A CLINICO-EPIDEMIOLOGICAL STUDY OF HAIR LOSS IN CHILDREN

By

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LIST OF ABBREVIATIONS USED

➢ α-KIF	α-Keratin intermediate filament molecules
➢ AA	Alopecia Areata
➢ AGA	Androgenetic Alopecia
➢ AEC	Ankyloblepharon-Ectodermal Defects
➢ ARIH syndrome	Autosomal recessive Ichthyosis with Hypotrichosis Syndrome
➢ BMP	Bone Morphogenic Protein
➢ CIE	Congenital Ichthyosiform Erythroderma
➢ DLE	Discoid Lupus Erythematosus
➢ EDA	Ectodysplasin
➢ EDAR	Ectodysplasin and its Receptor
➢ FD	Folliculitis Decalvans
➢ IRS	Inner Root Sheath
➢ IFAP	Ichthyosis Follicularis, Congenital Atrichia and Photophbia
➢ KPAF	Keratosis Pilaris AtrophicansFaciei
➢ KID syndrome	Keratitis, Ichthyosis and Deafness Syndrome
➢ LAS	Loose Anagen Syndrome
➢ LEKTI	Lymphoepithelial Kazal-type related inhibitor``
➢ ORS	Outer Root Sheath
➢ NS	Netherton Syndrome
➢ SPINK5	Serine Peptidase Inhibitor Kazal-type 5 (gene)
➢ TE	Telogen Effluvium
➢ T:V	Terminal to vellus hair ratio
➢ WNT	Wingless-type Integration Site

ABSTRACT

BACKGROUND:

Hair loss or alopecia is a common complaint in dermatology OPD and is associated with significant cosmetic concerns. Most cases of hair loss are reported in adults and are a relatively rare event in children. Hair loss in children may be caused by a number of conditions, which according to its mode of presentation, can be broadly grouped as congenital or acquired. Ethnicity, hair type, environment and cultural factors significantly affect hair loss. Though there are many studies on alopecia in adults, there is paucity in literature and research on causes of hair loss in children.

AIMS:

(i)To clinically evaluate all children presenting with hair loss and describe their clinical pattern.

(ii)To identify and document the different etiological factors of hair loss in children.

MATERIALS AND METHODS:

The present study was a hospital based descriptive study carried out in R L Jalappa Hospital and Research Centre, Tamaka, Kolar, from January 2016 to July 2017. All patients less than 18 years presenting with hair loss to the outpatient department of Dermatology, Venereology and Leprosy at the above-mentioned hospital were included in the study. A written informed consent was taken from the patient's parents or guardian. A detailed history of the patient including name, age, sex, history of presenting illness, hair grooming pattern, habits & tics, nail changes, other skin changes, systemic diseases, birth history, developmental history, family history of similar complaints and drug intake was taken.

General physical examination, scalp examination, examination of hair shaft and root, tests for hair anchorage and fragility were done in all cases. Relevant tests and laboratory investigations were done when necessary.

RESULTS:

Among 75 cases presenting with hair loss, 38 were male and 37 were female. The mean age of the study group was 8.2 ± 4.9 years. Majority of the children with hair loss were in school going age group (6 – 10 years) and adolescent age group (10 – 18 years), contributing to 30.7 % (n=23) and 28% (n=21) respectively. 61.3% of the children had hair loss of less than one month duration. 56 patients (76.6%) presented with asymptomatic hair loss. Itching was an associated complaint in 15 (20%), scaling in 7 (9.3%), pain in 3 (4%) and swelling in 3 (4%). The hair loss was localized in 80% of cases and diffuse in 20%. 8 out of 75 children had congenital form of hair loss. There were four cases of nevus sebaceous, one case of keratosis follicularis spinulosa decalvans, one case of X linked ichthyosis, one case of congenital aplasia cutis and one case of Darier's disease. The acquired alopecia was further sub-classified into scarring and non – scarring alopecia. Out of the 67 cases of acquired alopecia, four cases were scarring and the remaining were non – scarring. Three of the four cases of scarring alopecia were following infections and one was a case of discoid lupus erythematosus. Out of the 63 cases of non- scarring acquired alopecia, tinea capitis formed the majority (30.6%), followed by alopecia areata (25.3%) and telogen effluvium (9.7%). The other uncommon causes of acquired non-scarring hair loss in our study were tractional alopecia (n=5), seborrheic dermatitis (n=4), trichotillomania (n= 3), scalp candidiasis(n=1) and androgenetic alopecia (n=1).

CONCLUSION:

Hair loss in children is relatively rare when compared to adults, but is associated with significant psychological stress in both parents and the child. Diagnosis is difficult because the clinical presentation and etiologies differ from that of adults. Tinea capitis is the most common cause of hair loss in children in developing countries. Early diagnosis and prompt treatment can help prevent further spread of the infection and can completely reverse the hair loss. Alopecia areata, telogen effluvium, tractional alopecia and trichotillomania are a few other common causes.

Though congenital alopecias and scarring alopecias are less common, it is important to consider these conditions in diagnosing hair loss in children. Malnutrition is an important contributory factor in hair loss in children. Detailed history and physical examination is imperative in every case. Additional tests like KOH mount, dermatoscopy, Wood's lamp, Gram staining, bacterial culture, fungal culture and skin biopsy help in diagnosis.

Keywords: Alopecia, children, hair loss, tinea capitis, alopecia areata, telogen effluvium.

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INTRODUCTION

Hair loss or alopecia is a common complaint in dermatology OPD and is associated with significant cosmetic concerns[1]. Most cases of hair loss are reported in adults and are a relatively rare event in children[2].

Hair loss in children may be caused by a number of conditions, which according to its mode of presentation, can be broadly grouped as congenital or acquired[2]. The causes of alopecia vary according to the age group[2]. In addition ethnicity[1], hair type[1], environment[3] and cultural factors[3] also play a significant role in hair loss.

Hair loss in children is associated with significant psychological stress that may affect the growth and development of the child[4]. Certain cases of alopecia may be associated with inapparent systemic anomalies and nutritional deficiencies[5].

Though there are many studies on alopecia in adults, there is paucity in literature and research for causes of hair loss in children. This study is an attempt to appraise our knowledge on the clinical presentations and various etiologies of alopecia in children. The ultimate goal is to provide a guide for evaluation of hair loss in children.

OBJECTIVES

AIMS AND OBJECTIVES

1. To clinically evaluate all children presenting with hair loss and describe their clinical pattern.

2. To identify and document the different etiological factors of hair loss in children.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

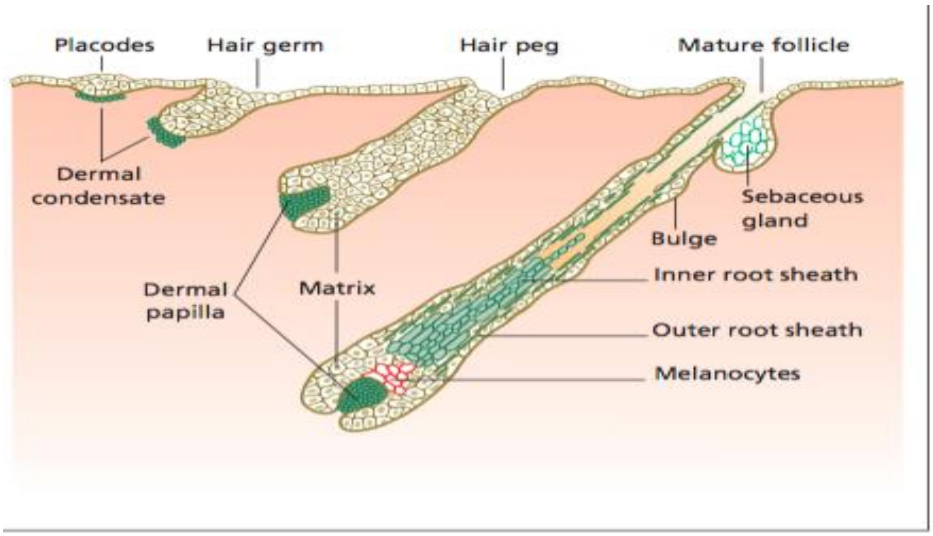
1. ANATOMY AND DEVELOPMENT OF HAIR

Hair is found only in mammals, serving varied functions like insulation from heat, sexual and social communication, and assisting in dispersion of scents secreted by complexes of sebaceous or apocrine glands.[6]

1.1 HAIR EMBRYOLOGY

In the human fetus, hair follicles develop from small collection of cells, called epithelial placodes (pre-term stage) by the crowding of basal keratinocytes. This first appears around 10 weeks of gestation.[7] The epithelial placode then expands to form the 'primary hair germ' whose progeny eventually generate the entire epithelial portion of the hair follicle. The epithelial placode induces the aggregation of formation of dermal papilla.[8]

Figure 1. Embryonic stages of hair follicle morphogenesis[9]



In the next stage of development, the bulbous peg or hair bud is formed by elongation of the hair germ diagonally into the mesenchyme. It becomes club shaped, with a concave tip surrounding the cluster of the mesodermal cells destined to be the dermal papilla.[10]

The hair follicle elongates and becomes bulbous at the lower end. The matrix is formed by the epithelial cells immediately surrounding the enclosed papilla, which gives rise to the hair shaft and inner root sheath. The outer root sheath is an extension of the epidermal basal layer which envelopes the entire hair follicle.[11]

Each primitive follicle bud consists of two or more smaller and oblique buds adjacent to it, that together form the primary follicle triad.[11] The first follicles are identified at 9 weeks of gestation and can be visible on the face in regions like eyebrows, upper lip and chin. Follicles are uniformly developed over the scalp by 10 weeks and marked angulations to the dermis develop by 11 weeks.[12] Embryonic hair shafts can be seen by 17 weeks on the eyebrows and over the entire scalp at 18 weeks. Follicle development spreads in the cephalocaudal direction.[13]

Various molecular pathways, growth factors, proteins and genes play an essential role in the growth of the hair follicle. Activation of canonical (β-catenin dependent) WNT (Wingless-type Integration Site) signals are important for the initiation of hair follicle formation. Ectodysplasin (EDA) and its Receptor (EDAR) are other important pathways that are involved in the placode stage of hair morphogenesis. Hence, formation of placodes in response to the first dermal signal involves activation of Ectodysplasin (EDA) and its Receptor (EDAR) signalling in the epithelium, followed by epithelial WNT signalling, and subsequent activation of BMP (Bone Morphogenic Protein) signalling.[14]

1.2 TYPES OF HAIR

There are three types of hair according to the hair follicle size:

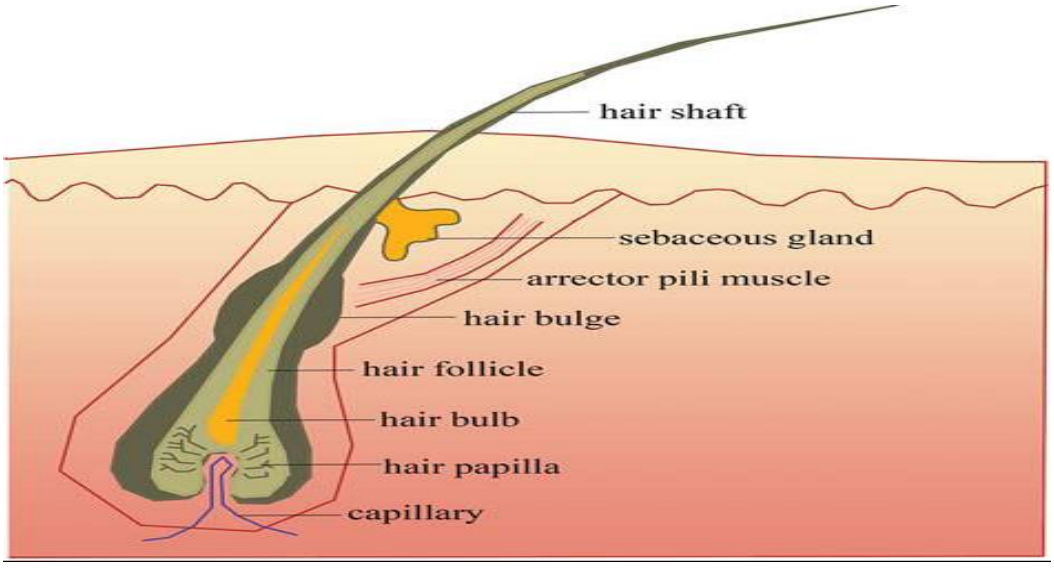
1. **Lanugo Hair:** It is the first hair fibre produced by a hair follicle. They are long, unpigmented and very fine hair. This type of hair is normally shed by the embryo at around 7–8 months' gestation or soon after birth.[15]
2. **Vellus Hair:** These are fine (less than 0.03 cm in diameter), unpigmented hair which are less than 2cm in length. They replace the lanugo hair at 7–8 months of gestation. Nose and cheeks are the common areas where vellus hair are seen in adults.[16]
3. **Terminal Hair:** These are long, coarse, pigmented, medullated hair seen on scalp, eyebrows and lashes at birth. Hair follicles around the genitals and axillae transform under the influence of hormones. Such changes are noted in the beard area and chest in men. Androgens cause the terminal hair to revert to vellus hair on the scalp in androgenetic alopecia.[16]

1.3 STRUCTURE OF HAIR

Hair consists of two distinct regions:

1. Hair shaft – Fully keratinized non-living part located above the skin[17]
2. Hair follicle - It is the living part located under the skin[17]

Figure 2. Diagram of an anagen follicle[17]



1.3.1 Hair Shaft

Hair shaft consists of three layers; cuticle, cortex and medulla.[17]

Cuticle

The hair shaft cuticle covers the hair with its integrity and properties greatly impacting the appearance of the hair. Proximally, the flat, square-shaped cuticle cells are tightly adherent to the cortex cells. The peripheral movement of the cuticle cells is responsible for the outward direction of growth of the distal free edge of the hair.[18] These cells also extensively overlap and interconnect with the cuticle cells of inner root sheath which contributes to the follicular anchorage of the growing hair. The cuticle cells have important protective functions against physical and chemical insults.[18]

Cortex

Cells migrate from the hair bulb to form the cortex. The shape of the cells is more fusiform. These cells are tightly bound and placed parallel to the axis of the shaft. Each cortex cell is packed with multiple axial keratin filaments (microfibrils) composed of hard α-Keratin intermediate filament (α-KIF) molecules.[19] Several cortex cells join together to form larger units called macrofibrils which represents about 50% of the cortex. The cortex forms the bulk of the hair shaft and also contains melanin.[19]

Medulla

The medulla consists of the coarser fibres that are located at the center of the shaft. The hair medulla consists of structural proteins that are markedly different from hair keratins and are rich in amino acid citrulline.[20]

1.3.2 Hair Follicle

The arrector pili muscle is attached to the bulge area of the hair follicle dividing the hair into two distinct parts:[21]

Upper Hair Follicle: This is distal to the attachment of arrector pili, and consists of the infundibulum and isthmus.[21]

Lower Hair Follicle: This is proximal to the attachment of arrector pili, and consists of the suprabulbar region and the hair bulb.[21]

The lower follicle regenerates with every hair follicle cycle but the upper follicle remains permanent.[21]

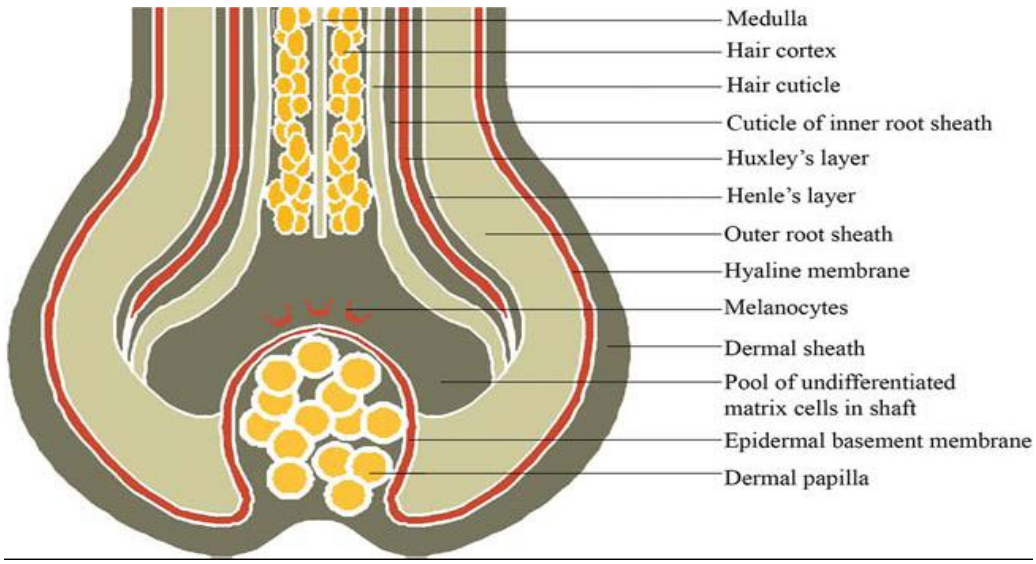


Figure 3. Diagram of proximal follicle[21]

Infundibulum

It is the uppermost part of the hair follicle that extends from the opening of the sebaceous gland to the skin surface. The upper part called the acroinfundibulum differentiates in a manner similar to epidermis, producing a granular layer and stratum corneum, which desquamates into the follicular lumen.[22]

Isthmus

It is the lower portion of the upper hair follicle located between the opening of the sebaceous gland and the insertion of arrector pili muscle. At this level, epithelium keratinization begins with the lack of the granular layer called 'trichilemmal keratinization'. Hair follicle stem cells are present in the bulge area on the isthmus close to the insertion of the arrector pili muscle.[23]

SuprabulbarRegion

It is the follicular region below the isthmus and above the hair bulb. It is composed of three layers from outermost to innermost: Outer root sheath, inner root sheath and hair shaft.[24]

- **Outer Root Sheath (ORS)**

This extends from the epidermis at the infundibulum to the hair bulb and its cells change considerably throughout the follicle. In the infundibulum, it is similar to the epidermis whereas in the isthmus, they begin to keratinize in a trichilemmal pattern. The ORS is multilayered in the region of upper hair bulb and becomes a single layer of cuboidal cells in the lower part of the hair bulb. Keratinocytes in ORS form the bulge area at the base of the isthmus where the arrector pili muscle is attached. In some follicles, there is a distinct single cell layer interposed between the outer and inner root sheaths known as the companion cells. Companion cell layer have numerous intercellular connections with the inner root sheath and migrate distally along with the inner root sheath to the isthmus region. It forms a plane of slippage between the inner and outer root sheaths. The ORS also contains melanocytes, Langerhan cells and Merkel cells.[25]

- **Inner Root Sheath (IRS)**

It contains three layers: Henle's layer (outer), Huxley layer (middle) and cuticle layer (inner). The cuticle layer cells interconnect with those of the hair shaft cuticle providing a tight anchorage between the hair shaft and the follicle. All three layers of the IRS undergo keratinization. The IRS keratinizes before the hair shaft within it and hence controls the definitive shape of the hair shaft. The IRS coats and protects the hair shaft up to the isthmus where it disintegrates.[26]

Hair Bulb

It is the expanded onion-shaped portion of the lower hair follicle, which includes the hair matrix and the follicular papilla. It is the active reproductive part of the hair. Follicular dermal papilla, mucopolysaccharide rich–stroma, nerve fibre and capillary loop are enclosed in the hair bulb. The matrix cells are confined to the lowermost portion of the follicle and surround all sides of the follicular papilla.[27]

The inner root sheath and hair shaft are derived from the matrix cells. The IRS is derived from the lower, laterally located matrix cells whereas the hair shaft originates from the upper, centrally located matrix. The matrix also produces hair keratins and other associated proteins. Melanocytes reside among the matrix stem cells and produce melanin, which are phagocytosed by surrounding matrix cells during differentiation. The hair colour depends on the amount and type of the phagocytized melanin pigment.[28]

1.4 Hair Growth Cycle

Hair development is a continuous cyclical process where all hair follicles go through different phases of growth cycle consisting of anagen (growth phase), catagen (regression

phase), telogen (resting phase) and exogen (shedding phase). The duration of the phases depends on the location of the hair, nutritional status, hormones and age.[29]

1.4.1 Anagen Phase

It is an active growth phase. During this phase, the hair follicle enlarges, becomes onion shaped and leads to the formation of hair fibre.[8] About 85–90% of all scalp hairs are in anagen phase.[30]

It is divided into six stages (I-VI). During anagen I-V (proanagen), hair cells proliferate, enclose the growing dermal papilla, grow downward into the skin and differentiate into the hair shaft and inner root sheath. The newly formed hair shaft starts elongating and becomes pigmented. During anagen VI (metanagen), there is a fully developed hair follicle, i.e., an epithelial hair bulb surrounds the dermal papilla deep in the subcutaneous tissue, and the hair shaft is visible on the skin surface. This phase lasts for several years in the hair follicles.[30]

1.4.2 Catagen Phase

This phase starts when the anagen growth phase comes to an end. The growth of the hair shaft stops in this phase. Initially, there will be a significant decrease in the differentiation and proliferation of the hair matrix keratinocytes and the pigment-producing activity of melanocyte stops. The hair follicle undergoes apoptosis-driven regression resulting in a reduction in about one-sixth of its diameter. The proximal end of the hair shaft keratinizes to form a club-shaped structure. The dermal papilla gets transformed into a group of quiescent cells adjacent to the regressing hair follicle epithelium and it moves upwards to eventually lie just below the level of the arrector insertion. This phase usually lasts for a few weeks.[31]

1.4.3 Telogen Phase

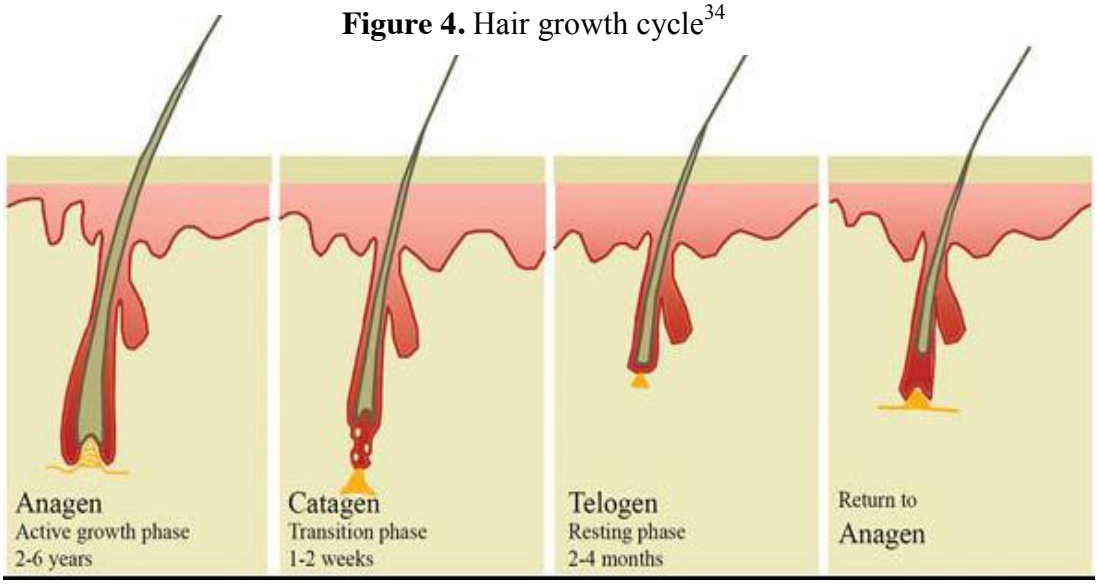
During this phase, the hair goes into the resting state and usually lasts for few weeks (eyelashes) to eight months (scalp hair). There is no growth during this phase and the dermal papilla stays in the resting phase. Telogen hair follicles are characterized by the lack of pigment-producing melanocytes and the inner root sheath. The dermal papilla of the telogen hair follicles is in close relation to a small cap of secondary hair germ (stem cells). Approximately about 10–15% of all hairs are in the resting phase.[32]

This dormant phase lasts for 2–3 months before the cycle begins again with the anagen phase.[33]

1.4.4 Exogen

This is the shedding phase. The shedding is believed to be an active process and is independent of telogen and anagen. There is less interest in the mechanism of hair shedding but from the patient's perspective, it is probably the most important phase of hair growth. It is not unusual for human telogen hairs to be retained in more than one follicular cycle and this suggests that anagen and exogen phases are independent.[34]

Figure 4. Hair growth cycle[34]



1.4.4 Bulk-Activation Hypothesis

It is given to explain the cyclical activity of the follicle. It proposes that stem cells of the follicle reside in the bulge region at the level of insertion of arrector pili muscle that marks the bottom of the permanent portion of hair follicles. [35]

The hypothesis emphasizes on the following points:

i. The bulge cells are morphologically undifferentiated and slow cycling under normal conditions. However, these stem cells have the potential to multiply at a high rate and differentiate into epidermis, hair follicle and sebaceous gland.[36]

ii. During anagen phase IV, these stem cells divide to form transient amplifying cells which multiply to form matrix cells.[36]

iii. The transient amplifying cells have limited mitotic activity. The variation in the proliferative capacity of the transient amplifying cells is responsible for the variation in length of anagen phase. Matrix cells, derived from bulge stem cells, are transient amplifying cells and thus have only a limited proliferative potential.[37]

iv. The upward movement of dermal papilla cells during late catagen phase is crucial to allow the subsequent physical interaction or activation of the resting bulge cells via signaling pathways and release of growth factors to start a new cycle of hair growth.[38]

3. ALOPECIA

Alopecia refers to loss of hair from any hair-bearing surface of the body[39]

There are 2 major forms of alopecia:

1) **Scarring Alopecia:** There is fibrosis following inflammation resulting in permanent loss of hair follicles. A smooth scalp without any visible follicular openings is usually observed.[40]

2) **Non-scarring Alopecia:** The hair shafts are gone, but the hair follicles are preserved, explaining the reversible nature.[40]

Scalp hair loss in pediatric age group can be classified as follows:

Table 1. Classification of hair loss

1. CONGENITAL ALOPECIA

A. <u>Diffuse congenital atrichia or hypotrichosis</u>

i. Hypotrichosis occurring alone or with other minor abnormalities
   a) Isolated congenital atrichia or hypotrichosis
   b) Marie Unna hypotrichosis
   c) Atrichia with papular lesions
   d) Congenital hypotrichosis with milia

ii. Hypotrichosis with juvenile macular dystrophy

iii. Hypotrichosis with ectodermal dysplasias

iv. Hypotrichosis with ichthyoses

v. Hypotrichosis with premature aging syndromes

vi. Hypotrichosis with immunodeficiency syndrome

vii. Hypotrichosis with genetic disorders of hair shaft[41]
   a) Increased fragility
      i. Trichorrhexis nodosa
      ii. Trichorrhexis invaginata
      iii. Monilethrix
      iv. Pseudomonilethrix

v. Pili torti

vi. Pili bifurcate

vii. Trichothiodystrophy

viii. Marie Unna hypotrichosis

ix. Mitrochondrial disorders[42]

b) No hair fragility

i. Pili annulati

ii. Woolly hair

iii. Uncombable hair[42]

B. <u>Congenital localized alopecia</u>

i. Neonatal occipital alopecia

ii. Scalp injury

iii. Temporal triangular alopecia

iv. Localized alopecia associated with other nevoid conditions

v. Localized alopecia associated with syndromes[42]

2. ACQUIRED ALOPECIA

A. <u>Non-scarring acquired alopecia</u>

i. Telogen effluvium

ii. Androgenetic alopecia

iii. Alopecia areata

iv. Tinea capitis

v. Traction alopecia [43]

B. Scarring acquired alopecia

i. Primary Cicatricial

a) Lichen planopilaris

b) Keratosis pilaris atrophicans

c) Discoid lupus erythematosus

d) Folliculitis decalvans

e) Dissecting cellulitis of the scalp

f) Acne keloidalis

g) Traumatic alopecia [44]

ii. Secondary Cicatricial

a) Localized scleroderma

b) Radiation

c) Pemphigoid

d) Chemical/physical injuries

e) Bacterial, viral or fungal infections[42]

3.1 CONGENITAL OR EARLY ONSET ALOPECIA

Alopecia that occurs at birth or during the early months of life can be diffuse or localized.[45]

3.1.1 DIFFUSE CONGENITAL ALOPECIA

Diffuse congenital alopecia may be hypotrichosis (reduction in hair follicles) or atrichia (complete absence of hair follicles) and may occur alone or as a part of a syndrome.[41]

3.1.1.1 Hypotrichosis occurring alone or with other minor abnormalities

a) Isolated Congenital Atrichia or Hypotrichosis

Within this group, several distinct genotypes are represented with dominant, recessive and X-linked inheritance pattern. The autosomal recessive inherited ones are the most severe and manifest at birth. Either there is complete absence of scalp hair or very sparse hair growth with histopathology showing absence of hair follicles or scattered miniaturized hair follicles respectively. The scalp, eyebrows, eyelashes and general body hair are affected. The extent of hypotrichosis may not be evident until the child is several years old due to the variation of hair cover in young children.[44]

b) Marie Unna Hypotrichosis

This is an autosomal dominant condition that is characterized by coarse, wiry and sparse hair in early childhood. The hair loss then continues to progress and is often aggravated during puberty. Microscopic examination demonstrates irregularly distributed twisting and longitudinal ridging. Histopathologically, there are reduced numbers of follicles with mild to moderate inflammatory infiltrate. There is no fibrosis and scarring. The condition is genetically heterogeneous with mutations being identified in both chromosome 8p21 and chromosome 1p21.[44] The eyebrows, eyelashes and axillary hairs are also affected.[45]

c) Atrichia with Papular Lesions

This is a rare, irreversible alopecia that is characterized by small white papular lesions. It has an autosomal recessive inheritance and the affected individuals present with a distinct pattern of total hair loss on the scalp, axilla and body. Also, there is patchy

involvement of the eyelashes and eyebrows. The hair is almost completely shed during the first weeks and months of life. The papular lesions are distributed diffusely on the face and scalp, and they erupt only during infancy. The papules represent keratin-filled follicular cysts. Histology demonstrates the tubular epithelial structures devoid of the hair bulbs resembling epidermoid cysts. This condition has been shown to be due to mutation in the hairless gene on chromosome 8p21. Atrichia with papular lesions also occur in the clinical setting of vitamin D-dependent rickets type IIA that results from mutations in the vitamin D receptor gene.[41]

d) Congenital hypotrichosis with Milia

This is an X-linked dominant disorder characterized by hypotrichosis with sparse, coarse hair and multiple milia.[42] It has some clinical similarity to atrichia with papular lesions. It presents at birth on the face, limbs and trunk.[41]

3.1.1.2 Hypotrichosis with Juvenile macular dystrophy

Hypotrichosis with juvenile macular dystrophy is a rare autosomal recessive disorder with sparse and short hair from birth. There is progression of macular degeneration within few years leading to blindness. It occurs as a result of a mutation in CDH3 encoding P-cadherin.[41]

3.1.1.3 Hypotrichosis with Ectodermal Dysplasias

Hypotrichosis is an important feature in many Ectodermal Dysplasias (ED) syndromes but often occurs only after the neonatal period. Some of these conditions include

hidrotic ED, hypohidrotic ED, Ankyloblepharon-Ectodermal Defects (AEC) and clefting syndrome, Rapp-Hodgkin syndrome and Bazex-Dupre-Christol syndrome.[46]

3.1.1.4 Hypotrichosis with Ichthyoses

a) Lamellar Ichthyosis

The newborn is born encased in the collodion membrane that sheds in 10 to 14 days. This condition is characterized by alopecia that usually occurs as a result of hair being shed with the large plates of scale which exfoliate from the scalp or as a result of secondary bacterial infection.[47]

b) Ichthyosis Follicularis, Congenital Atrichia and Photophobia (IFAP)

It is an X-linked trait, characterized by atrichia or severe hypotrichosis, keratotic follicular papules and photophobia since birth. IFAP is now thought to be a cell-to-cell adhesion disorder. Gingival erythema, periorificial erosions, keratotic plaques, nail dystrophy and recurrent infections are the other features of IFAP. Oral retinoids can be used to improve the cutaneous features but not in the treatment of alopecia or photophobia.[42]

c) Peeling Skin Syndrome

Peeling skin syndrome is a rare disorder characterized by continuous shedding of stratum corneum. It starts either at birth or early childhood. It is inherited in an autosomal recessive pattern. The skin involvement is generalized but may spare the face, palms and soles. There is a single case report of a child with the inflammatory variant of the peeling skin syndrome that had associated hair shaft abnormalities like trichorrhexis invaginata, irregular twisting of the hair and hair shaft narrowing.[46]

d) Keratitis, Ichthyosis and Deafness Syndrome (KID Syndrome)

KID syndrome is a congenital ectodermal defect that appears to be genetically heterogeneous. It is caused by mutations in the GJB2 (connexin 26) or the connexin 30 genes. Severe hypotrichosis of scalp, eyebrows and eyelashes is present at birth and persists throughout life. The features of KID syndrome include the spiny follicular plugging, widespread indurated erythematous plaques, perioral furrowing, reticulate hyperkeratosis of palms and soles, keratitis and sensorineural hearing loss.[41]

e) Congenital Ichthyosis, Follicular Atrophoderma, Hypotrichosis and Hypohidrosis

This combination of traits have been described as a new autosomal recessive genodermatoses and all the features are present from the neonatal period.[43]

f) Autosomal Recessive Ichthyosis with Hypotrichosis (ARIH) Syndrome

ARIH syndrome is characterized by corneal involvement, congenital ichthyosis and abnormal hair, caused by alteration in the STI4 gene which encodes the serine protease matriptase.[41]

3.1.1.5 Hypotrichosis with Premature Aging Syndromes

In these conditions, the onset of hypotrichosis is usually delayed until several years of age. There is appearance of sparse hair in early infancy in some cases of Hutchison-Gilford progeria, Rothmund-Thomson syndrome and Cockayne syndrome. In case of severe neonatal progeroid syndrome, features like sparse anterior scalp, thin eyebrows and absence of eyelashes are evident at birth, along with redundant skin, absence of subcutaneous fat and presence of prominent blood vessels.[41]

3.1.1.6 Hypotrichosis with Immunodeficiency Syndrome

Hair loss is a remarkable feature of the heterogeneous group of congenital immunodeficiency conditions presenting in early infancy with erythroderma, failure to thrive and diarrhea. It includes severe combined immunodeficiency-associated congenital graft-versus-host disease and Omenn syndrome. Sparse scalp, eyebrow and eyelash hair in the neonatal period is seen in cartilage hair hypoplasia syndrome, associated with short limbs and growth failure.[47]

3.1.1.7 Hypotrichosis with Genetic Disorders of the Hair Shaft

Due to increased fragility of hair, many of the genetic disorders of the hair shaft can lead to early alopecia.[47]

a) **Monilethrix**

It is characterized by hair shafts with elliptical nodes at regular intervals with intervening, non-medullated tapered fragile constrictions. As a result of breakage, hairs grow less than 1–2 cm in length and results in a stubby appearance. It usually presents in early childhood, but there is one case report of onset in the second decade of life.[43]

It is an autosomal dominant disorder caused by mutations of the human basic hair keratin hHb6 and hHb1, which causes disruption of the keratin formation in the hair shaft cortex. Autosomal recessive monilethrix is caused by heterozygous mutation in the desmoglein 4 gene which contributes to the disruption of desmosomes in the patient's hair shaft. The diagnosis is confirmed by examining hairs under light microscopy. The extent of hair breakage is variable in monilethrix, and vellus hair also shows the abnormality in severely affected patients.[44]

b) Pseudomonilethrix

It is a developmental defect of the hair, characterized by irregular nodes along the hair shaft resulting in increased fragility, breakage and partial baldness. It is inherited as an autosomal dominant trait with incomplete penetrance. This condition is seen in both the sexes. Pseudomonilethrix involves only the scalp hairs. The hairs are dry, brittle, irregular in length and difficult to groom. The abnormality is seen in all shades of fair, brown and black hair. The diagnosis is elucidated by light and electron microscopy.[43]

c) Pili Torti

Pili torti is characterized by hair shafts which are flat and twisted 180° degrees along its axis, at irregular intervals. Pili torti usually presents in the first two years of life. Inheritance patterns can be autosomal dominant, autosomal recessive or sporadic. The scalp, eyebrows and eyelashes have hairs that do not grow long and are easily broken.[43] A recent dermatoscopic study found that pili torti was reported in approximately 57.1% of cases with primary cicatricial alopecia.[44]

d) Trichorrhexis Invaginata (Netherton Syndrome)

Netherton Syndrome (NS) is an autosomal recessive disorder characterized by a triad of ichthyosis linearis circumflexa, trichorrhexis invaginata and atopic diathesis. It is usually seen in infancy and progresses with growing age.[43] Clinically, the scalp hair is sparse, brittle and short.[44] The skin findings in NS ranges from ichthyosis linearis circumflexa to non-bullous Congenital Ichthyosiform Erythroderma (CIE). Newborns with NS will have collodion membrane, generalized scaling or erythema. Atopic dermatitis, hay fever, angioedema, urticaria, allergic rhinitis, hypereosinophilia, recurrent skin infections and increased levels of immunoglobulin E are seen. Short

stature, growth retardation and mental deficits occur in many patients. Microscopically, in trichorrhexis invaginata (bamboo hair) the distal hair shaft infolds into the proximal hair shaft. When the hair breaks at this area of invagination, only the proximal invaginated hair shaft can been seen which is referred to as 'golf-tee hair'. A defect in the Serine Peptidase Inhibitor Kazal-type 5 (SPINK5) gene on chromosome 5q32 that encodes the serine protease inhibitor LEKTI (Lymphoepithelial Kazal-type related inhibitor) causes Netherton syndrome.[43]

e) Trichothiodystrophy

The term trichothiodystrophy is used to describe cysteine-deficient brittle hair.[44] It is defined as a clinically diverse autosomal recessive neuroectodermal disorder with brittle hair and low sulfur content of hair. This is caused by mutation of the regulatory gene involved in the transcription of DNA and involves all the body hairs. The sulfur and cysteine content of the hair is reduced by 50% in both the cuticle and the cortex. The characteristic hair shaft defect in trichothiodystrophy is a clean transverse fracture called trichoschisis. But it is a heterogeneous disorder which consists of more than hundred variable features. Under polarized light, 'tiger tail' pattern of alternating bright and dark diagonal bands are seen in most of the trichothiodystrophy individuals.[43]

f) Trichorrhexis Nodosa

Trichorrehexis nodosa is the most common hair shaft anomaly. It can be congenital or acquired. In congenital trichorrhexis nodosa, inherited as an autosomal dominant trait, the hair is usually normal at birth but is replaced with abnormal fragile hair within few months. The acquired trichorrhexis nodosa is a distinctive response of the hair shaft to

external injury. On light microscopy, there are nodular fractures and splaying of hair, producing an appearance suggestive of the ends of two brushes pushed together. This condition is commonly associated with other hair shaft disorders, particularly pili annulati. Trichorrhexis nodosa may rarely present as a result of systemic causes such as metabolic disorders or malnutrition.[44]

g) Woolly Hair

Woolly hairs are hair shaft anomalies that clinically present as tightly curled hair.[48] It usually occurs in non-African ancestry. Hairs are tightly curled, with an average curl diameter of 0.5 cm and can also contain wide twists over several millimeters along its own longitudinal axis. Hair shafts may be ovoid, flattened or irregular. It is associated with increased fragility, trichoschisis and pili annulati.[48] Woolly hair is differentiated into three types based on the clinical features and inheritance: The hereditary woolly hair, the familial woolly hair and the woolly hair nevus.[48]

- **Hereditary Woolly Hair:** It is inherited in an autosomal dominant pattern and affects the entire scalp. It is seen at birth or within the first few months of life. It is usually not associated with prominent hair loss. It usually occurs alone but has been reported with pili torti, pili annulati, ocular problems and keratosis pilaris.[48,49] There can be variation in hair colour. There is improvement in manageability of hair with age.[49]
- **Familial Woolly Hair:** It is inherited in an autosomal recessive pattern and is associated with hypotrichosis (thinning of the hair).[49] The hair does not grow beyond the length of a few centimeters and this may be secondary to a shortened anagen phase.[49] The hairs are thin, sparse, short and light colored.[48] The hair changes are noticeable at birth.[48] Dermatoscopy of the woolly hair shows hair shafts resembling a

crawling snake with short wave cycles. The hair shaft shows longitudinal twisting and fractures on light microscopy.[49]

- **Woolly Hair Nevus:** It is a rare, non-hereditary, focal, nevoid condition characterized by confined patch of unruly and tightly curled hair with an altered texture, associated with epidermal or melanocytic nevus. It equally affects both the sexes and can appear in childhood or adolescence. It gets coarser with age giving an unsightly appearance. It may be present in one or several areas of the scalp.[49]

h) Uncombable Hair (Pili trianguli et canaliculi)

It is characterized by light silvery-blond, dry and unruly hair. This condition presents in childhood but shows spontaneous improvement with age. Dermatoscopic examination is very useful in the diagnosis of uncombable hair. Triangular or reniform hair shaft with longitudinal grooving or flattening is seen on dermatoscopy. Scanning electron microscopy is considered as the gold standard for the diagnosis of uncombable hair.[50]

i) Pili Annulati

It is an autosomal dominant hair shaft disorder characterized by alternating light and dark bands in the hairs. On dermatoscopy, the light bands correspond to air-filled cavities within the cortex, whereas, on light microscopy the cavities appear as dark patches. This condition is more susceptible to weathering than normal hair and there are reports of association with alopecia areata.[51]

3.1.2 CONGENITAL LOCALIZED ALOPECIA

3.1.2.1 Neonatal Occipital Alopecia

There is development of well-defined patch of alopecia in the occipital area during the initial months of life. For a long time, it was speculated to be caused by scalp friction because of rubbing of the back of the head to the bedding. But now it has been proved to be caused by the entry of occipital hair into telogen phase.[52]

3.1.2.2 Birth Injury on Scalp

Temporary alopecia occurs in areas of scalp which may be damaged by instrumentation such as the forceps, vacuum extractor, scalp monitors and also over cephalohematomas.[52]

3.1.2.3 Temporal Triangular Alopecia

This is characterized by well-circumscribed triangular or lance-shaped area of non-scarring alopecia in the fronto-temporal area. The terminal hairs are replaced with vellus hairs in the affected area. In about 80% of the cases, it is unilateral. This condition is commonly seen in neonates and has been attributed to forceps trauma during birth. However, few cases of onset in early childhood and adulthood have been reported. On histopathologic examination, majority of the hair follicles have vellus hairs. There is no change in the number of follicles but a decrease in follicle size has been noted.[53]

3.1.2.4 Localized Alopecia Associated with Other Nevoid Conditions

a) **Congenital Melanocytic Nevus**

These nevi are usually associated with hypertrichosis but large nevi causing a cutis verticis gyrata appearance may have sparse hair or even complete absence of hair.[54]

b) Sebaceous Nevus of Jadassohn

These nevi seen at birth have no hair on the surface and are usually seen on the scalp, but may occur on forehead, face or neck. It may be very flat and delicate at birth that it may go unnoticed. It increases in size during adolescence.[54]

c) Aplastic Nevus

In this nevoid condition there is complete absence of the skin appendages in an area of otherwise normal skin.[54]

d) Aplasia Cutis

Alopecia is a feature of both the common form of aplasia cutis congenital and the membranous aplasia cutis. In the common form, irregularly shaped erosion that eventually heals with hypertrophic scarring is seen. An oval or round, smooth, hairless membrane surrounded by a collar of hair is seen in membranous cutis aplasia. Aplasia cutis producing a localized congenital alopecia may be a feature of various syndromes including Adams-Oliver syndrome and TorielloOcculo-ectodermal syndrome.[55]

e) Cranial Meningoceles, Encephaloceles and Heterotropic Meningeal or Brain Tissue

This condition is characterized by the presence of tumors or cysts which are either hairless or have sparse overlying hair. A surrounding collar of long hair that produces the 'hair collar sign' is often present.[55]

3.1.2.5 Localized Alopecia associated with Syndromes

a) Hallermann-Streiff Syndrome

In this condition, hair loss occurs typically in the frontal and parietal area over the cranial sutures in the early months of life, associated with atrophic facial skin, craniofacial anomalies and ocular abnormalities.[56]

b) X- Linked Dominant Conditions

Several rare syndromes caused by X-linked dominant genes interfere with the hair growth and form a mosaic pattern of alopecia in affected females. The homozygous males with these conditions rarely survive. Focal dermal hypoplasia (Goltz syndrome), incontinentia pigmenti, oro-facial digital syndrome and X-linked dominant chondrodysplasia punctata are a few examples. The hair loss seen in these conditions is often patchy, but may be linear or spiral at times when it follows the lines of Blaschko.[57]

c) Hypotrichosis with congenital forms of cutis verticis gyrata

Cutis verticis gyrata is characterised by marked folding of scalp skin covered by sparse hair. It may occur at birth with melanocytic nevi or may be associated with certain syndromes. In Turner's syndrome, it is believed that the redundant skin results from resolved intrauterine lymphedema. In Beare-Stevenson syndrome, it is due to a mutation in fibroblast growth factor receptor gene, and is associated with craniofacial anomalies. There is excessive production of various types of connective tissue in Michelin-tire baby syndrome, which causes generalized folding of the skin and marked hypotrichosis of the scalp. In Shar Pei dog syndrome, multiple folds of redundant skin occur all over the body due to an accumulation of hyaluronic acid. Several forms of cutis verticis gyrate develop later in life.[58]

3.2 ACQUIRED ALOPECIAS

3.2.1 NON-SCARRING ACQUIRED ALOPECIA

3.2.1.1 Alopecia Areata

Alopecia areata (AA)is a recurrent, non-scarring type of hair loss. This occurs most commonly in children and young adults, with 30–48% of the patients affected below 20 years of age. The risk of developing alopecia areata throughout life is 1.7% with a prevalence of 0.1%.[59]

It occurs in patchy, confluent or diffuse pattern. Sudden patchy hair loss is the most common presentation. Scalp is the most common site of involvement. The patches of alopecia are round or oval, well defined and appear smooth. In the diffuse type of alopecia areata, there is widespread sudden hair loss with 'overnight graying' of hair as it spares the gray hair.[59]

It is an autoimmune disorder with genetic basis and environmental triggers. It is associated with several genes that control the activation and proliferation of T cells. These include interleukin-2 (IL-2) gene, human leukocyte antigen and the Unique Long 16(UL16)-binding Protein (ULBP) that encodes activating ligands of natural killer cells. Hence, the immune system plays an important role in the pathogenesis of alopecia areata.[60]

On histopathology, the anagen hair follicles at margins of the expanding patches of alopecia areata characteristically show a perifollicular and intrafollicular inflammatory cell infiltrate. The infiltrate is composed mainly of activated T lymphocytes, with few macrophages and Langerhan's cells.[61]

Topical steroids, topical dithranol, topical minoxidil, intralesional steroids, contact immunotherapy, systemic steroids and photochemotherapy are a few of the treatment modalities available.[62]

3.2.1.2 Telogen Effluvium

Kligman coined the term telogen effluvium. It refers to abnormality in normal hair growth cycle where the hair follicles undergo premature termination of anagen, precipitating telogen.[63] It is a reaction pattern to various physical or mental stressors. Few of the common causes are:

- High fever[63]
- Hospitalization[63]
- Surgery under general anesthesia[63]
- Emotional stress[63]
- Crash diets[63]
- Thyroid disorders[63]
- Vaccinations[63]
- Drugs[63]

Patients with this condition experience a sudden increase in the shedding of hair diffusely over the scalp 3 to 4 months after the inciting event. For some patients, the duration of hair loss lasts for more than 6 months and this is referred to as chronic telogen effluvium. The anagen re-growth takes place over the next 6 to 12 months after the loss of telogen hair. If inciting events are not treated, the new anagen hairs growing are pushed into telogen and the shedding process will continue.[64]

The diagnosis can be made by hair fall count, hair pull test, trichogram, unit trichogram, scalp biopsy and dermatoscopy. Treatment of TE is primarily reassurance and counseling. The hair fall is expected to cease within 3–6 months.[63]

3.2.1.3 Androgenetic alopecia

Androgenetic Alopecia (AGA) is the most common type of hair loss in adults. AGA is characterized by stepwise miniaturization of the hair follicle due to alteration in the hair

cycle dynamics under the influence of dihydrotestosterone. Both testosterone and genetic predisposition are necessary for development of AGA in men. The age of onset is variable, but the first signs of AGA can be seen only after adrenarche.[65]

In AGA, there is a gradual decrease in duration of anagen phase and increase in that of telogen phase. As the duration of anagen phase determines the hair length, the hair becomes shorter in length than its precursor (miniaturization) causing a bald appearance.[65]

The essential feature in AGA in both sexes is patterned balding over the crown. Terminal hairs are progressively replaced by shorter hairs which may also appear less pigmented. In male pattern baldness, there is fronto-temporal recession with thinning at the vertex. Widening of the central parting that may resemble a 'Christmas tree' pattern is seen female pattern hair loss.[66]

Trichoscopy is used as an important tool in the diagnosis of androgenetic alopecia.[19] Hair diameter diversity is the characteristic feature on trichoscopy. Scalp biopsy is required rarely in the clinically atypical cases. Terminal to vellus hair ratio (T:V) less than four, on horizontal sectioning, is considered diagnostic.[67]

Topical minoxidil appears to be safe and effective in adolescents with AGA. Finasteride and other antiandrogens are usually reserved for older patients.[67]

3.2.1.4 Trichotillomania/Trichotillosis

Trichotillomania is classified as an impulse control disorder with a compulsion to pull own hair. This is a self-induced and recurrent type of hair loss. It is more common in children and even persists in adulthood. Multiple genes have been implicated in causing increased vulnerability to trichotillomania. There is mutations in SLITRK1 (SLIT and NTRK [neutrophictyrosinase kinase receptor type1]) gene and serotonin 2A receptor genes. There

is sudden onset of patchy, geometric pattern of hair loss with hairs of different lengths. It can involve scalp, upper eyelashes, eyebrows, and even pubic hair.[68]

3.2.1.5 Tractional Alopecia

It is caused by damage to the hair follicle by constant pulling or tension over the hair. It often occurs in children and adults who wear tight braids, ponytails or other tight hairstyles that cause tension, pulling and breakage of hair. Tractional alopecia is more common in children and teenagers of African descent population because of their hairstyling practices. Constant pulling or tension on the hair follicle damages the dermal papilla that is the source of new cells in the hair follicle for hair growth.[69]

In tractional alopecia, thinning of hair is noted along the hairline particularly the frontal and temporal areas. A sterile pustule is often seen around the pulled hair follicle because of the inflammation due to the tension. [70]

3.2.1.6 Tinea Capitis

Tinea capitis is a superficial fungal infection of the scalp, hair follicles and hair shaft, caused by dermatophytes. Tinea capitis is mainly caused by fungi of species of the genera *Trichophyton* and *Microsporum*. Direct inoculation causes the growth of fungal hyphae centrifugally in stratum corneum and into the hair follicle. The zone of involvement is visible above the skin surface by 12–14 days. Endothrix infections are characterized by arthroconidia (spores) within the hair shaft and there is no destruction of the cuticle. Whereas in ectothrix infections, the hyphae and arthroconidia are outside the hair shaft destroying the cuticle.[71]

Tinea capitis is a dermatophyte infection that mainly affects the children (3–7 years of age). The features include scale, some degree of redness and alopecia therefore has diffuse scale,

patch, black dot, diffuse pustular and kerion varieties. Tinea capitis with zoophilic fungi leads to intense inflammation. Both zoophilic and anthropophilic fungi lead to kerions that are swollen and sometimes purulent and boggy-appearing plaques.[72]

3.2.1.7 Loose Anagen Hair Syndrome

Loose Anagen Syndrome (LAS) is a hair disorder in which the hair is loosely anchored and can be painlessly pulled from the scalp. This hair disorder has been classically described in children, predominantly in blonde girls. Few cases have been reported in adults. [73]

LAS should be suspected in patients with sparse fine hair, often tangled hair at the back of the head, that can be painlessly and easily removed from the scalp.[73]

LAS have been categorized into 3 groups: LAS type A presents with sparse, short hair. LAS type B presents with curly, patchy and difficult to control hair. LAS type C is the adult type, where hair presents with normal thickness, but falls out excessively.[74]

3.2.1.8 Anagen effluvium:

The term anagen effluvium is considered misleading because the abnormal anagen hairs in this condition are usually broken off rather than shed. Anagen effluvium is categorized into two types: The common dystrophic anagen effluvium and the loose anagen syndrome.[75]

Anagen effluvium presents with abrupt shedding of much of or all of hair on the scalp and often the entire body including eyebrows, eyelashes and body hair.[76] It may leave the scalp partially or completely bald. The main cause of anagen effluvium is infection, drugs, toxins, radiation and autoimmune disease.[77]

Diagnosis of anagen effluvium is made by taking a careful history, particularly of recent medicines, and by examining the scalp and shed hair. In anagen effluvium, the hair end is

tapered, narrowed, irregular or broken off. Other tests may be arranged to rule out other causes of hair loss, including iron deficiency, thyroid disease, systemic lupus erythematosus and infections.[77]

Treatment for anagen effluvium includes topical minoxidil solution, scalp cooling during chemotherapy and cosmetic camouflage for eyebrows.[77]

3.2.2 SCARRING ACQUIRED ALOPECIA

3.2.2.1 Keratosis Pilaris Atrophicans

Keratosis Pilaris Atrophicans is a genetic skin condition. It develops when hair follicles get plugged by keratin debris, particularly on the face. It is a rare subtype of keratosis pilaris which is characterized by the small follicle oriented papules, usually on the arms and legs.[78]

Keratosis pilaris atrophicans is caused by abnormal keratinization of the follicular infundibulum, resulting in obstruction of the growing hair shaft and inflammation. Chronic inflammation leads to fibrosis, atrophy, shrinkage of the hair bulb and alopecia.[28]

Keratosis pilaris atrophicans presents with follicle oriented papules on the forehead and brow region, associated with erythema. The skin feels like sandpaper on palpation.[79]

The diagnosis of keratosis pilaris atrophicans is made by detailed history, dermatoscopy, wood's lamp examination, complete blood count , peripheral blood smear and biopsy.[80]

The treatment for keratosis pilaris atrophicans includes avoiding hot dry environment and use of emollients. The other common medications used include lactic acid, vitamin D and salicylic acid, topical retinoid and steroids.[80]

3.2.2.2 Folliculitis Decalvans

Folliculitis Decalvans (FD) is an uncommon condition characterized by neutrophilic inflammation of the hair follicles causing primary cicatricial alopecia. It typically manifests

as recurrent painful follicular pustules and exudate.[81] This leads to eventual destruction of the follicle and consequent permanent hair loss.[82]

Folliculitis Decalvans can cause an area of the scalp to become itchy and painful. The diagnosis is often made by examining the skin and performing skin biopsy. Treatment is usually a combination of anti- inflammatory and antibacterial shampoos, solutions or creams with oral antibiotics. Steroid cream/lotion/ointments are often used.[81]

3.2.2.3 Dissecting Cellulitis of Scalp

Dissecting cellulitis of the scalp is a rare inflammatory scalp condition. Pustules, nodules and abscesses develop with subsequent hair loss over the affected area. Hair loss is permanent due to the inflammation that destroys the hair follicles and leaves scar tissue. The causes of cellulitis of scalp are abnormal blockage of the hair follicles that leads to inflammation and secondary infection. Symptoms include painful pustules and abscesses with oozing pus.[82]

The condition is usually be diagnosed by examining the scalp and biopsy of scalp may be required in some cases. The topical treatment includes antiseptic washes and shampoos as well as topical antibiotics. Oral medications include long-term courses of oral antibiotics that are often used to treat this condition.[83]

3.2.2.4 Acne Keloidalis

Acne keloidalis is an uncommon form of folliculitis and cicatrical alopecia that affects the nape of the neck. Folliculitis keloidalis is also referred to as acne keloidalis nuchae or acne keloidalis. . It is a mechanical form of folliculitis due to constant injury during haircuts or use of razor. The ingrown hair shafts irritate the wall of the hair follicle resulting in inflammation, destruction of follicle and scarring.[84]

The treatment for acne keloidalis includes use of antimicrobial cleansers, topical or injectable steroids.[84]

3.2.2.5 Pseudopelade of Brocq

Pseudopelade of Brocq is a rare cause of permanent hair loss from the scalp. pseudopelade of Brocq is more of an atrophic condition rather than scarring.[81]

The signs and symptoms include:

- Alopecia
- Lichenification
- Sparse scalp hair
- Papules
- Recurrent skin infections
- Abnormality of the nail
- Aplasia/hypoplasia of the eyebrow
- Cheilitis[81]

Pseudopelade of Brocq is difficult to treat. Combination of topical corticosteroids and corticosteroids injected directly into the scalp lesions may be beneficial. Additional treatment options include oral steroids, hydroxychloroquine, isotretinoin and mycophenolate mofetil.[81]

3.2.2.6 Alopecia Mucinosa

Alopecia mucinosa is also referred to as follicular mucinosa, was first reported by Pinkus in 1957. The face, the neck and the scalp are the most frequently affected sites, although lesions may appear on any part of the body.[82]

Bald patches with prominent hair follicles characterize alopecia mucinosa. Alopecia mucinosa is classified into:[82]

- A primary and acute disorder occurring in children and adolescents (Pinkus type)
- A primary and chronic disorder occurring in people older than 40 years
- A secondary disorder associated with benign or malignant skin disease
- Urticaria-like follicular mucinosis (rare)[83]

Usually primary and acute alopecia mucinosa occurring in children resolves spontaneously. The treatment may include topical, intralesional and systemic corticosteroids, oral antibiotics, dapsone, indomethacin, interferons, photochemotherapy, radiation therapy.[81]

3.2.2.7 Lichen Planopilaris

Lichen planopilaris is a type of scarring hair loss that occurs with a relatively common skin disease, known as lichen planus. Lichen planus is an autoimmune disorder in which white blood cells attack and destroy skin cells expressing unknown antigens. Triggering factors may include pharmacologic agents, contact sensitizers or infectious agents. [84]

Lichen planopilaris typically causes an intensely itchy scalp. The vertex is most commonly affected and symptoms of pain, burning and scalp tenderness may occasionally be present. Gradually, areas of hair loss may be noticed. Lichen planus can also affect the skin, mouth, genitals and nails.[84]

Treatments of lichen planopilaris includes topical corticosteroids, steroid injections, topical calcineurin inhibitor creams and ointments.[84]

3.2.2.8 Discoid Lupus Erythematous

Discoid Lupus Erythematosus (DLE) is a chronic inflammatory and scarring skin condition favoring the photo-exposed areas. The lesions are commonly seen on face, ears and scalp, but may involve the other body areas also.[85]

Discoid lupus commonly presents with erythematous, scaly papules and plaques occurring typically on sun-exposed areas. But 50% of discoid lupus lesions are found on areas of hair bearing scalp that are protected from the sun. Discoid lupus can occur at all ages and among all ethnic groups. It occurs more frequently in women than in men.[86]

DLE is a scarring autoimmune disease. The treatment of DLE may include topical corticosteroids, intralesional steroids, antimalarials. Other drugs used for the treatment of DLE are methotrexate, cyclosporinA, tacrolimus, azathioprine.[86]

3.2.2.9 Morphea

Morphea or localized scleroderma is a skin condition characterized by excessive collagen deposition in dermis, subcutaneous tissues or both leading to thickening of skin.[87]

Morphea is a skin condition that causes an oval patch or multiple patches of discoloured and thickened skin on the face, neck, hands, torso or feet. The patch is initially erythematous with outer lilac edge. The centre of the patch gradually becomes white or yellow in color.[87]

Complications of morphea are seen in deep lesions, lesions on the face or neck, or widespread lesions. The complications of morphea are:

• Restricted joint mobility

• Joint pain

• Cosmetic deformities

• Permanent eye damage in children

• Hair loss[87]

The treatment is based on the type of morphea. Majority of patients are managed with topical medications and phototherapy. Systemic therapy with methotrexate and/or glucocorticoids is needed for only those with active and deep morphea.[87]

MATERIALS & METHODS

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The present study was a hospital based descriptive study carried out at R.L. Jalappa Hospital and Research Centre, Tamaka, Kolar, from January 2016 to June 2017. All patients below 18 years of age, presenting with hair loss to the outpatient department of Dermatology, Venereology and Leprosy at the above mentioned hospital were included in the study.

CRITERIA FOR SELECTION

Inclusion criteria:

- All patients below 18 years of age with complaint of hair loss.

Exclusion criteria:

- All cases of neonatal telogen effluvium (physiological).

Methods of data collection:

All children presenting to R L Jalappa hospital with hair loss and fulfilling the inclusion criteria were included in the study.

A written informed consent was taken from the patients parents or guardian.

A detailed history of the patient including name, age, sex, history of presenting illness, hair grooming pattern, habits & tics, nail changes, other skin changes, systemic disease, birth history, developmental history, family history of similar complaints and drug intake was taken.

General physical examination, scalp examination, examination of hair shaft and root, tests for hair anchorage and fragility was done in all cases. In addition, wood's lamp examination, microscopic examination of hair and dermatoscopy were done when indicated. Relevant laboratory investigations were done.

Statistical analysis:

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test** was used as test of significance for qualitative data. Continuous data was represented as mean and standard deviation.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram and Pie diagram.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

RESULTS

OBSERVATION AND RESULTS

Table 2: Age distribution of patients in the study

		Count	%
Age	Neonates (<1 month)	1	1.3%
	Infants (1month – 1 year)	7	9.3%
	Toddlers (1– 3 years)	7	9.3%
	Preschool (3 – 6 yrs)	16	21.3%
	School Going (6-10 yrs)	21	28.0%
	Adolescents (10 – 18 yrs)	23	30.7%

In the study majority of subjects were adolescents (30.7%) followed by 28% of school going children, 21.3% of preschool children, 9.3% of toddlers, 9.3 % of infants and 1.3% of neonates.

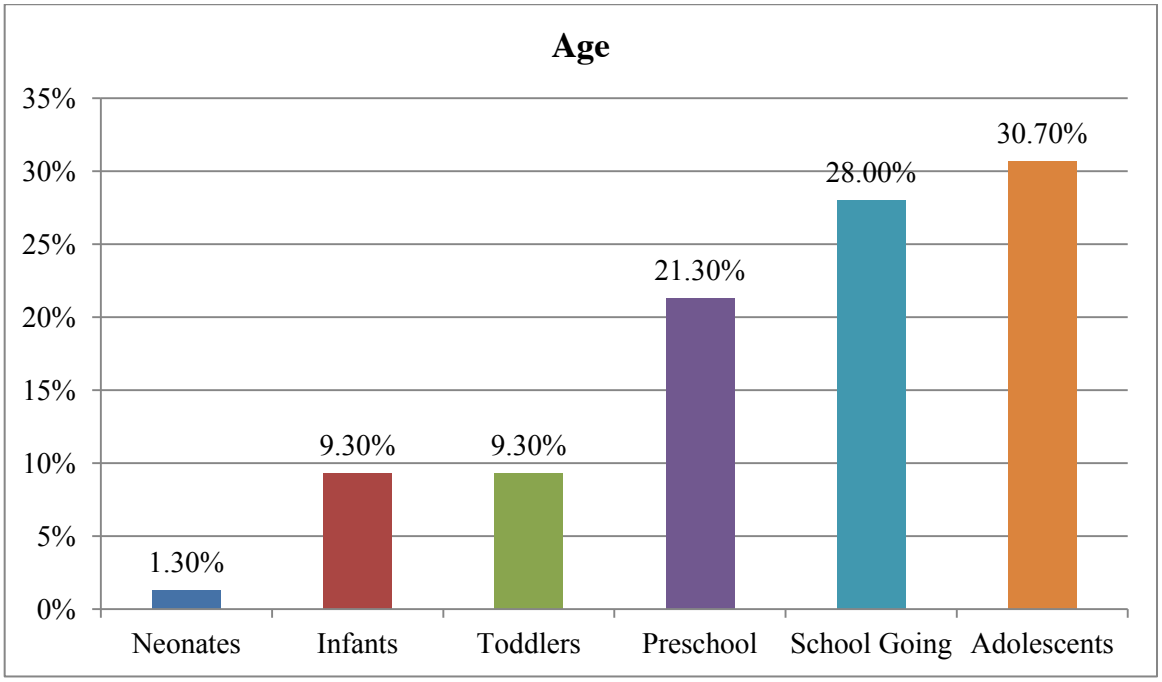

Figure 5: Bar diagram showing age distribution of patients in the study

Table 3: Gender-wise distribution of patients in the study

		Count	%
Gender	Female	38	50.7%
	Male	37	49.3%

In the study, 50.7% were females and 49.3% were males.

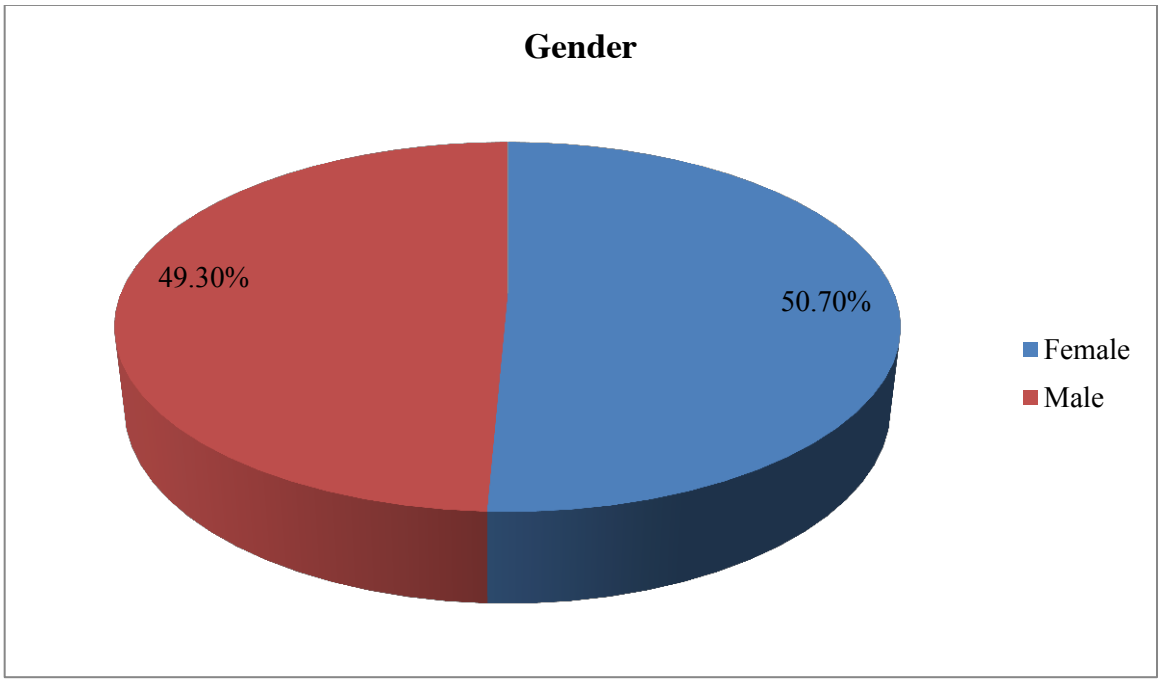

Figure 6: Pie diagram showing Gender-wise distribution of patients in the study

Table 4: Pattern of Hair Loss among patients in the study

		Count	%
Pattern of Hair Loss	Diffuse	15	20.0%
	Localized	60	80.0%

In the study 80% had localized hair loss and 20% had diffuse hair loss.

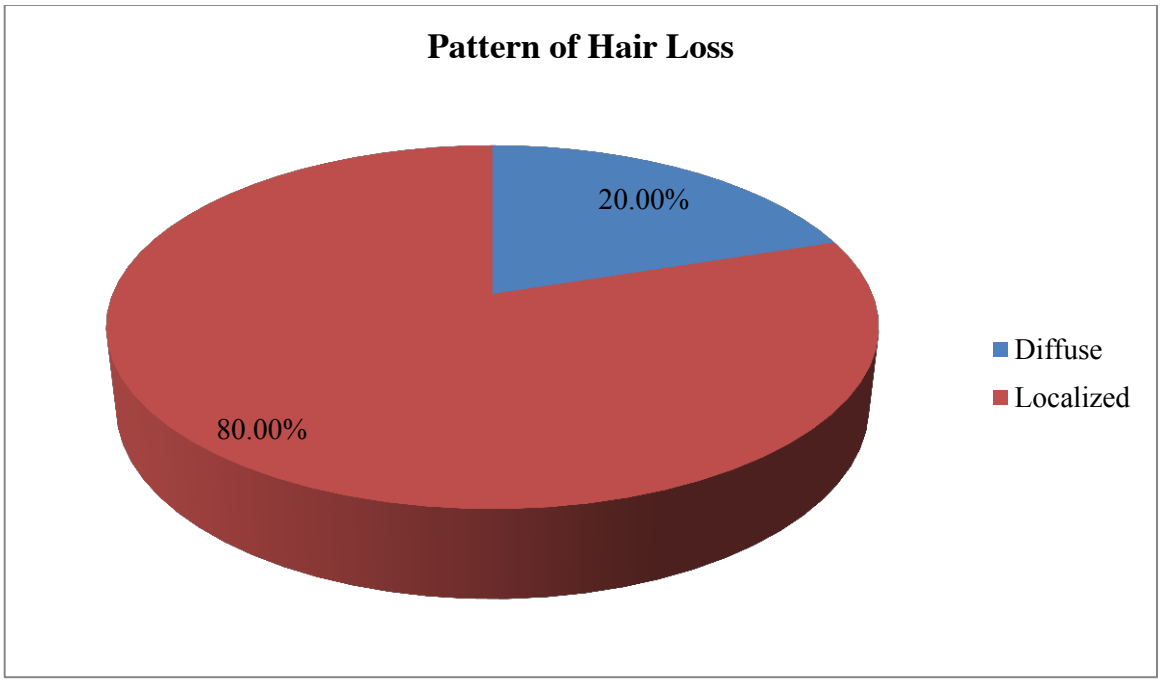

Figure 7: Pie diagram showing pattern of hair loss distribution among patients in the study

Table 5: Duration of Hair Loss among patients

		Count	%
Duration of Hair loss	<1 Month	46	61.3%
	1 to 3 months	16	21.3%
	4 to 6 months	4	5.3%
	6 to 12 months	5	6.7%
	> 12 months	4	5.3%

In the study majority had hair loss of duration of less than a month (61.3%). 21.3% had hair loss of duration 1 to 3 months, 5.3% had duration of 4 to 6 months, 6.7% had duration of 6 to 12 months and 5.3% had duration of more than 12 months.

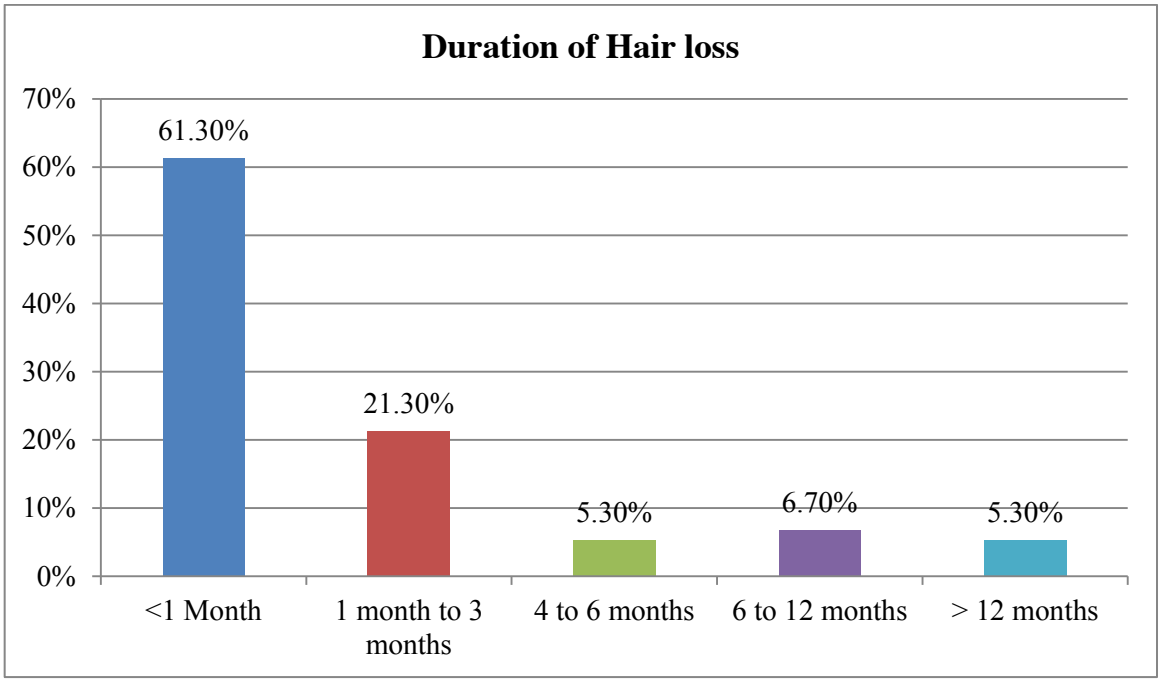

Figure 8: Bar diagram showing duration of hair loss among patients

Table 6: Associated symptoms among patients

In the study 76.6% of patients presented with asymptomatic hair loss. 20% had itching, 9.3% had scaling and 4% had pain and swelling respectively as associated symptoms.

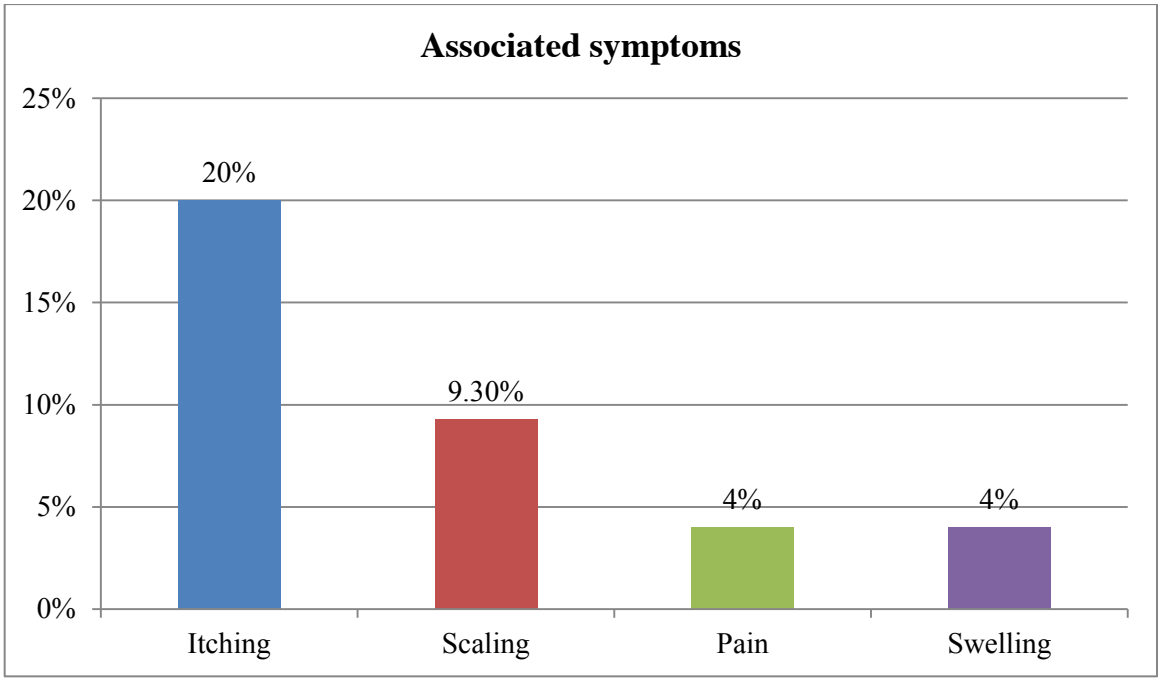

Figure 9: Bar diagram showing associated symptoms among patients

Table 7: Mode of onset of hair loss

		Count	%
Mode of onset of Lesion	Acquired	67	89.3%
	Congenital	8	10.7%

In the study, 10.7% had congenital hair loss and in 89.3% had acquired hair loss.

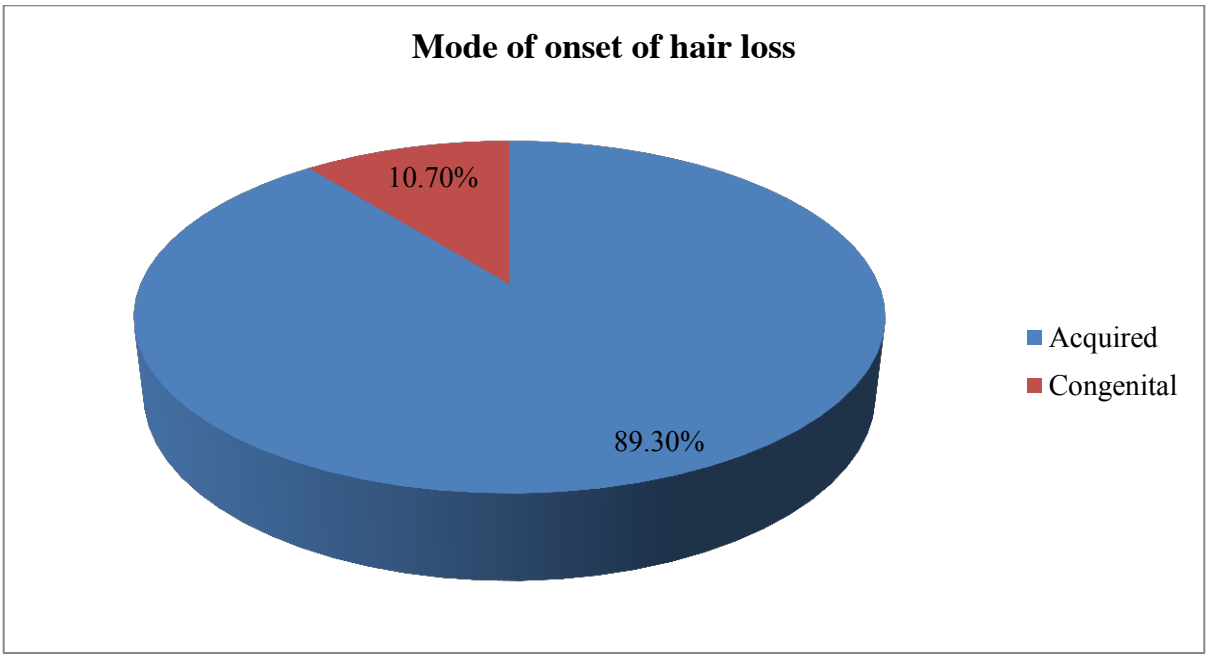

Figure 10: Pie diagram showing mode of onset of hair loss among patients

Table 8: Type of acquired hair loss

		Count	%
Type of Lesions	Non scarring	63	94.1%
	Scarring	4	5.9%

In the study, 94.1% of the acquired hair loss were non-scarring and 5.9% were scarring.

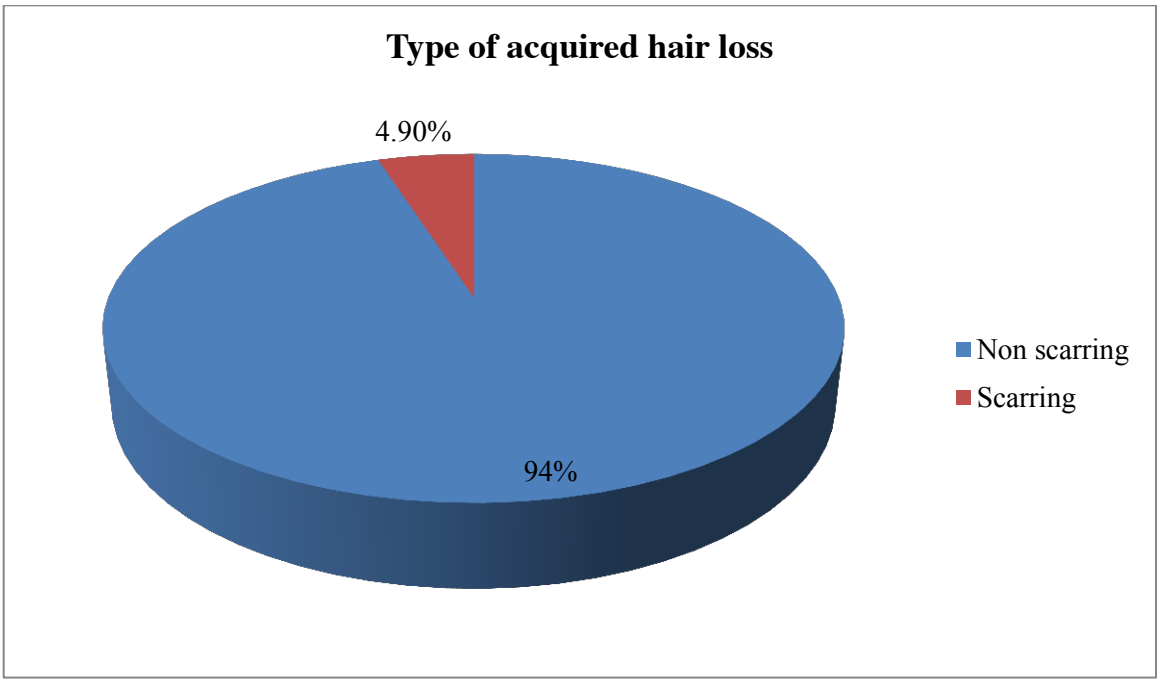

Figure 11: Bar diagram showing type of acquired hair loss

Table 9: Nail changes among patients

			Count	%
Nail changes	Absent		58	77.3%
	Present	Leuconychia	14	18.7%
		Long Ridging	1	1.3%
		Koilonychia	1	1.3%
		Pitting	1	1.3%

In the study 18.7% had leuconychia, 1.3% had koilonychia, 1.3% had longitudinal ridging and 1.3% had pitting respectively.

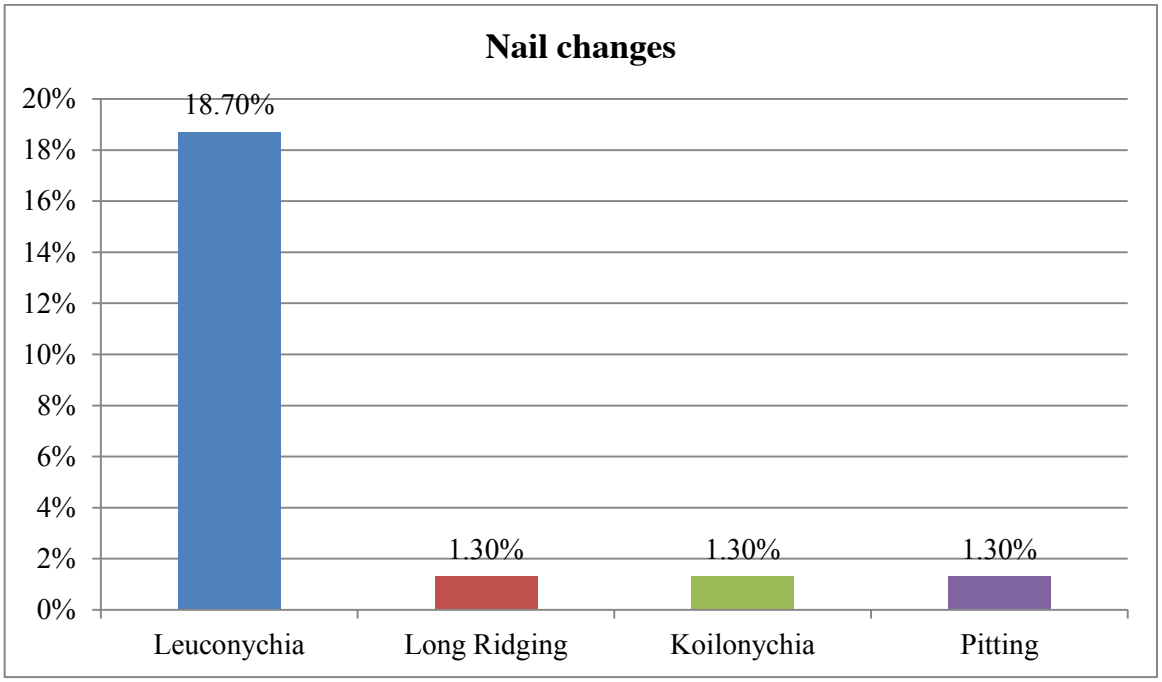

Figure 12: Bar diagram showing Nail changes among patients

Table 10: Other skin changes among children with hair loss

Out of the 75 children, 3 patients had other associated skin changes as follows:

			Count	%
Other Skin Changes	Absent		72	96.0%
	Present	Tinea Faciei	1	1.3%
		Vitiligo	1	1.3%
		Vitiligo + LP	1	1.3%

In the study 1.3% had Tinea Faciei, Vitiligo and Vitiligo with Lichen Planus respectively.

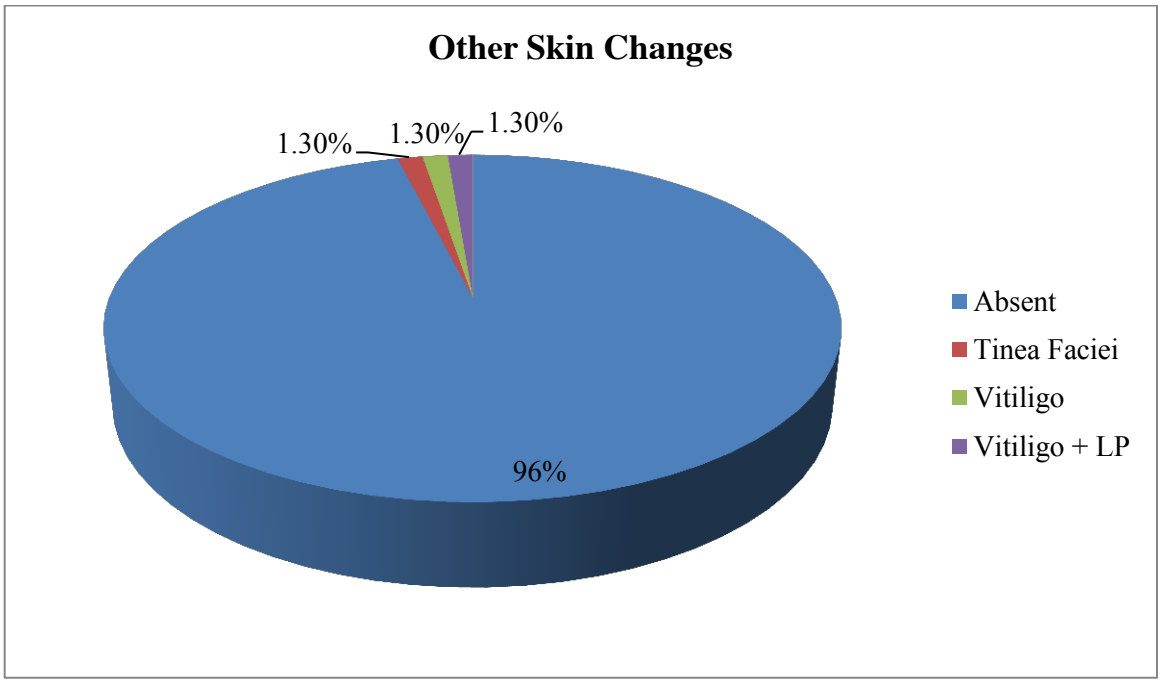

Figure 13: Pie diagram showing other skin changes among patients

Table 11: Associated systemic disorders among children with hair loss

Out of 75 patients with hair loss, there were 7 patients with associated systemic disorders

			Count	%
Associated systemic disorders	Absent		68	90.7%
	Present	Anaemia	2	2.7%
		Hypothyroidism	3	4.0%
		Depression	1	1.3%
		PCOS	1	1.3%

In the study 4% had hypothyroidism, 2.7% had anemia, 1.3% had depression and PCOS respectively.

Table 12: Malnutrition among patients

Malnutrition was calculated for all children less than 10 years of age using the WHO weight for age charts. Out of the 54 children less than 10 years of age in the study, malnutrition was noted in 8 children.

		Count	%
Malnutrition	Normal	46	61.3%
	Grade 1	7	9.3%
	Grade 2	1	1.3%

In the study, 9.3% had grade 1 and 1.3% had grade 2 malnutrition.

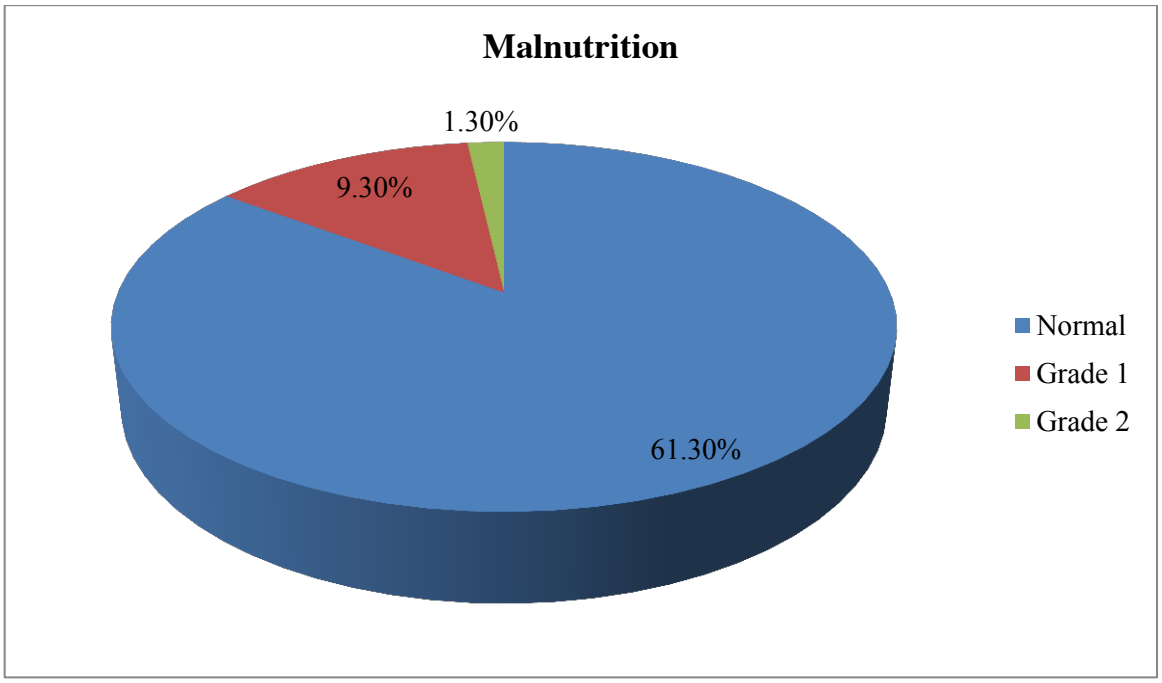

Figure 14: Pie diagram showing Malnutrition among patients.

Table 13: Other relevant history among patients

		Count	%
Past H/O Hair Loss	Absent	70	93.3%
	Present	5	6.7%
Family H/O Hair Loss	Absent	70	93.3%
	Present	5	6.7%
H/O Atopy	Absent	64	85.3%
	Present	11	14.7%

In the study 6.7% had past history of hair loss and family history of hair loss respectively.

14.7% had history of atopy.

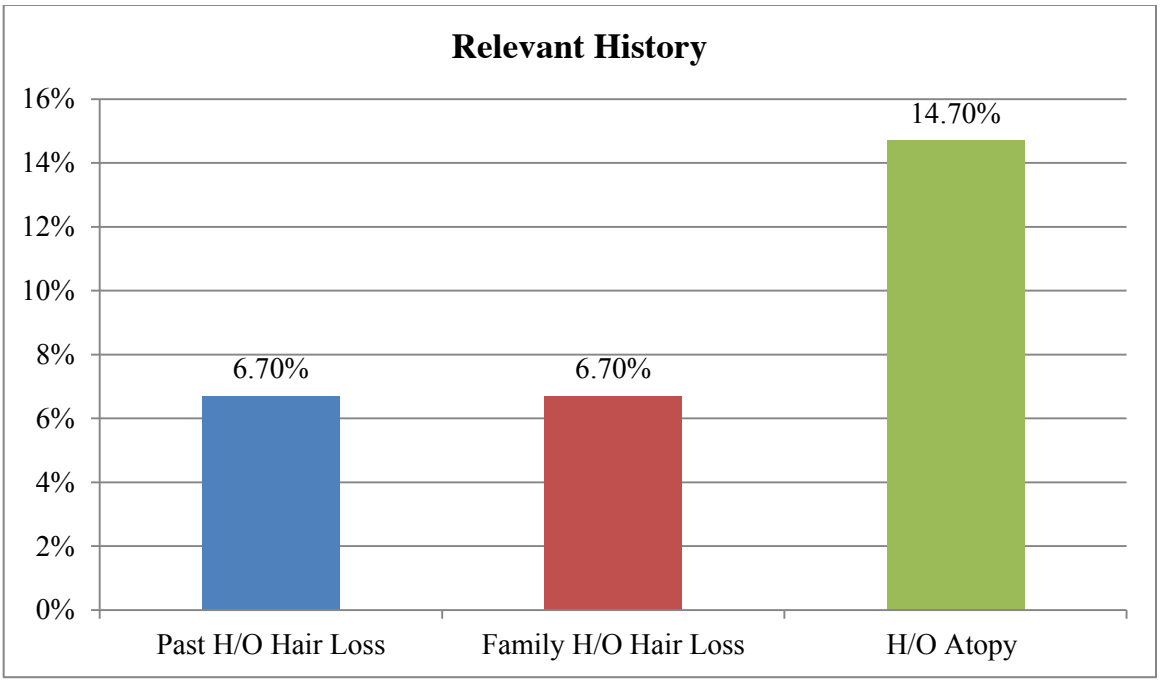

Figure 15: Bar diagram showing Relevant History among patients

Table 14: Event Preceding Hair Loss among patients

			Count	%
Event Preceding Hair Loss (Precipitating factors)	Absent		57	76.0%
	Present	Psychological Stress	6	8.0%
		Tonsuring	6	8.0%
		Fever	4	5.3%
		Surgery	2	2.7%

In the study, 8% had history of Psychological Stress, 8% had tonsuring, 5.3% had fever and 2.7% had undergone surgery preceding the hair loss.

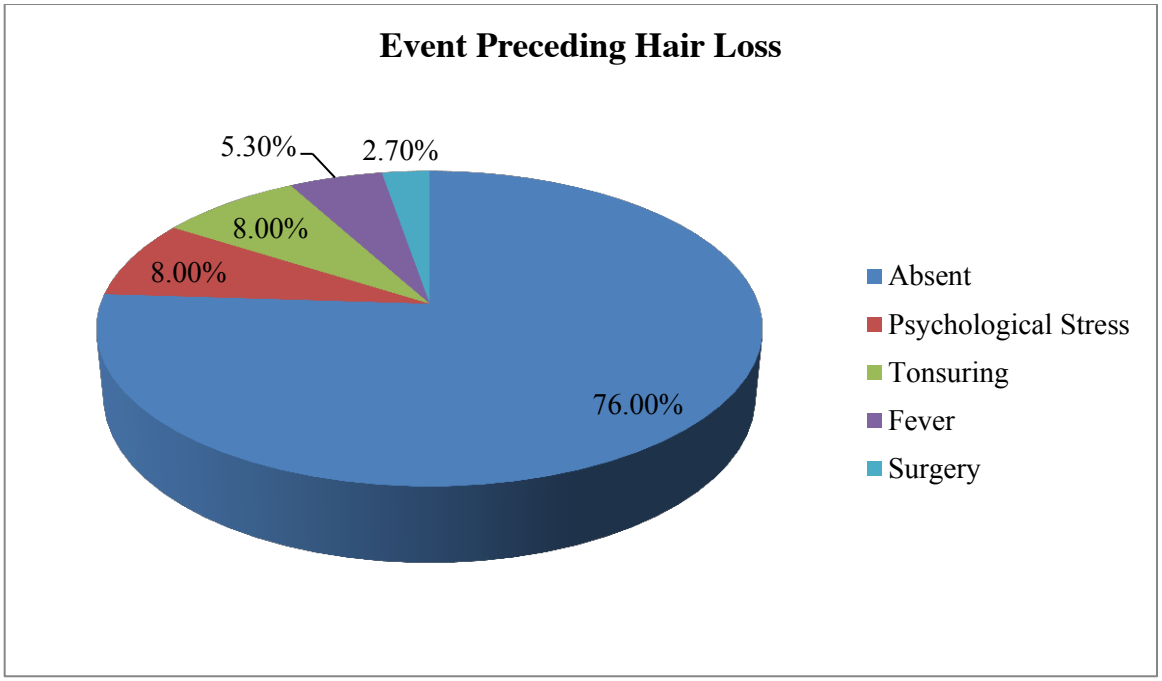

Figure 16: Pie diagram showing event preceding hair loss among patients

Table 15: Poor Grooming Practices among patients

Poor grooming practices like infrequent washing of hair, sharing of combs and towels, using hot styling tools and combing of wet hair were seen in 18 of the 75 cases.

		Count	%
Poor Grooming Practices	Absent	57	76.0%
	Present	18	24.0%

In the study, 24% had poor grooming practices.

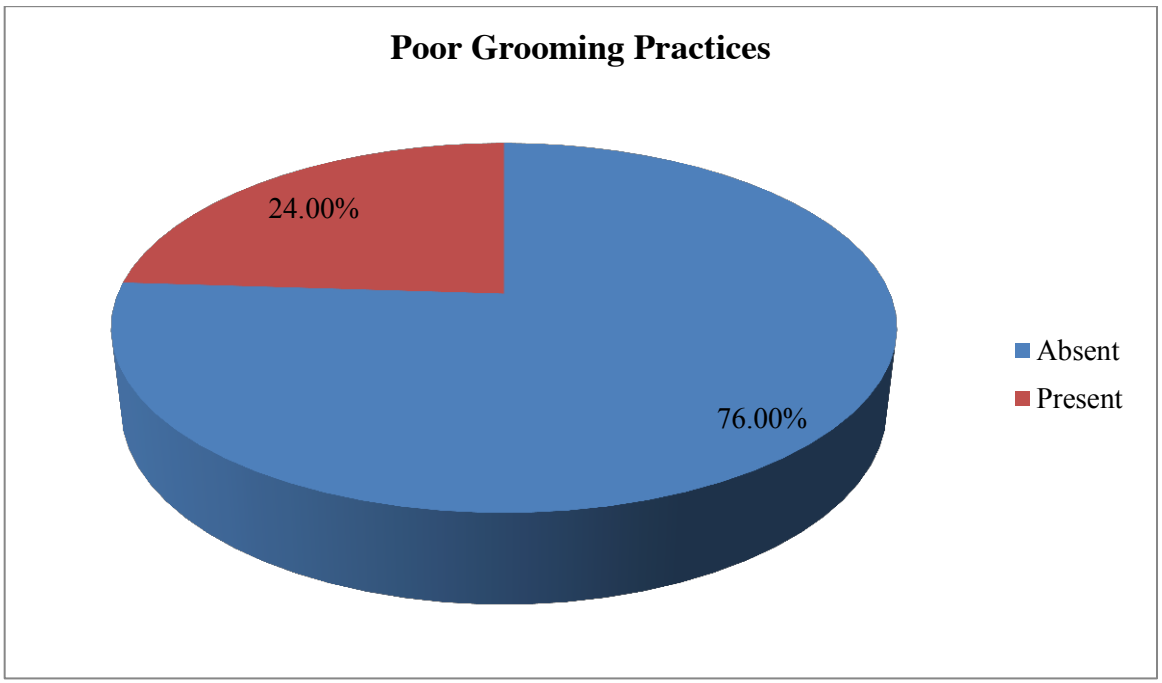

Figure 17: Pie diagram showing Poor Grooming Practices among patients

Table 16: Final diagnosis among patients

			Count	%
Final Diagnosis	Congenital	Nevus Sebaceous	4	5.3%
		Keratosis Follicularis Spinulosa Decalvans	1	1.3%
		X Linked Ichthyosis	1	1.3%
		Aplasia Cutis	1	1.3%
		Dariers Disease	1	1.3%
	Acquired Non Scarring	Tinea Capitis	23	30.6%
		Alopecia Areata	19	25.3%
		Telogen Effluvium	7	9.3%
		Tractional Alopecia	5	6.7%
		Seborrheic Dermatitis	4	5.3%
		Trichotillomania	3	4.0%
		Scalp Candidiasis	1	1.3%
		Androgenetic Alopecia	1	1.3%
	Acquired Scarring	Following Infections	3	4.0%
		Discoid Lupus Erythematosus	1	1.3%
		TOTAL	75	100%

In the study, the most common diagnosis was Tinea Capitis in 29.3%, followed by Alopecia Areata in 24%, Telogen Effluvium in 9.3%, Tractional Alopecia in 6.7% and others as shown in the above table.

Table 17: Profile and findings among Tinea capitis cases.

Out of 75 patients in the study , 23 patients had tinea capitis making it the most common cause for hair loss in children in this study.

		Count	%
Age	< 1 month	0	0%
	1 month to 1 year	1	4.3%
	1 year to 3 years	4	17.4%
	3 to 6 years	5	21.7%
	6 to 10 years	10	43.5%
	10 to 18 years	3	13.1%
Gender	Female	7	27.3%
	Male	16	72.7%
Duration of Hair Loss	<1 Month	18	78.3%
	1 month to 3 months	5	21.7%
	4 to 6 months	0	0.0%
	6 to 12 months	0	0.0%
	> 12 months	0	0.0%
Associated Symptoms	Itching	9	39.1%
	Scaling	2	8.7%
	Pain	2	8.7%
	Swelling	3	13.0%
Type of Lesion	Inflammatory	5	21.8%
	Non Inflammatory	18	78.2%
Clinical Type	Black Dot	4	17.4%
	Grey Patch	12	52.2%
	Kerion	5	21.7%
	Mixed	2	8.7%
Other Sites Tinea	Absent	22	95.7%
	Tinea Faciei	1	4.3%
Family H/O Tinea	No	21	91.4%

	Yes	2	8.7%
Event Preceding	No	17	73.9%
	Tonsuring	6	26.1%
H/O Oil Application	No	12	52.2%
	Yes	11	47.8%
Poor Grooming Practices	Present	9	39.3%
	Absent	14	60.7%

Majority of subjects with tinea capitis were in the age group of 6 to 10 years (43.5%). Males were more affected (72.7%). The patients commonly presented within duration of hair loss of less than 1 month (78.3%). Itching was an associated symptom in 39.1%.

78.2% of the tinea capitis were non inflammatory while 21.8% were inflammatory. Grey patch of tinea capitis (52.2%) was the most common type followed kerion (21.7%), black dot (17.4%) and mixed clinical type (8.7%) . 1 patient had associated tinea faciei. Family history of tinea was seen in 8.7% of the cases. 26.1% had preceding history of tonsuring. 52.2% gave history of oil application and 39.3% gave history of poor grooming practices.

Table 18: Profile and findings among Alopecia areata cases

A total of 19 patients had alopecia areata making it the second most common cause for hair loss in the study.

		Count	%
Age	< 1 month	0	0%
	1 month to 1 year	0	0%
	1 year to 3 years	2	10.5%
	3 to 6 years	3	15.8%
	6 to 10 years	3	15.8%
	10 to 18 years	11	47.8%
Gender	Female	10	52.6%
	Male	9	47.4%
Duration Of Hair Loss	<1 Month	14	73.6%
	1 Month To 3 Months	5	22.4%
	4 To 6 Months	0	0.0%
	6 To 12 Months	0	0.0%
	> 12 Months	0	0.0%
Clinical Pattern	Ophiasis	2	10.5%
	Patchy	16	84.2%
	Reticulate	1	5.3%
Grade	Mild	13	68.4%
	Moderate	4	21.1%
	Ophiasis	2	10.5%
Other Hair Bearing Sites	Eyebrows	1	5.3%
	No	18	94.7%
Nail Changes	No	14	73.6%
	Long Ridging	1	5.3%
	Leuconychia	3	15.8%
	Pitting	1	5.3%
Other Skin lesion	No	18	94.7%
	Vitiligo + LP	1	5.3%

Associated systemic diseases	Absent	17	89.5%
	Hypothyroidism	2	10.5%
Preceding Event	Absent	16	84.2%
	Fever	1	5.3%
	Psychological Stress	2	10.5%
H/O Atopy	No	14	73.7%
	Yes	5	26.3%
Family History	No	18	94.7%
	Yes	1	5.3%

Majority of subjects with alopecia areata were in the adolescent age group (47.8%). Females were slightly more affected (52.6%). The duration of hair loss was <1 month in 73.6%.

The clinical patterns observed were patchy pattern in 84.2%, ophiasis in 10.5% and reticulate pattern in 5.3% . 68.4% had mild, 21.1% had moderate and 10.5% had ophiasis clinical grade. 5.3% had involvement of eyebrows as well. The nail changes were leuconychia in 15.8 %, longitudinal ridging in 5.3% and pitting in 5.3% respectively. 1 case of alopecia areata had associated vitiligo and lichen planus. Hypothyroidism was noted in 2 cases. 10.5% gave history of psychological stress and 5.3% gave history of fever prior to onset of hair loss. History of atopy was seen in 26.3% and 5.3% had family history of alopecia areata. .

Table 19: Profile and findings among Telogen Effluvium cases

A total of 7 cases of telogen effluvium were seen, third most common cause for hair loss in the study.

		Count	%
Age	< 1 month	0	0%
	1 month to 1 year	0	0%
	1 year to 3 years	0	0%
	3 to 6 years	1	14.3%
	6 to 10 years	1	14.3%
	10 to 18 years	5	71.4%
Gender	Female	7	100.0%
Duration of Hair Loss	<1 Month	1	14.3%
	1 Month To 3 Months	2	28.6%
	4 To 6 Months	3	42.9%
	6 To 12 Months	1	14.3%
	> 12 Months	0	0.0%
Type	Acute	4	57.1%
	Chronic	3	42.9%
Nail Changes	Leuconychia	4	57.1%
	No	3	42.9%
Associated systemic diseases	Anaemia	1	14.3%
	Hypothyroidism	1	14.3%
	No	4	57.1%
	PCOS	1	14.3%
Preceding Event	Absent	1	14.3%
	Fever	2	28.6%
	Psych Stress	2	28.6%
	Surgery	2	28.6%
PEM	Grade 1	1	14.3%

Majority of subjects with Telogen Effluvium were in the adolescent age group (71.4%). 100% of the cases were females. 57.1% had acute telogen effluvium and 42.9% had chronic telogen effluvium. 57.1% had leuconychia of nails. 14.3% had anemia, hypothyroidism and PCOS respectively as associated systemic features. 28.6% had fever, psychosocial stress and surgery as preceding event respectively. 14.3% of the cases had grade 1 PEM.

PHOTOGRAPHS

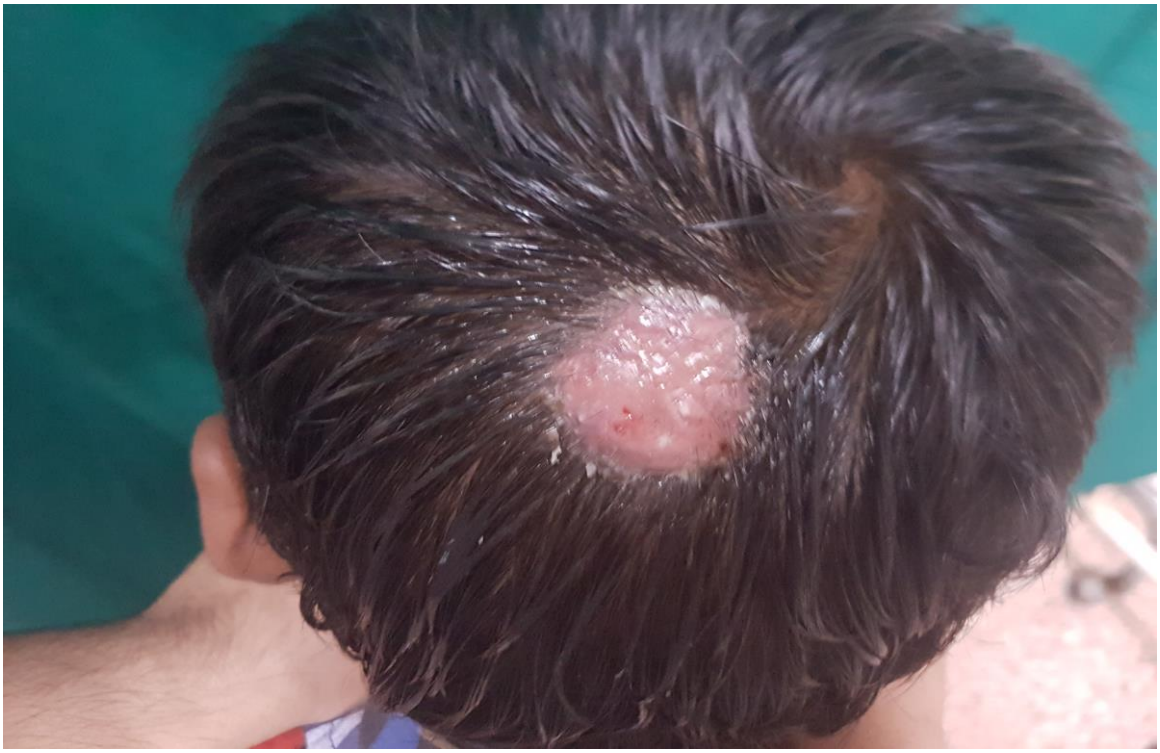

Figure 18: Tinea capitis, kerion type.

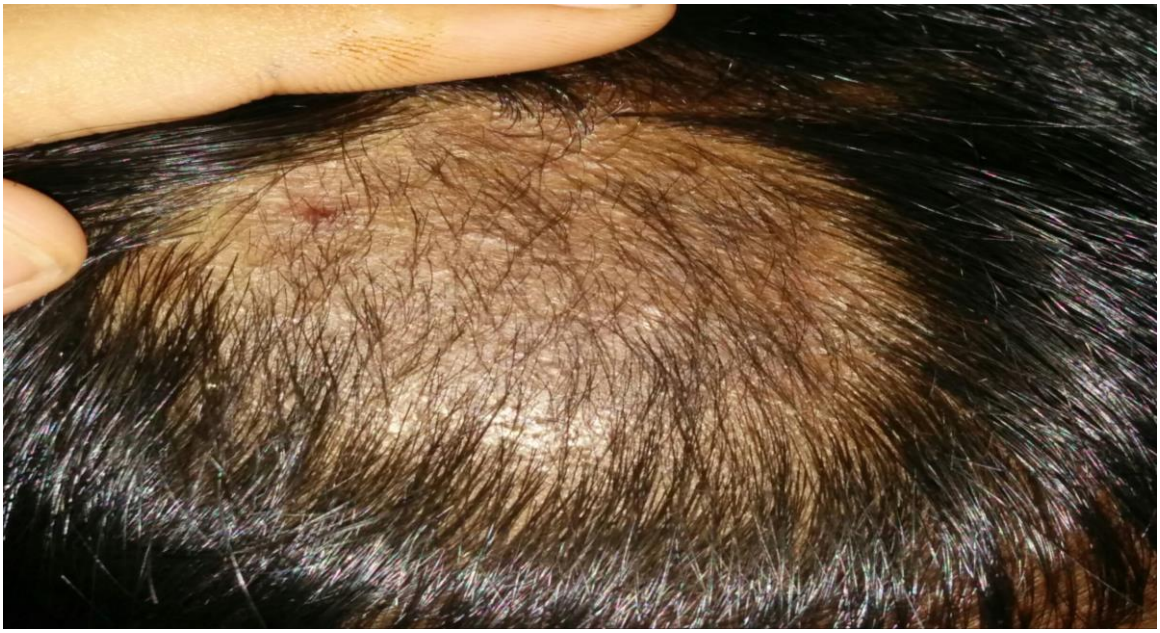

Figure 19 : Tinea capitis, grey patch type.

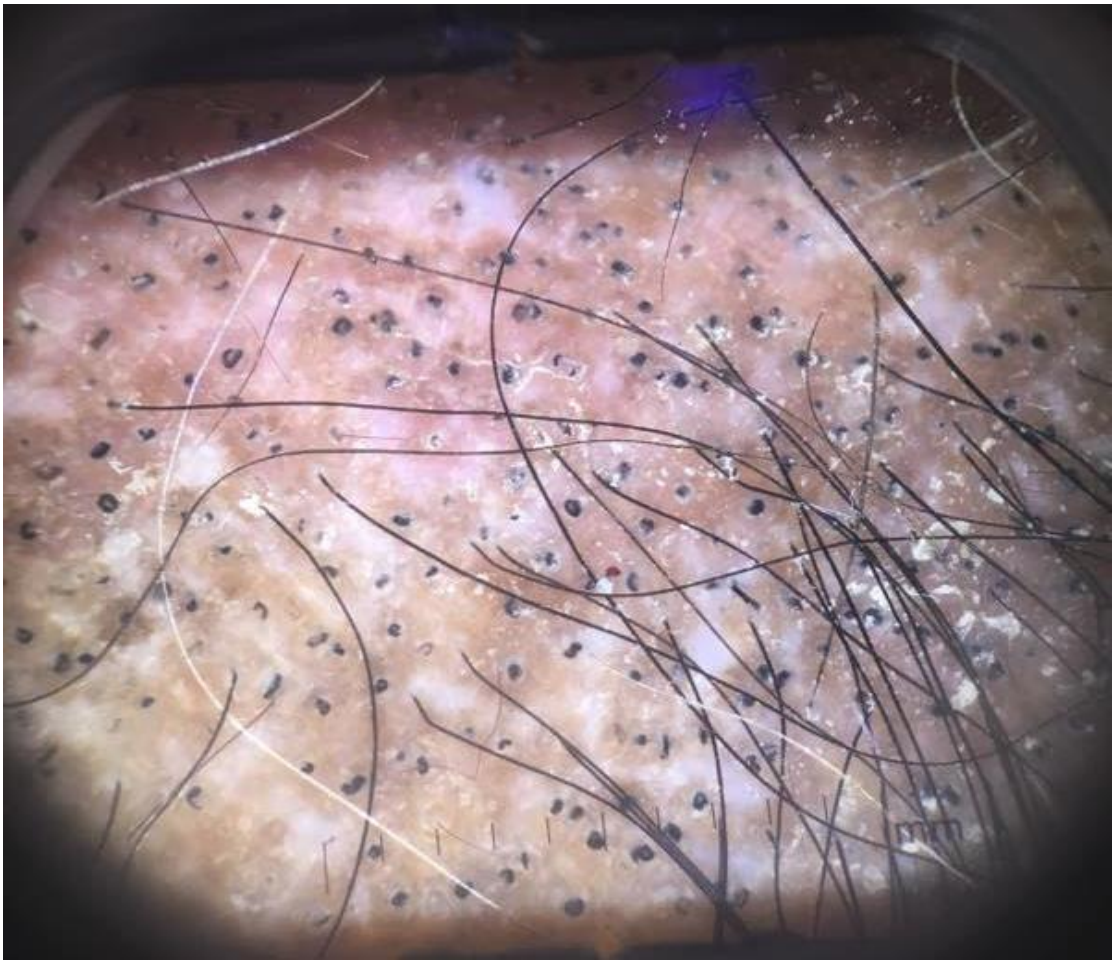

Figure 20: Dermascopic image of tinea capitis showing black dots, broken hair comma shaped hair and few areas of scarring.

Figure 21: Alopecia areata, patchy type.

Figure 22: Dermascopic picture of alopecia areata showing exclamation hair and pigtail hair.

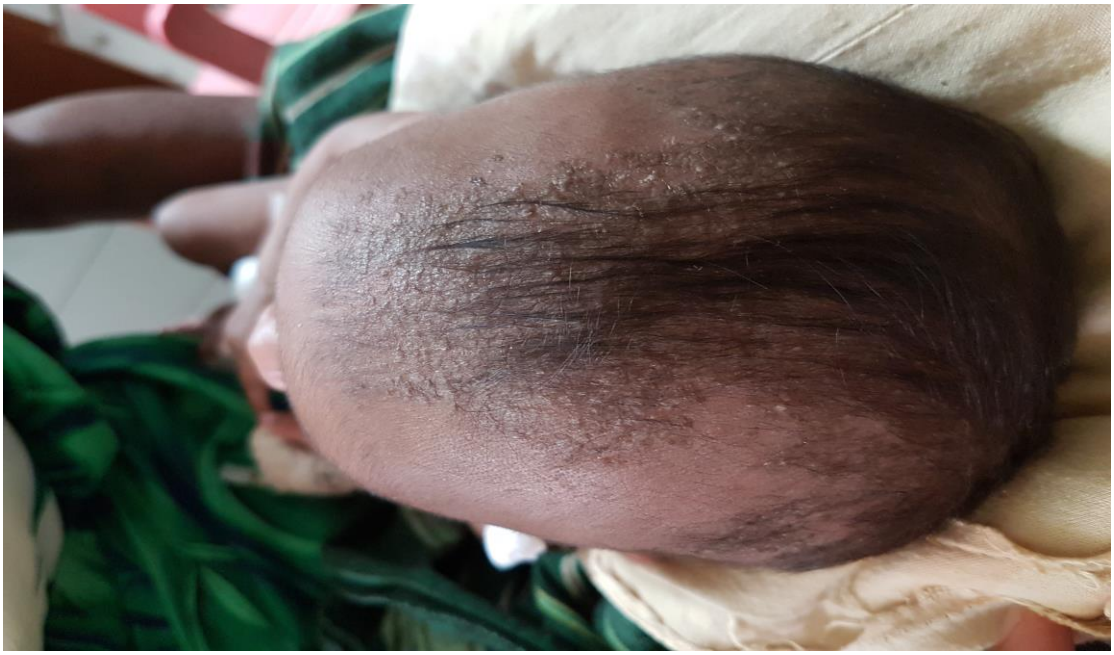

Figure 23: Seborrheic dermatitis

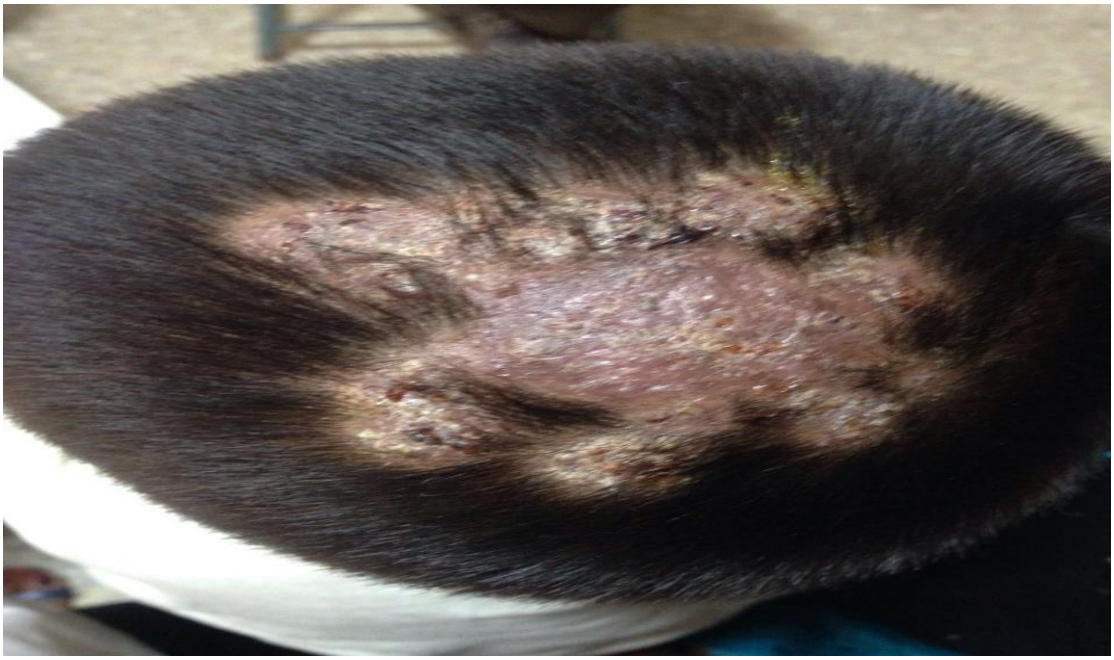

Figure 24: Scalp candidiasis

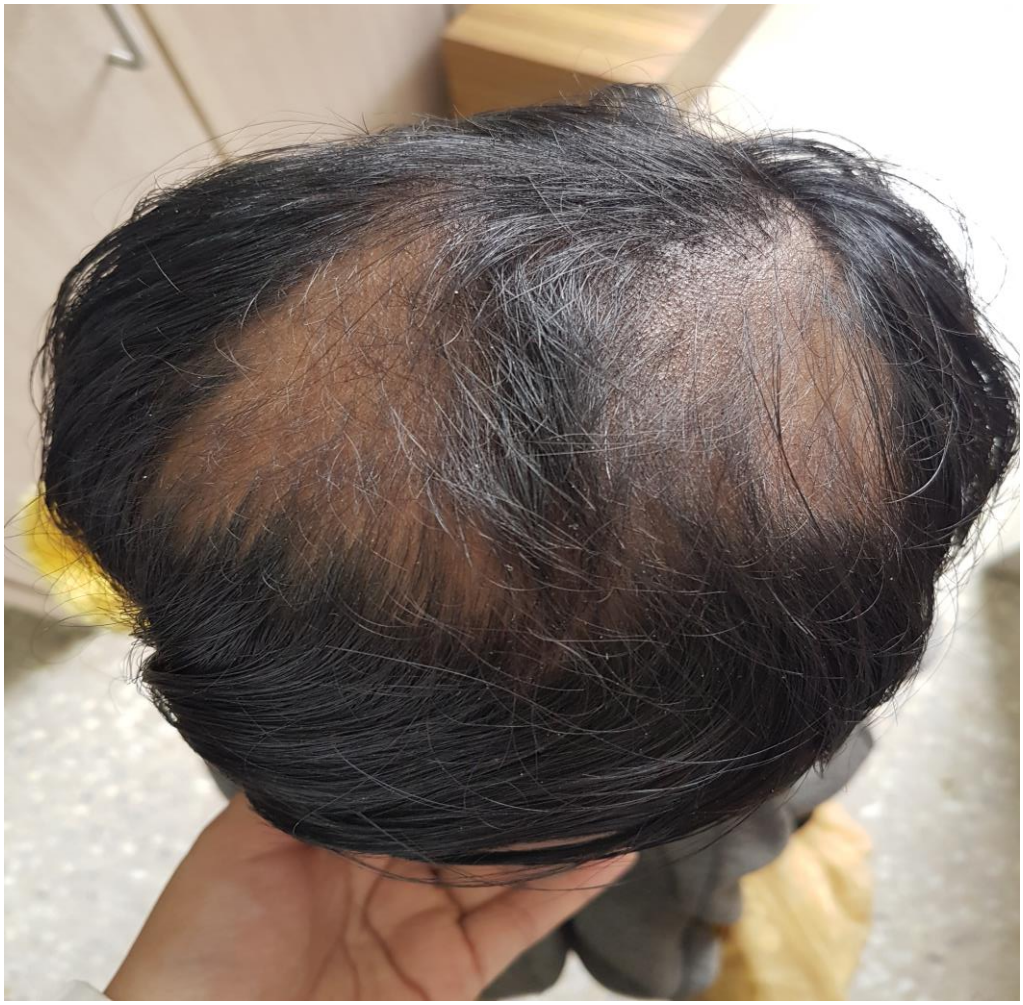

Figure 25: Trichotillomania

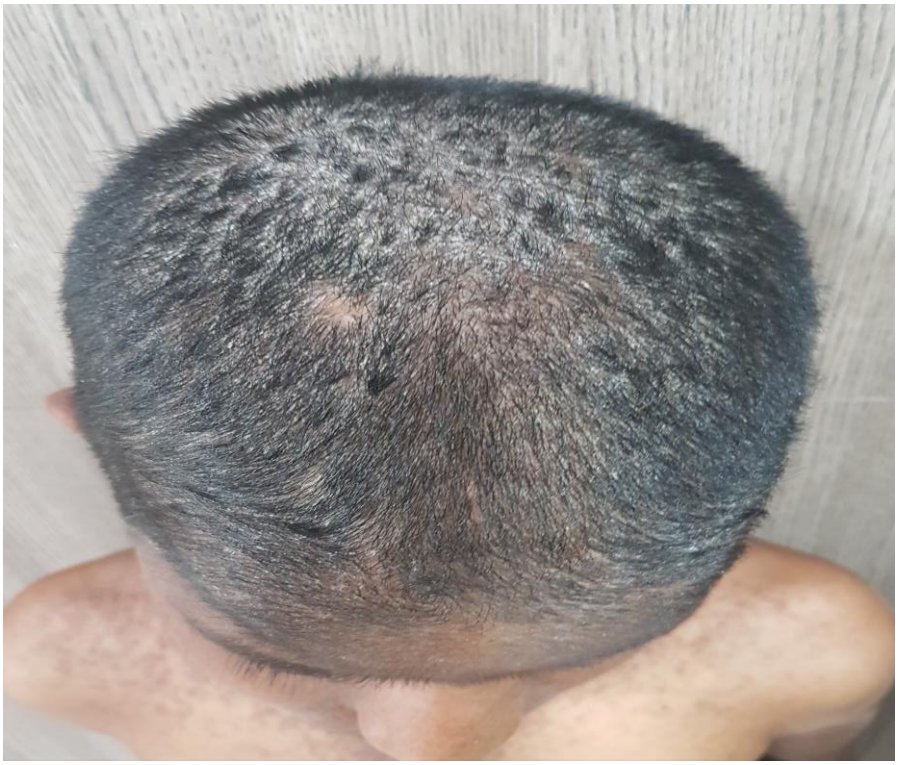

Figure 26: Darier's disease

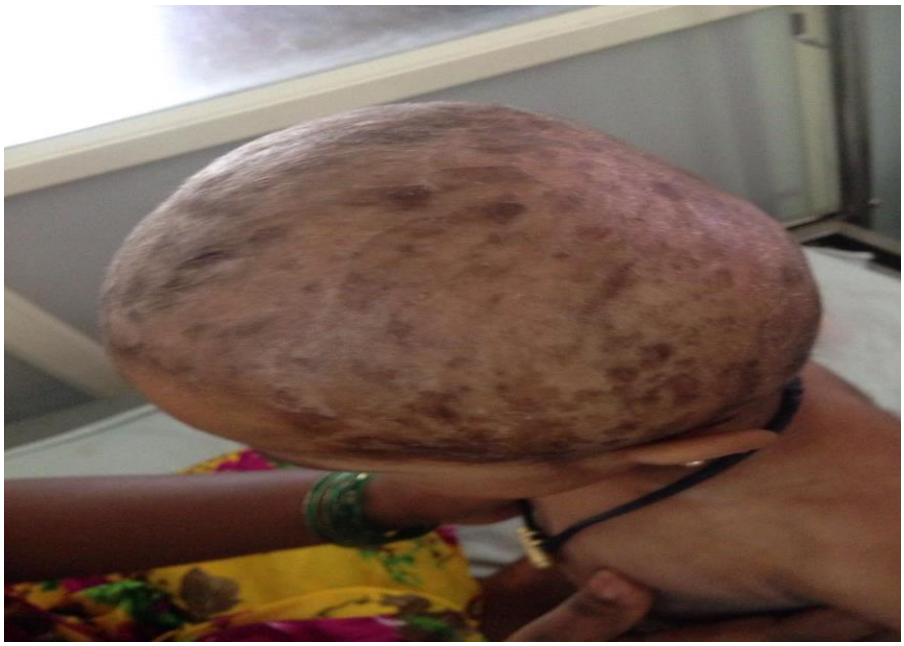

Figure 27: X- linked ichthyosis

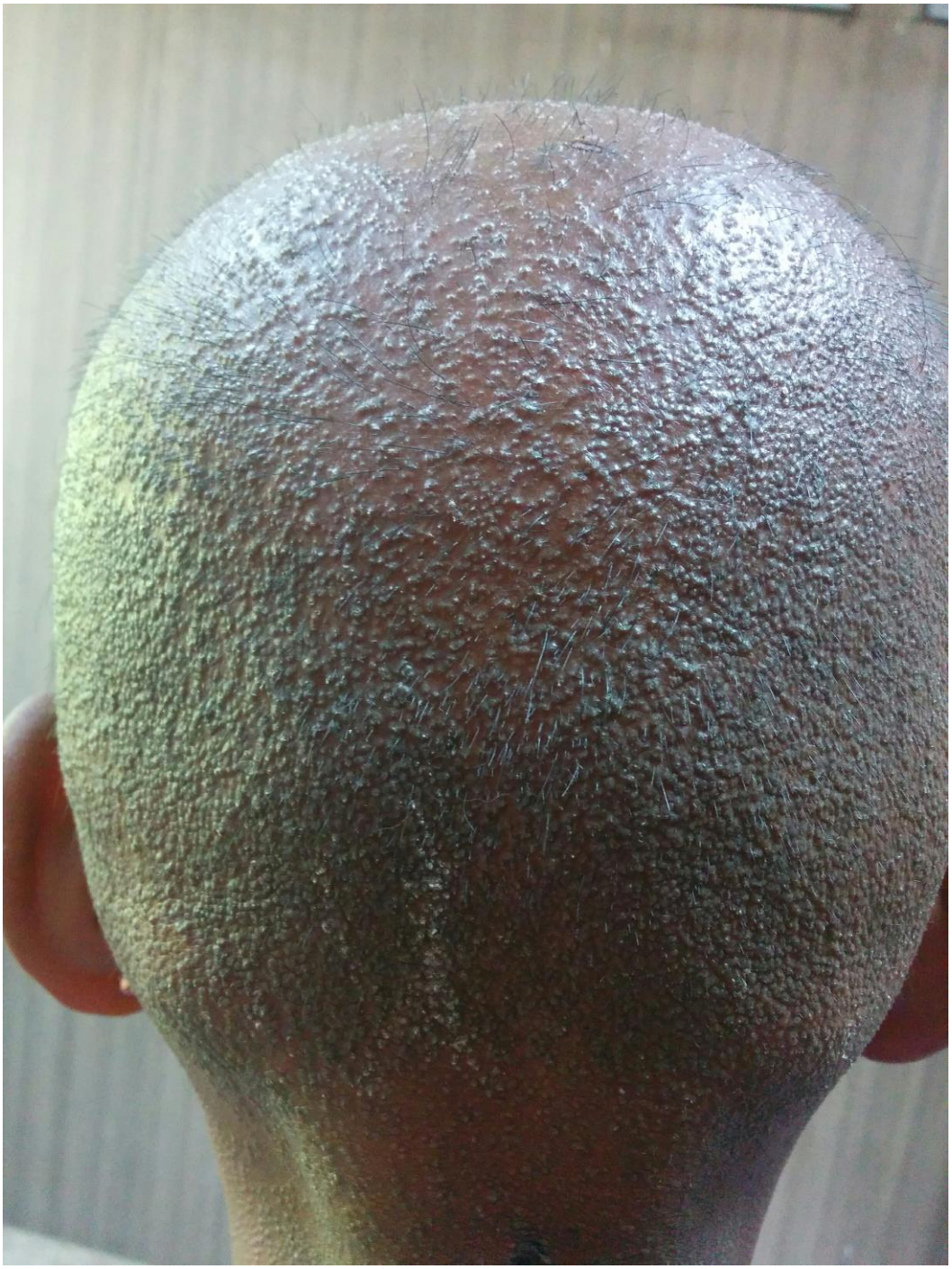

Figure 28: Keratosis folliculiaris spinulosa decalvans

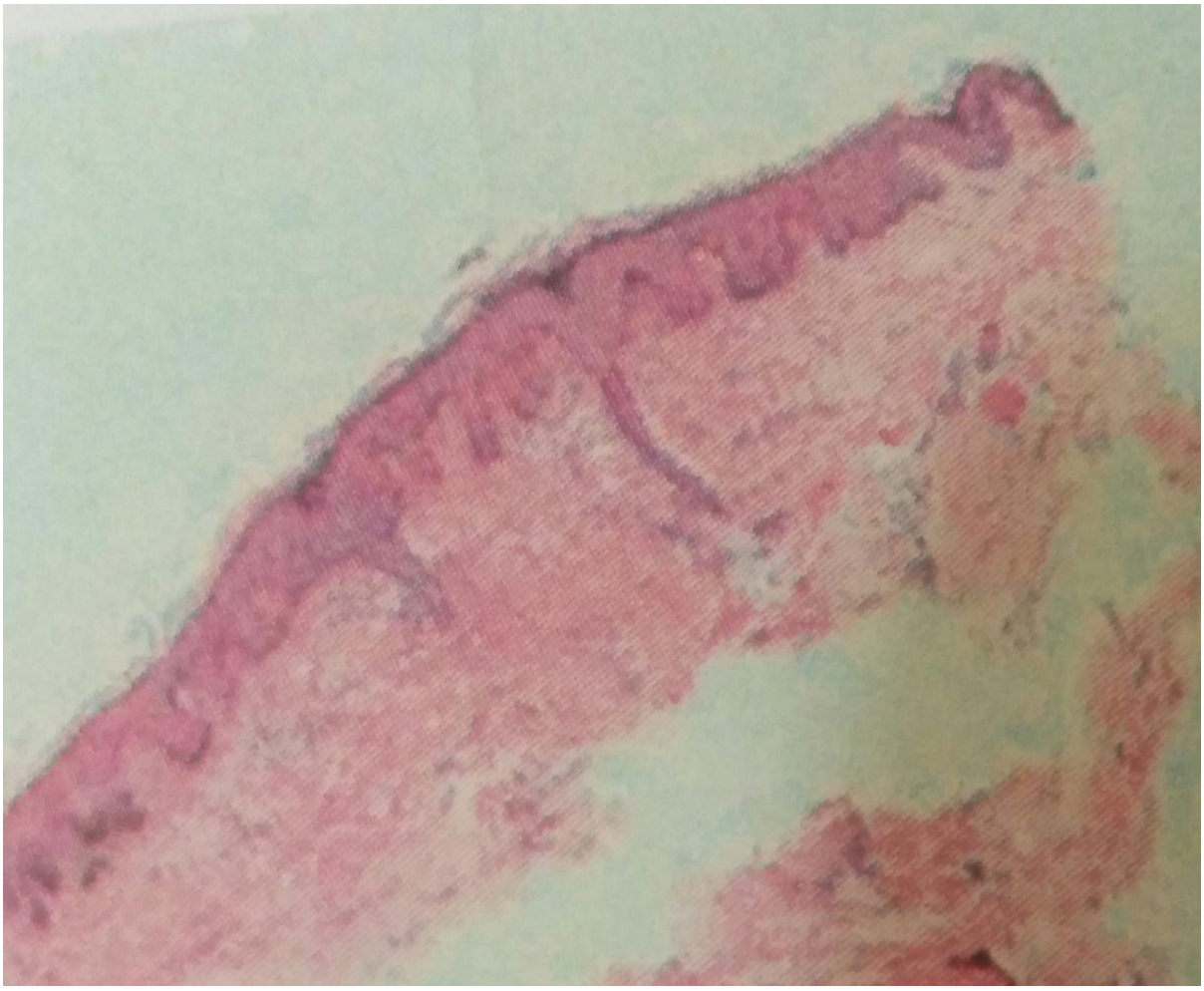

Figure 29: Histopathology of keratosis follicularis spinulosis decalvans. Epidermis shows mild hyperkeratosis and acanthosis. Follicular plugging and mild perifollicular lymphocytic infiltration with mild fibrosis.

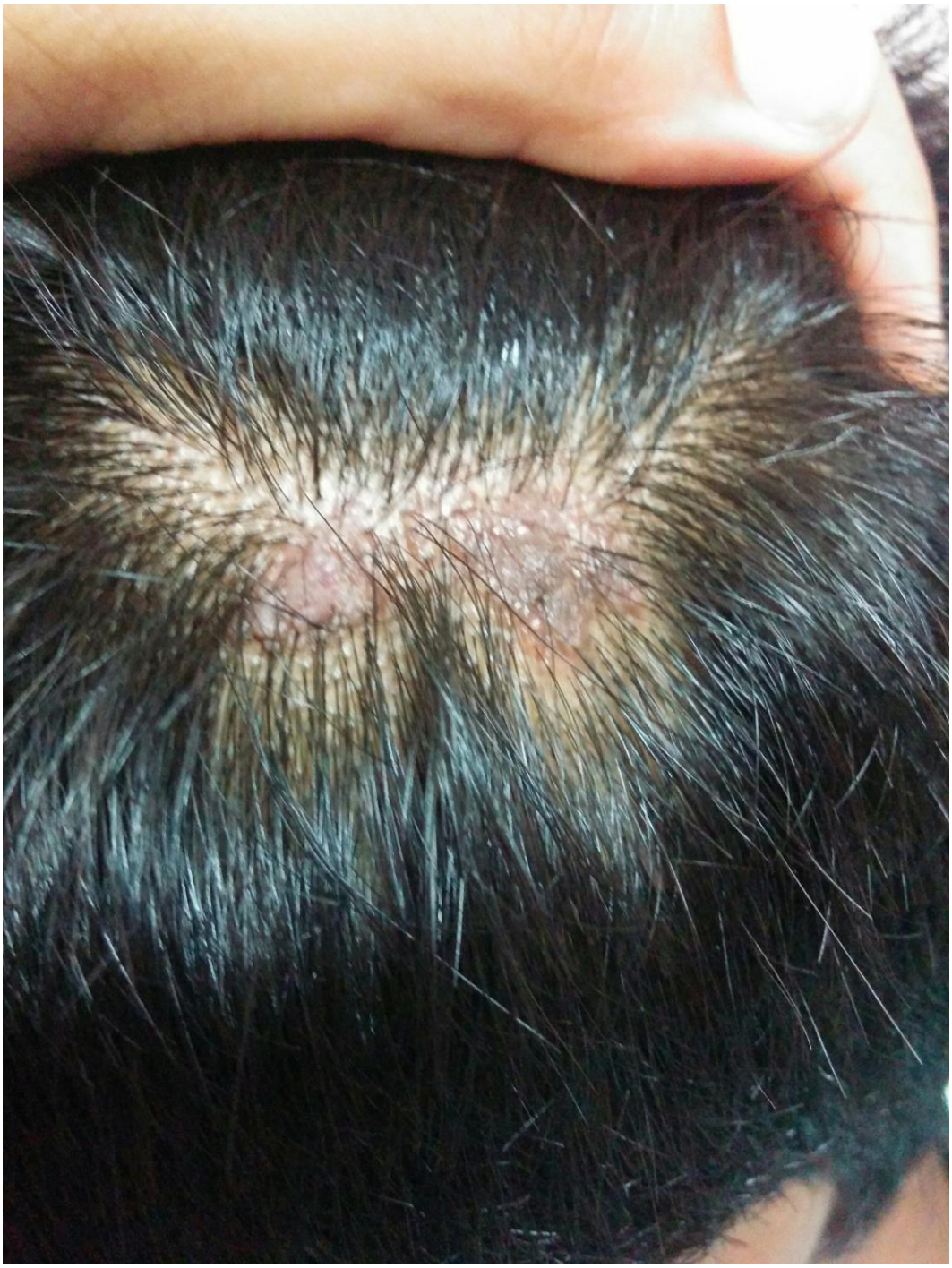

Figure 30: Nevus sebaceous

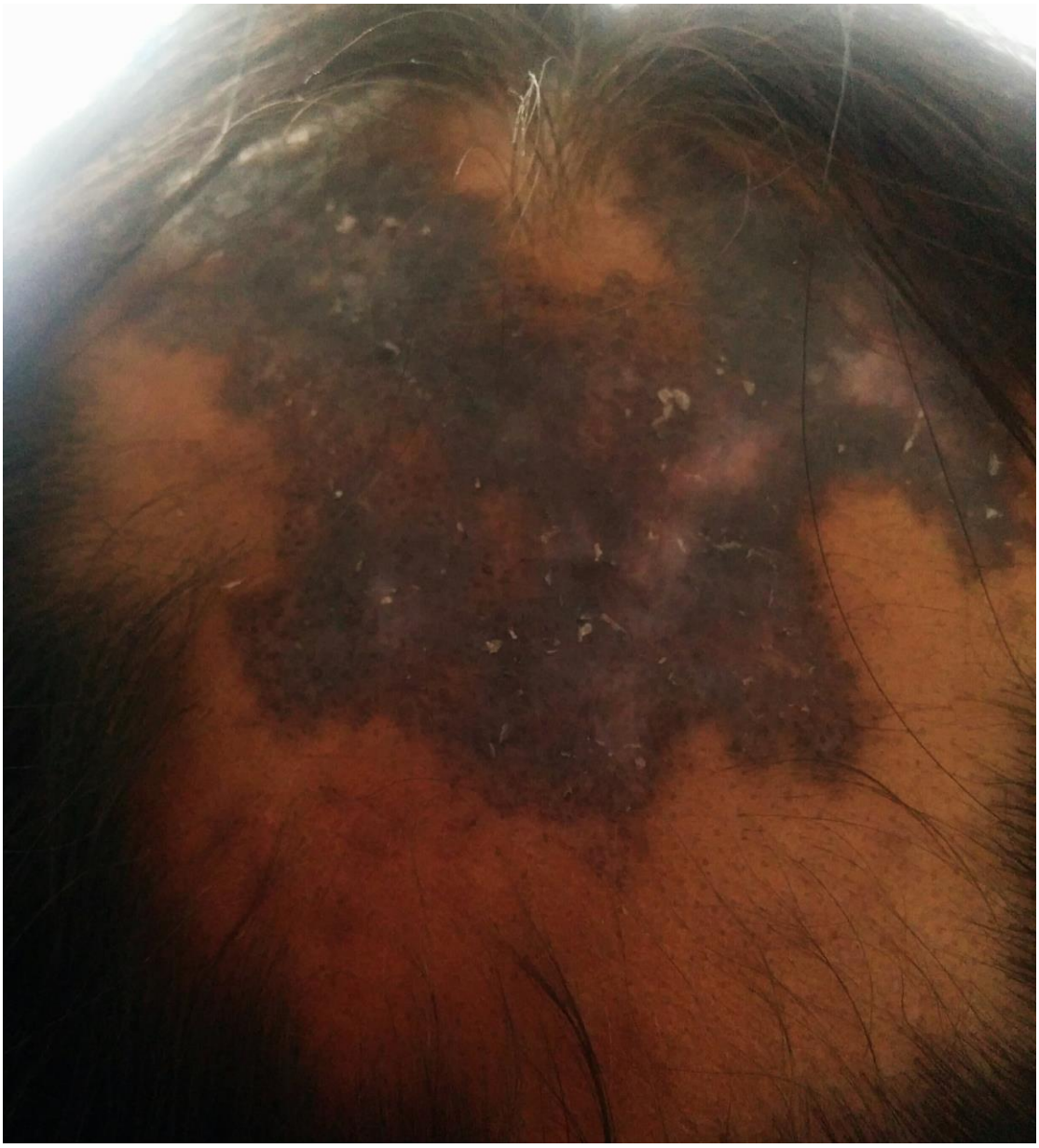

Figure 31: Discoid lupus erythematosus

DISCUSSION

DISCUSSION

Hair loss in children is challenging in terms of diagnosis and treatment.

The age of presentation in our study ranged from four days to eighteen years. Majority of the children with hair loss were in school going age group (6 – 10 years) and adolescent age group (10 – 18 years), contributing to 30.7 % (n=23) and 28% (n=21) respectively. This was similar to a study in South – East Nigeria, which had maximum cases between 7 to 12 years.[1] However our study differed from the above mentioned study by having a child as young as four days presenting with hair loss, whereas in their study the youngest child was three months.[1]

A similar study showed that hair loss in children was more common in males (62.8% males vs 37.2% females),[1] but in our study the gender distribution was almost equal with 38 male patients and 37 female patients. Congenital form of hair loss occurred equally in males and females contributing to 50% of the cases each and acquired hair loss had 33 males (49.25%) and 34 females (50.75%).

Out of the total 75 cases, 56 patients (76.6%) presented with aymptomatic hair loss. Itching was an associated complaint in 15 (20%), scaling in 7 (9.3%), pain in 3 (4%) and swelling in 3 (4%). A study in Jordon also showed that asymptomatic hair loss was the most common presentation (85.6%), but it had scaling as second most common complaint (36.3%) in contrast to itching in our study (20%).[3]

The duration of hair loss ranged from less than a month to more than a year. 61.3% of the children had hair loss of less than 1 month duration, 21.3% had 1 to 3 months duration, 5.3% had 4 to 6 months duration, 6.7% had 6 to 12 months duration and 5.3% had hair loss of more than 12 months duration. This was in accordance with another study.[3]

The duration of hair loss in the acquired group ranged from 5 days to 9 months whereas in the the congenital group it ranged from as early as 4 days to 6 years. This could be probably be attributed to the early health care seeking behavior in parents of school going age group.

The pattern of hair loss was localized in 80% of our cases and diffuse in 20%, similar to other studies. Acquired form of hair loss formed the majority contributing to 89.3% of the cases in comparison to the congenital type which contributed to only 10.7% of the cases.

8 out of 75 children had congenital form of hair loss. There were four cases of nevus sebaceous, one case of keratosis follicularis spinulosa decalvans, one case of X linked ichthyosis, one case of congenital aplasia cutis and one case of Darier's disease.
A study reported conditions like melanocytic nevus, Netherton syndrome and hair shaft disorders contributing to congenital alopecia,[1] which is not seen in this present study. However, we encountered certain conditions like keratosis follicularis spinulosus decalvans and Dariers's disease that were not reported in various other studies. [1,3]

Acquired alopecia was seen in 67 of the 75 children in the study. Acquired causes were classified into scarring and non-scarring types. 94.1% of acquired cases belonged to non-scarring group and 5.9% belonged to the scarring group similar to another study which showed 89.9% of non-scarring alopecia and 1.7% of scarring alopecia.[1] This highlights the

fact that acquired non-scarring alopecia is the commonest cause of hair loss in children which may be attributed to high number of scalp infections.

Non scarring acquired alopecia was seen in majority of the children (n=63). Tinea capitis (n=23) was the most common cause in this group, of hair loss accounting for 30.6% of overall cases, followed by alopecia areata 25.3% (n=19) and telogen effluvium 9.3% (n=7). These findings were similar another study where tinea capitis accounted for majority of the cases i.e 38.1%, followed by alopecia areata 23.9% and telogen effluvium 9.7%.[3]

The other uncommon causes of acquired non-scarring hair loss in our study were tractional alopecia (n=5), seborrheic dermatitis (n=4), trichotillomania (n= 3), scalp candidiasis (n=1) and androgenetic alopecia (n=1). Certain conditions like atopic dermatitis, psoriasis and loose anagen hair syndrome were reported in another study,[3] but were not encountered in our study. This may be due to different racial factors.

i. Tinea capitis

This was commonly seen in preschool and school going children in our study contributing to 21.7% (n=5) and 43.5% (n=10) of the cases respectively. The children in these age groups are commonly affected as they engage in several activities of close contact such as rubbing of heads while playing, sharing of hair combs and caps. Though it is said that tinea capitis is rare after puberty, 3 children had tinea capitis in the adolescent age group (10 – 18 years). The remaining 5 cases were observed in less than 3 years of age.

Out of 23 cases, 16 were male and 7 were female which shows that shorter hair in males facilitates easier mode of spread of spores compared to long hair in females.

12 cases (52.2%) were of grey patch type, 5 (21.7%) kerion, 4 cases (17.4%) black dot and 2 cases (8.7%) presented with more than one type of aforementioned pattern or the mixed type. This is in contrast to another study which reported black dot to be the most common type.[88] Favus type of tinea capitis was not observed in our study.

1 patient (4.3%) had previous episode of tinea capitis and 2 patients (8.7%) gave history of tinea capitis in family members, reflecting on the contagious nature of the condition.

ii. Alopecia areata:

A total of 19 cases of alopecia areata were seen in our study, more commonly in the adolescent age group (10-18 years). 2 patients were found between 1-3 years, 3 patients between 3-6 years, 3 patients between 6-10 years and the remaining 11 patients were in the adolescent group. The earliest age of presentation was 2 years. Similarly, a study on childhood alopecia areata in Iran showed that the commonest age groups affected were 6-8 years and 15-16 years, with the earliest age of presentation being 1 year.[89]

Out of 23 cases of alopecia areata, 11 were males and 12 were females i.e slightly higher incidence in females. This was unlike another study which reported a higher incidence in males (59.3% males versus 40.7% females). [89]

16 patients (84.2%) of alopecia areata presented with the patchy clinical pattern, followed by 2 cases (10.5%) of ophiasis and 1 case (5.3%) of reticulate pattern. No cases of sisaphio, alopecia totalis or alopecia universalis were noted in our study. Another study reported similar incidence of the various clinical patterns but also had sisaphio (2.4%) and alopecia totalis (2.4%). [89]

Involvement of other hair bearing areas such as eyebrows, was seen in 1 patient (5.3%). Another study reported other sites involvement in 10% of the cases, a higher percentage than our study.[69] Nail involvement (26.4%) was much higher in our study in comparison to the above mentioned study which showed only 10% of the cases affected.[89]

26.3% of the cases gave a positive history of atopy, similar to another study.[90] It is believed that atopic dermatitis increases the risk for cutaneous autoimmune conditions like vitiligo and alopecia areata by activation of common inflammatory pathways like thymic stromal lymphopoietin.[91]

1 patient (5.3%) had multiple associated autoimmune conditions like vitiligo, lichen planus and autoimmune thyroiditis. However, another study showed a lower incidence of associated autoimmune conditions with 2% of patients associated with vitiligo and 1% with hypothyroidism. [89]

iii. Telogen effluvium:

Out of 7 cases of telogen efluvium, 5 cases (71.4%) were observed in adolescent age group (10-18 years), 1 case between 3-6 years and 1 case between 6- 10 years. The mean age of presentation was 16 years. All 7 patients were females and none were male. Similarly, a study in Jordon also reported a higher incidence in females.[3] This was probably because females find the hair shedding more troublesome and seek medical attention earlier. [64]

4 patients presented with acute telogen effluvium and the other 3 had chronic telogen efluvium. The common causes in our study were fever, stress and surgery. A similar study attributed majority of cases of telogen effluvium to fever followed by iron deficiency anemia.[3]

Acquired scarring alopecia was seen only in 4 cases of the 67 cases of acquired hair loss. 3 cases (75%) of scarring following infections and 1 case (25 %) of discoid lupus erythematosus were seen. One study reported folliculitis de calvans and lichen planopilaris causing scarring alopecia. Both these conditions were not seen in our study. [3]

Poor hair grooming practices such as infrequent washing of hair, sharing combs, towels and caps and brushing wet hair were found in 24% (n=18) of our patients. All 18 belonged to acquired group indicating that acquired hair loss is influenced by hair care practices.

Malnutrition was calculated for all children less than 10 years of age using the WHO weight for age charts. Out of the 54 children less than 10 years of age in the study, malnutrition was noted in 8 children. 9.3% had grade 1 and 1.3% had grade 2 malnutrition. This highlights the fact that nutritional deficiency is an important contributing factor for hair loss in children.

CONCLUSION

CONCLUSION

Hair loss in children is relatively rare when compared to adults but is associated with significant psychological stress in both the parents and the child. Ethnicity, hair type, environment and cultural factors play an important role in hair loss. Diagnosis of alopecia in children is difficult because the clinical presentation and etiologies differ from that of adults.

Tinea capitis is the most common cause of hair loss in children in the developing countries. Early diagnosis and treatment can help prevent further spread of the infection and can completely reverse the hair loss. Alopecia areata, telogen effluvium, tractional alopecia and trichotillomania are a few other common causes. These conditions require ample psychological counseling along with specific therapy.

Congenital alopecias like aplasia cutis, nevus sebaceous, melanocytic nevus and hair shaft disorders are rare. Similarly, scarring alopecias like discoid lupus erythematosus and lichen planopilaris are less common. But it is important to consider these conditions in diagnosing hair loss in children.

Poor grooming practices like sharing of combs and towels, combing wet hair and using hot styling tools influence hair loss, hence it is important to increase awareness of healthy hair care practices among patients. Since malnutrition is a major contributory factor in hair loss in children, adequate supplementation of nutrients is essential.

SUMMARY

SUMMARY

- A total of 75 children (less than 18 years) presenting with hair loss to the department of Dermatology, Venereology and Leprosy, R.L.Jalappa Hospital and Research Centre, attached to Sri Devaraj Urs Medical Collge, Tamaka, Kolar, between June 2016 and July 2017 were enrolled in the study.
- Detailed history was taken, thorough clinical evaluation and relevant laboratory investigations were done.
- Among 75 cases, 38 were male and 37 were female.
- The youngest subject in the study was four days old and the oldest subject was eighteen years old. The mean age of the study group was 8.2 ± 4.9 years. It was most common in school going age group (6-10 years).
- The patients commonly presented in the first three months of onset of hair loss
- The hair loss was localized in 80% of cases and diffuse in 20%.
- The patients commonly presented with asymptomatic hair loss (76.6%). Associated complaints like itching, scaling, pain and swelling were seen in the remaining patients.
- Out of the 75 cases, 8 cases were congenital alopecia and 67 cases were acquired alopecia.
- The congenital alopecia cases were : four cases of nevus sebaceous, one case of keratosis follicularis spinulosa decalvans, one case of X linked ichthyosis, one case of congenital aplasia cutis and one case of Darier's disease.
- The acquired alopecia was further sub-classified into scarring and non – scarring alopecia. Out of the 67 cases of acquired alopecia, four cases were scarring and the remaining were non – scarring.

- Three of the four cases of scarring alopecia were following infections and one was a case of discoid lupus erythematosus.
- Out of the 63 cases of non- scarring acquired alopecia, tinea capitis formed the majority (n=23), followed by alopecia areata (n=19) and telogen effluvium (n=7).
- The other uncommon causes of acquired non-scarring hair loss in our study were tractional alopecia (n=5), seborrheic dermatitis (n=4), trichotillomania (n= 3), scalp candidiasis (n=1) and androgenetic alopecia (n=1).
- Tinea capitis was more common in school going (6-10 years) and preschool (3-6 years) children. It showed a male preponderance and was significantly associated with poor hair grooming practices and oil application on hair. Grey patch was the most common clinical type, followed by black dot , kerion and mixed type. Favus was not seen in our study.
- Alopecia areata was the second most common cause. It was more common in adolescents and almost equally distributed in males and females. Out of the 19 cases, 16 had patchy clinical pattern, followed by 2 cases of ophiasis and 1 case of reticulate pattern. No cases of sisaphio, alopecia totalis or alopecia universalis were noted in our study.
- Telogen effluvium was noted only in females in our study and most commonly in adolescents. Out of the 7 cases, 4 presented as acute telogen effluvium and the remaining 3 presented as chronic telogen effluvium. It was commonly seen after fever, stress and surgery.
- Past history of hair loss was seen in 5 of the 75 cases. Family history of hair loss was also noted in 5 cases.
- History of atopy was seen in 11 cases, 5 of these had alopecia areata, highlighting the association between autoimmune conditions and atopy.

- Poor hair grooming practices such as infrequent washing of hair, sharing combs, towels and caps and brushing wet hair were found in 24% of our patients.
- Malnutrition was seen in 8 of the 75 children. Six children had grade 1 malnutrition and two had grade 2 malnutrition.

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ANNEXURES

ANNEXURES

PROFORMA

SL. No: **Date:**

Name: **Height:**

Age **Weight:**

Sex: **Address:**

OP/ IP No:

HISTORY OF HAIR LOSS:

- Onset
- Duration
- Progression
- Hair regrowth – Present/Absent
- Other associated symptoms
    - itching
    - burning sensation
    - swelling
    - pain
- Other sites involved

Antenatal and natal history:

Developmental history:

Past history:

Family history of alopecia:

Medication history:

Diet history:

Hair care practices:

Treatment taken:

OTHER COMPLAINTS:

H/O STRESS: **>** Difficulty in school >Social adjustment problems

>Life changes

ON EXAMINATION:

GENERAL PHYSICAL EXAMINATION:

Vitals:

Head to Toe examination:

Systemic examination:

Skin:

Mucous membranes:

Nails:

Teeth:

Eyes:

HAIR EXAMINATION:

1) **HAIR APPEARANCE:**

- Texture
- Lustre

2) **HAIR LOSS**

- extent & distribution:
    - patchy

      no of patches
    - generalised
    - central
- hair line
- residual hair at site of alopeca
    - terminal
    - vellus
- loss of hair at other sites:
    - eyelashes
    - eyebrows
    - beard
    - limbs
    - axilla
    - pubic area

- **SCALP ASSESSMENT**
- follicular ostia
    - present/absent/diminished
    - distended(patulous)/dusky
- fringe sign

- colour of affected scalp
    - pink/peach coloured
    - skin coloured
- erythema

  perifollicular /patches /diffuse
- scaling

  perifollicular/patches/diffuse
- pustules

  crusts

  telengiectasia

  atrophy

  hypo/hyper- pigmentation

- **HAIR SHAFT ASSESSMENT**
- hair card test
    - presence of miniaturized test
    - new hair/broken hair
    - temporal recession

- hair pull test
    - negative /positive
    - if positive, anagen/telogen hair
- hair tug test

  positive/ negative

OTHER CLINICAL TESTS:

INVESTIGATIONS:

1. Hair mount
2. Wood's lamp examination
3. Skin biopsy
4. Dermography
5. Complete haemogram
6. Thyroid function test
7. RFT
8. S.iron
9. S.zinc
10. ANA
11. KOH mount/ fungal culture
12. Bacterial culture

PROVISIONAL DIAGNOSIS:

PATIENT INFORMATION SHEET

Study title: A clinico-epidemiological study of hair loss in children.

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

Aim: To study the clinical patterns and different etiological factors of hair loss in children.

Hair loss or alopecia is a common complaint in dermatology OPD and can be caused by a number of conditions. Though majority of the cases are seen in adults, a significant number is seen in children as well. The causes of hair loss vary according to the age group. In addition ethnicity, hair type, environment and cultural factors also play a significant role in hair loss.

Hair loss in children is associated with significant psychological stress which may affect the development of the child.

This study will help to know the common presentations and different etiological factors of hair loss in children. Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study we will collect information (as per proforma) from you. Relevant blood investigations (Complete haemogram, urine routine, renal function tests serum iron, serum zinc, thyroid function tests, anti-nuclear antibodies, VDRL, urine routine, KOH mount and fungal culture, bacteriological studies) will be carried out if required. This information collected will be used for dissertation and publication only.

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

not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For any further clarification you can contact the study investigator:

Dr. Amulya. Ramamurthy

Mobile no: 8050080010

E-mail id: amulya.bingi@gmail.com

CONSENT FORM

Study title: A CLINICO-EPIDEMIOLOGICAL STUDY OF HAIR LOSS IN CHILDREN.

PG Guide:: Dr. RAJASHEKAR T.S

PG Co-Guide: Dr. DR K.N.V.PRASAD

Principal investigator: DR. AMULYA.RAMAMURTHY

Name of the subject:

Age :

Address :

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

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

Parents's signature

Signature of the witness: Date:

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

Chief Researcher/ Guide signature Date:

KEY TO MASTER CHART SHEET

A: Serial Number

B: Hospital Number

C: Age

D: DAYS

M: MONTH/MONTHS

Y: YEAR/YEARS

E : Gender

M: MALE

F: FEMALE

F: Pattern of hair loss :

L: LOCALISED

D: DIFFUSE

G: Duration of hair loss :

D: DAYS

M: MONTH/MONTHS

Y: YEAR/YEARS

H: Associated Complaints :

P: PAIN

Sw: SWELLING

I: ITCHING

S: SCALING

C: CRUSTING

A: ASYMPTOMATIC

I: Other hair bearing sites involved:

E: EYEBROWS

B: BODY

J: Type of hair loss :

C: CONGENITAL

A: ACQUIRED

K: If acquired hair loss:

S: SCARRING

N: NON-SCARRING

L: Associated nail changes:

P: PITTING

L: PATCHY LEUCONYCHIA

LR: LONGITUDINAL RIDGING

K: KOILONYCHIA

M: Other associated skin changes:

V: VITILIGO

LP: LICHEN PLANUS

TF: TINEA FACIEI

N : Associated medical conditions:

H: HYPOTHYROIDISM

P: PCOS

A: ANAEMIA

D: DEPRESSION

O: Malnutrition grading according to WHO weight for age charts (applicable in children less than ten years):

N: NORMAL

1: GRADE 1

2: GEADE 2

P: Past History of hair loss

Q : Family History of hair loss

R: History of atopy in self or family members

S: History of any preceding event

T: TONSURING

F: FEVER

S: SURGERY

P: PSYCHOLOGICAL STRESS

T: History of poor grooming habits like infrequent washing of hair, sharing combs, combing wet hair, using hot styling tool etc.

U: Final Diagnosis:

T: TINEA CAPITIS

AA: ALOPECIA AREATA

TE: TELOGEN EFFLUVIUM

TA: TRACTIONAL ALOPECIA

TR: TRICHOTILLOMANIA

SD: SEBORRHEIC DERMATITIS

AGA: ANDROGENETIC ALOPECIA

SC: SCALP CANDIDIASIS

FI: FOLLOWING INFECTIONS

DLE: DISCOID LUPUS ERYTHEMATOSUS

KFSD: KERATOSIS FOLLICULARIS SPINULOSA DECALVANS

AC: APLASIA CUTIS

NS: NEVUS SEBACEOUS

Sl.No	H. NO	Age	Gender	Pattern of HL	Duration of HL	Assoc symptpms	Other HB sites	Congenital/ Acquired	Scarring /Non - scarring	Nail changes	Other skin changes	Assoc syst dis	Malnutrition	Past H/O HL	Fam H/O HL	H/O Atopy	Event preceding HL	Poor grooming habits	Final Diagnosis
1	411759	5	M	L	15 D	P, Sw		A	N				N					YES	TC
2	388207	1	F	L	20 D	I, S		A	N				N				T	YES	TC
3	297669	3	M	L	10 D	A		A	N				N			YES		YES	TC
4	283860	11	M	L	2 M	I		A	N								T		TC
5	403190	8	F	L	1 M	I		A	N	L			1						TC
6	403354	8	M	L	20 D	A		A	N				N						TC
7	403718	3	M	L	1 M	I, S		A	N				N				T	YES	TC
8	405782	5	M	L	1 M	A		A	N				1					YES	TC
9	406296	7	F	L	2 M	A		A	N				N						TC
10	407942	6	M	L	1 M	I		A	N				N						TC
11	408960	5	M	L	2M	A		A	N	L			N			YES			TC
12	410648	6	F	L	1 M	I		A	N				N						TC
13	410816	9	M	L	15 D	A		A	N				N					YES	TC
14	410888	2	M	L	1 M	P, Sw		A	N				N					YES	TC
15	368547	7	F	L	2 M	I		A	N		TF		N				T		TC
16	359266	16	M	L	15 D	A		A	N					YES					TC
17	412638	11	M	L	15 D	A		A	N	K		A							TC
18	385775	8	M	L	1 M	A		A	N				N				T		TC
19	414636	6	M	L	10 D	A		A	N				N						TC
20	415608	9	M	L	5 D	I		A	N	L			N					YES	TC
21	418907	2	F	L	1 M	Sw		A	N				1			YES	T	YES	TC
62	421642	9	F	L	2 Y	A		A	S				N			YES			TC
22	419955	10	M	L	7 D	I		A	N				N						TC
24	418784	14	M	L	2 M	A		A	N								F		AA
25	323058	14	F	L	1 M	A		A	N										AA
26	311306	6	F	L	1 M	A		A	N	L	V, LP	H	N			YES			AA
27	403217	2	M	L	15 D	A		A	N	LR			1			YES			AA
28	403310	11	F	L	1 M	A		A	N										AA
29	465472	18	M	L	7 D	A		A	N										AA
30	403722	2	F	L	20 D	A		A	N				N						AA
31	200300	14	F	L	2 M	A		A	N				N						AA

32	406203	6	M	L	1.5 M	A		A	N				N				P	YES	AA
33	402941	9	F	L	20 D	A		A	N				N						AA
34	401919	8	M	L	1 M	A		A	N	P			N						AA
35	402686	4	F	L	10 D	A		A	N				N			YES			AA
36	416833	6	M	L	2 M	A		A	N				N			YES			AA
37	405653	11	M	L	10 D	A		A	N							YES			AA
38	406683	7	M	L	15 D	A		A	N	L			N					YES	AA
39	406824	11	F	L	1 M	A	E	A	N					YES					AA
40	407869	12	M	L	2 M	A		A	N										AA
41	421642	9	F	L	2 Y	A		A	S				N			YES			AA
41	407540	17	F	L	1 M	A		A	N			H			YES		P		AA
43	375208	18	F	D	6 M	A		A	N	L		P			YES		P		TF
44	463114	16	F	D	1 M	A		A	N			A							TF
45	412954	15	F	D	6 M	A		A	N	L						YES	S		TF
46	413156	12	F	D	2 M	A		A	N	L							F	YES	TF
47	304404	10	F	D	3 M	A		A	N				1				S		TF
48	392938	3	F	D	4 M	A		A	N	L			N		YES		F		TF
49	375411	17	F	D	9 M	A		A	N			H		YES			P		TF
50	323415	MONT	F	L	20 D	A		A	N				N					YES	TA
51	383135	5	F	L	20 D	A		A	N				N					YES	TA
52	243547	MONT	F	L	1 M	A		A	N	L			1						TA
53	460059	7	F	L	2 M	A		A	N	L	V		2					YES	TA
54	402534	MONT	M	L	15 D	A		A	N				N						TA
55	311015	MONT	F	D	15 D	I, S		A	N				N						SD
56	322364	5	F	D	1 M	I, S		A	N				N					YES	SD
57	411065	MONT	M	D	15 D	I, S		A	N				N						SD
58	288623	14	F	D	15 D	I, S		A	N				N						SD
59	332878	8	F	L	15 D	A		A	N			D	N			YES	P		TR
60	415784	8	F	L	15 D	A		A	N				N				P		TR
61	214211	8	M	L	20 D	A		A	N				N						TR
62	412388	18	M	D	2 M	A		A	N					YES	YES				AGA
63	266437	6	M	L	1 M	P, S		A	N	L			N	YES			F	YES	SC
64	425012	10	M	L	9 M	A		A	S				N						FI
65	425631	4	M	L	3 M	A		A	S				N						FI
66	423924	6	F	L	2 M	A		A	S				N		YES			YES	FI
67	280856	15	F	L	8 M	A		A	S										DLE

68	425634	9	F	L	8 M	A		C					1					NS
69	429602	8	F	L	3 M	A		C		L			N					NS
70	311306	12	F	L	1 Y	A		C										NS
71	429241	10	M	L	6 M	A		C					N					NS
72	284192	13	F	D	6 Y	I	E, B	C										KFSD
73	296352	1	M	D	1 M	A		C					N					XLI
74	326684	12	M	D	4 Y	I, C		C		L								DD
75	459503	4 DAY	M	L	4 D	A		C					N					AC