PSORIASIS
24	881356	B	M	AGRICULTURIST	1	3	SCALP	T		-	-	-	-	B+C	C	A	B	H	Y	-	AGA	B	T	SEBORRHOEIC DERMATITIS
25	881418	B	M	BUSINESS	1	3	SCALP	S		-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
26	881466	B	F	HOUSEWIFE	1	3	SCALP	-		-	-	-	-	F	C	A	B	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
27	881484	A	M	BUSINESS	3	2	SCALP	-		-	-	-	-	C	D	-	A	E	Y	-	-	B	-	FOLLICULITIS
28	887514	B	M	TEACHER	1	3	SCALP	U+T+L		E	-	-	-	A+B+C	C	A	A	H	Y	-	-	B	U+T+LL	CHRONIC PLAQUE PSORIASIS
29	854536	E	M	BUSINESS	1	3	SCALP	-		E	-	-	H	A+B+C	C	A	A	H	Y	-	-	G	-	SCALP PSORIASIS
30	620894	A	F	HOUSEWIFE	1	3	SCALP	-		-	-	-	-	F	-	A	B	-	Y	-	AA	B	-	P.SICCA
31	885010	D	M	BUSINESS	1	3	SCALP	T		B	-	-	D	B+C	C	A	B	H	-	-	BALD SCALP	B	T	SEBORRHOEIC DERMATITIS
32	881573	E	M	BUSINESS	1	3	U	S+U+T+L		B	-	-	H,JHD	F	C	A	A	H	-	-	BALD SCALP	G	U+T+L	CHRONIC PLAQUE PSORIASIS
33	846417	A	M	STUDENT	1	2	SCALP	POSTAURICULAR,TRUNK		-	-	-	-	F	C	A	B	H	Y	-	-	B	POSTAURI,T	SEBORRHOEIC DERMATITIS
34	791439	E	F	HOUSEWIFE	1	3	U,T	S+U+T+LL		-	-	-	H	C	C	B	A	H	Y	-	-	G	U+T+L	BULLOUS PEMPHIGOID
35	885913	B	M	AGRICULTURIST	4	3	U+T	S		-	-	-	-	F	A	-	B	D	Y	-	-	LEUCOTRICHIA	U+T	PEMPHIGUS VULGARIS
36	888913	A	F	STUDENT	1	3	SCALP	-		E	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	-	SCALP PSORIASIS
37	889309	B	M	AGRICULTURIST	1	3	SCALP	U+T+L		E,D	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	U+T+LL	CHRONIC PLAQUE PSORIASIS
38	888930	A	M	STUDENT	1	3	SCALP	-		-	-	-	-	C+D	C	A	B	H	Y	-	-	-	-	SEBORRHOEIC DERMATITIS
39	793196	C	M	AGRICULTURIST	1	3	ELBOWS	S+U+T+L		E	-	-	-	A+B+C	C	A	A	H	Y	-	-	-	U+T+LL	CHRONIC PLAQUE PSORIASIS
40	891349	A	F	STUDENT	1	3	SCALP	-		E	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	-	CHRONIC PLAQUE PSORIASIS
41	891671	C	F	TEACHER	1	3	SCALP	U		-	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	U	CHRONIC PLAQUE PSORIASIS
42	892226	C	M	BUSINESS	1	3	SCALP	-		B	-	-	-	F	-	A	B	-	Y	-	-	-	-	P.SICCA
43	891756	C	F	HOUSEWIFE	1	3	U	S+U		-	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	U	CHRONIC PLAQUE PSORIASIS
44	891804	B	M	BUSINESS	1	3	U	S+U		-	-	-	-	B+C	C	A	B	H	Y	-	-	-	S+U	SEBORRHOEIC DERMATITIS
45	891882	D	M	AGRICULTURIST	1	3	SCALP	U+T+L		E	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	S+U+L	CHRONIC PLAQUE PSORIASIS
46	889379	B	M	AGRICULTURIST	1	3	SCALP	EYEBROWS, POSTAURI		-	-	-	-	B+C	C	A	B	H	Y	-	-	-	EYE,POSTAURI	SEBORRHOEIC DERMATITIS
47	732767	A	F	STUDENT	3	2	SCALP	-		-	-	-	-	B	D	-	A	E	Y	-	-	-	-	FOLLICULITIS
48	735699	A	F	STUDENT	4	-	U	S+U+LL		-	-	-	-	B+C	A	-	B	D	Y	-	-	LEUCOTRICHIA	U+T+LL	VITILIGO VULGARIS
49	892593	B	F	AGRICULTURIST	1	3	SCALP	U		E	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	S+U	CHRONIC PLAQUE PSORIASIS
50	892199	C	M	AGRICULTURIST	1	3	SCALP	U+L		E	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	U+LL	CHRONIC PLAQUE PSORIASIS
51	866262	C	M	AGRICULTURIST	1	3	SCALP	RETROAUR		B	-	-	-	C+D	C	A	B	H	Y	-	-	B	RETROAUR	SEBORRHOEIC DERMATITIS
52	761357	C	M	POOJARI	1	3	UL	S,T,L		E,C	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	U+T+LL	CHRONIC PLAQUE PSORIASIS
53	915534	C	M	BUSINESS	1	3	SCALP	-		B,C,D	-	-	H	A+B+C	C	A	A	E	Y	-	-	-	-	SCALP PSORIASIS
54	923055	A	F	HOUSEWIFE	1	3	SCALP	U+T+L		_	_	_	-	A	C	A	A	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
55	879716	E	M	AGRICULTURIST	1	3	SCALP	U+T+LL		C	-	-	-	A+B+C	C	A	A	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
56	915259	A	M	ENGINEER	1	3	-	S+T+LL		-	-	-	-	A+B	C	A	A	E	Y	-	-	B	U+T+LL	CHRONIC PLAQUE PSORIASIS
57	910262	B	M	BUSINESS	1	3	SCALP	U+T+LL		C	-	-	-	A+B+C	C	A	A	E	Y	-	_	B	U+T+LL	CHRONIC PLAQUE PSORIASIS
58	868156	B	M	OFFICER	1	3	SCALP	-		B	-	-	H	A+B	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
59	859307	A	M	WELDER	1	3	SCALP	-		-	-	-	-	A+B	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
60	862369	D	M	AGRICULTURIST	1	3	SCALP	U+L		E	-	-	-	B+C	C	A	B	H	Y	-	BALD SCALP	B	-	SEBORRHOEIC DERMATITIS
61	871724	A	M	STUDENT	1	3	SCALP	U+L		E	-	-	-	A+B+C	C	A	A	E	Y	-	-	B	U+L	CHRONIC PLAQUE PSORIASIS
62	871723	D	M	AGRICULTURIST	1	3	U	S+U+LL		E	-	-	H	A+B+C	C	A	A	E	Y	-	-	G	U+T+LL	CHRONIC PLAQUE PSORIASIS
63	905481	C	M	TEACHER	1	3	SCALP	U+L		B	Y	-	-	A+B	C	A	A	E	Y	-	-	B	U+L	CHRONIC PLAQUE PSORIASIS
64	891349	A	F	STUDENT	1	3	U	S+T+LL		D	-	-	-	A	C	A	A	E	Y	-	-	B	T+LL	CHRONIC PLAQUE PSORIASIS
65	933096	E	M	SECURITY GUARD	1	3	SCALP	-		C	-	-	-	A+B	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
66	933220	C	M	COOLIE	1	3	SCALP	U+T		-	-	-	-	A+B	C	A	A	E	Y	-	-	B	U+T	CHRONIC PLAQUE PSORIASIS
67	862835	D	M	AGRICULTURIST	1	3	SCALP	TROAUR, NASOLABIAL,TRUI		B	-	-	H	A+B	C	A	B	H	Y	-	-	B	AUR,NASOLABIAL	SEBORRHOEIC DERMATITIS
68	892593	B	F	TECHNICIAN	1	3	SCALP	U+T+L		D	-	-	-	A+B	C	A	B	H	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
69	915534	C	M	BUSINESS	1	3	U	S+T+LL		C	-	-	-	A+B+C	C	A	B	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
70	857035	A	F	STUDENT	1	3	SCALP	-		D	-	-	-	B+C	C	A	B	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
71	857736	E	M	AGRICULTURIST	1	3	SCALP	U+T		A	-	-	-	C+D	C	A	A	E	Y	-	-	B	U+T	CHRONIC ACTINIC DERMATITIS
72	851338	C	M	AGRICULTURIST	4	3	SCALP	U+T		-	-	-	-	A+B	A	-	A	D	Y	-	-	LEUCOTRICHIA	U+T	VITLIGO VULGARIS
73	859340	E	F	AGRICULTURIST	4	3	LIPS,SCALP	U+T		-	-	-	-	A+B+C	A	-	B	D	Y	-	-	LEUCOTRICHIA	U+T	VITILIGO VULGARIS
74	870233	A	F	AGRICULTURIST	4	3	U	S+T+LL		-	-	-	-	A+B+C	A	-	B	D	Y	-	-	LEUCOTRICHIA	T+LL	VITILIGO VULGARIS
75	859204	C	M	GUARD	1	3	SCALP	-		C	-	-	-	A+B+C	C	A	A	E	Y	-	-	-	-	SCALP PSORIASIS
76	845956	E	M	AGRICULTURIST	1+3	3	U	F+U+T+L		G	Y	Y	-	B+C	C	A	B	H	Y	-	-	-	F+T+U+L	DRUG HYPERSENSITIVITY SYN TO CARBAMAZEPINE
77	813032	D	M	BUSINESS	1	3	U	S+U+T+L		-	Y	-	-	F	C	A	A	H	Y	-	-	-	U+T+L	CHRONIC PLAQUE PSORIASIS
78	829768	A	F	AGRICULTURIST	3	3	ORAL CAVITY	S+U+T		-	Y	-	-	V+C	C	B	A	H	Y	-	-	-	O+U+T+L	PEMPHIGUS VULGARIS
79	837023	B	M	AGRICULTURIST	1	3	SCALP	-		-	-	-	-	F	-	A	B	-	Y	-	-	-	-	PITYRIASIS SICCA
80	863602	C	M	AGRICULTURIST	1	3	SCALP	-		-	Y	-	-	A+B	C	A	A	H	Y	-	-	-	-	SCALP PSORIASIS
81	CAMP	C	M	AGRICULTURIST	1	3	SCALP	U+L		-	Y	-	-	B+C	C	A	A	E	Y	-	-	-	U+L	CHRONIC PLAQUE PSORIASIS
82	CAMP	C	M	AGRICULTURIST	1	3	SCALP	RETROAUR,TRUNK		-	-	-	-	A+B+C	C	A	B	H	Y	-	-	-	RETRO,TRUNK	SEBORRHOEIC DERMATITIS
83	865250	D	M	AGRICULTURIST	4	3	U	S+T+U+L		-	-	-	-	A+B	A	-	B	D	Y	-	-	LEUCOTRICHIA	U+T+L	VITILIGO VULGARIS
84	858505	C	M	AGRICULTURIST	1	3	SCALP	TROAUR, NASOLABIAL,TRUI		-	-	-	-	A+B	C	A	B	H	Y	-	-	-	ETROAUR,NASO	SEBORRHOEIC DERMATITIS
85	671879	B	F	AGRICULTURIST	1	3	SCALP	-		-	-	-	-	F	-	A	B	-	Y	-	AA	-	-	P.SICCA
86	731230	E	M	AGRICULTURIST	5	1	SCALP	FACE		-	-	-	D,H,JHD	LEFT SIDE	A	-	A	PURPURIC	-	-	BALD SCALP	-	FACE	PORT WINE STAIN
87	632184	B	M	BUSINESS	3	3	ORAL CAVITY	U+S+T+L		-	Y	Y	-	F	C	C+E	A	H	-	-	BALD SCALP	-	U+O+T+L	PEMPHIGUS VULGARIS
88	729431	C	M	AGRICULTURIST	3	3	U	S+T+U+L		-	Y	Y	-	A+B	C	C+E	A	H	Y	-	-	-	O+U+T+L	PEMPHIGUS VULGARIS
89	726071	A	M	STUDENT	1	3	SCALP	-		-	Y	Y	-	V+B	B	ATROPHY	A	H	Y	-	LOSS OF HAIRV+F	-	-	SCARRING ALOPECIA
90	CAMP	E	M	AGRICULTURIST	3	3	SCALP	-		-	-	-	-	O	D	-	A	E	Y	-	-	G	-	FOLLICULITIS
91	CAMP	C	M	AGRICULTURIST	1	3	SCALP	U		-	Y	-	-	A+B+C+V	C	A	A	E	Y	-	-	B	U	CHRONIC PLAQUE PSORIASIS
92	763665	C	M	BUSINESS	1	3	U	S+T+LL		-	Y	-	-	D	D	A	A	E	Y	-	-	B	U +T+L	PUSTULAR PSORIASIS
93	542677	B	M	AGRICULTURIST	1	3	SCALP	U		B	-	-	-	A+B	C	A	A	E	Y	-	-	B	U	CHRONIC PLAQUE PSORIASIS
94	843130	A	M	TEACHER	1	3	SCALP	-		-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
95	892180	B	M	AGRICULTURIST	1	3	SCALP	-		-	-	-	-	C+D	C	A	A	H	Y	-	-	-	-	SEBORRHOEIC DERMATITIS
96	896806	A	M	DRIVER	3	3	U	O+S+U+T		-	Y	Y	-	V+C	C	B	A	H	Y	-	-	B	U+O+T	PEMPHIGUS VULGARIS
97	CAMP	C	M	AGRICULTURIST	1	3	SCALP	-		B	-	-	-	A+B+C	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
98	902108	D	M	AGRICULTURIST	3	3	T	U+S+O+L		-	Y	Y	-	B+C	C	B+C	A	H	Y	-	-	B	O+U+T+L	PEMPHIGUS VULGARIS
99	902090	C	F	AGRICULTURIST	3	3	SCALP	U+O+T		-	-	-	-	B+C	C	B	A	H	Y	-	-	B	O+U+T+L	PEMPHIGUS VULGARIS

100	900213	D	M	AGRICULTURIST	1	3	SCALP	U+T+L	-	Y	Y	-	F	C	A	A	E	Y	-	-	G	U+T+L	CHRONIC PLAQUE PSORIASIS
101	904258	C	F	AGRICULTURIST	4	3	SCALP	T	-	Y	-	-	F	A	-	B	D	Y	-	-	LEUCOTRICHIA	T	VITILIGO VULGARIS
102	905481	C	M	TEACHER	1	3	SCALP	U+L	C	Y	-	-	A+B+C	C	A	A	E	Y	-	-	-	U+L	CHRONIC PLAQUE PSORIASIS
103	846763	A	F	STUDENT	1	3	SCALP	-	D	Y	-	-	A+B	C	A	A	E	Y	-	-	-	-	SCALP PSORIASIS
104	905532	C	F	HOUSEWIFE	1	3	SCALP	-	D	-	-	-	A	C	A,ATROPHY	A	D	Y	Y	-	-	-	DLE
105	833696	B	M	clerk	3	2	SCALP	-	C	-	-	-	D	B	-	A	E	Y	-	-	-	-	FOLLICULITIS
106	829999	E	M	AGRICULTURIST	1	3	SCALP	U+L	E	-	-	-	A+B	C	A	A	E	N	-	BALD SCALP	B	U+L	CHRONIC PLAQUE PSORIASIS
107	901924	C	M	AGRICULTURIST	1	3	U	S+T+U+L	E	Y	-	-	A+B	C	A	A	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
108	910262	B	M	AGRICULTURIST	1	3	U	S+T	E	Y	-	-	A+B+C	C	A	A	E	Y	-	-	B	T+U	CHRONIC PLAQUE PSORIASIS
109	909052	B	M	BUSINESS	1	3	SCALP	-	C	Y	-	-	B	B+D	A	A	E	Y	Y	-	B	-	SCARRING ALOPECIA WITH CONTACT DERMATITIS
110	895943	C	M	AGRICULTURIST	1	3	LL	S+U	E	Y	-	-	B+C	C	A	A	E	Y	-	-	B	U+L	CHRONIC PLAQUE PSORIASIS
111	913270	C	M	BUSINESS	1	3	SCALP	RIGHT PINNA	-	-	-	-	A	C	A,ATROPHY	A	E	Y	Y	-	B	RIGHT PINNA	DLE
112	914446	D	M	BUSINESS	1	3	SCALP	U+T+L	-	Y	-	D	A+B	C	A	A	E	Y	-	-	B	U+T+L	CHRONIC PLAQUE PSORIASIS
113	914275	A	F	STUDENT	1	3	U	S+T+u+L	-	Y	Y	-	F	C	A	A	E	Y	Y	-	B	U+T+L+F	ERYTHRODERMA SECONDARY TO PSORIASIS
114	CAMP	C	M	AGRICULTURIST	4	3	U	SCALP	-	-	-	-	B	A	-	A	D	Y	-	-	LEUCOTRICHIA	U	SEGMENTAL VITILIGO
115	CAMP	A	M	STUDENT	3	3	SCALP	-	-	-	-	-	B	E	-	A	E	Y	-	-	B	-	PYOGENIC GRANULOMA
116	CAMP	C	M	AGRICULTURIST	1	3	SCALP	-	-	Y	-	-	C+D	C	A	A	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
117	917207	A	M	AGRICULTURIST	1	3	SCALP	T	-	-	-	-	B+C	C	A	B	H	Y	-	-	B	T	SEBORRHOEIC DERMATITIS
118	917432	A	F	STUDENT	4	3	EYES	S+T	-	-	-	-	D	A	-	A	D	Y	-	-	LEUCOTRICHIA	T	VITILIGO VULGARIS
119	917791	D	F	HOUSEWIFE	3	3	SCALP	-	F	-	-	-	V	D	-	A	E	Y	-	-	-	-	FOLLICULITIS
120	CAMP	C	F	HOUSEWIFE	4	3	U	S+T+LL	-	Y	-	-	B+C	A	-	A	D	Y	-	-	LEUCOTRICHIA	T+U+L	VITILIGO VULGARIS
121	CAMP	C	F	HOUSEWIFE	3	3	SCALP	U	-	Y	-	-	C	D	-	A	E	Y	-	-	-	U	FOLLICULITIS
122	CAMP	C	F	HOUSEWIFE	3	3	SCALP	-	-	-	-	-	C	D	-	A	E	Y	-	-	-	-	FOLLICULITIS
123	CAMP	C	F	HOUSEWIFE	1	3	SCALP	-	-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
124	921876	A	M	clerk	1	3	SCALP	-	-	-	-	-	A+B	C	A	B	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
125	919494	C	F	HOUSEWIFE	1	3	SCALP	-	E	-	-	-	A+B	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
126	CAMP	D	F	HOUSEWIFE	1	3	SCALP	-	-	-	-	-	A+B+C	C	A	B	H	Y	-	-	G	-	SEBORRHOEIC DERMATITIS
127	CAMP	D	F	AGRICULTURIST	1	3	SCALP	-	D	-	-	-	A+B	C	A	B	H	Y	-	-	G	-	SEBORRHOEIC DERMATITIS
128	CAMP	E	F	AGRICULTURIST	3	3	SCALP	-	-	-	-	-	B	D	-	A	E	Y	-	-	G	-	FOLLICULITIS
129	CAMP	D	F	HOUSEWIFE	4	3	T	S	-	-	-	-	C	A	-	A	D	Y	-	-	LEUCOTRICHIA	T	VITILIGO VULGARIS
130	936385	A	F	STUDENT	1	3	SCALP	RETROAUR,	E	Y	-	-	D	C	A	A	E	Y	-	-	B	RETROAUR	SCALP PSORIASIS
131	947967	A	F	STUDENT	1	3	SCALP	-	-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
132	932660	A	M	STUDENT	3	3	SCALP	-	-	-	-	-	C	D	-	A	E	Y	-	-	B	-	FOLLICULITIS
133	927084	A	M	AGRICULTURIST	3	3	SCALP	-	-	-	-	-	B	D	-	A	E	Y	-	-	B	-	FOLLICULITIS
134	952868	A	F	STUDENT	1	3	SCALP	-	-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
135	CAMP	B	F	AGRICULTURIST	1	3	SCALP	-	-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
136	942347	C	M	AGRICULTURIST	1	3	U	S+T+U+L	A	-	-	-	A	C	A	B	H	Y	-	-	B	U+T+LL	CHRONIC ACTINIC DERMATITIS
137	942370	A	M	OFFICER	1	3	SCALP	-	-	-	-	-	B+C	C	A	B	E	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
138	ATTENDER	D	F	ATTENDER	5	3	SCALP	-	-	-	-	-	B+C	E	-	A	SKIN COLORED	Y	Y	-	B	-	DERMOID CYST
139	CAMP	E	F	AGRICULTURIST	5	3	SCALP	-	-	-	-	-	B	E	-	A	SKIN COLORED	Y	Y	-	G	-	DERMOID CYST
140	ATTENDER	A	F	ATTENDER	4	3	SCALP	-	-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
141	ATTENDER	C	F	ATTENDER	1	3	SCALP	-	-	-	-	-	C	C	A	B	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
142	796906	C	F	TEACHER	1	3	SCALP	-	-	-	-	-	B	C	A	B	H	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
143	942452	A	M	STUDENT	1	3	SCALP	-	E	-	-	-	A+B	C	A	A	E	Y	-	-	B	-	SCALP PSORIASIS
144	796679	C	F	AGRICULTURIST	1	3	SCALP	-	-	-	-	-	B	C	A	B	E	Y	-	-	B	-	SEBORRHOEIC DERMATITIS
145	792278	B	M	AGRICULTURIST	1	3	SCALP	-	-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
146	615875	B	F	HOUSEWIFE	1	3	SCALP	-	-	-	-	-	F	-	A	B	-	Y	-	-	B	-	P.SICCA
147	CAMP	C	F	AGRICULTURIST	1	3	SCALP	RETROAUR	-	-	-	-	B+C	C	A	B	H	Y	-	-	B	RETROAUR	SEBORRHOEIC DERMATITIS
148	606038	C	F	COOLIE	4	3	U	S+T	-	Y	-	-	B	A	-	A	D	Y	-	-	LEUCOTRICHIA	-	VITILIGO VULGARIS
149	798211	A	F	STUDENT	4	3	T	S+T	-	-	-	-	V	A	-	A	D	Y	-	-	LEUCOTRICHIA	T	SEGMENTAL VITILIGO
150	641570	C	M	AGRICULTURIST	1	3	SCALP	-	-	-	-	-	B	C	A	B	H	Y	-	-	-	-	VITILIGO VULGARIS
151	CAMP	C	M	AGRICULTURIST	4	3	T	S+U	-	-	-	-	V	A	-	A	D	Y	-	-	LEUCOTRICHIA	U+T+LL	VITILIGO VULGARIS
152	CAMP	C	M	AGRICULTURIST	1	3	SCALP	-	-	-	-	-	C	C	A	B	H	Y	-	-	-	-	SEBORRHOEIC DERMATITIS
153	615768	A	F	HOUSEWIFE	1	3	SCALP	-	-	-	-	-	B	C	A	B	H	Y	-	-	-	-	SEBORRHOEIC DERMATITIS
154	CAMP	E	F	HOUSEWIFE	5	3	FACE	S	-	-	-	-	B	C	-	A	H	Y	-	-	B	FACE	SEBORRHOEIC KERATOSES
155	508027	D	M	AGRICULTURIST	1	3	T	S+F+U+T	A	Y	-	-	D	C	A	A	E,H	Y	-	-	B	F+U+T	CHRONIC ACTINIC DERMATITIS
156	615959	C	F	HOUSEWIFE	5	3	SCALP	T	-	-	-	-	A	B	-	A	CHERRY RED	Y	-	-	G	TRUNK	CHERRY ANGIOMA
157	537230	C	M	AGRICULTURIST	1	3	U	F+S+T+U+L+O	G	Y	Y	S	A+B+D	C	A,EXFOLIATION	B	E	Y	-	-	B	O+F+U+T+L	DRUG REACTION (TEN) SEC TO PHENYTOIN
158	CAMP	C	M	BUSINESS	3	3	U	O+S+U+T	-	Y	Y	-	B+C+D	C	B	A	E	-	-	AGA	B	U+T	PEMPHIGUS VULGARIS
159	953316	D	F	HOUSEWIFE	4	3	T	S+U+O	-	-	-	-	B	A	-	A	D	Y	-	-	LEUCOTRICHIA	U+T+O	VITILIGO VULGARIS
160	562591	B	M	OFFICER	4	3	ORAL CAVITY	T+S	-	Y	-	-	C	A	-	A	D	Y	-	-	LEUCOTRICHIA	U+O+T	VITILIGO VULGARIS
161	787397	A	F	BANK WORKER	1	3	SCALP	-	-	-	-	-	F	-	C+E	B	-	Y	-	-	-	-	P.SICCA
162	508332	E	M	AGRICULTURIST	1	3	U	S+T+LL	-	Y	Y	-	A+B	C	A	B	H	-	-	BALD SCALP	-	F+U+T+L	ACQUIRED ICTHYOSES
163	542402	B	M	OFFICER	4	3	U	S+T	-	Y	-	-	A	A	-	A	D	Y	-	-	B	U+T	VITILIGO VULGARIS
164	603552	A	F	HOUSEWIFE	1	3	SCALP	-	-	-	-	-	-	A	-	B	-	Y	-	-	B	-	P.SICCA
165	623949	A	F	STUDENT	1	3	SCALP	-	-	-	-	-	-	A	-	B	-	Y	-	-	B	-	P.SICCA
166	CAMP	A	M	STUDENT	5	1	U+T	ALL	-	Y	-	-	A+B	C	A	B	H	Y	-	-	B	F+U+T+L	X-LINKED RECESSIVE ICTHYOSES
167	562591	B	M	AGRICULTURIST	1	3	SCALP	-	-	-	-	-	A+B	C	A	B	H	Y	-	-	B	T	SEBORRHOEIC DERMATITIS
168	614903	C	F	HOUSEWIFE	3	2	SCALP	-	F	Y	-	-	V	C	D	A	-	Y	-	-	B	-	TRAUMATIC ULCERATION
169	CAMP	C	F	HOUSEWIFE	1	3	SCALP	-	F	-	-	-	V+T	C	A	A	H	Y	-	MATTING	B	-	P.AMIANTIACEA
170	5033397	B	F	HOUSEWIFE	1	3	SCALP	T	-	-	-	-	B+C	C	A	B	H	Y	-	-	B	T	SEBORRHOEIC DERMATITIS
171	619683	E	M	AGRICULTURIST	1	3	SCALP	U+L	-	Y	-	-	A+B	C	A	A	H	-	-	BALD SCALP	-	U+L	CHRONIC PLAQUE PSORIASIS