TRICHOSCOPIC FEATURES IN FEMALE PATTERN HAIR LOSS- ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY"

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ABBREVATIONS

AGA	Androgenic alopecia
BIB	Breaking in between
BPH	Benign prostatic hyperplasia
BPPS	Brown peripilar sign
CNDPA	Caffeine, niacinamide, pathenol, dimethicone, acrylate polymer
DHEA	Dehydroepiandrosterone
DHEAS	Dehydroepiandrosterone sulphate
DHT	Dihydrotestosterone
FA	Focal atrichia
FAGA	Female androgenetic alopecia
FPHL	Female pattern hair loss
FSH	Follicle stimulating hormone
FU	Follicular unit
FUE	Follicular unit extract
FUT	Follicular unit transplant
GI	Gastro intestinal
HCP	Honey comb pigmentation
HDD	Hair diameter diversity
HFS	Scalp hair follicle
HPT	Hair pull test
LH	Luteinising hormone
LLLT	Low level laser therapy
LSESr	Liposterolic extract

LW I	Ludwig’s I
LW II	Ludwig’s II
LW III	Ludwig’s III
MPHL	Male patterned hair loss
MT	Male type
NIR	Near infra red
NO	Nitric oxide
NT-3	Neurotrophin -3
OPD	Outpatient department
PHL	Patterned hair loss
PRP	Platelet rich plasma
PSO	Pumpkin seed oil
R	Roots
R/BIB	Roots + breaking in between
RH	Recession of hairline
RH/T	Recession of hair line+ thinning
RH/T/S	Recession of hair line+ thinning+ shedding
SHBG	Sex hormone binding globulin
S+T	Shedding + thinning
UAT	Unit area trichogram
WPPS	White peripilar sign
YD	Yellow dots

ABSTRACT

INTRODUCTION: Patterned hair loss (PHL) is a common cause of hair loss in both men and women, and a condition for which patients commonly seek dermatologist consultation. PHL in females is known as female pattern hair loss (FPHL) and is frequently responsible for psychological distress and impaired social functioning.

FPHL is a non-scarring alopecia characterised by miniaturisation of the hair follicles, wherein terminal hairs are converted into vellus hairs, resulting in a decrease in hair density and thinning of hair over the frontal, mid-frontal, and vertex regions while retaining the frontal hair line.

The exact patho-physiology of female pattern hair loss is unknown with genetic factors, hair cycle defects, hormonal and environmental factors being implicated in its cause. It may occur alone or may be a part of constellation of androgen related conditions. Early diagnosis and initiation of treatment are desirable because treatments are more effective to avoid the progression of hair loss than stimulating regrowth. Typically, a diagnosis of FPHL can be confirmed by review of a patient's medical history and a physical examination, scalp biopsy is diagnostic but usually not required.

Trichoscopy- “dermoscopy of scalp” is a non-invasive method (vs. scalp biopsy) for diagnosis of hair loss using videodermoscopy of hair and scalp at higher magnifications (10x–140x) and allows measurement of hair diameter diversity (HDD), which is a characteristic feature of FPHL, recently termed as “Anisotrichosis”. Trichoscopy has been widely used as a diagnostic as well as a prognostic tool to measure anisotrichosis in cases of overt androgenetic alopecia

(AGA) and FPHL. Trichoscopy can also be used as a tool to diagnose FPHL in early cases.

OBJECTIVE: To study the trichoscopic features of female pattern hair loss.

MATERIALS AND METHOD: This was a one-year hospital based cross-sectional study on 110 patients who were clinically diagnosed as female pattern hair loss in the age group of 18-60 years with any grade of hair loss. The trichoscopic images were recorded using a videodermoscope Dinolite AM7915MZT-USB 2.0 Microscope, 5M pixels, 10 - 140 magnification model and relevant photographs were reorded.

RESULTS- The mean age of patients was 32.94 ± 9.26 years with 50.46% patients with duration of hair loss more than 9 months and maximum patients having strong family history of hair loss. Most patients presented with Ludwig-I grade followed by Ludwig-II, Ludwig-III and least with Male type fronto-temporal (MT) recession. Statistically significant difference was noted between stage and duration of hair loss. Trichoscopic features observed were HDD in 97.27% patients, brown peripilar sign (BPPS) in 25.45%, white peripilar sign (WPPS) in 30.91%, focal atrichia in 28.18%, one hair per follicular unit in 89.09%, 2-3 hair per follicular unit in 22.73%, yellow dots in 2.73%, scaling in 54.55%, white dots in 54.55% and honey-comb pigmentation in 40.91% patients respectively.

CONCLUSION - From our study we can conclude that trichoscopy could be excellent tool, which is simple, non-invasive cost effective for diagnosing FPHL using combination of findings like HDD, BPPS, WPPS, focal atrichia, white dots, single hair per follicular unit, honey-comb pigmentation and comparing with relatively spared occipital region.

Diagnosis of FPHL is mainly clinical and trichoscopy may aid in diagnosing FPHL differentiating it from other condition like chronic telogen effluvium and obviating the need of painful procedures like scalp biopsy. Our study lacks histo-pathological and hormonal investigations however past studies done have already established correlation of trichoscopic and histo-pathological changes.

Keywords: Hair, alopecia, trichoscopy, videodermoscopy, female pattern hair loss.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF PHOTOGRAPHS

INTRODUCTION

A female's hair is valuable to her splendour and sexuality. Long robust and shining hair stands for vitality, youth and, the health of lady and for this reason hair loss in lady produces extra psychological misery than in guys.

Greek phrase '*Alopex*' stands for the origin of the word 'alopecia' meaning fox. Alopecia refers to loss of hair in areas in which hair could have been found in most cases. The trouble of baldness in humans has been described for a while. (1)

Patterned hair loss is one of the most common and common hair problems for which both men and women seek specialist advice (PHL). (2)

Hair loss was designated as 'diffuse alopecia' in females. Role of male hormones was proved by Hamilton in 1942 in patterned baldness in men and so androgenetic alopecia (AGA) was the term coined for the condition. In women, patterned alopecia is considered similar to that of men, so ''female androgenetic alopecia'' or ''female pattern baldness'' is used. (1)

Some researchers use the label AGA interchangeably with female pattern hair loss. Nevertheless, the role of androgen in causing this entity is not properly set up, despite the fact that women with hyperandrogenism have excessive prevalence FPHL (up to 86%) and it remains uncertain, but new androgen independent mechanisms are being recognized.(2,3)

With advancing age, hair loss in ladies may be a very widely recognized entity. Classification for hair loss in female sex is given with the aid of special scales like Ludwig, Sinclair, Olsen, Hamilton & Norwood.(4)

FPHL is non-scarring alopecia, marked by miniaturization of hair follicles, conversion of terminal hair to vellus hairs, lower in hair density and thinning of hair over frontal, mid-frontal, and vertex region with retention of frontal hair line. [2] [3]

The exact pathophysiology of female pattern hair loss is unknown. Various elements are implicated in the causation of disorder consisting of genetic factors, hair cycle defects, hormonal, and environmental elements.[1]

FPHL can occur either alone or as a part of other androgen-associated conditions. The diagnosis of FPHL calls for a history, clinical examination, laboratory work up, and frequently a scalp biopsy to differentiate it from other causes.[5]

Trichoscopy- "dermoscopy of scalp" has won momentum in the subject of dermatology within the closing one decade. Its use in diverse scalp and hair disorders is nicely set up, but the literature is lacking in the field of FPHL.

Trichoscopic features for FPHL that are described in literature includes difference in hair shaft diameter, increased in the number of vellus hairs, yellow dots, white and brown peripilar sign, white dots, honeycomb pigmentation, areas of focal atrichia, and single hair per follicular unit.[6]

Owing to the paucity of literature towards FPHL in India, this study became proposed to scrutinize the trichoscopic findings in ladies with FPHL presenting with various grades as per Ludwig's scale.

OBJECTIVE

To study the trichoscopic features of female pattern hair loss.

REVIEW OF LITERATURE

HISTORY

Hair is only found in mammals, and its principal function during evolution was to provide insulation and protection from the elements. In humans, however, hair's principal job is to play a vital aid in social relationships. [7]

Fossilized casts and imprints in coprolites and pellets from the Late Palaeocene strata of Inner Mongolia provide the first direct evidence of hair in animals. [8]

EMBRYOLOGY

Hair follicle development on the chin, upper lip, and eyebrow is seen at 9 weeks of embryonic life in the womb [8]

The development of the hair apparatus/ pilosebaceous unit from the primitive epithelium is known as hair morphogenesis. There are several stages to it:

i. **Pre-germ stage**- At third month of fetal life primary hair germ develops with crowding of basal keratinocytes.

ii. **Hair germ stage**- This epidermal thickening then grows downward toward the dermis with the underlying mesenchymal cells showing signs of activity.

iii. **Hair peg stage**- It gradually elongates into a column pushing the underlying dermal cells with it.

iv. **Bulbous peg stage**- As the column elongates, its tip forms a cup (the future bulb) that comes to enclose the group of dermal cells underneath.[9] (Figure.1)

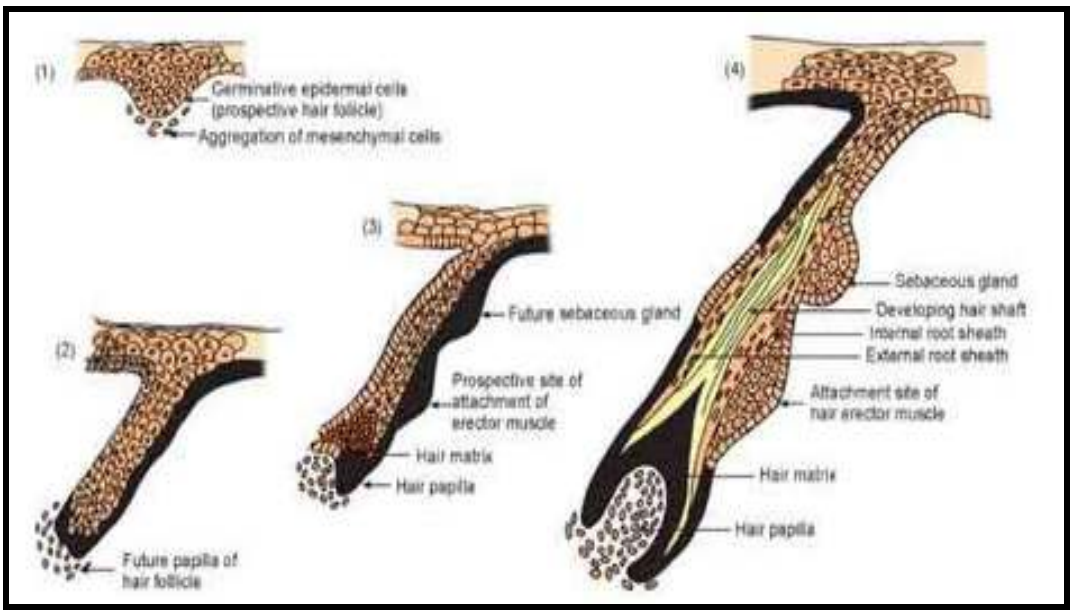


Figure. 1: Development of pilo-sebaceous unit[9]

The cells at the bulb's base begin to differentiate at 12 weeks, the outer cells form the inner root sheath, and the inner cells forms the eventual hair shaft. A hair canal is formed when a differentiated hair "cone" coated by the inner root sheath penetrates the hair column. The hair germs become divided as the skin develops, and new secondary germs sprout between them. [8]

HAIR FOLLICLE BIOLOGY

Hair is a tube-like structure with the epidermis at one end as well as being a keratinized outcome of the hair follicle. The follicles in the dermis are slanted, and the longer ones extend into the subcutaneous layer. [7] Hair follicles have two parts on histology in longitudinal section. [10]

i. **Upper segment**- consisting of

   a. Infundibulum- Stretches from the hair follicle's ostium to the opening of sebaceous duct

b. Isthmus- Stretches from the sebaceous duct above to connection of arrector pili muscle.

ii. **Lower segment**- Extends from the arrector pili's attachment to the follicle's base.

a. Stem- From the connection of arrector pili muscle above to Adamson's fringe (keratogenous zone ends below).

b. Bulb- From Adamson's fringe to the base of follicle.

During hair cycle, the lower follicle experiences multiple bouts of regression and regeneration, but the upper follicle maintains its form. [7] Three-layered hair shaft has: an inner layer (medulla), a middle layer of keratin fibres (cortex), and an outside layer of shingle-like cells (cuticle). The dermal papilla, the hair bulb, the inner and outer root sheaths, the point of attachment of the arrector pili muscle, the region of the hair bulge, the entryway of the sebaceous gland, and the opening to the skin's surface make up the follicle from the bottom up (Figure 2).

The hair shaft and the inner root sheath are lamellarly intertwined. At the opening of the hair follicle, the outer root sheath unites with the epidermis. [7, 9]

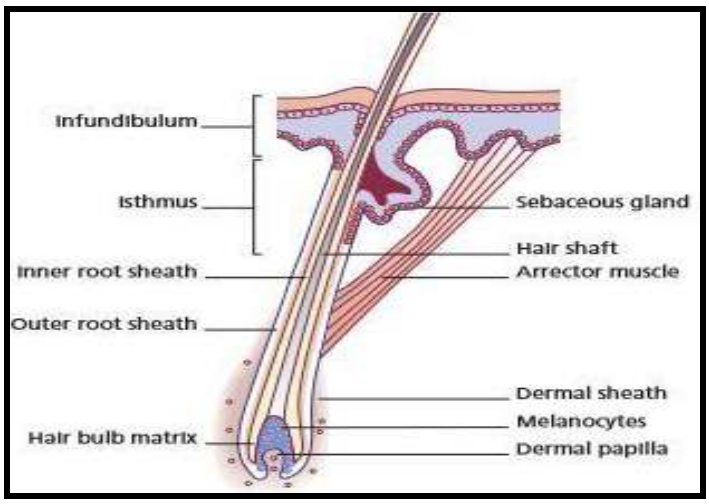


Figure. 2: Anatomy of hair follicle. [7]

From outside inward, cells of the follicular matrix can be distinguished in seven separate lines.

I. The outer sheath.
II. Henle's layer: Cornifies first & one cell thick.
III. Huxley's layer of the inner sheath and two cells thick.
IV. Cuticle of the inner sheath.
V. Cuticle.
VI. Cortex; and
VII. Medulla.

The papilla exits from the bulb's distal end through a tiny outlet, becoming one with a connective tissue sheath that envelops the entire follicle. [7,8,9] (figure.3)

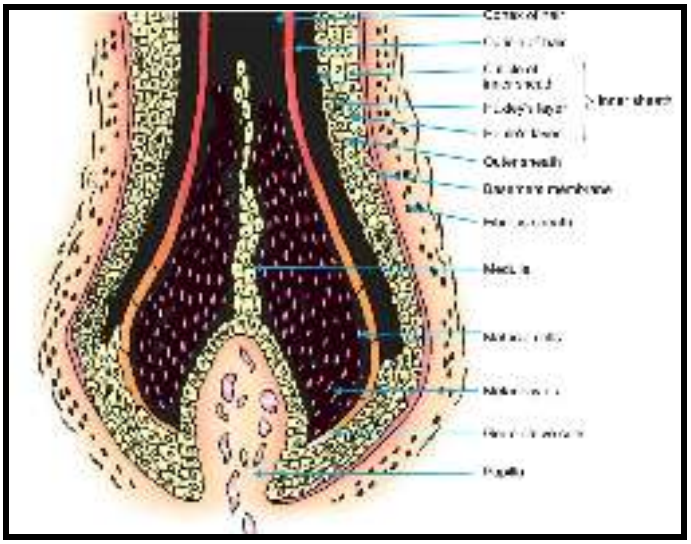


Figure. 3: Differentiation of cells of follicular matrix [9]

A thick network of nerves surrounds hair follicles, composed of sensory and autonomic nerve fibres. [6,7]

Free nerve endings, pilo- Ruffini nerve endings, and Merkel nerve endings are all forms of nerve endings found near hair follicles. [11] A dense perifollicular network of big longitudinal arterioles and smaller transverse anastomoses supplies blood to the follicle. The papilla has a vascular plexus that begins in the subcutaneous vessel plexus. [10] Dermal papilla is the hair follicle's command centre made up of mesenchymal cells, primarily fibroblasts. Thickness of hair depends on hair papilla size which directly correlates the size of hair bulb. [10]

TYPES OF HAIR

Based on the shaft thickness, hair is graded as [9]

i. Vellus hair (<0.03 mm, < 1cm in length)- Thin, short, unmedullated, and unpigmented covering almost entire of the body surface. The hair bulb of vellus hair in anagen phase resides in reticular dermis.

ii. Terminal hair (>0.06 mm, > 1cm in length) – Thick, longer, medullated & dark covering the scalp, beard, moustache, axilla, and pubis. The hair bulb resides in subcutis in anagen phase.

iii. Intermediate hair (0.03–0.06 mm)- Terminal hairs with hair bulbs residing in the reticular dermis rather than the subcutis and finer than the classic terminal hairs which are in the process of miniaturization.

iv. Lanugo (wool-like) hairs- Thin, unmedullated, and lightly pigmented but lengthier than vellus hair. Covers the fetal skin and shed at age of 8–9 months or post birth.

v. Sebaceous hair follicles: May develop distinct hair shaft types, changing from lanugo to vellus to terminal, as in the axilla or pubis, or from lanugo to terminal to vellus, as over the scalp.[11](figure.4)

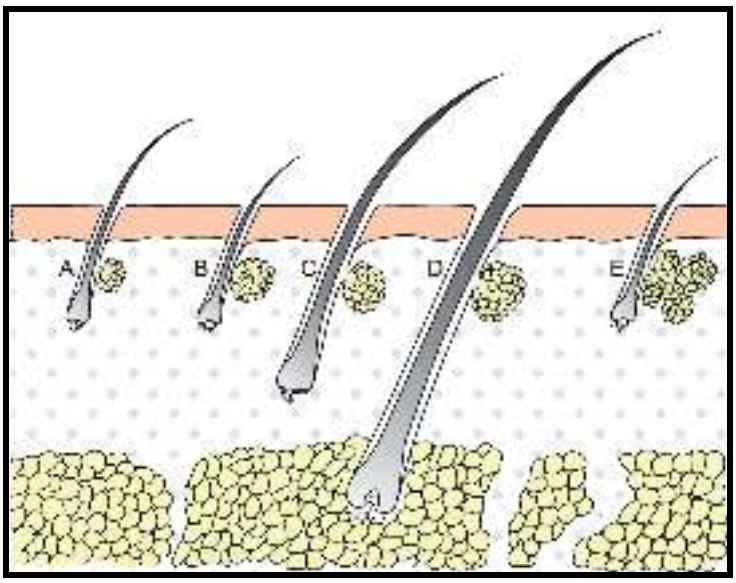


Figure. 4-Types of hair follicles: A. Lanugo. B. Vellus. C. Intermediate. D. Terminal. E. Sebaceous [9]

Ante puberty, terminal hair is usually only seen on the scalp, brows, and eyelashes. Under plethoric androgens, post puberty secondary sexual hair emerges from vellus hair. In androgenetic alopecia, terminal hair follicles in the scalp evolve to vellus-like or miniaturised hair follicles. [9]

HAIR CYCLE

- Cyclical phases of activity and inactivity occurring in hair follicles is known as hair cycle [7] which consists of five phases(figure.5)

  i. Growth phase (anagen I-VII)- 2-8 years
  ii. Regression phase (catagen)- about 2 weeks
  iii. Resting phase (telogen)- 2-4 months
  iv. Shedding phase (exogen)
  v. Lag phase (kenogen)

Anagen phase is further divided into seven stages: [13]

i. Stage I— The dermal papilla grows and the germ-like covering epithelium begins to mitotically divide.

ii. Stage II— Surrounding the dermal papilla, bulb matrix cells evolve and descent along the fibrous streamer.

iii. Stage III— All follicular elements are distinguished from bulb matrix cells.

iv. Stage IV—Reactivation of matrix melanocytes.

v. Stage V—Hair shaft emerges and telogen hair is dislodged.

vi. Stage VI—New hair shaft shows up from skin surface.

vii. Stage VII—Stable growth.

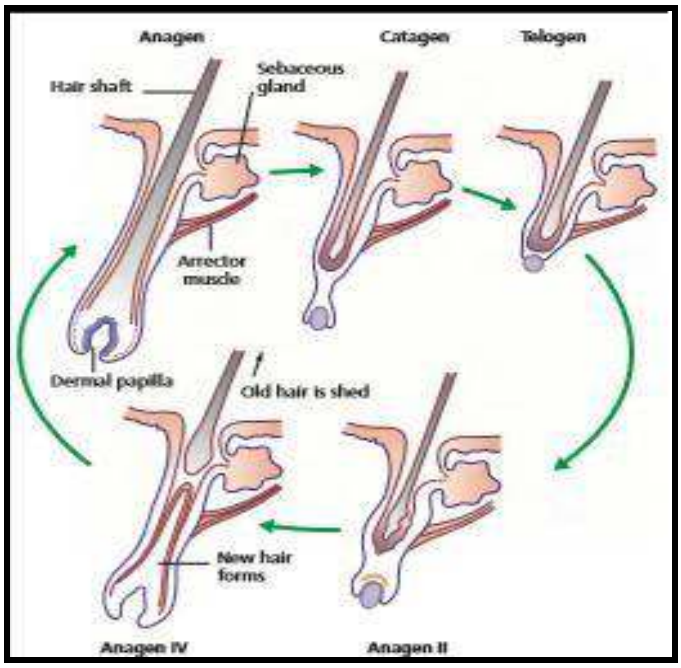


Figure 5: Hair cycle [8]

Hair grows during anagen phase. The onset of mitotic activity in epithelial cells overlying the dermal papilla will set up entry of a resting hair follicle into anagen phase. The span of anagen phase regulates the final hair length and it varies according to the different sites of the body [6,7]. Scalp hair has protracted anagen phase, lasting anywhere from 2 to 8 years. [7]

Epithelial cell division slows and ceases at the conclusion of anagen, and the follicle enters catagen, an involutionary phase. The catagen phase settles in about two weeks. During catagen state, the proximal end of the hair shaft keratinizes to create a club-shaped structure, while the bottom section of the follicle involutes due to apoptosis. The follicle's base, together with its dermal papilla, rises until it reaches just below the level of the arrector insertion.

The telogen phase is the time between the end of follicular regression and blooming of new anagen phase. 2–4 months is the usual duration of the telogen phase lasts. [6,7,9]

During the telogen phase, the dermal papilla shrinks to its smallest size. The follicle follows the papilla along the fibrous tube during a fresh anagen cycle, and the papilla enlarges once again. [8,9] (figure.5)

Club hair is lost by an active mechanism designated as exogen. Kenogen marks the latency phase post loss of club hair. [6,7]

Normally, the phases of the hair follicle's development cycle are independent of one another. [6,7]

Hair cycle distribution for terminal scalp hair.

- Anagen: 85–90% Telogen: 10–15% Catagen: <1%

RATE OF HAIR GROWTH

The rate of hair growth varies from species to species, and within one species from region to region, as well as with sex and age. Rates of scalp hair growth in humans vary between 0.3 and 0.5 mm per day, slightly faster in adult women than men but greater in pre-pubertal boys than girls. Male beard growth has been recorded at 0.27–0.38 mm/day. Vellus hair grows at a slower rate: 0.03 millimeters a day on the forehead. [6]

FEMALE PATTERN HAIR LOSS

Androgenetic alopecia (AGA) or pattern hair loss accounts most cases of hair loss in both men and women. Role of androgens in causation of hair loss in men is well elucidated. However, its role in FPHL is not known exactly and also distribution of hair loss is varied. [1] FPHL is a habitual cause of hair loss for which women seek dermatological treatment. The prevalence of this entity increases with age and affects quality of life.

BACKGROUND

EPIDEMIOLOGY

Hair thinning and loss is a typical concern among women who visit dermatological out patient department (OPD). The oftenness varies on the demographic and with advancing age (i.e., post-menopause due to hormonal

influence). As exact criteria to define FPHL are lacking prevalence studies are also few in literature.[1] [2]

According to Chinese research, the prevalence of FPHL was 6.0 %, ranging from 1.3 % in the 18-29-year-old group to 10.3 % in the seventh tenner and 11.8 % thereafter, with a 19.2 % positive family history. In a scrutiny of 1006 Caucasian women, FPHL was found to be prevalent in the late twenties and to peak beyond 50 years of age. The Indian studies have no data provided. [2]

AGE

FPHL can present at any time after puberty although there are 2 main periods of onset-early onset (teens-20 years) or late onset (40-50 years). [1] Women who acquire FPHL early in life are more likely to develop severe MPHL. [18] 3% of people acquire clinically identifiable FPHL by the age of 29, 13% by the age of 49, 8% by the age of 69, and another 6% beyond the age of 70. [7]

The female's average age with FPHL was 34.4±10.6 years in research by Zhang X et al. [12] Rakowska et al. [13] found that the average age of FPHL was 36.2 years, with a range of 18-59 years.

MORBIDITY

Impact of FPHL on health is psychological. Loss of hair in female sex causes low self- appraisal and decreased quality of life.[14] Baldness in women is usually unexpected and unwelcomed at any point of life.[15] Hair is part of the defining characteristic in human and it has great psychological and sociological role in making identity and appearance of one self.[16] Psychological deterioration is more apparent in women than in men who are more concerned about their body image. [17] The impact

of FPHL is influenced by both disease related factors like extent of hair loss, visibility of scalp as well as on psychological factors like coping up, beliefs, and treatment adherence. Also emotional aspects are associated with FPHL such as jealousy of other women with thick long hair, low esteem and lack of confidence in social gatherings etc.[18]

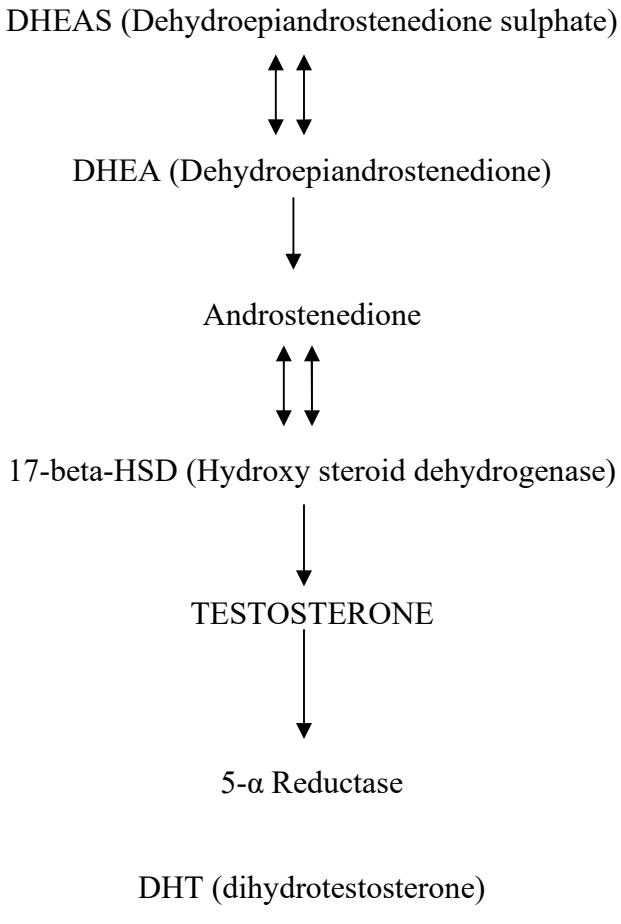
In survey by Sawant et al[19] major concern faced by women with pattern hair loss were getting aged, becoming bald, unfavourable consequences on communal life and symptoms of gloom. Similar negative effects on social life was given by study done by Gupta et al.[16]

ETIOLOGY AND PATHOGENESIS

Exact aetiology in causation of FPHL is not known. Role of various factors in causation of FPHL like genetic, hormonal, defect in hair cycle and environmental variables have a role. [1]

I. ANDROGENS

Androgens cause androgenetic alopecia (AGA) in genetically sensitive men and women. Patients with AGA have higher 5 α-reductase action and higher amounts of dihydrotestosterone in their hair follicles (DHT). DHT is formed by peripheral conversion of testosterone by 5 α-reductase enzyme.[20,21,22]



Both Type 1 and 2 5α-reductase iso-enzymes differ in their distribution in the body. The type 1 iso-enzyme is frequently identified in sebaceous glands, notably after puberty and in acne-prone zones namely the face. The type 2 iso-enzyme can be detected in neonatal hair follicles, foetal genital skin, and adult male prostate. In the companion and epidermal granular stratum of the hair follicle and within the hair follicle, the type 2 iso-enzyme was identified. In vulnerable individuals, DHT links to the androgen receptor, and the hormone-receptor complex promotes the genes that drive the gradual transformation of bigger terminal follicles into miniaturised follicles. [23,24,25,26]

The androgen receptor, also known as "caspase," is a particular protein that binds DHT. They expose the DNA linking station and modify the expression of certain androgen dependent genes after conformational changes in the hormone-

receptor complex. [27]. When compared to women who do not have hair loss, those with pattern hair loss had greater 5α reductase (5αR) and more androgen receptors. [28]

II. ESTROGENS

The significance of estrogens in hair development on the scalp is less understood. The job of estrogens in FPHL is debatable. Estrogens have a role in androgen metabolism by lessening DHT concentrations through direct inhibition of 5α-reductase or estrogen-induced conversion of testosterone to weaker steroids. The increasing incidence of FPHL following menopause elucidates estrogen's stimulatory function. Similarly, a drop in oestrogen levels during the postpartum period may explain for the simultaneous conversion of hair follicles to the telogen phase, resulting in telogen gravidarum. [14] 40% lesser androgen receptors (AR) seen in female frontal scalp than men.

Women's frontal hair follicles had 3- and $3^{1/2}$times lower amounts of 5α-reductase I and II, each. Women instead have six times the amount of aromatase in their frontal hair. [14] In follicles, aromatase plays a protective function. [8] This might explain why males and females have different clinical manifestations of PHL. [14]

III. RELATIONSHIP BETWEEN THYROID LEVELS AND HAIR LOSS

Thyroid hormone is vital for hair follicle growth and maintenance. However, the specific process remains unknown. However, it has been hypothesised that hypothyroidism deducts sex hormone binding globulin (SHBG) production, resulting in elevation in free testosterone levels. [27]

Thyroid dysfunction was linked to all kinds of alopecia as people became older, according to research by Vincent M et al [29] Zhang X et al [12], on the other

hand, observed no link between the gravity of FPHL and the laboratory value of gonadal profiles.

IV. ROLE OF PROLACTIN IN HAIR LOSS

In study done by Schmidt et al[30] in 1989 hyperprolactinemia was concerned as a probable cause of androgenetic alopecia. Prolactin interferes with androgen metabolism in causation of hair loss.

According to Foitzik et al[31] , prolactin and prolactin receptors are expressed over routine human scalp hair follicles (HFS) and human skin. Prolactin and prolactin receptor activity found to be elevated during the switch from anagen to catagen in hair follicles. It has also been hypothesised that prolactin aids as an autocrine hair growth regulator, inhibiting hair growth.

1. GENETICS

Role of genetics in FPHL is uncertain. Patients with FPHL frequently have a family history of the disease and an incomplete penetrance with polygenic pattern within 40-54 % of cases. [1] Pattern hair loss in 1st degree male relatives >30 years was 54 % in women with FPHL and 21% in female relatives >30 years. [32] The AR/ectodysplasin A2 receptor, which is found on the chromosome X and predisposes to FPHL, has been discovered. [14]

2. IMMUNE SYSTEM AND HEALTH

The various cytokines secreted by the perifollicular macrophages and mast cells unpleasantly affect the hair cycle. Interlukin-1(IL-1), Tumor necrosis factor-α (TNF-α), and fibroblast growth factor-5 (FGF-5) secreted by macrophages induce

catagen. Perifollicular mast cell degranulation has been observed at the onset of anagen–catagen and telogen–anagen transformation.[33]

Follicles switch from terminal to vellus-like hair in androgenetic alopecia, popularly termed as pattern hair loss. Traditionally, this process was expected to sustain many follicular cycles to execute. [34] With each cycle, the anagen duration of AGA decreases, resulting in tiny, finer hair. (figure7)

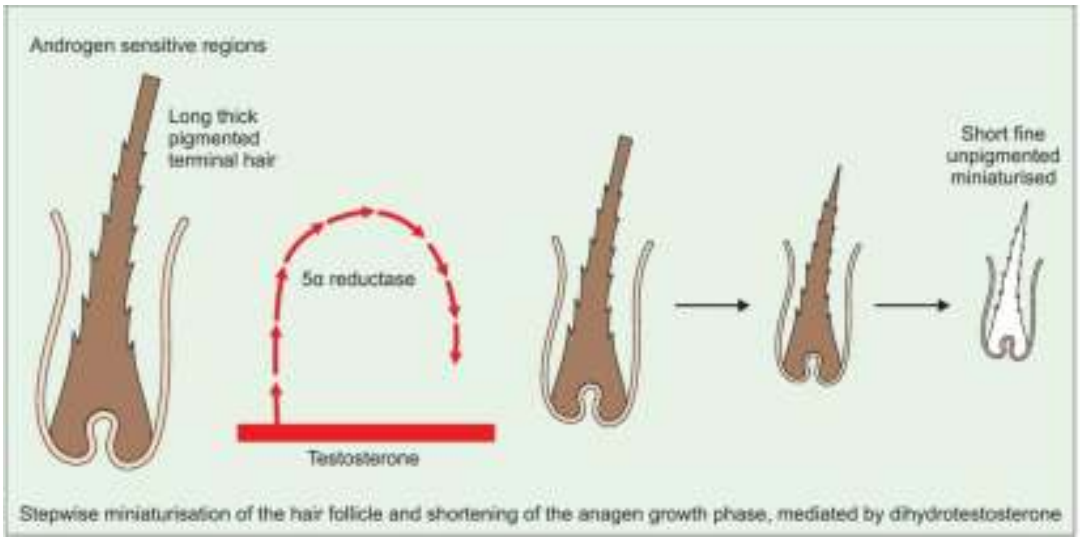


Figure.7: Miniaturization of hair [9]

As anagen phase duration shortens, whereas telogen phase lengthens or stays the same, resulting in a reduction in anagen: telogen ratio from about 10-12:1 to 5:1. As its anagen phase which regulates hair length, each successor cycle results in the novel anagen hair being shorter than previous. Eventually the anagen gets so short that the advanced hair does not have enough time to grow long enough to reach the surface. Telogen hair makes up the maximus of total hair and is less tightly attached to the follicle than anagen hair, resulting in greater hair loss.

Finally, the latency between late telogen hair shedding (exogen) and the commencement of new hair growth (anagen) prolongs, resulting in more follicles without hair and a fall in scalp hair density. [35] However, the number of follicles per unit area stays steady. [36]

The hair follicle is a multifaceted structure that is constantly active. Early termination of the anagen phase hallmark's FPHL. Follicular regression is driven by diffuse apoptosis of follicular keratinocytes. The precise function of cytokines along with growth factors in follicular keratinocyte apoptosis has yet to be uncovered. The hair follicle diameter is influenced by the magnitude of the dermal papilla.

Secondary to reduction in papillae volume, the miniaturisation process occurs. Hair thickness maintained in anagen phase so no miniaturization during it. As a result, it is clear that the miniaturisation process takes place between the catagen aspect and the development of new hair. The elements which directly cause a drop in papillae volume are uncertain, and they should be the major focus of future FPHL interventions and preventive measures. [1]

The loss of papilla cells owing to apoptosis is one proposed reason for follicular constriction. The failure of anti-apoptotic systems as a critical aspect in the causations of FPHL should be explored. [1]

1. ENVIRONMENTAL FACTORS

Environmental variables are still thought to play a subsidiary role in the development of FPHL. Psychological stress, hypertension, diabetes, smoking, multiple marriages, lack of photoprotection, economic prosperity, and diminished physical activity have all been implicated to the ailment. [1]

2. NEURAL SYSTEM AND HAIR

Hair formation and regeneration are aided by a variety of neurotrophins and their receptors. Neurotrophin 3 (NT-3) triggers the catagen phase and aids hair genesis. The hair cycle is also modulated by glia-derived neurotrophic factors, neurturin, and brain-derived neurotrophic factors. [9]

CLINICAL FEATURES

The onset is sluggish, as per history. Diffuse thinning of the crown is prevalent in females. [37] In women, the phenomenon is typified by an expansion of the scalp's central parting. [38] Women, on the whole, preserve their frontal hair line. Women report bitemporal recession to a limited degree than males. The changeover from pigmented vellus hairs to non-pigmented terminal hairs is protracted. [39] Only a tiny fraction of hair follicles is afflicted by FPHL. Therefore women usually do not become completely bald and there is only diffuse thinning of hair.[37]

PATTERNS OF HAIR LOSS

There are 3 variant patterns of hair loss in females:

- A generalized thinning of the crown region with the frontal hair line unaffected.

This pattern is explained by two scales:

i. Commonly used Ludwig scale, a three-point scale (figure. 8) [5,40]
ii. Sinclair's five-point scale (figure. 9) [37,41,42]

The three patterns in Ludwig's categorization equate to stages or progressive variations of female androgenetic alopecia (FAGA), as follows:

F-AGA Degree I (Minimal): it heralds onset of FAGA. Nominal part width amplification with detectable thinning of hair from the anterior portion of the crown.

F-AGA Degree II (moderate):

Because of a surge in thin and short hairs, thinning in the crown area becomes more noticeable.

F-AGA Degree III (intense)

With a substantial widening of the part width, the crown area becomes denuded, but the frontal hairline is sustained.

F-AGA.M (Female Androgenetic Alopecia of Male Pattern): Ludwig also documented female hair loss and bitemporal regression, which in itself is akin to the male pattern of alopecia. It is sub-categorized based on Ebling's or Hamilton-Norwood's classification.

F-AGA.M may be outlined in the following diseases:

i. Persistent adrenarche syndrome
ii. Alopecia secondary to adrenal or ovarian tumour
iii. Post-hysterectomy and as
iv. Involutive alopecia.[27]

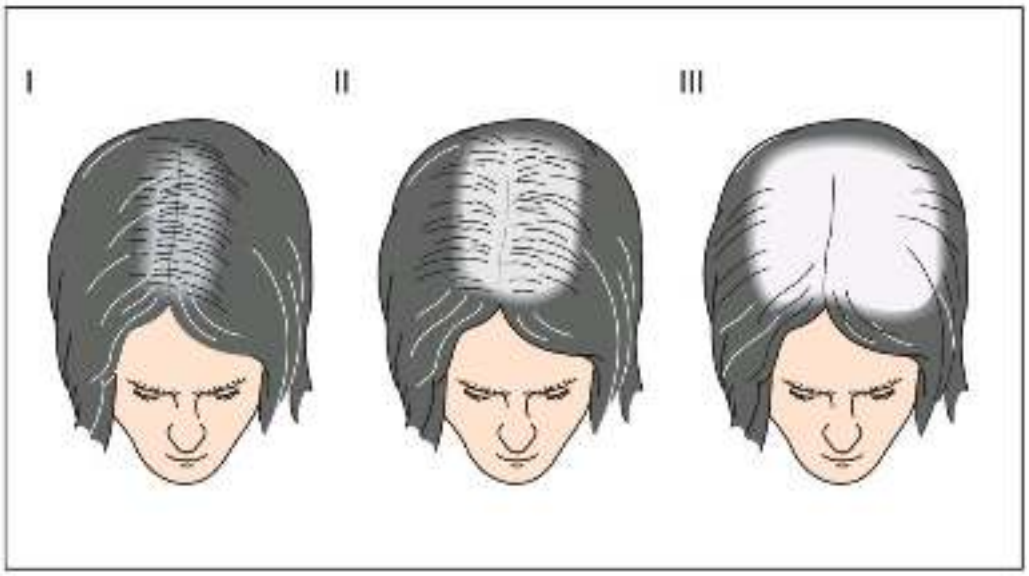


Figure. 8: Ludwig's grading of hair loss. [8]

Sinclair scale, divides FPHL into 5 grades:

Grade I: Normal

Grade II: Central part width widening.

Grade III: Thinning of hair on both side of the central part with its widening.

Grade IV: Diffuse hair loss over scalp.

Grade V: Advanced hair loss

There are 2 periods of onset of FPHL:

i. Immediate post-puberty to the 3rd decade
ii. 5th to 6th decade.

Women with early onset FPHL may have hyperandrogenism. The late onset FPHL corresponds to peri-menopause or menopause.[40]

During the initial progression of FPHL, upsurge in hair shedding may occur, which is perceived as positive hair pull in the affected sites on clinical evaluation.

However as disease progresses the hair shedding tends to stabilize and hair pull may not be positive.[40]

HISTOPATHOLOGY

The easiest approach to tell the disparity betwixt diffuse alopecia areata and AGA is to take a scalp biopsy. For a comprehensive analysis of alopecia, a punch biopsy or incisional biopsy stretching into fat to include terminal hair bulbs is essential. [43,44] Some dermatologists recommend having biopsy samples from at least two distinct sites:

a. One for horizontal sections: this permits for the identification and quantification of follicle stage (anagen, catagen, telogen), follicle diameter (to discern amongst terminal and vellus hair), and any other elements that could affect hair development findings (e.g., Fibrosis, inflammation) [42]

b. One for vertical sections. (Figure.12) [43,45]

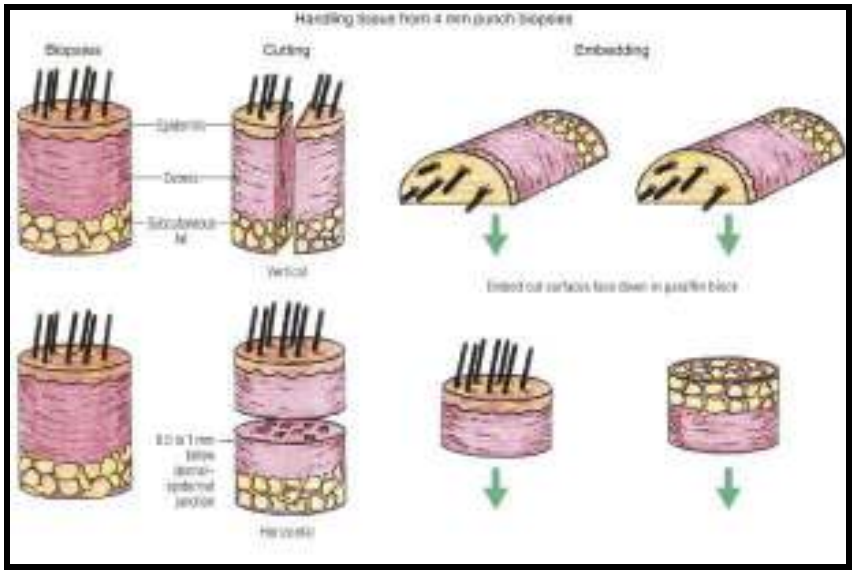


Figure.12: Histopathology of AGA [43]

Only a transverse slice allows for quantitative measurement of AGA, allowing for a satisfactory assessment. Furthermore, samples with a diameter of less than 4 mm possess insufficient follicles to generate relevant findings. [46]

The histologic hallmarks of AGA are follicular loss, or miniaturisation. Horizontal sections up to the snuff of the lower infundibulum of terminal hair follicles should be scrutinized to define the number of miniaturised follicles, as slices underneath this may lack the vellus hair whose bulbs are present in the upper in dermis.

Terminal hair has a shaft diameter of ≥ 0.06 mm, intermediate/ indeterminate hair has a shaft diameter ranging 0.06 - 0.03 mm, and vellus hair owns a diameter ≤ 0.03 mm, according to Headington. Terminal-to-vellus hair index < 3:1 is diagnostic of male pattern AGA. [43,44]

DIAGNOSIS

A correct diagnosis based on clinical history, clinical examination and biochemical investigations are essential for successful treatment.

I. The clinical history will include a thorough evaluation of any variables that may cause or worsen alopecia, such as chronic illness, dietary changes, metabolic and endocrinological aberrations, and recent surgical and pharmacological therapies. The time of initiation of hair loss, the site, whether increased shedding was evident at the time of onset and persists, and any shifts in lifestyle within 6 months dawn to the onset of hair loss should all be questioned. [45] History of topical application of any drug or intake of steroids should be taken.

- In clinical examination, after clicking baseline photograph, all areas of the scalp examined and looked for any hair loss patch, focal atrichia, broken hair, erythema, scaling, or atrophy. [45,47]

- Non-invasive

- Semi-invasive and

- Invasive specific methods may be undertaken.

NON-INVASIVE:

1. **Daily hair count**: In this method, patient is instructed to collect hair shed in 24 hours, count them, and congregate in plastic bags. Patient is instructed to collect all shed hair in the sink or comb. Hair counts are taken every day for seven days. On shampoo days, anticipate more hair to fall out. [9,48]

   If >100 hair/day are lost, microscopic examination for hair shaft defects and abnormalities of hair bulb should follow.

2. **Standardised wash test**: After 5 days of not rinsing his or her hair, the patient rinses it and gathers any hair shed in a gauze set over the drain in the washbasin. The hair is next assessed and grouped into length categories: >5 cm, 3-5 cm, and < 3 cm. Placed on correctly fitted glass slides, lengthy and dwarf hairs are subjected to microscopic inspection. The goal of this examination is to scrutinize the state of the root and assess the hair diameter. The results are built on hypothesis that typical hair grows 1 cm monthly. The diagnosis of AGA is made when over 10% of the hair shorter than 3 cm are miniaturized, as hair miniaturization is progressive in this disorder.[49]

In AGA, the anagen phase gets shorter and shorter, producing more miniaturised hair. [48,50] The global number of shed hair shows the degree of hair loss (telogen effluvium when >100) and the percentage of vellus hair depicts the severity of androgenetic alopecia. The approach aids in the diagnosing telogen effluvium alongwith AGA, as well as determining which of the two illnesses is more severe and should be treated first. [50]

3. **60-S hair count test (wash test)**: The four phases in this approach are as follows: Comb your hair for 60 seconds before rinsing over a pillow or cover of contrasting colour to your hair, commencing at the back of the scalp and proceeding ahead towards frontal sector of scalp. Repeat it for three consecutive days with comb or brush. Reckon and record the quantity number of hairs in the comb or brush and onto pillow. Execute the method above monthly and report the findings to your dermatologist. [45,50]

Merits and demerits

It is tiresome and time-taking for the patient. But it can be performed by patients on their own and monitor their progress. The method is very subjective. The number 100 is arbitrary.

Diagnostic definitions cannot be put forth for wash test as vellus hair count on the surface does not correlate with actual vellus hair on scalp biopsy, as many vellus hair follicles do not make up to reach the surface.

Nonetheless, the patient finds it cumbersome to go 7 days without shampooing. Hair breaking during combing might explain the falsely increased numbers in 'daily hair counts' and '60-s hair counts.' Although simple and inexpensive to

execute, these evaluations are neither standardised nor diagnostic. They do, however, provide the doctor with an estimate of how much hair is shed.[50]

4. Hair pull test (Pull-out test, Sabouraud's sign, traction test)

A valuable auxiliary, qualitative test for assessing the extent of continued hair loss. Grabbing around 50–60 hairs, evaluator tugs them from the proximal to distal ends. A positive pull test and vigorous shedding are indicated by the removal of > 10% of the hair. The patient must not shampoo for at least a day prior to the pull test. Negative test in AGA, except at times in which there is substantial telogen hair loss in a pattern distribution. [51,52] Hair in anagen should stay anchored in place, but hair in telogen should fall out readily. Based on number of hairs plucked out, an estimate of proportion of hair follicles in a telogen stage can be made. So, if we pull on 20 hairs and only two come out, the percentage of telogen hair follicles is 10%. As a general rule, a telogen frequency of 10% is great, up to 25% is usual, and more than 35% is an issue [48]

Merits and demerits

It is based on the notion of 'gentle' hair tugging to promote telogen hair loss. It can be used to determine the extent and location of hair loss. In telogen effluvium (TE), anagen effluvium, loose anagen syndrome, early instances of patterned alopecia, and at the expanding fringe of alopecia areata (AA), the test is positive. The test is affirmative throughout the entire scalp in instances of acute TE, while it is positive over the thinning region in situations of AGA. [52] Even if the pull test does not indicate greater hair loss, the extraction of anagen hair with thicker root

sheaths clearly supports cicatricial alopecia. If anagen hair is present, the test is always labelled pathological. (53)

Hair pull tests vary from person to person. It is a very crude method and is not standardized as it is subjected to so much inter-individual variation among investigators. The pulling force may not be uniform. Negative tests do not exclude the diagnosis.(54)

5. The Fold Sign or Jacquet's Sign

Folding an area of the scalp in between the two thumbs forms the fold sign. The test is affirmative if multiple folds can be easily produced, indicating the lack of hair fibres in some, many, or all follicles, the presence of miniaturised hair (AGA), or the dearth of follicles (scarring alopecia). (55)

6. **Instrument-Aided Inspection**

A. Hair Growth Window: Using a razor or thin scissors and a perforated ruler, a 1-cm^2 patch of hair is shaved or clipped as near to the scalp as feasible. After a week, the length of the re-grown hair in this region is measured. Hair grows at typical pace of 2.5 mm each week (1 cm a month). This test determines hair growth, as well as to encourage patients that their hair is growing appropriately. Patients who dye on a customary basis can acquire the same information by measuring the distance between the roots and the point where the colour changes. (48)

B. Capillary Tube Method: The hair length is defined using a calibrated capillary tube encasing a hair shaft.

C. Global photography: Canfield's method has lately been validated. The use of a stereotactic positioning device, which fixes subjects chin and forehead and mounts a specific camera and flash device, ensures that the view, magnification, and illumination are consistent between study visits. In order to repeat baseline hair separating and combing in subsequent follow-up visits, keep the same hair style and colour alongwith same coordinators. The use of four conventional perspectives (vertex, midline, frontal, and temporal) is recommended. [56]

D. Dermoscopy and Videodermoscopy:

Scalp dermoscopy, termed 'trichoscopy,' a simple and non-invasive method that employs magnification using dermoscope for better assessment of scalp skin and hair. Dermoscopy optimizes diagnostic accuracy far more than basic clinical examination. With this methodology, clinical signs may be investigated in higher detail. [57] Videodermoscopy allows for quick, high-resolution viewing at a number of magnifications (up to 1000 times with advanced versions), as well as the potential to digitally record and store the examined photographs for subsequent application. [58]

Trichoscopic equipment: Trichoscopic equipment includes a handheld dermatoscope and a video-dermatoscope for trichoscopy. Ten-fold magnification is achieved using hand-held dermoscope magnification, while the video dermoscope provides magnification of up to 1000x. Handheld dermatoscopes are efficient in terms of both time and money. However, they lack greater magnifications and picture storage. To improve clarity and visibility, use water, ultrasound gels, aqueous gels, liquid paraffin, oil, and

alcohol. Individual preference determines which device to use and how much fluid to immerse in.

2–4 terminal hairs/ unit follicle alongwith 1–2 vellus hairs of consistent thickness and colour are present in a healthy scalp. The thickness and colour of a typical terminal hair are consistent over its entire length. White dots are the follicular apertures and eccrine sweat gland duct openings.

Dermatoscopic features definitive of AGA are [59]:

a) Hair diameter variance of > 20%.
b) In early AGA, peripilar sign appears as a brown depressed halo around the follicular opening.
c) In mature cases, sebum and keratin deposits within dilated follicular infundibula would appear as yellow dots.
d) As disease progresses, a honeycomb pigmented network can be beheld in solarexposed sites.

Criteria for FAGA laid down by Rakowska et al study includes:[13]

Major criteria:

1. at 70 x magnification > 4 yellow dots in 4 images in frontal area
2. average hair thickness in frontal is low as compared to occipital area
3. > 10% of thin hair in frontal region (< 0.03mm)

Minor criteria:

1. Frontal: occiput fraction of single hair per unit is >2:1.
2. Frontal: occiput fraction of number of vellus hair >1.5:1.
3. Frontal: occiput fraction of hair follicle with perifollicular discoloration >3:1.

Based on trichoscopy, two major or one major with two minor criteria are indicated for F-AGA diagnosis. The established trichoscopy criteria in this study allow for a 98 % specific diagnosis of FAGA. [13]

Findings in AGA with their significance includes following:[60]

1. Hair diameter diversity: characteristic of AGA and reflects transition of terminal hair into vellus hairs.
2. Brown peripilar sign (BPPS): brown halo of ~1mm around the hair follicle seen in former levels of AGA.
3. White peripilar sign (WPPS): white halo of ~1mm around the hair follicle, seen in severe grades.
4. Yellow dots (YD): corresponding to empty hair follicle with distended sebum, sighted in early along with severe grades, seen as yellow round or polycyclic dots.
5. Focal atrichia (FA): seen in later stages, designates areas of total hair loss, seen as pencil erased areas.
6. Honeycomb pigmentation (HCP): seen in all grades, appears as hypomelanotic zones bordered by hyperchromic lines (melanin in rete ridges).

Hu et al.[61] observed that HDD in 100% of patients in their study. HDD was also detected in the affected region of all AGA and FAGA instances by Inui et al., suggesting that HDD is an important aspect. [62] In study done by Ramatulasi et al[63] 93% of patients were reported to have HDD.

BPPS signifies histopathological presence of lymphocytic infiltrates around hair follicles.[12] Inui et al [62]reported peripilar signs in 20% of FAGA patients. In their work, Hu et al [61] discovered BPPS in 44.5 % of females with AGA. Zhang et al.

[12] found BPPS in 31.7% of patients and WPPS in 26.7% of cases in their study. Yellow dots in AGA represent sebum. Yellow dots were seen in 10% of FAGA patients, according to Inui et al. [62] The number of yellow dots in FAGA was restricted to 10 on the gross hair loss area.

Hu et al. [61] noted 24.0 % of female AGA patients had a varied number of yellow dots of several dimensions. Yellow dots could be seen in 1.67 % of patients, as per Zhang et al. [12] Focal atrichia, specified as zones of size of a pencil eraser, was shown to be positively associated with the advancement of AGA. Focal atrichia is caused by atrophic follicles, which were observed in 56.5 % of FAGA patients by Hu et al [61] In a study by Zhang et al [12], 56.7 % of the participants had focal atrichia. Also Zhang et al[12] correlated this finding with advanced stages of FPHL. Honeycomb pigmentation (HCP) on the scalp is caused by sun exposure and is characterised by hypomelanotic regions (less so in overlying dermal papillae) surrounded by hyperchromic lines (melanin of rete ridges), which is most frequently perceived in thinning or completely balding areas. HCP was reported in 30.5 % of female AGA patients, as per Hu et al. [61] HCP was documented in 61.7 % patients in a report by Zhang et al. [12]

PPS, Scalp pigmentation, focal atrichia, and white dots are all characteristic of PHL, as according to Zhang et al [12], and WPPS is a bad predictor of treatment. WPPS, scalp pigmentation, and FA have all been attributed to progressed FPHL.

E. Hair weight: Hairs in a specific target zone are snipped close to the scalp at reference, then permitted to regrow for a timeframe before being snipped close to the scalp, collected, and weighed to measure hair weight. [50]

F. Contrast felt examination: This technique is used to detect scalp hair that is short and tiny. Each side of an index card with black and white felt is used. The index card is held along the scalp after separating the hair. The margins of fine short hair with broken or tapering distal ends protrude. The androgen-dependent regions of both men and women with androgenetic alopecia may be seen with these miniature hair.[50,53]

G. Phototrichogram: alias Photographic trichogram. A baseline image is taken after all hair in a $2cm^2$ region is trimmed 1mm from the skin surface. This very same area is taken a week following, and the hairs are trimmed once more. This process is continued until there are enough images to compare. By comparing the baseline images, one can see which hairs have grown (anagen) and which have not (telogen), as well as the pace of hair development (the length of hair in 7 days).[64]

Procedure:

a. Hair clipping: 1 cm 2 stencils are used to outline the preferred region with a permanent marker. With curved surgical scissor, hairs in the targeted region are then cut in approximation to the scalp surface.

b. Hair diameter measurement: Clipped hair are placed over glass slide and dry mounted with a clear adhesive tape so that its diameter can be quantified under a microscope at a 40x resolution. Micrometre calibrated scale with a reading of 0.01 mm is used. Using the measuring eyepiece, the diameter of hair is measured closest to its base.

c. Contrast enhanced phototrichogram: Hair is dyed black right before the treatment begins. This approach is more sensitive for less pigmented and thin hair since the momentarily coloured hairs provide a stronger contrast against the white scalp.

d. Scalp immersion proxigraphy: Adding immersion oil to the region of interest and covering it with cover slip improves hair visualisation.

e. Trichoscan: In this method, the entire process of phototrichogram is computerised and the analysis is automated. An inbuilt dermoscope with an 20x-40x magnification is utilized for examination of hair. A defined area is shaved and growth of hair and other parameters as in phototrichogram are noted.

H. Confocal Microscopy of Hair: This is a non-invasive technique that projects high quality three-dimensional optical real time images of a transverse section of the skin and hair. This instrument has been mainly explored by researchers in the study of hair morphogenesis, and assessing the effects of various hair cosmetics.[65,66]

SEMI-INVASIVE METHODS

1. **Trichogram/ Hair pluck test**: On the fifth day after shampooing, hair is removed from specific places to do this test. Hairs around the region are fixed using clips and 60-80 hairs are kept in place using a haemostat coated with rubber. By twisting & lifting hair shafts in emerging direction, hairs are plucked. Hair shafts are clipped just 1 cm above the sheaths of root and are stacked on a slide side by side and taped together. [67]

To successfully complete the test, 50-100 hairs from the temparo-parietal, occipital, and vertex regions must be observed (around 25 hairs from each area).

Darkly pigmented triangle or delta-shaped bulbs with an angle to the hair shaft, as well as the presence of an inner root sheath, characterise anagen hair bulbs. Telogen hair has a club-shaped hair bulb and no inner root sheath and is less pigmented. Anagen and telogen hair are differentiated, and anagen to telogen ratios are determined. [68].

2. **Unit area trichogram (UAT):** A uniform fibre tip pen is used to indicate an area on the scalp. To minimise damage to the hair bulb, every hair inside and on the scribed line was epilated using forceps/tweezers in the direction of hair growth.

   UAT is more accurate than a traditional trichogram since it considers hair density and diameter. However, there are a few flaws. Hair plucking is a painful technique that causes hair destruction, resulting in dystrophic hair that is difficult to understand. Because of their tiny size, early anagen and vellus hair are often overlooked. [69,70]

3. **Trichometry:** Trichometry refers to the measurement of hair diameter as an objective way of assessing patients with hirsutism, hypertrichosis, or alopecia. Measurements of hair diameter above the ORS are more constant, and it is preferable to measure it in the proximal 40 mm of hair. Disparities in diameter measurements are common because of the oval cross-sectional area of the hair shaft and because of swelling of shafts due to moisture. Trichometry can be

accomplished with the use of dermoscopy and laser beam diffraction. A rough indication of hair diameter can be obtained by calculating the vellus hair index (vellus/terminal hair number) from shavings obtained from a standard sized area.[65]

INVASIVE TECHNIQUES

Transverse/horizontal section scalp biopsies are used for non-cicatrizing alopecia rather than longitudinal/vertical section biopsies. The transverse sectioning enables for the examination of a higher number of hair follicles. [71]

Under local anaesthesia using lignocaine with 1:1,00,000 adrenaline with biopsy punch of at least 4 mm diameter, which gives an effective diameter of 12.6 mm^2, the biopsy is completed. Deep biopsy including entire follicular unit, including some subcutaneous fat is considered adequate. Usually, biopsy involves a depth of 4 mm. In transverse section, sectioning is done at level 1 mm just below the dermo-epidermal junction, which corresponds to the opening of sebaceous gland ducts into the follicle, where the numbers of vellus hair are highest.[72]

Progressive miniaturization is the hallmark of AGA. Sebaceous glands seems to be enlarged in relation to the miniaturized hair follicles. The sequential deduction in anagen phase duration causes a relative increase in telogen hair.[44]

LABORATORY EVALUATION

i. Complete hemogram
ii. Total leucocyte count
iii. Differential count
iv. PCV

v. MCHC

vi. MCH

vii. Platelet count

viii. Serum ferritin

ix. T3, T4, TSH

x. Evaluation must also include serum free testosterone, serum DHEAS, prolactin, LH, FSH in women with irregular menses and hirsutism.

The role of biochemical profile in FPHL is debatable and not clear. Hormonal abnormalities usually are absent in most women with PHL. Androgen excess to be ruled out in cases with early onset and advanced stages of hair loss. The 2011 European consensus mentions free androgen index and prolactin as screening tests.

MANAGEMENT

The treatment options for FPHL are: [73]

1. Pharmacological drugs
2. Cosmetic aids
3. Surgical treatments
4. Experimental

A. PHARMACOLOGICAL

1. **Minoxidil**: When administered systemically for hypertension, minoxidil, a piperidinopyrimidine derivative, was found to produce hypertrichosis. It causes arteriolar dilatation through potassium channel-opening activity of minoxidil sulfate (active moiety).

   Minoxidil acts via:

- Vasodilatory effects
- Angiogenic effects
- Anti-androgenic effects
- Immunosuppressive effects
- Potassium channel opening capability
- Suppression of collagen synthesis
- Enhanced cell proliferation and DNA synthesis.[74]

2% topical minoxidil received approval by the Food and Drug Administration (FDA) for women with FPHL.[45,74,75,76]. The use of 5% minoxidil is recommended in women who failed to respond to 2% formulations and also in women who wants aggressive treatment.[76]

Minoxidil solution should be applied to the scalp rather than applying superficially over the hair. Twice daily application 12 hrs apart with a maximum of 1 mL at a time is recommended. The scalp should not be washed for at least 4 hrs. after application. The hands should be thoroughly washed after application.[75]

Patients are informed that a transient telogen effluvium may develop in the first 2-8 weeks, resulting in greater hair loss. But they are also reassured that the condition is self-limiting and wanes with anagen re-growth. Patients are advised to avoid discontinuing the treatment because of the temporary increased shedding.[73]

Contact dermatitis (dryness, scaling, and/or redness), facial hypertrichosis (fine hair) on the cheeks and forehead, and intensification of pre-existing dermatoses (seborrheic dermatitis, psoriasis) are all not uncommon adverse effects following use of molecule. It was found that most of the cases of allergic dermatitis were due to *propylene glycol*, one of the vehicles of the solution[73,75]. Newer foam vehicles have reduced propylene glycol content and are preferred by the patients because of less irritation and lack of sticky residues which are associated with current formulations.[77] Hypertrichosis fades post 4 months of discontinuation. [73] With only 1% to 4 % systemic absorption, systemic adverse effects usually unfollow. However, it should be used with caution in patients with hypertension and cardiac disease.[78] It belongs to category C pregnancy medication and there have been scattered reports of fetal malformations when it is used during pregnancy and hence it is better avoided during pregnancy and lactation.[79] Minimum 12 month treatment is must for efficacy appraisal. [74]

2. **Tretinoin**: Tretinoin works by lengthening the anagen phase and three-folding the percutaneous absorption of minoxidil, consequently it's been used in combination with minoxidil to treat AGA. There is a drastic improvement in cosmetic scalp coverage with the use of tretinoin (58% with tretinoin 0.025% alone; 66% with tretinoin 0.025% combined with 0.5% minoxidil).[80]

3. **Azelaic Acid**: 5α-reductase inhibition is the proposed mechanism and hence tried in AGA.[81]

4. **Ketoconazole**: Ketoconazole 2% has been found to disrupt the testosterone metabolism in the skin and hence can be a therapeutic agent for AGA. However, there are limited studies of its efficacy in AGA.[82]

5. **Topical finasteride**: Topical finasteride possess finer safety profile than oral finasteride. Once daily use of its 0.25% solution inhibits scalp DHT and may minimize the unsought sexual adverse effects associated to systemic DHT reduction. [83]

6. **Latanoprost**: Latanoprost, a prostaglandin analogue, is thought to promote hair growth by extending the anagen phase. Lengthening of eyelashes and eyebrows has been observed when latanoprost is used topically for glaucoma. Latanoprost vastly increases hair density against baseline.[74]

7. Topical formulations with limited efficacy evidence in AGA include – Carpronium chloride, t-flavanone, Cytopurine/pentadecane, Adenosine and Cepharanthine.[84]

SYSTEMIC THERAPY

1. **Finasteride**: Finasteride by blocking enzyme 5-α reductase II, prevents DHT formation from testosterone. Finasteride 1mg/day is frequently well accepted by postmenopausal women with FPHL. Breast soreness and increased libido are infrequent adverse effects that are more likely in the first year of therapy, and are reversible with dosage adjustment, and lessen with time with sustained usage.[73] It is contraindicated in pregnant women and women of childbearing age in general as it may lead to external genital

abnormalities and feminization of male fetus. It is pregnancy category X drug.[73,74,75]

2. **Cyproterone acetate**: It blocks DHT- androgen receptor binding and has progesterone and anti-gonadotrophic properties.[45] Treatment doses are varied but the most effective dose is 100mg/day given during 5-15th day of menstrual cycle supplemented by 50mcg ethinyl estradiol on days 5-25th. Menstrual cycle alterations, increased weight, reduced sexual desire, depression, soreness in breast, GI upsets.[73]

3. **Spironolactone**: It belongs to anti-androgen class of drugs, a competitive inhibitor of DHT- receptor binding and nuclear translocation. Major metabolites are canrenone and potassium canrenone with half-life of nearly 20 hours and are eliminated via biliary route.[45]

   The minimal effective dose is 100mg/day a dosage based more on the treatment of hirsutism than alopecia. There are no studies to address a dose effective in FPHL and published results of >100mg/day are primarily on cohorts of fewer than 10 women.[45]

   Postural hypotension, electrolyte imbalances, menstrual alterations, fatigue, urticaria, breast soreness and haematological disturbances are the possible adverse effects.[45,73]

4. Flutamide: A potent anti-androgen with its metabolite 2-hydroxy-flutamide, it inhibits binding of testosterone to the androgen receptor. Flutamide when given at as 250mg/day for a year could show greater

improvement in hair loss. Side effects includes breast tenderness and hepato-toxicity.[45,73]

5. Estrogens: Both oral and topical are tried in women although no controlled studies have been done. Topical 0.025% 17-alpha-estradiol preparation appeared to stabilize hair loss when applied for 6 months by 7 women with AGA compared to 2 female control.[45] [75]

B. COSMETIC AIDS

AGA patients can employ hair extensions, prosthetics, and wigs to improve their cosmesis. Prosthetic hair implant with artificial hair is avoided in fear of increased adverse effects. The fibre diameter is one of the most critical aspects in the aesthetic look of thinning hair. A topical mixture of caffeine, niacinamide, panthenol, dimethicone, and an acrylate polymer (CNDPA) has been demonstrated to enhance hair fibre diameter, which can assist individuals with thinning hair improve their aesthetic look.[84]

C. NEWER AGENTS

1. **Botulinum toxin**: Improvement in androgenic alopecia was noted when botulinum toxin 150 units (dose divided over 30 injection sites) was injected into the muscles around the scalp in few research. Botulinum toxin is thought to relax scalp muscles, lowering pressure on the perforating vasculature, and enhancing blood flow and oxygen concentration. This improves AGA because low-oxygen settings favour DHT formation whereas high-oxygen settings favour estradiol formation from testosterone.[85]

2. **Melatonin** (N-acetyl-5-methoxytryptamine): It was initially discovered as a pineal gland-produced and released circadian-rhythmed neuro-hormone. Melatonin demonstrates protective and anti-apoptotic effects due to its potent antioxidant properties and proclivity to trap free radicals, preserving the functional capability of non-neoplastic cells. Melatonin is produced by human hair follicles, and melatonin receptors are expressed and influence hair cycles. When compared to placebo, a double-blind, placebo-controlled pilot study using topical 1 mL of a 0.1% melatonin-alcohol solution reported a marked increase in detectable anagen hair in the occipital and frontal region after 6 months in women and men with early stage AGA and diffuse alopecia. [86]

3. **Botanical extracts**

I. Saw palmetto (*Serenoa repens*): The berries of the palm tree saw palmetto (alias Serenoa repens (SR), Serenoa serrulata, or Sabal serrulata) are used to make saw palmetto extract. Belonging to Arecaceae family, a medicinal herb in the United States, and native to the West Indies, it is a 5α-reductase (5R) inhibitor occurring naturally. It cannot be found in India and must be imported from the United States. Plentiful Carotenoids, lipases, tannin, sugars, and fatty acids are found in the pure extract of SR, which includes 85-90% fatty acids and sterols. The mode of action is similar to that of finasteride, in that it suppresses 5α-reductase. DHT absorption by the hair follicle is also decreased by SR, resulting in less DHT binding to androgenetic receptors. Saw palmetto's liposterolic extract (LSESr) with beta sitosterol combination might improve AGA. [87]

Saw palmetto lotion and pills are given for a duration of six months. There are two different sorts of herb palmetto supplements in the market. One type is dried saw palmetto berries, while another is saw palmetto extract in tablet form. The suggested dose is 160 mg twice daily. With satisfactory results, treatment might be maintained for up to six months. (88)

In a three-month trial, topically administered SR extract in lotion and shampoo base showed a 35% increase in hair density and a 67% reduction in sebum. (89)

In another research, all patients (100%) reported an improvement in symptomatology related with AGA, with 84% reporting cosmetically significant changes.(87)

II. Reishi (Ganoderma lucidum): Red reishi, also known as Ling Zhi in China. It is a mushroom which has multiple health benefits.

Significant 5-α reductase inhibition and thus preventing potent androgen formation by reishi mushroom is suggested by various studies. (90)

III. Pumpkin seed oil (PSO): It is a kind of vegetable oil derived from pumpkin seeds. It limits 5α -reductase activity and has anti-androgenic qualities in rats. Phytosterols, which block 5α-reductase, are responsible for its activity. PSO supplementation had a favourable anabolic impact on hair growth throughout a 24-week randomised, double-blind, placebo-controlled study. (91)

IV. Green Tea (Camellia sinensis): Epigallocatechins in green tea blocks the 5α-reductase conversion of testosterone to DHT, making it a powerful

anti-androgen. This anti-androgen mechanism might help prevent benign prostatic hyperplasia (BPH), acne, and balding. [90]

V. Lauric acid and myristic acid: These belong to the category of inhibitors of enzyme 5*α*-reductase. Lauric acid is a 12-carbon atom chain saturated fatty acid produced from plants such as coconut oil, is a 5R I and II inhibitor. Myristic acid a 14-carbon saturated fatty acid that may be found in botanicals like nutmeg butter and coconut oil. It's also a 5α-reductase II inhibitor. [92]

4. Low – level laser (light) therapy (LLLT)

It is the use of low-power light (between 1mW and 500mW) to boost tissue regeneration, decrease inflammation, and alleviate pain in a diseased location. The light has a small spectral bandwidth and a power density of 1mw-5W/cm^2 in the red or near infrared (NIR) spectrum (600-1000 nm). Hairmax Laser Comb was recognized by the US FDA in 2007 and granted 510 K clearance in 2011 as a safe therapy for the treatment of male AGA and female AGA.[93] Laser phototherapy excites anagen hair re-entry into the telogen hair follicles, lengthens the duration of anagen phase, also increases the rates of proliferation in active anagen and prevents development of premature catagen hair. LLLT may boost anagen hair via nitric oxide (NO) released from cytochrome *c* oxidase by photodissociation.[94]

5. Platelet–rich plasma (PRP)

PRP is a concentrate of human platelets in small volume of plasma. It has 4-7 times higher concentration of platelets than the baseline. PRP is obtained from

the patient's own blood by centrifugation and is injected into the sites of alopecia subcutaneously. Ubel conducted an experiment in 2005 where he studied 23 patients of hair transplant after enriching hair root grafts with PRP and without PRP. After one year region implanted with PRP enriched grafts showed yield of 18.7 FU/cm^2 as opposed to 16.4 FU/cm^2 of region without PRP. Therefore it concluded as a safe cheap and non-allergic adjuvant in management of AGA.[95]

6. Stem cells

Role of stem cells in AGA is still being evaluated. While stem cell therapy is already being used in many places, but there is no significant data to support clinical use of stem cells at present. Animal studies have shown that bio-engineered hair follicles obtained from stem cells are efficacious. Adipose-derived stem cells produce a variety of cytokines that encourage hair development. Studies have been done using adipose-derived stem cell-conditioned medium to treat alopecia.[96]

D. SURGICAL TREATMENTS:

1. Hair transplantation

2. Scalp flaps:

a) Rotation flaps.

b) Transposition flaps.

c) Temporo-parietal-occipital (pedicled)/Jari flaps.

d) Temporo-parietal-occipital (free/microvascular flaps).

e) Lateral scalp flaps.

f) Temporal vertical flaps and other flaps.

3. Alopecia reduction / Scalp reduction

a) Simple alopecia reduction.

b) Major alopecia reduction or scalp lift.

c) Major alopecia reduction with prior scalp extension (non-volumetric).

d) Alopecia reduction with prior tissue expansion (volumetric).

e) Alopecia reduction with intra-operative stretching (volumetric and non-volumetric).

Hair transplantation[97]: In 1984, Headington published a paper on follicular units which provided a logical basis for future hair transplantation techniques and methods. He established that hair does not grow in single strands, but in clusters known as follicular units.

Each unit consists of:

A. Terminal follicles (1-4).

B. Vellus follicles (1, rarely 2).

C. Sebaceous lobules and insertions of arrectores pili.

D. Perifollicular vascular plexus and neutral net.

E. Perifollicular collagen.

Follicular unit transplantation (FUT) has become the gold standard for hair transplantation as an outcome of Headington's observations. FUT is a hair restoration operation in which hair is grafted in its natural form, in individual follicular units (FUs).

Newer methods of hair transplantation have been developed. These include[97]:

a) Follicular unit extraction (FUE): The basic principle of FUE is to extract an intact unit through a hole which is as small as possible which would heal with minimal and inconspicuous scarring. This is also referred to as suture less method of transplantation.

b) Trichophytic closure: It is the stereo surgeon’s response to FUE to meet the demand of a better donor scar.

c) Body hair transplantation: First reported by Wools and depends on principle of recipient influence as suggested by Tommy Hwang. He demonstrated that transplanted body hair would grow longer and thicker in scalp.

d) Use of Robotics: Since the hair transplant surgery is repetitive, it has been thought that the procedure is ideally suited for a robotic application. Recently a machine called ARTAS has been introduced to do robotic assisted Follicular Unit Extraction.

Women with good hair density in the donor site over the occipital scalp and substantial hair loss or thinning of the frontal scalp are ideal surgical candidates for hair transplantation. The procedure is usually done under local anaesthesia and single

session involves transplantation of 00-1200 grafts. In women 1-3 sessions spaced around 6 months apart are undertaken depending on the hair loss severity and donor sites availability.[73]

Local complications of the procedure include:[73]

i. Facial edema

ii. Scalp erythema

iii. Crusting

iv. Post-operative bleeding

v. Infection

vi. Sterile folliculitis

vii. Transient headaches and numbing of the scalp

viii. Abnormal scarring of the graft

ix. Occasionally with densely packed grafts, temporary telogen effluvium may occur for a few weeks after the procedure.

Hair transplantation is frequently disregarded in FPHL and is not as popular as it is in male AGA, although professional opinion suggests that many women can anticipate outcomes that are at least as excellent as those found in early male AGA .[73]

MATERIALS & METHODS

The details of the methodology are described below:

- **Study source**: The study was conducted in the Department of Dermatology, Venereology and Leprosy, in tertiary care hospital, Belgaum as a part of the MD academic curriculum.
- **Study duration**: The study was conducted between 1st January 2020 to 31st December 2020
- **Ethical clearance:** Clearance was taken from the Ethical Committee of the institute.
- **Study design:** Hospital based cross-sectional study.
- **Sample size:** Sample size was calculated from the formula below:

$$n = \frac{p(100-p)Z^2}{E^2}$$

- n is the sample size required, **p** is the percentage occurrence of a state or condition (proportion or prevalence), **E** is the percentage maximum error required, **Z** is the value corresponding to level of confidence required.
- There is a prevalence of 15.3% of diffuse hair loss among women, this prevalence is used to calculate sample size. With 95% confidence level and 10% of maximum error sample size required is,

$$n = \frac{15.3 \times (100-15.3) \times (1.96)^2}{10^2}$$

$$n = 49.78368 \approx 50$$

- **Hence minimum sample size required is 50 with the prevalence of 15.3%.**
- Hence, according to the sample size and also considering the total number of patients attending the OPD during the study period a total of 110 patients were taken into the study.

A complete history was obtained, and a comprehensive medical examination was done for every patient and recorded in a specially prepared proforma. Photographic records were maintained. Permission from institutional ethical committee and informed consent was procured from patients participating in the study.

Inclusion criteria:

- All consenting female patients with grade I,II,III of Ludwig's grade of FPHL and male type of fronto-temporal recession (FAGA-M).
- Age group 18-60yrs

Exclusion criteria:

- Any active infection of the scalp, malignancy, on chemotherapy or radiotherapy, suffering from acute illness
- Non -consenting patients.

PROCEDURE OF THE STUDY

After obtaining the informed consent and ethics committee permission, all patients presenting the Dermatology OPD who satisfied the specified inclusion and exclusion criteria were recruited for the study. Patients showing thinning of hair over

the crown, temporal area, recession of hair line, and widening of the central partition were classified to have female pattern hair loss.

CLINICAL HISTORY

All patients had a full history taken using the standard questionnaire, with an emphasis on the onset and duration of hair loss, any related symptoms (itching, discomfort in the scalp, scaling of the scalp), and any exacerbating variables. A complete history of lifestyle changes was taken, including addiction histories and sleep patterns.

Diabetes, hypertension, hypothyroidism, hyperthyroidism, and anaemia were all mentioned in the past. History of intake of any oral or topical medications was asked. Family history of hair loss amongst mother, sister and aunt was obtained. An organised proforma was used to document everything.

CLINICAL EXAMINATION

A detailed clinical examination was done and presence of pallor, icterus, clubbing, cyanosis, edema, lymphadenopathy, seborrheic dermatitis, folliculitis, other scalp lesion or infection was looked for.

Acanthosis nigricans, acne and hirsutism were checked as markers of hyperandrogenism.

Pattern of hair loss was looked for and graded using the Ludwig scale into Ludwig I (LW I), Ludwig II (LW II), and Ludwig III (LW III), as well as male type fronto-temporal recession (MT).

After subjecting each patient to the traction or the hair pull test, trichoscopy using a non-contact polarized dermoscope at 50-fold magnification was followed at hair loss sites in all enrolled females and the results were related to the occipital region (served as control region). The relative dermoscopic variations in variables in frontal and occipital region areas were only recorded as pattern hair loss more often than not affects the frontal scalp.

Patients with identical occipital and frontal dermoscopic findings were presumed to have telogen effluvium instead of FPHL and were therefore omitted from the analysis.

Trichoscopic assessment of patterned hair loss should be carried in the fronto-parietal area, about at the intersection between the nose line and ear implantation line.[98] Trichoscopic assessment of the temporal region, according to Rakowska et al., may be avoided in practice. [13] In addition, the frontal area had greater pattern hair loss–trichoscopic alterations than the occipital region. At 50x magnification, the frontal and occipital scalps were investigated in the center (sagittal plane) 3 cm just above hairline. All the images evaluated for the hair changes are presented below.

Details of criteria used to describe trichoscopic findings

1. Variability in hair diameter- Presence of hairs of distinction width i.e., small, medium, and terminal hair in one field of vision. (As image is 2-dimensional width of hair shaft is used instead of diameter)
2. Peripilar sign- Brown peripilar sign (BPPS)

   White peripilar sign (WPPS)

   Halo of ~1mm around the hair follicle

3. White dots- Pin point white dots
4. Single hair per follicular unit
5. 2-3 hairs per follicular unit
6. Yellow dots- Empty follicles seen as yellow dots
7. Focal atrichia ~ Size of pencil eraser which represents area of total hair loss over scalp
8. Honeycomb pigmentation
9. Scaling.

RESULTS AND OBSERVATIONS

METHODS:

R 4.0.3 and Excel statistical softwares are being used for analysis of data. Continuous variables are represented by mean ± SD form and categorical variables by a frequency table. Chi square test is used to see the association between two categorical varaibles. Kruskal Wallis test is used to compare distributions between more than two groups. p-value less than or equal to 0.05 indicates the significance.

Table 1: Age distribution

Age group	No of Cases	Percentage
18 – 30	51	46.36
31 – 40	30	27.27
41 – 50	27	24.55
>50	2	1.82

Graph 1: Distribution of subjects by age group

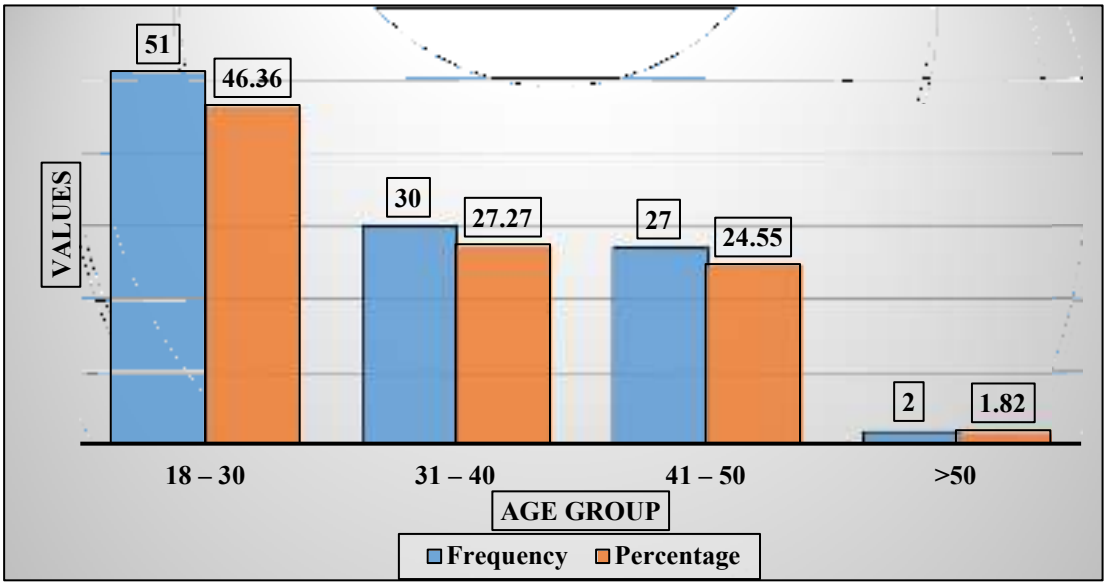


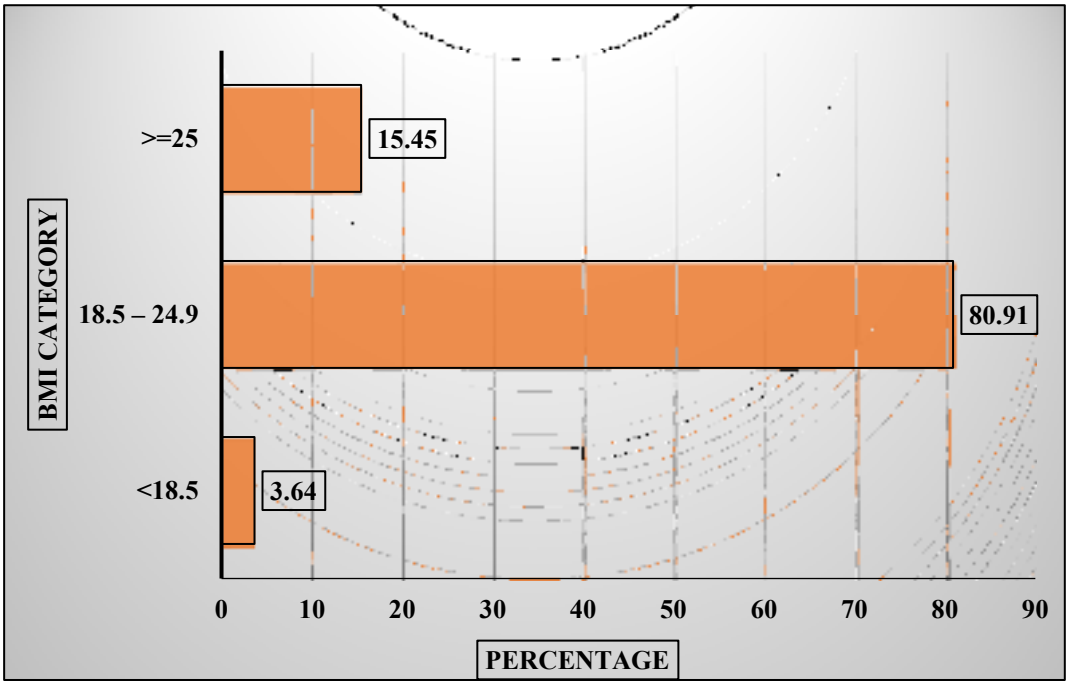
Table 1 and Graph 1 suggests that out of 110 patients 51 patients (46.36%) were in the age group of 18-30 years, 30 patients (27.27%) were in the age group of 31-40 years, 27 patients (24.55%) were in the age group of 41-50 years and 2 patients (1.82%) were in the age group >50 years. Thus, majority of patients were clustered in 18-30 years of age group.

The study had candidates with age **32.94 ± 9.26 years**. In our study youngest patient was 18 years old while oldest was 52 years old

Table 2: BMI distribution

BMI category	No of Cases	Percentage
<18.5	4	3.64
18.5 – 24.9	89	80.91
>=25	17	15.45

Graph 2: Distribution of subjects by BMI

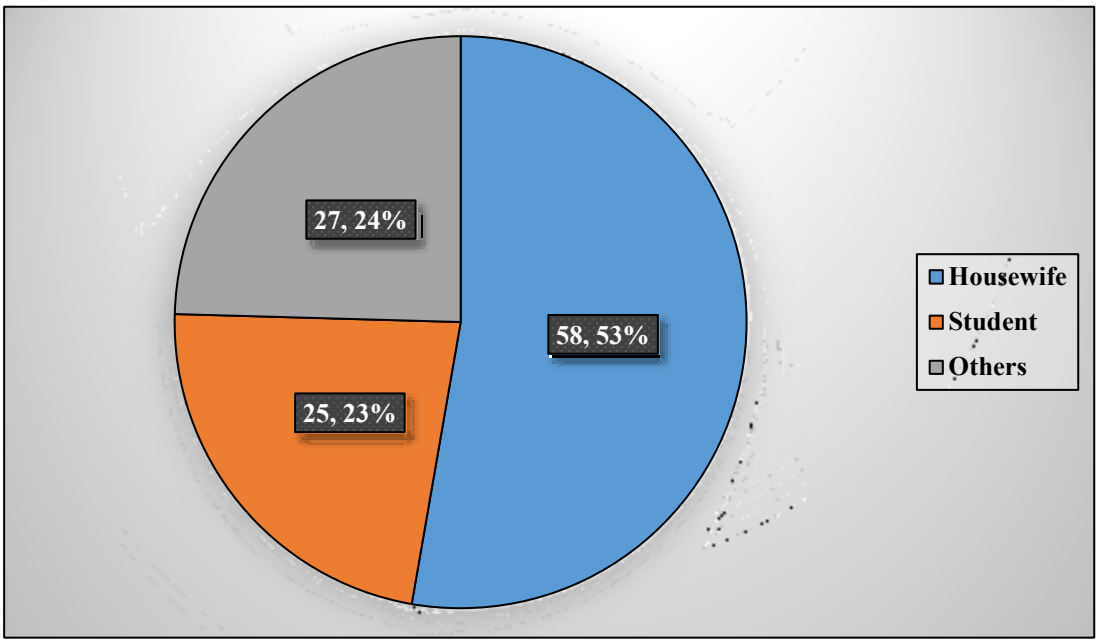


Out of 110 patients 4 patients (3.64%) were in the BMI category of <18.5, 89 patients were (80.91%) in the BMI category of 18.5 – 24.9, 17 patients (15.45%) were in the BMI category of ≥25. Thus, majority of patients were clustered in 18.5 – 24.9 BMI category. The mean BMI was found to be 21.97±2.71

Table 3 Occupation Distribution

Occupation	No of Cases	Percentage
Housewife	58	52.73
Student	25	22.73
Others	27	24.55

Graph 3: Distribution of subjects by Occupation

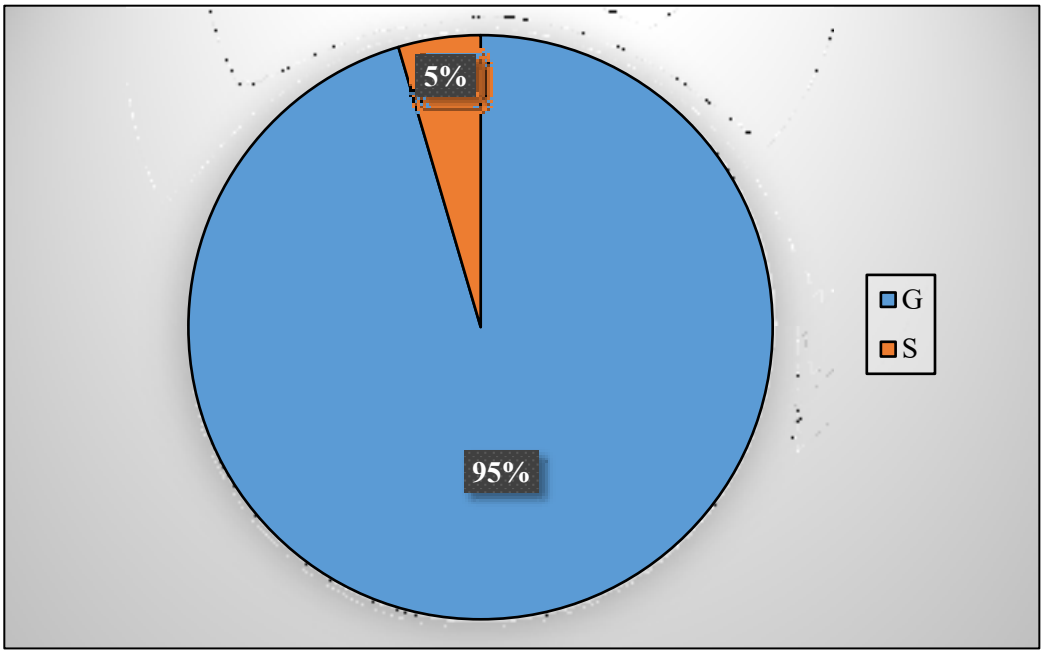


Above data (table 3 and graph 3) suggests that majority of patients 58 patients (52.73%) were housewives, followed by students 25 patients (22.73%) and rest 27 patients (24.55%) were engaged to other occupations.

Table 4: Onset of hair loss

Onset of hair loss	No of Cases	Percentage
Gradual	105	95.45
Sudden	5	4.55

Graph 4: Distribution of subjects by Onset of hair loss.

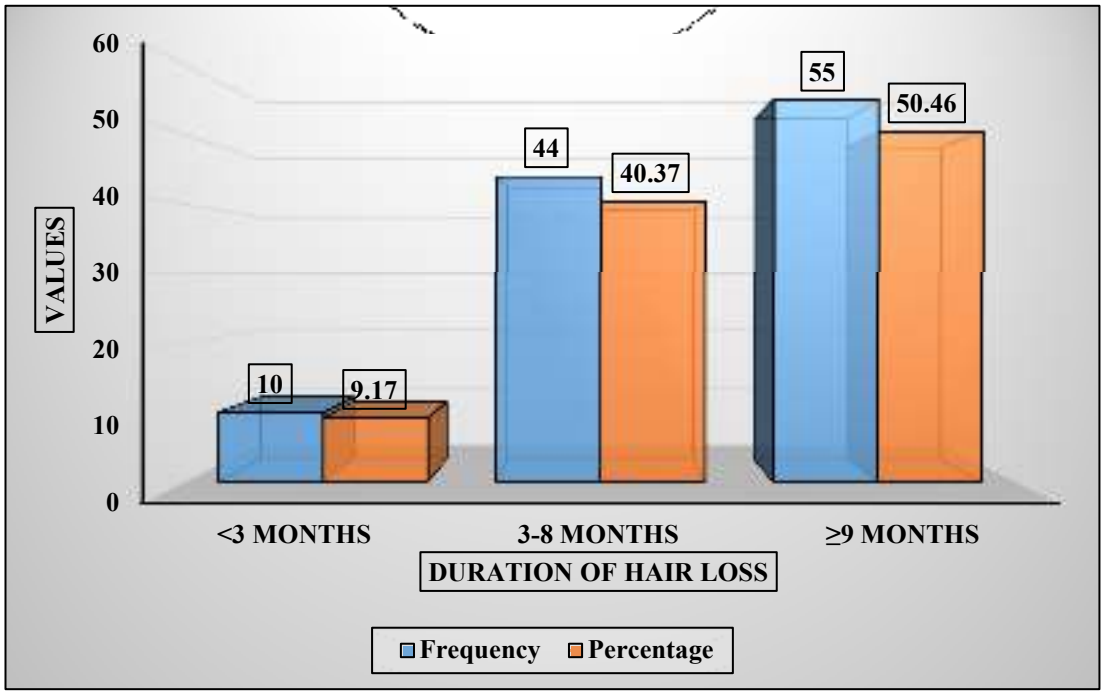


Above data (table 4 and graph 4) depicts in 105 patients (95.45%) had gradual onset of hair loss and 5 patients (4.55%) had sudden onset of hair loss.

Table 5: Duration of hair loss

Duration of hair loss	No of Cases	Percentage
< 3 months	10	9.17
3-8 months	44	40.37
≥ 9 months	55	50.46

Graph 5: Distribution of subjects by duration of hair loss

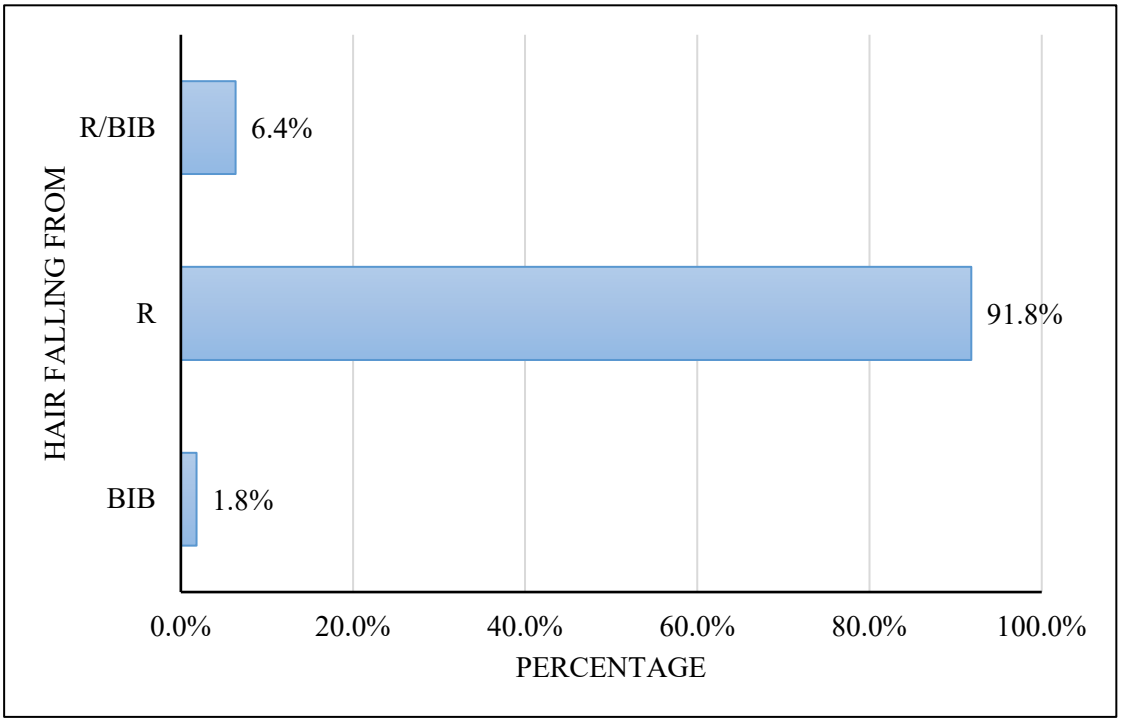


Above data (table 5 and graph 5) suggests that 44 patients (40.37%) had 3-8 months duration of hair loss, 55 patients (50.46%) had duration of > 9 months and 10 patients (9.17%) had duration of less than 3 months.

Table 6: Site of hairfall

Hair falling from	No of Cases	Percentage
BIB	2	1.82
R	101	91.82
R/BIB	7	6.36
(*BIB- breaking in between, R-roots)		

Graph 6: Distribution of subjects by Hair falling from.



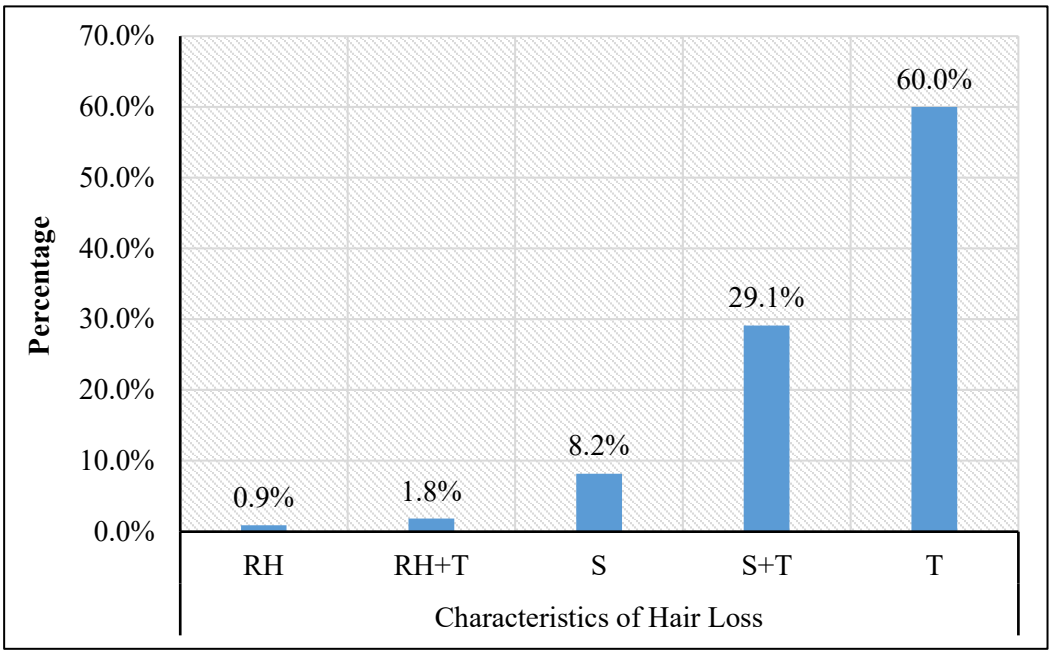
Above data (table 6 and graph 6) highlights those 101 patients (91.82%) complained of hairs falling from roots and 7 patients (6.36%) complained of both hairs falling from roots and breaking in between and only 2 patients (1.82%) complained of hair breaking in between alone.

Table 7: Characteristic of hair-loss

Characteristic of hair loss	No of Cases	Percentage
RH	1	0.91
RH+T	2	1.82
S	9	8.18
S+T	32	29.1
T	66	60.00

(*RH- recession of hair line, T-Thinning, S shedding)

Graph 7: Distribution of subjects by Characteristics of Hair loss.

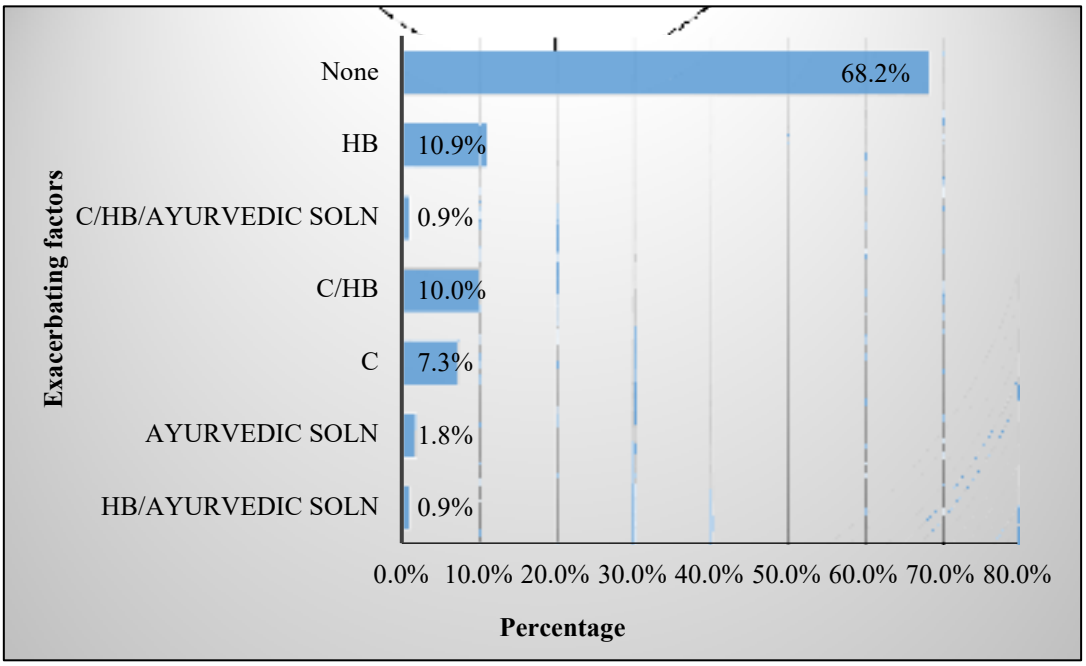


Above data (table 7 and graph 7) suggest majority of 66 patients (60%) complained of hair thinning followed by 32 patients (29.09%) complained of both shedding and thinning, 9 patients (8.18%) had complained of shedding alone.

Table 8: Exacerbating factors

Exacerbating factors	No of Cases	Percentage
C	8	7.27
C/HB	11	10.00
C/HB Ayurvedic SOLN	1	0.91
HB	12	10.91
HB Ayurvedic SOLN	1	0.91
N	75	68.18
N Ayurvedic SOLN	2	1.82
(*C- combing, HB- head bath, soln- solution)		

Graph 8: Distribution of subjects by exacerbating factors.

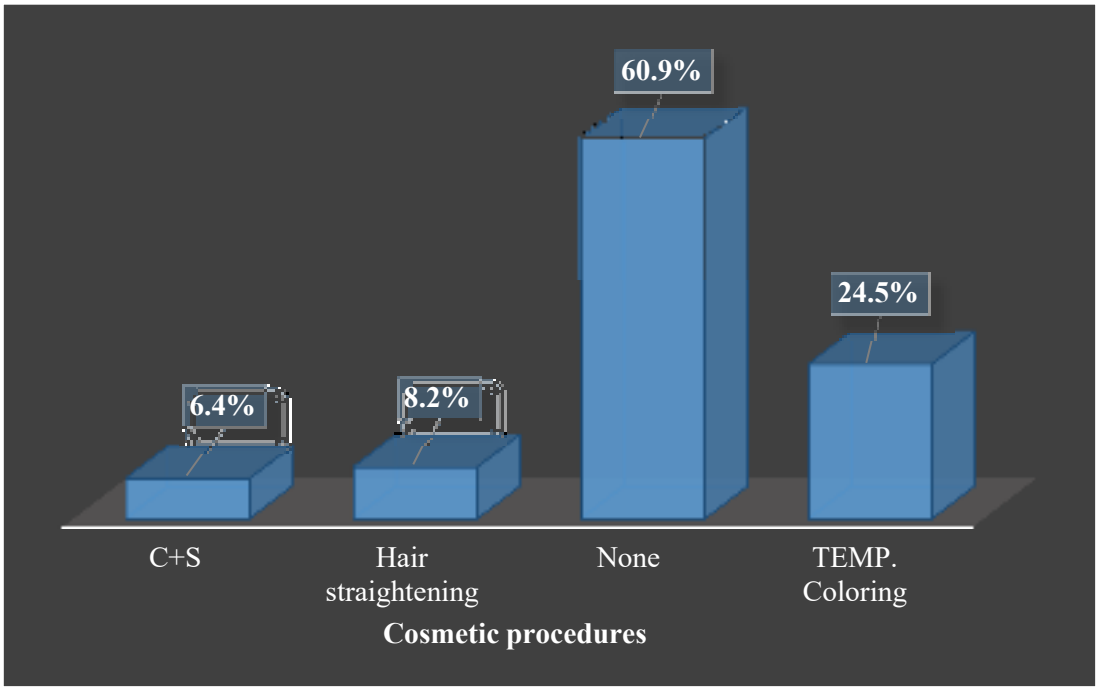


Above data (table 8 and graph 8) suggests that 75 patients (68.18%) studied had no exacerbating factors; however small proportion i.e., 8 patients (7.27%) witnessed increase hair loss while combing hair or during head bath.

Table 9: Cosmetic procedures

Cosmetic procedures	No of Cases	Percentage
C+s	7	6.36
Hair straightening	9	8.18
None	67	60.91
Temp. Coloring	27	24.55

Graph 9: Distribution of subjects by cosmetic procedures.

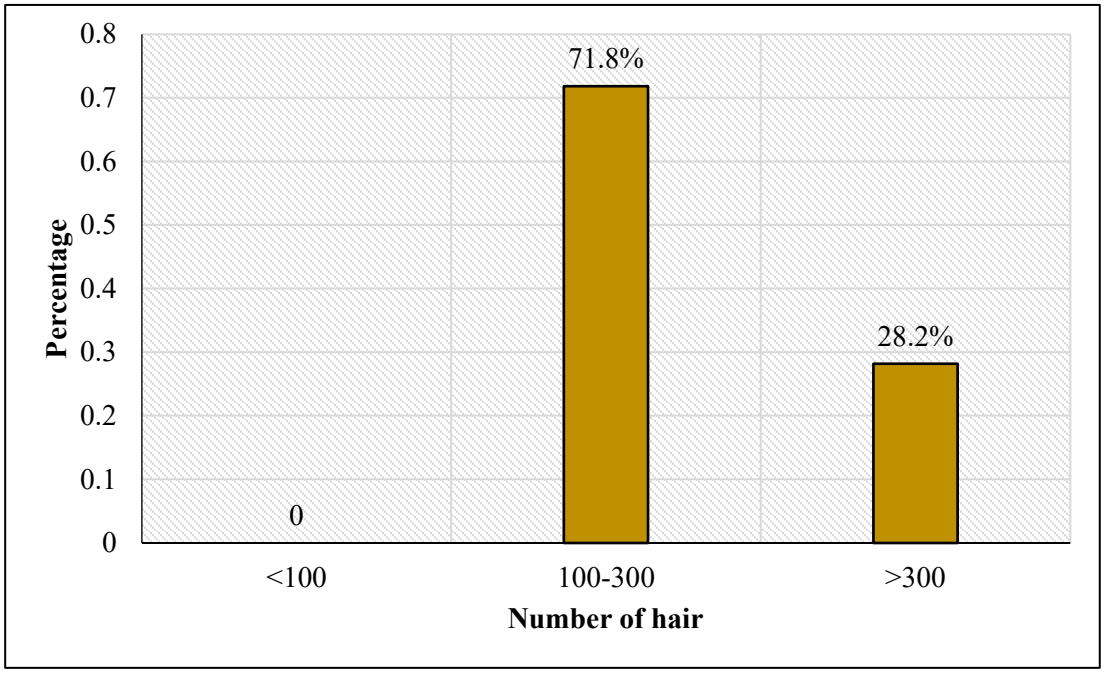


Above data (table 9 and graph 9) suggests that 67 patients (60.91%) studied did not indulge in any sort of parlour activities like hair colouring or straightening, 27 patients (24.55%) were indulged in temporary colouring of hairs and 9 patients (8.18%) were undergoing hair straightening. 7 subjects (6.36%) had undergone both colouring & straightening.

Table 10: Number of hair lost per day

Number of hair lost per day	No of Cases	Percentage
<100	0	0
100-300	79	71.82
>300	31	28.18

Graph 10: Distribution of subjects by No. of hair lost per day

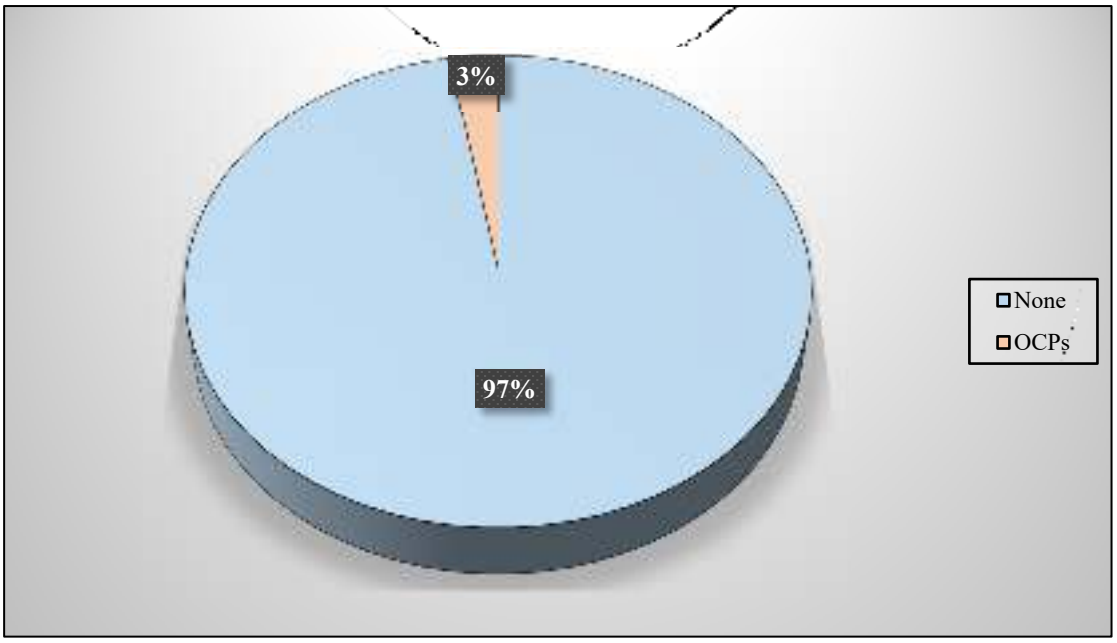


Above data (table 10 and graph 10) shows that majority 79 patients (71.82%) complained that on an average they lose about 100-300 hairs per day and 31 patients (28.18%) witnessed more than 300 hair loss per day.

Table 11: Drug history

Drug history	No of Cases	Percentage
N	107	97.27
OCPS	3	2.73
OCPS: Oral contraceptive pills		

Graph 11: Distribution of subjects by Drug history.

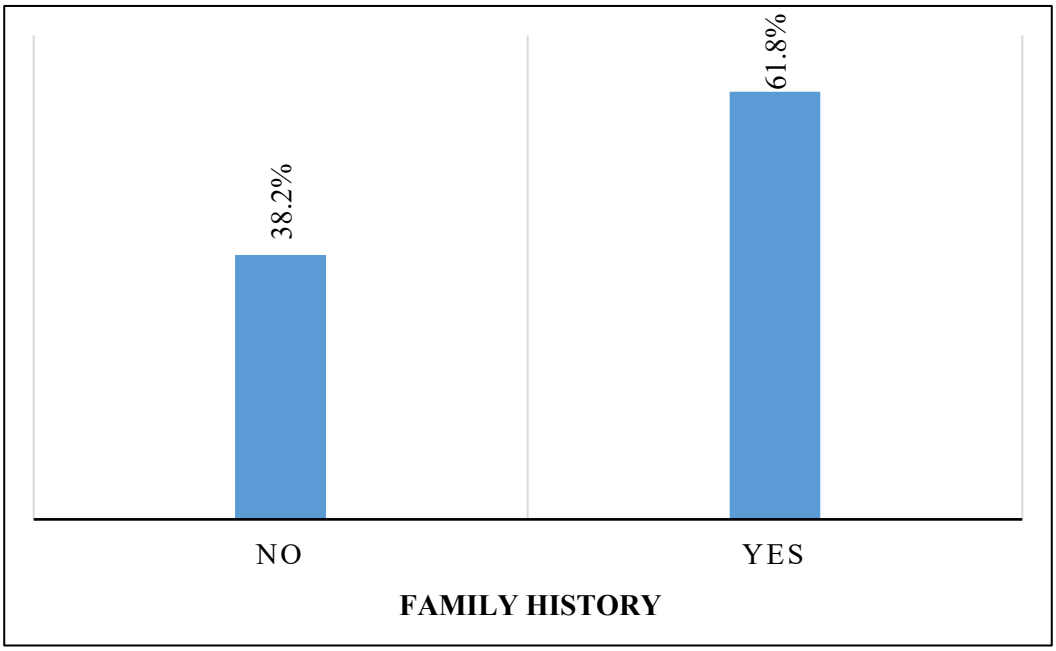


Above data (table 11 & graph 11) depicts majority of patients studied 107 patients (97.27%) were not on any drug treatment, 3 patients (2.73%) were on OCPs.

Table 12: Family history of hair loss

Family history	No of Cases	Percentage
No	42	38.18
Yes	68	61.82

Graph 12: Distribution of subjects by family history of hair loss.

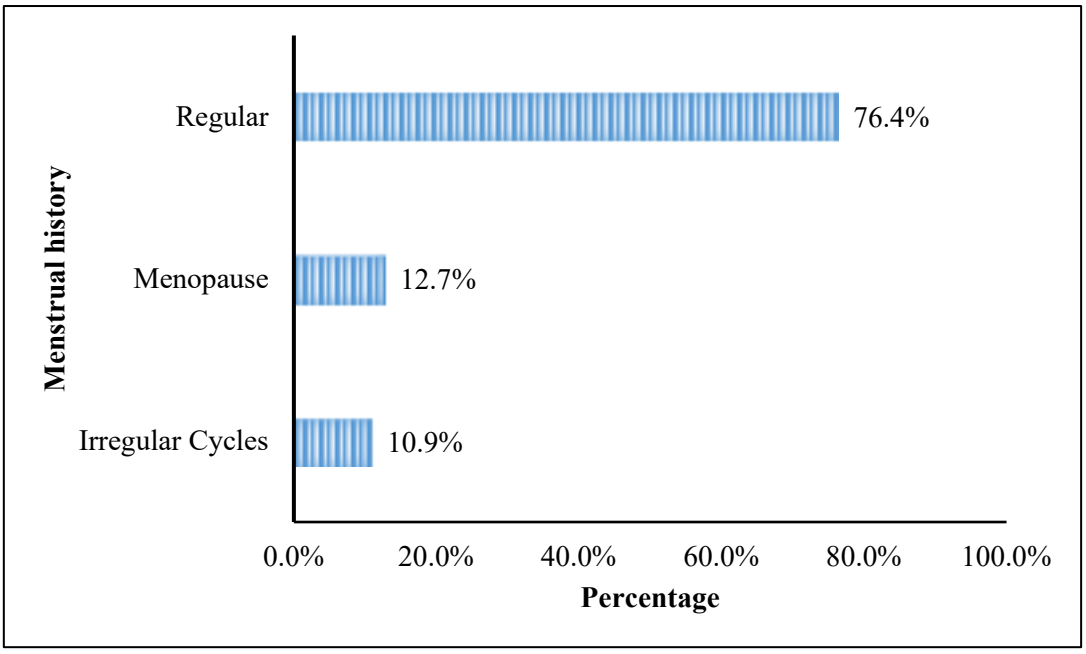


Above data (table 12 & graph 12) suggests that 68 patients (61.82%) had positive family history of hair loss and 42 patients (38.18%) did not report any familial history.

Table 13: Menstrual status

Menstrual status	No of Cases	Percentage
Irregular Cycles	12	10.91
Menopause	14	12.73
Regular	84	76.36

Graph 13: Distribution of subjects by menstrual status.

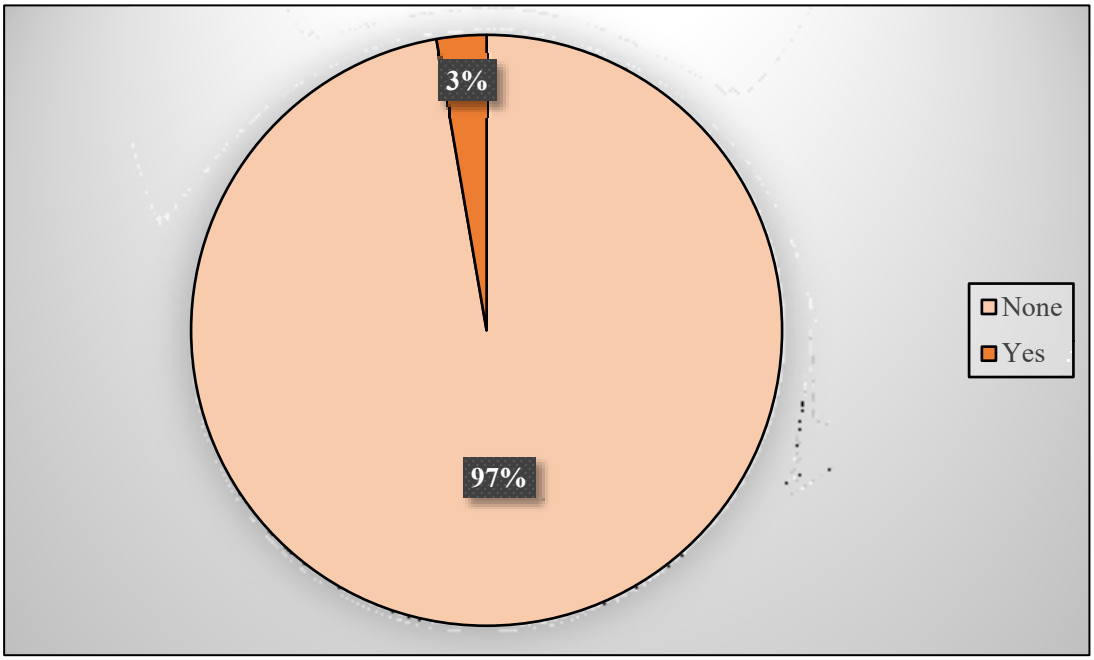


Above data (table 13 & graph 13) indicates majority of patients studied, 84 patients (76.36%) had regular menstrual cycles, 12 patients (10.91%) had irregular cycles and 14 patients (12.73%) had attained menopause.

Table 14: History of Infertility

Infertility	Cases	Percentage
No	67	60.91
Not applicable	40	36.36
Yes	3	2.73

Graph 14: Distribution of subjects by history of infertility.

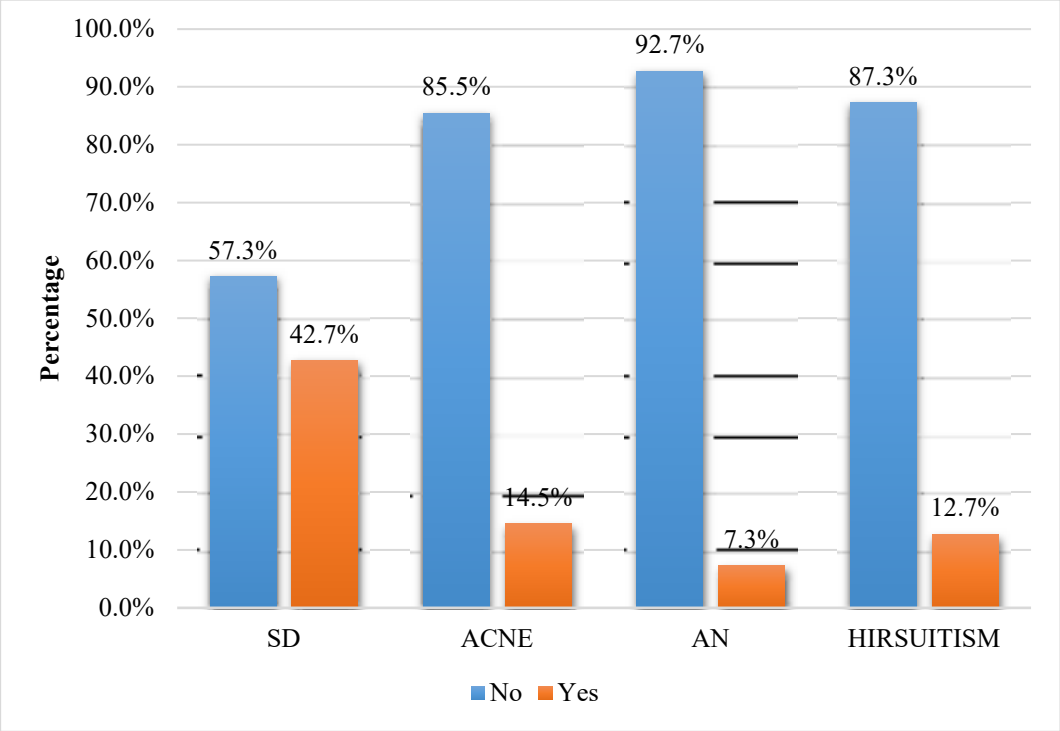


Above data (table 14 and graph 14) suggests that only 3 patients (2.73%) had history of infertility, 40 patients (36.36%) were unmarried and the rest did not have any history suggestive of infertility.

Table 15: Associated Clinical features.

Seborrheic dermatitis (SD)	Cases	Percentage
No	63	57.27
Yes	47	42.73
Acne		
No	94	85.45
Yes	16	14.55
Acanthosis Nigricans (An)		
No	102	92.73
Yes	8	7.27
Hirsutism		
No	96	87.27
Yes	14	12.73

Graph 15: Distribution of subjects by Associated clinical features.

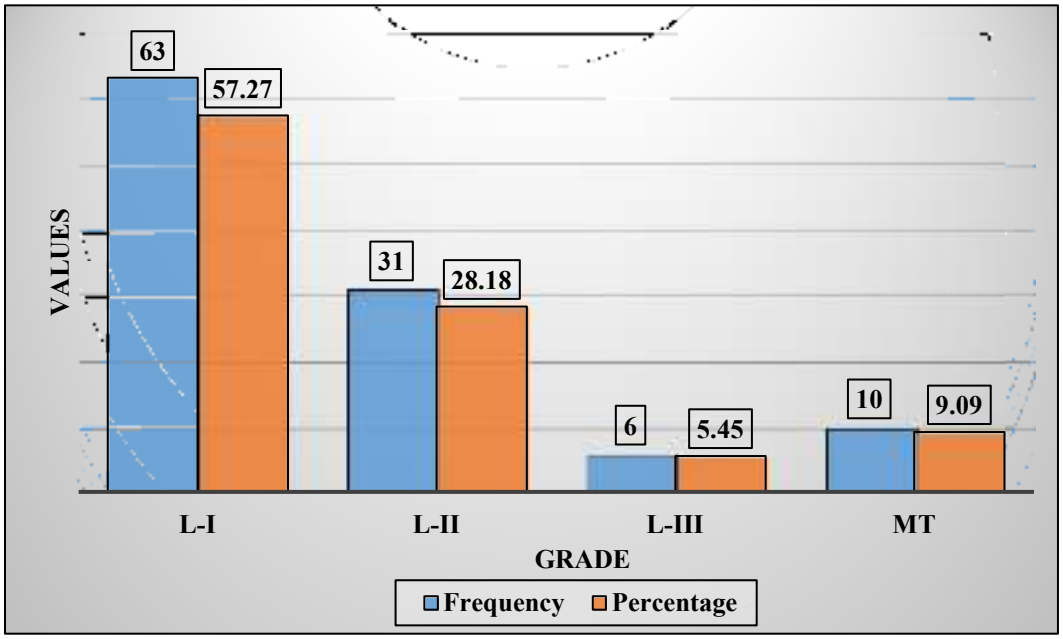


Above data (table 15 & graph 15) suggests that 47 patients (42.73%) had associated seborrheic dermatitis, 16 patients (14.55%) had acne, 8 patients (7.27%) had acanthosis nigricans and 14 patients (12.73%) had hirsutism.

Table 16: Grading of Hair Loss

Grade		
L-I	63	57.27
L-II	31	28.18
L-III	6	5.45
MT	10	9.09
L-I: Ludwig I, L-II: Ludwig II, L-III: Ludwig III, MT: Male Type		

Graph 16: Distribution of subjects by Grade of hairloss.

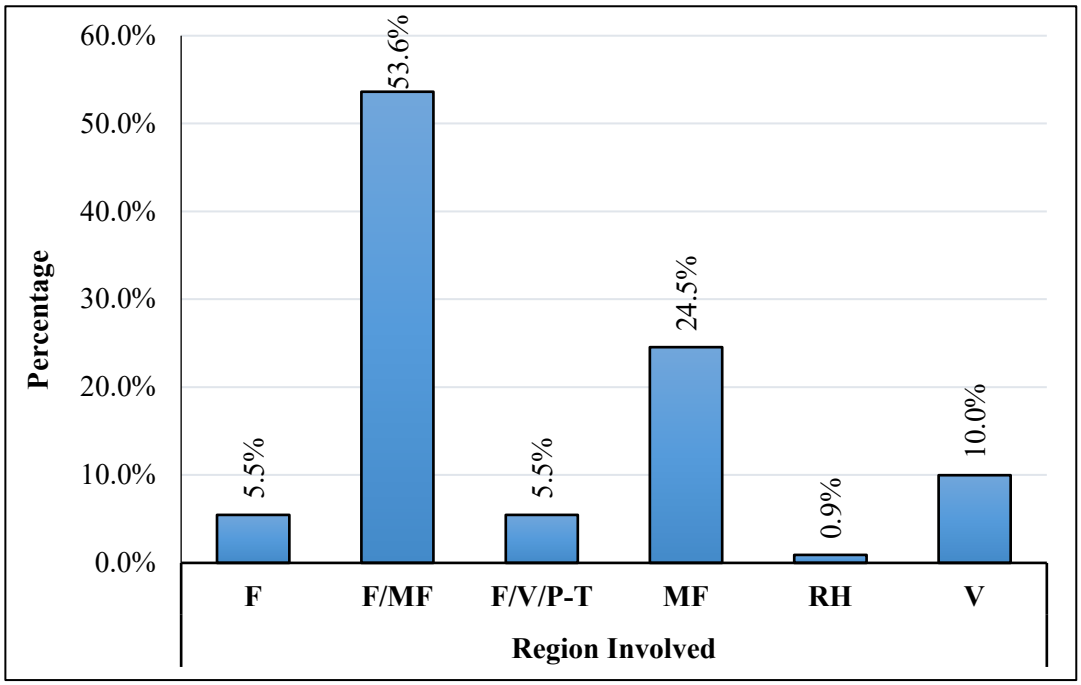


Above data (table 16 & graph 16) suggests that majority of patients studied clustered in Ludwig's stage I – 63 patients (57.27%) followed by Ludwig's stage II-31 patients (28.18%) and 6 patients (5.45%) were of Ludwig's stage III and 10 patients (9.09%) had male pattern baldness.

Table 17: Region Involved

Region involved	Cases	Percentage
F	6	5.45
F/MF	59	53.64
F/V/P-T	6	5.45
MF	27	24.55
RH	1	0.91
V	11	10.00
F-frontal, MF- Mid-frontal, V- Vertex, P-T- Parieto-temporal, RH- recession of hair line		

Graph 17: Distribution of subjects by region of Hair loss.

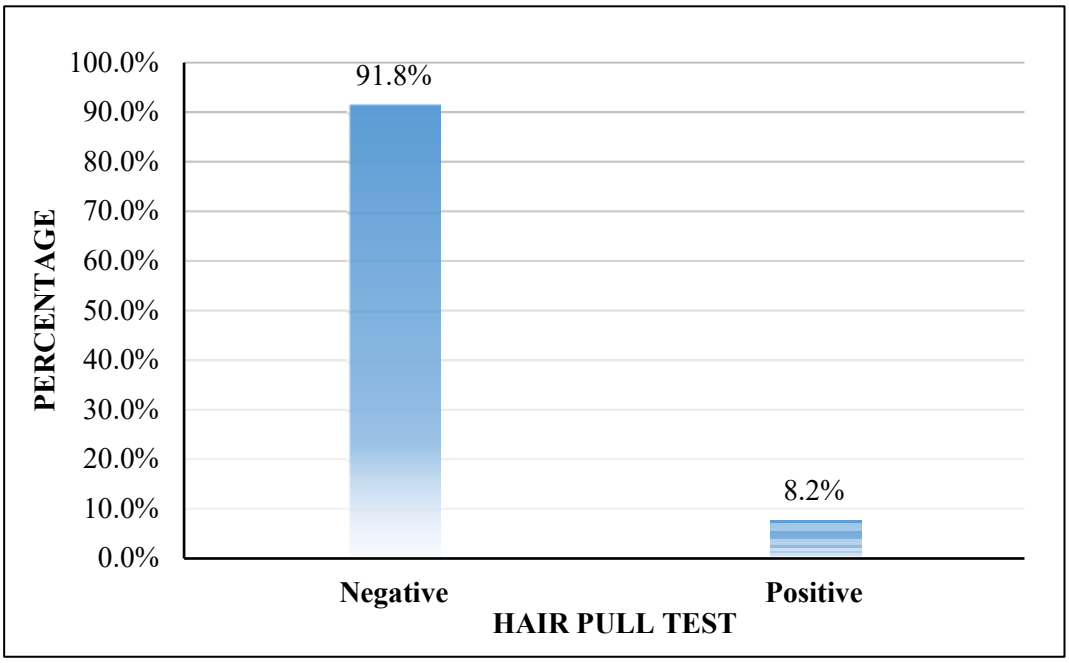


Above data (table 17 and graph 17) suggests that most common region of scalp involved in patients in our study was combination of frontal and mid-frontal region in 59 patients (53.64%) followed by mid-frontal in 27 patients (24.55%).

Table 18: Hair Pull test

Hair pull test	Cases	Percentage
Negative	101	91.82
Positive	9	8.18

Graph 18: Distribution of subjects by hair pull test

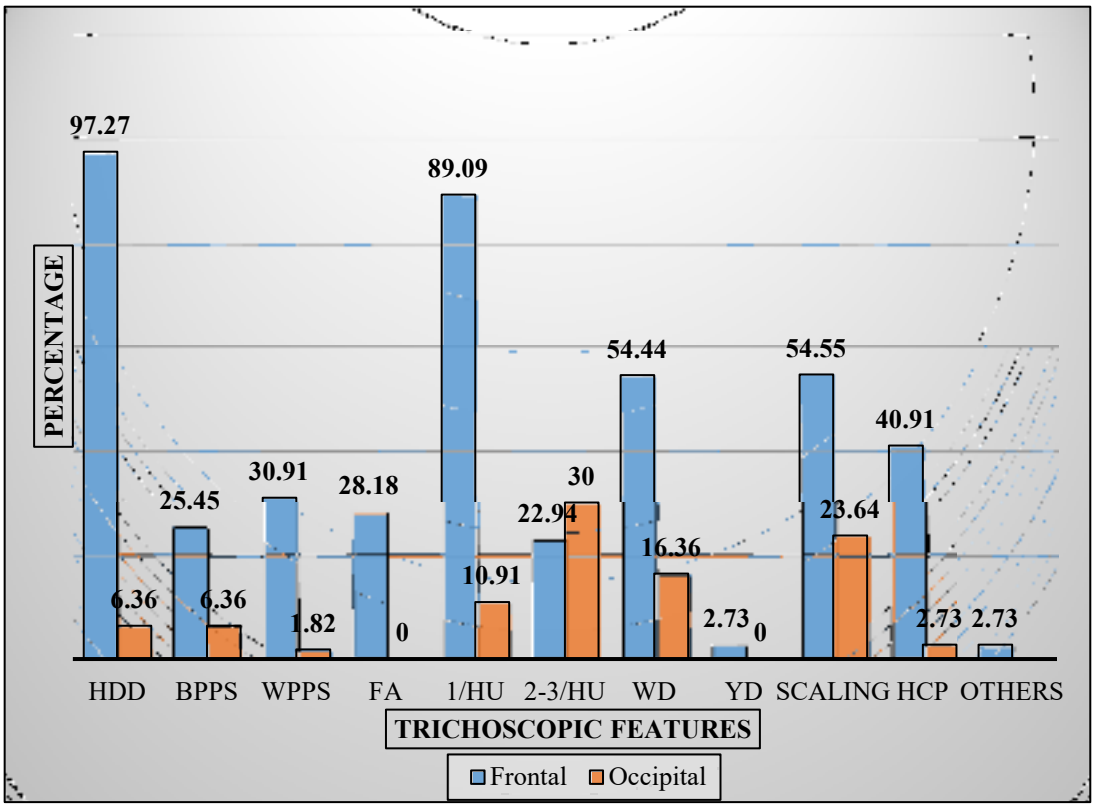


Above data (table 18 & graph 18) indicates that 101 patients (91.82%) studied had negative hair pull test and 9 patients (8.18%) negative hair pull test.

Table 19: Trichoscopic features in Frontal and occipital regions

Variables		Frontal	Occipital
HDD	No	3 (2.73)	103 (93.64)
	Yes	107 (97.27)	7 (6.36)
BPPS	No	82 (74.55)	103 (93.64)
	Yes	28 (25.45)	7 (6.36)
WPPS	No	76 (69.09)	108 (98.18)
	Yes	34 (30.91)	2 (1.82)
FA	No	79 (971.82)	110 (100)
	Yes	31 (28.18)	0 (0)
1/HU	No	12 (10.91)	98 (89.09)
	Yes	98 (89.09)	12 (10.91)
2-3/HU	No	84 (77.06)	77 (70)
	Yes	25 (22.94)	33 (30)
WD	No	50 (45.45)	92 (83.64)
	Yes	60 (54.55)	18 (16.36)
YD	No	107 (97.27)	110 (100)
	Yes	3 (2.73)	0 (0)
Scaling	No	50 (45.45)	84 (76.36)
	Yes	60 (54.55)	26 (23.64)
HCP	No	65 (59.09)	107 (97.27)
	Yes	45 (40.91)	3 (2.73)
Others	No	107 (97.27)	110 (100)
	Yes	3 (2.73)	0 (0)

Graph 19: Distribution of subjects by Trichoscopic features



Above result depicts observed trichoscopic features in patients included in our study and it includes: hair diameter diversity 97.27% (107/110) in frontal and 6.36% (7/110) in occipital, brown peripilar sign 25.45%(28/110) in frontal and 6.36% (7/110) in occipital, white peripilar sign 30.91% (34/110) in frontal and 1.82% (2/110) in occipital, focal atrichia 28.18% (31/110) in frontal, one hair per follicular unit 89.09% (98/110) in frontal and 10.91% (12/110) in occipital, 2-3 hair per follicular unit 22.94% (25/110) in frontal and 30% (33/110) in occipital, yellow dots in 2.73% (3/110) in frontal, scaling in 54.55% (60/110) in frontal and 23.64% (26/110) in occipital, white dots in 54.55% (60/110) in frontal and 16.36% (18/110) in occipital, honey comb pigmentation 40.91% (45/110) in frontal and 2.73% (3/110) in occipital.

Table 20: Comparison of age in each stage of hair loss

Grade	Mean (SD)	Median (IQR)	P value
L-I	32.16 (9.30)	30 (24.50, 39.50)	0.198
L-II	35.58 (8.45)	36 (29.00, 45.00)	
L-III	29.00 (8.37)	28 (22.00, 35.50)	
MT	32.00 (11.19)	29 (22.25, 42.00)	

By Kruskal Wallis test, there is no significant difference in the distribution of age with Stage of hair loss with Grades. ($p>0.05$)

Table 21: Association of different factors with grades of hair loss

Variables		L -I	L -II	L -III	MT	P value
Family history	No	27 (42.86)	12 (38.71)	0	3 (30.00)	0.217
	Yes	36 (57.14)	19 (61.29)	6 (100)	7 (70.00)	
Menstrual history	Irregular cycle	7 (11.11)	2 (6.45)	1 (16.67)	2 (20.00)	0.872
	MP	8 (12.70)	5 (16.13)	0	1 (10.00)	
	Regular	48 (76.19)	24 (77.42)	5 (83.33)	(70.00)	
SD	No	36 (57.14)	17 (54.84)	5 (83.33)	5 (50.00)	0.637
	Yes	27 (42.86)	14 (45.16)	1 (16.67)	5 (50.00)	
ACNE	No	53 (84.13)	28 (90.32)	5 (83.33)	8 (80.00)	0.864
	Yes	10 (15.87)	3 (9.68)	1 (16.67)	2 (20.00)	
AN	No	58 (92.06)	28 (90.32)	6 (00)	10 (100.00)	0.817
	Yes	5 (7.94)	3 (9.68)	0	0	
Hirsuitism	No	55 (87.30)	28 (90.32)	5 (83.33)	8 (80.00)	0.869
	Yes	8 (12.70)	3 (9.68)	1 (16.67)	2 (20.00)	

By chi square test, there is no association between distinct factors with grades of hair loss. ($p>0.05$)

Table 22: Trichoscopic findings in Frontal region and its association as per grading of hair loss.

Frontal Variables		L -I	L -II	L -III	MT	P value
HDD	No	3 (4.76)	0	0	0	0.712
	Yes	60 (95.24)	31 (100)	6 (100)	10 (100.00)	
BPPS	No	50 (79.37)	23 (74.19)	4 (66.67)	5 (50.00)	0.231
	Yes	13 (20.63)	8 (25.81)	2 (33.33)	5 (50.00)	
WPPS	No	43 (68.25)	19 (61.29)	5 (83.33)	9 (90.00)	0.327
	Yes	20 (31.75)	12 (38.71)	1 (16.67)	1 (10.00)	
FA	No	54 (85.71)	17 (54.84)	1 (16.67)	7 (70.00)	<0.001*
	Yes	9 (14.29)	14 (45.16)	5 (83.33)	3 (30.00)	
1/HU	No	8 (12.70)	1 (3.23)	1 (16.67)	2 (20.00)	0.343
	Yes	55 (87.30)	30 (96.77)	5 (83.33)	8 (80.00)	
2-3/HU	No	46 (74.19)	28 (90.32)	5 (83.33)	5 (50.00)	0.046*
	Yes	16 (25.81)	3 (9.68)	1 (16.67)	5 (50.00)	
WD	No	35 (55.56)	8 (25.81)	2 (33.33)	5 (50.00)	0.043*
	Yes	28 (44.44)	23 (74.19)	4 (66.67)	5 (50.00)	
YD	No	61 (96.83)	30 (96.77)	6 (100)	10 (100.00)	0.999
	Yes	2 (3.17)	1 (3.23)	0	0	
Scaling	No	28 (44.44)	13 (41.94)	5 (83.33)	4 (40.00)	0.295
	Yes	35 (55.56)	18 (58.06)	1 (16.67)	6 (60.00)	
HCP	No	44 (69.84)	14 (45.16)	4 (66.67)	3 (30.00)	0.022*
	Yes	19 (30.16)	17 (54.84)	2 (33.33)	7 (70.00)	
Others	NAD	61 (96.83)	30 (96.77)	6 (100)	10 (100.00)	0.999
	Pigment	2 (3.17)	1 (3.23)	0	0	

By chi square test, there is significant association present between FA, 2-3/HU, WD and HCP with grades of hair loss. ($p<0.05$)

By chi square test, There is no association present between HDD, BPPS, WPPS, 1/HU, YD, Scaling and others with Frontal and occipital. ($p>0.05$)

Table 23: Trichoscopic findings in occipital region and its association as per grading of hair loss

Occipital Variables		L -I	L -II	L -III	MT	P value
HDD	No	61 (96.83)	29 (93.55)	4 (66.67)	9 (90.00)	0.045*
	Yes	2 (3.17)	2 (6.45)	2 (33.33)	1 (10.00)	
BPPS	No	61 (96.83)	29 (93.55)	5 (83.33)	8 (80.00)	0.121
	Yes	2 (3.17)	2 (6.45)	1 (16.67)	2 (20.00)	
WPPS	No	62 (98.41)	31 (100)	6 (100)	9 (90.00)	0.347
	Yes	1 (1.59)	0	0	1 (10.00)	
1/HU	No	57 (90.48)	30 (96.77)	2 (33.33)	9 (90.00)	0.001*
	Yes	6 (9.52)	1 (3.23)	4 (66.67)	1 (10.00)	
2-3/HU	No	46 (73.02)	23 (74.19)	3 (50.00)	5 (50.00)	0.297
	Yes	17 (26.98)	8 (25.81)	3 (50.00)	5 (50.00)	
WD	No	53 (84.13)	24 (77.42)	6 (100)	9 (90.00)	0.496
	Yes	10 (15.87)	7 (22.58)	0	1 (10.00)	
Scaling	No	52 (82.54)	21 (67.74)	5 (83.33)	6 (60.00)	0.228
	Yes	11 (17.46)	10 (32.26)	1 (16.67)	4(40.00)	
HCP	No	61 (96.83)	30 (96.77)	6 (100)	10 (100.00)	0.999
	Yes	2 (3.17)	1 (3.23)	0	0	

By chi square test, there is significant association present between HDD and 1/HU with grades of hair loss. ($p<0.05$)

By chi square test, there is no signficant association present between BPPS, WPPS, 2-3/HU, WD, Scaling and HCP with Frontal and occipital. ($p>0.05$)

Table 24: Comparison of Trichoscopic findings between Frontal and occipital region.

	Frontal	Occipital	p-value
HDD	**107 (97.27%)**	**7 (6.36%)**	**<0.0001***
BPPS	**28 (25.45%)**	**7 (6.36%)**	**0.0002273***
WPPS	**34 (30.91%)**	**2 (1.82%)**	**<0.0001***
FA	**31 (28.18%)**	**0 (0%)**	**<0.0001***
1/HU	**98 (89.09%)**	**12 (10.91%)**	**<0.0001***
2-3/HU	**25 (22.73%)**	**33 (30%)**	**0.2841**
WD	**60 (54.55%)**	**18 (16.36%)**	**<0.0001***
YD	**3 (2.73%)**	**0 (0%)**	**0.245**
SCALING	**60 (54.55%)**	**26 (23.64%)**	**<0.0001***
HCP	**45 (40.91%)**	**3 (2.73%)**	**<0.0001***

By two sample proportion test, we can observe that there is significant difference in the distribution of all dermoscopic findings except 2-3/HU and YD between frontal and occipital sides.

Table 25: Comparison of duration of hair loss in each grade of hair loss.

Grade	Mean (SD)	Median (IQR)	P value
L-I	9.46 (8.55)	6 (4, 12)	0.00111*
L-II	20.9 (17.5)	18 (6, 30)	
L-III	32 (19.96)	30 (19.5, 45)	
MT	15.1 (12.19)	9 (5, 24)	

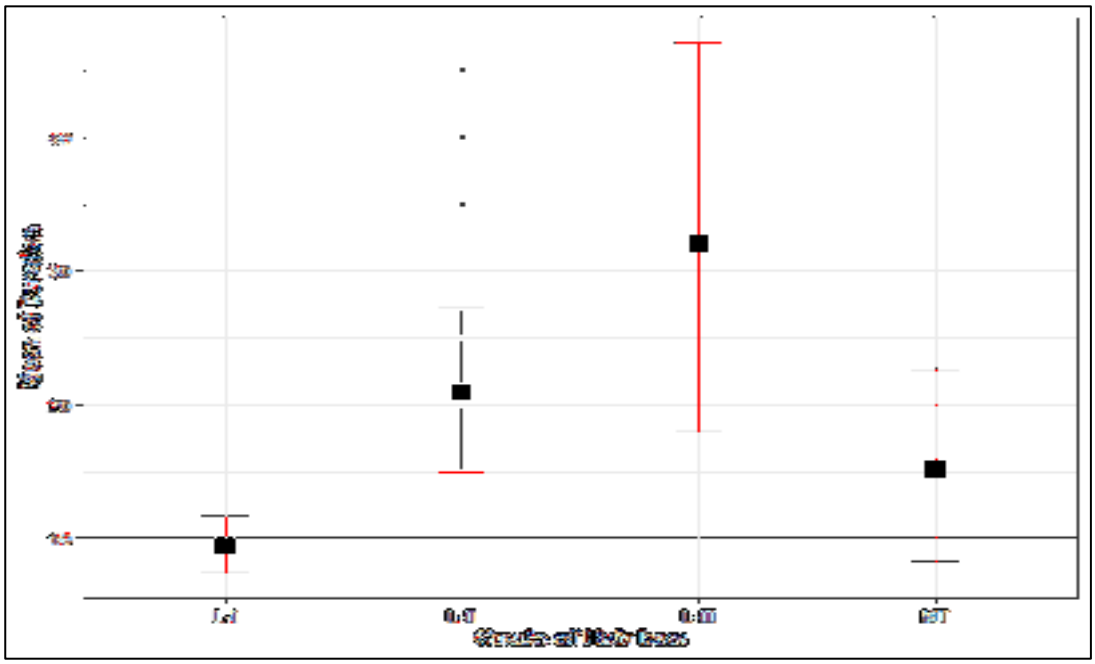
By Kruskal-Wallis test, there is significant difference in the distribution of duration over grades of hair loss. Below table gives the result of post-hoc analysis. Dunn's test is used for post-hoc analysis.

Table 26: Post-hoc analysis of duration of hair loss in each grade of hair loss.

Grades of hair loss		p-value
L-I	**L-II**	0.0005*
L-I	**L-III**	0.0009*
L-I	**MT**	0.094
L-II	**L-III**	0.082
L-II	**MT**	0.228
L-III	**MT**	0.042*

By Dunn's test, we can observe that there is significant difference in the distribution of duration between L-I and L-II, L-I and L-III, L-III and MT.

Graph 20: Comparison of Duration of hair loss with grades of hair loss.



DISCUSSION

The present study is a cross-sectional study done over a period of 12 months from January 2020 to December 2020 in the Department of Dermatology, Venereology and Leprology, KAHERS Dr Prabhakar Kore Hospital, Belagavi.

In our study we aimed to analyze the trichoscopic features with regards to severity of hair loss and facilitate its diagnosis using a simple non-invasive technique.

Various etiological factors play a role in causation of FPHL and the role of hormonal factors in causation of patterned hair loss is still debated and is controversial. Till date none of the studies have put forth definite role of endocrinal factors in etio-pathogenesis of FPHL and data in literature is sparse.

Video-dermoscopic criteria for diagnosing FPHL are recently given by Rakowska et al[13] which also helps to differentiate FPHL from telogen effluvium. But to diagnose patients as FPHL using these criteria require specialized instrument and dedicated software. Also due to cost factors these sophisticated instruments are not available with most of the dermatologist.

Using a USB dermoscope at 50x magnification, we planned to analyze and compare the frontal and occipital scalps of patients, as the occipital region would not be impacted in patterned hair loss. This would help us in diagnosing FPHL early with convenience and at low cost dermoscope.

In our study majority of patients clustered in age group between 18-30 years which could be probably due to the fact that these age group females are cosmetically more aware and thus they seek medical attention.

In study done by Ramatulasi et al[63] majority proportion of patients were in age group of 29-38 years. Similarly results of study done by Tandon et al[99] showed majority of patients were in 28-37 years of age. Also Lee et al [100]demonstrated 252 out of 445 patients in 3rd decade of their life which was in contrast to our study. This can be explained by racial difference and different sample size.

The mean age of presentation in our study was 32.94 ± 9.26 years. In study done by Ummiti A et al [60] mean age was 37 years and in study done by Zhang X et al[12] it was 34.4±10.6 years which was in agreement with our study.

In our study maximum patients (50.46%) had duration of hair loss more than 9 months. In study done by Zhang X et al[12] and in study by Tandon et al[99] the mean duration was 4.49±3.76 years and 5.1 years respectively.

In our study 57.27% (63/110), 28.18% (31/110), 5.45% (6/110) and 9.09% (11/100) of patients had hair loss as per L-I, L-II, L-III and Male type fronto-temporal recession, respectively with corresponding age of onset of 32.16±9.3 years, 35.58±8.45 years, 29±8.37 years and 32 ±11.19 years.

However, duration of hair loss among 4 groups were different (L-I→ 9.46±8.55 months; L-II→ 20.9±17.5 months; L-III→ 32±19.96 months; MT→ 15.1±12.19 months). There was statistically significant difference between stage of hair loss and duration of hair loss in our study.

In our study 61.82% of patients had strong family history of hair loss. Shilpashree et al[101] reported (51%) in their study. In a Chinese study strong family history was found in 19.2-32.4% of patients. Study done by Zhang X et al [12] incidence of family history was in 45% and study by Ramatulasi et al [63] was 56%

which was nearly matching with our study. Difference incidences of positive family history could be explained by underlying genetic factors which have a role in aetiology of FPHL due to difference in ethnicity.

Cutaneous signs of hyperandrogenism which was observed in our patients includes seborrheic dermatitis (42.73%), hirsutism (12.73%), acanthosis nigricans (7.27%) and acne (14.55%). Cutaneous hyperandrogenism is defined as increased formation of potent androgens that is testosterone and DHT from their precursors in skin.[102]

In study done by Ramatulasi et al[63] incidence of hirsutism was 40%, acanthosis nigricans was 37% and acne was 28.5%. Hirsutism was reported in 66.6% of cases in study done by Tandon et al.[99] Acne was observed in 36.6% in study done by Tandon et al[99] and 41.6% in study done by Moltz et al.[103] Higher incidence of acanthosis nigricans was outlined in study done by Tandon et al[99] (43.3%) which was much more than observed in our study.

In our study family history, menstrual history, associated findings like seborrheic dermatitis, acne, acanthosis nigricans and hirsutism did not have any statistical significance as per the grading of hair loss.

Results of studies suggesting role of hyperandrogenism in FPHL are discordant and thus dermatologist prefers to use term 'Female Pattern Hair Loss' instead of androgenetic alopecia. However, many women with features of hyperandrogenism complains of scalp hair loss indicating role of androgens in FPHL.

Hyperandrogenism can be explained clinically as well as biochemically. Clinical hyperandrogenism is defined as cutaneous signs and symptoms like acne, hirsutism

etc. whereas biochemically it is defined as elevation in the serum free testosterone, total testosterone or DHEAS above normal laboratory reference range which was omitted because of financial constraint in our study.

Molly Quinn et al[105] showed that there is no difference in hyperandrogenaemia with or without AGA. However Cela et al [104] reported significant role of testosterone, androstenedione in causation of AGA.

Biochemical hyperandrogenism is challenging aspect in evaluation of FPHL as many studies demonstrate that patients with FPHL don't possess elevated androgens levels. Also, wide variations of androgens level do exist in normal population and different reference range used by various laboratories.

Interestingly serum androgen levels do not reflect local androgen role in growth of hair follicle thus implicating that serum level of androgens may not be elevated in all subjects.[109,110]

The increase prevalence of FPHL subsequent to menopause and also increased level of estrogen in pregnancy suggests the stimulatory role of estrogen on hair growth. In our study 14 patients had attained menopause and no patients were pregnant. Out of 14 patients who had attained menopause 8 patients had L-I pattern, 5 had L-II pattern, 1 patient had Male type fronto temporal recession and none had L-III pattern. However, future researches are needed to prove role of estrogen in FPHL as data in literature is sparse.[112,113]

Observed trichoscopic features in patients included in our study were: HDD 97.27% (107/110), BPPS 25.45%(28/110), WPPS 30.91% (34/110), focal atrichia 28.18% (31/110), one hair per follicular unit 89.09% (98/110), 2-3 hair per follicular

unit 22.73% (25/110), yellow dots in 2.73% (3/110), scaling in 54.55%(60/110), white dots in 54.55%(60/110) and honey comb pigmentation 40.91% (45/110).

Rakowska et al[13] have established hair shaft diameter diversity as one of the major criteria to diagnose FPHL on trichoscopy. Also termed as 'anisotrichosis' and it correlates histo-pathologically with miniaturisation of hair. In our patient it was observed in 97.27% of cases and results were in agreement with findings by Ramatulasi et al[63] where this finding was present in 93% of patients. Bhamla et al [114] in their study suggested that solitary parameter of hair diameter diversity can diagnose Ludwig's-I FPHL in 75% if this is present in > 20% hairs. Hair diameter diversity was statistically significant when frontal scalp was compared to that of occipital. Thus, occipital area of patient's scalp can serve as control to compare and use these criteria as screening tool for FPHL.

Peripilar sign is halo of ~ 1mm surrounding follicular opening. Colour of the halo varies from white to brown. BPPS was seen in 25.45% of our patients which was similar finding in study done by Inuis et al [62] (20%). In contrast to our finding Ummiti A et al[60] and Hu et al [61] described this finding in 40% and 44.5% respectively. In study done by Zhang et al[12] in Chinese population BPPS was reported in 31.7% cases and Ramatulasi et al[63] reported this observation in 47% of cases. Deloche et al[115] described BPPS in highest number of cases(86%). According to literature peripilar sign is unique to AGA but in our study, we found it in 25.45% of patients only. It could be due to concealment of finding due to colour of scalp.

WPPS in our study was reported in 30.91% of cases. Incidence of this finding varied from 15-68% in different studies done.[12,60,61]

Brown Peripilar sign histo-pathologically correlates with peri-follicular infiltrate and melanogenesis and WPPS correlates perifollicular fibrosis. Thus, BPPS is present in preliminary stages whereas WPPS is seen in advanced cases of FPHL.

In our study we reported only 2.73% of patients had yellow dots which was similar to study done by Zhang et al[12] (1.67%). However this was contradictory to study done by Ramatulasi et al[63] (57%) and Ummiti A et.al[60](88%). Ummiti A et.al[60] suggested that difference in incidence in this observation could be secondary to diverse phenotypes of skin with variation in scalp pigmentation and activity of sebaceous gland. In addition Zhang et al[12] suggested that this could be due to easy recognition of white dots as compared to yellow dots.

Yellow dots represent dilated infundibulum of follicle which contains keratinous material with sebum. Yellow dots are one of major criteria on trichoscopy suggested by Rakowska et al[13].

Focal atrichia was seen in 28.18% patients in our study. Similar findings were observed by Ramatulasi et al[63] (18.6%) and Ummiti A et.al[60] (24%). Whereas Zhang et al[12] and Hu et al[61] reported this finding in majority of their patients. It correlated with advanced stage of FPHL in study done by Zhang et al[12].

In our study HCP was reported in 40.91% of patients. Ramatulasi et al[63] and Zhang et al[12] reported this finding in 60% and 61.7% of their patients. Also study done by Ummiti A et al[60] observed this finding in 80% and Hu et al [61]in 30.5%.

White dots were seen in 54.55% and single hair per follicular unit in 89.09% of our patients. The normal scalp each follicular unit comprises one to four terminal hairs with one to two vellus hair. However, majority of our patients showed increased

single hair per follicular units with predominant prevalence in the frontal area suggesting increase thinning of hair and helps in diagnosis of FPHL.

White dots were reported by Zhang et al[12] as white spots of size 0.2-0.3mm uniformly distributed; histo-pathologically they corresponds to eccrine gland opening on scalp. Sweat glands have highest activity of 5α-reductase thus they can undergo hypertrophy under influence of elevated androgen level. Zhang et al[12] also suggested that this feature could be due to hypertrophy of sweat glands under influence of elevated androgen level thus suggesting role of androgen in causation of FPHL.

These white dots can be seen in healthy individuals also with Fitzpatrick skin photo-types IV– VI[116] as they become evident on the contrasting background of the pigmented network.

In our study on comparison of frontal and occipital (control) group, apart from the incidence of yellow dots, 2-3 hairs per unit follicle (P=>0.05), incidence of HDD, BPPS, WPPS, one hair per follicular unit, white dots, scaling and HCP were statistically significant (p=<0.001).

In our study Focal atrichia, 2-3 hairs/HU, white dots, HCP correlated with grade of hair loss. (P<0.05)

CONCLUSION

FPHL is a little-known phenomenon, despite the fact that it is a widespread contributor for loss of hair in the general population. Various aetiologies have been proposed, some of which may overlap. Increased levels of androgens are frequent in candidates with features of hyperandrogenism, although not all patients with FPHL have them. When levels of androgen are within accepted range, solitary FPHL should not be thought as a symptom of hyperandrogenism.

After stimulation with corticotrophins, the best biochemical indicators in isolated FPHL or FPHL with limited evidence of hyperandrogenism would be a drop in serum SHBG, a rise in Basal 3-alpha-estradiol, and an exaggerated DHEA response (not necessary for clinical practice).[117]

Higher androgen receptors or increased androgen sensitivity may cause FPHL in persons with normal androgen levels.[108] Androgen levels which share an important part in the cause was not investigated in our work due to financial constraints.[108]

Investigating androgens in FPHL should be commenced in few cases with clues of hyperandrogenism and after ruling out the hormonal imbalances with suitable controls.

The probability of underlying altered gonadal profile should be considered as it can present without any manifestations. It is recommended to do hormonal investigations in patients especially with early onset and advanced hair loss and

treating clinician should be aware of possibility of underlying endocrinological conditions which may affect the hair re-growth in FPHL.

Various findings on Trichoscopy like HDD, BPPS, WPPS, focal atrichia, White dots, HCP can be utilized as non-invasive aid in diagnosis of FPHL and differentiate it from chronic telogen effluvium (CTE).

As pattern hair loss spares occipital region, we can conclude that patients own occipital region can serve as control for diagnosing FPHL.

Our study lacks histo-pathological and hormonal investigations however past studies done have already established correlation of trichoscopic and histo-pathological changes. Also, study needs to be done in larger population to validate it further.

Thus, from our study we can conclude that trichoscopy could be excellent tool, which is simple, non-invasive cost effective for diagnosing FPHL using combination of findings like HDD, BPPS, WPPS, focal atrichia, white dots, single hair per follicular unit, HCP and comparing with spared occipital region.

Diagnosis of FPHL is clinical and trichoscopy may aid in diagnosing FPHL differentiating it from other condition like CTE.

SUMMARY

In our study period of 12 months from 1st January 2020 to 31st December 2020, a total of 100 patients were enrolled. Maximum patients belonged to the age group of 18-30 years in our study due to fact of more cosmetic awareness and thus higher demand for treatment. Majority of patients were housewives. The mean duration of disease before consultation was 14 months.

Significant difference was found between stage of hair loss and duration of hair loss.

Majority had gradual onset of hair loss with frontal and mid-frontal region being most commonly involved. Majority of patients complained of increased thinning of hair and hairs falling from roots in our study.

Majority of patients did not have any exacerbating factors and least patients were indulged in parlour activities. On an average 100-300 hair loss was complained by majority of patients.

Family history was present in 61.82% of cases however it did not correlate with grading of hair loss.

Associated clinical features included seborrheic dermatitis, acne, acanthosis nigricans and hirsutism which did not had any association with grading of hair loss neither with elevated serum testosterone levels.

In our analysis we did not seek to investigate endocrinal abnormality due to financial constraint, thus further studies need to be done to evaluate the same.

Trichoscopic features in our study includes-HDD, BPPS, WPPS, and Focal Atrichia, 1 hair per follicular unit, yellow dots, scaling, honeycomb pigmentation, and white dots.

HDD, BPPS, WPPS, Focal Atrichia, 1 hair per follicular unit, scaling, HCP, and white dots achieved significant results when compared with the occipital scalp of subject. Focal atrichia, 2-3 hair/FU, white dots, HCP correlated with grading of hair loss in our study.

Although most trichoscopic findings does not demarcate according to stage and duration of hair loss, trichoscopy could be used to diagnose or differentiate FPHL from CTE using various above findings.

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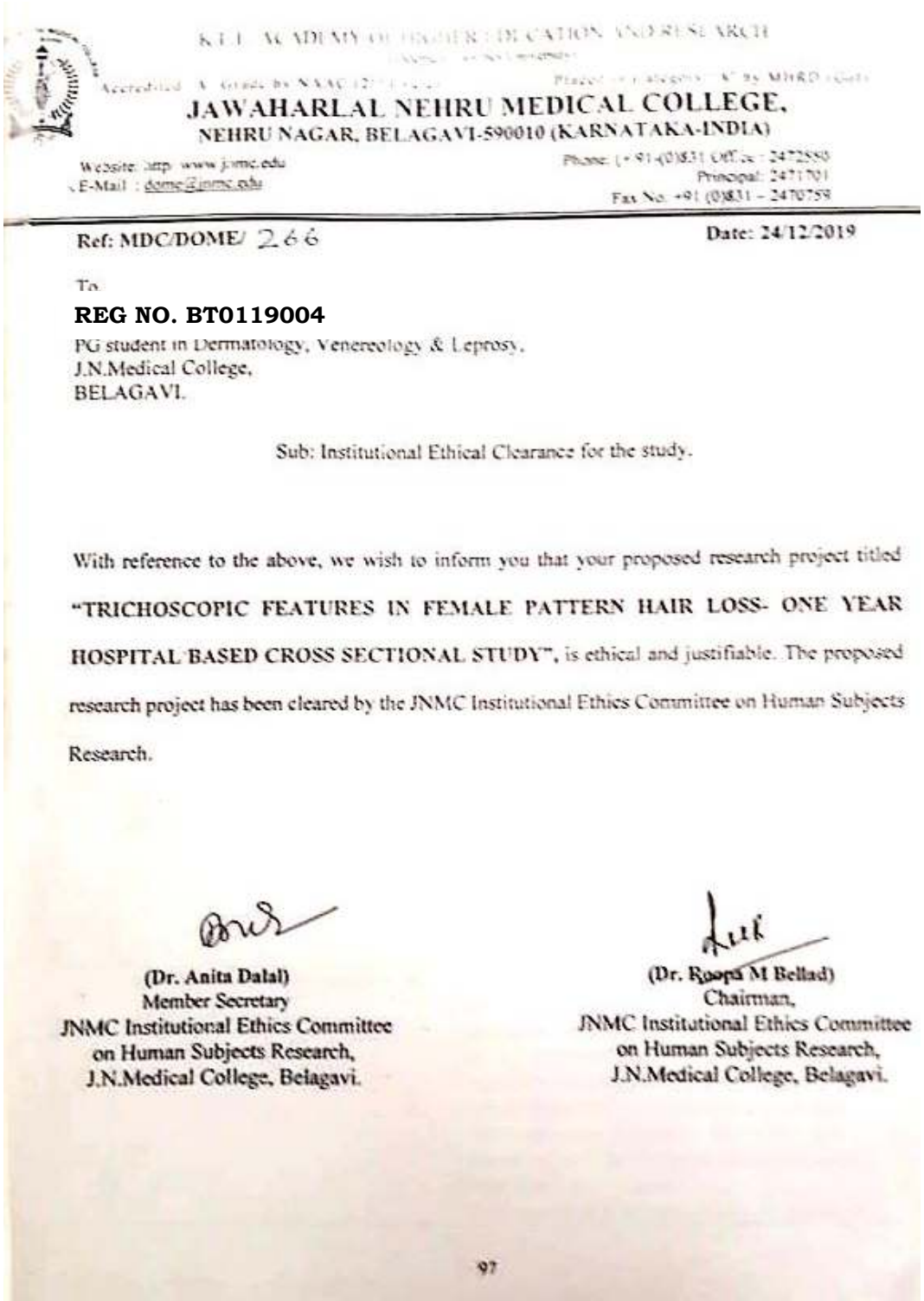
ANNEXURE-I- ETHICAL CLEARANCE LETTER



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH

Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (Govt.)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

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E-Mail : dome@jnmc.edu

Phone: (+91-(0)831 Off.ce : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 266 Date: 24/12/2019

To

REG NO. BT0119004
PG student in Dermatology, Venereology & Leprosy,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"TRICHOSCOPIC FEATURES IN FEMALE PATTERN HAIR LOSS- ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

97

ANNEXURE-II

INFORMED CONSENT FORM

I.D.NO.

Title of the study. : "TRICHOSCOPIC FEATURES IN FEMALE PATTERN HAIR LOSS- ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY" attending KLE's Dr Prabhakar Kore Hospital & MRC, Belagavi

The study is conducted by _____, Post Graduate (M.D) student in Dermatology under the guidance of _____**,** Associate Professor, Department of Dermatology, Venereology and Leprosy, JNMC, BELAGAVI.

Respected Sir/Madam,

We invite you to participate in our study as you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study:

Female pattern hair loss is a common condition causing hair loss wherein the changes can be seen using an instrument called dermatoscope/trichoscope. Hence this study intends to observe those changes/findings using the dermatoscope. You are being requested to participate in this study because you have been diagnosed to have FEMALE PATTERN HAIR LOSS.

Procedure:

You will be asked to give a detailed history of your disease, undergo a physical examination and dermoscopic examination along with the clinical pictures.

Risks and Benefits:

The result of you taking part in this research would help health care providers towards a better understanding of the disease, and thus we will be able to provide improved patient care.

Alternatives:

If you decide not to participate in this study, you will still be receiving the usual standard care for your disease.

Privacy and confidentiality:

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Relations with the Institutional policy:

The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this project.

Financial incentives:

You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results:

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation:

In case you need further information regarding your rights as a study participant, you may please contact **Dr.ROOPA M BELLAD,** chairman of the ethical committee, J N Medical College, Belagavi.

STATEMENT OF CONSENT

I.D.NO: | | | |

I Mr/Ms/Mrs ---------- volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

ANNEXURE III - PROFORMA

TRICHOSCOPIC FEATURES IN FEMALE PATTERN HAIR LOSS- ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY

CASE ID DATE

OPD NO

NAME AGE
SEX

ADDRESS PHONE NO

OCCUPATION

COMPLAINTS

HISTORY

Duration of hair loss ☐ < 3months ☐ 3-12 months ☐ > 12months

Pattern of hair loss: ☐ Diffuse ☐ Patchy

Itching over scalp ☐ Present ☐ Absent

Loss of hair over other regions ☐ Present ☐ Absent

If present mention areas (Axillary hair / Pubic Hair / Eye lashes / Eyebrows or any body hair)

Any treatment taken for Hair Loss Yes/ No

If Yes mention

History of any other drug intake Yes/No

If yes mention

Hair care & use of hair cosmetics Yes/No

If yes mention

Past history:

History of ☐ DM ☐ HTN ☐ Thyroid Abnormality

If yes Mention, duration and treatment

Any other serious illness in the past, if yes mention

Family history

Personal history

Diet: Vegetarian / Non- vegetarian

Menstrual history/ obstetric:

1. Cycles Regular / Irregular
2. If irregular mention
3. Obstetric history :
4. If Menopause

General examination

Built

Pallor / icterus /cyanosis/clubbing/lymphadenopathy

CUTANEOUS EXAMINATION

Hair pull test

Ludwig's Grade

Pattern & density

Scaling and Erythema

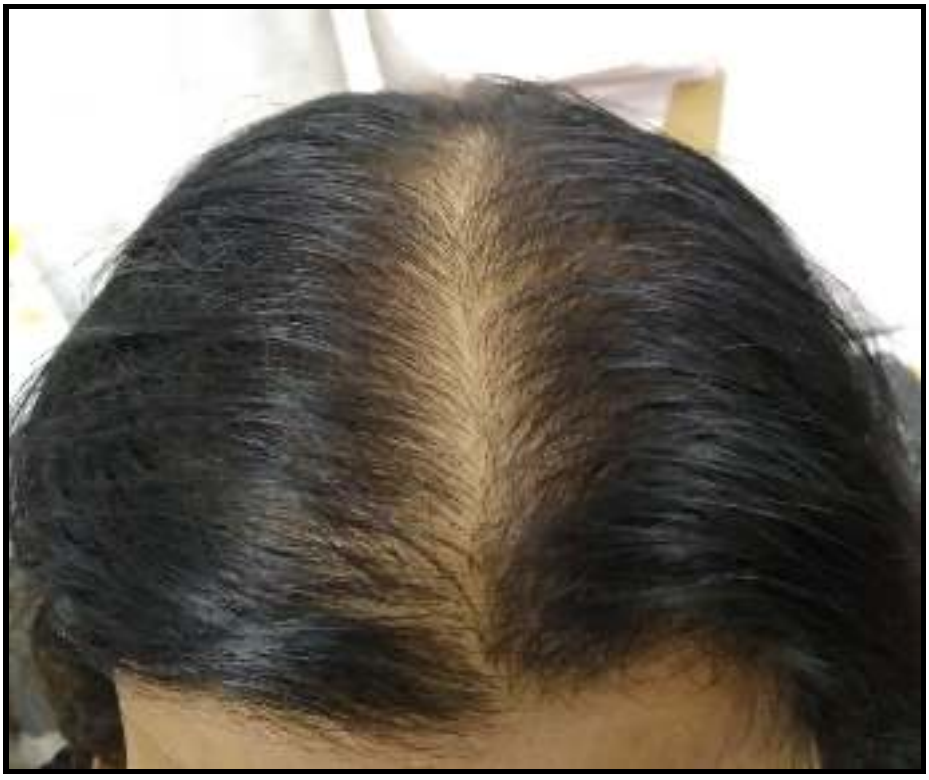
Any other features

Clinical diagnosis :

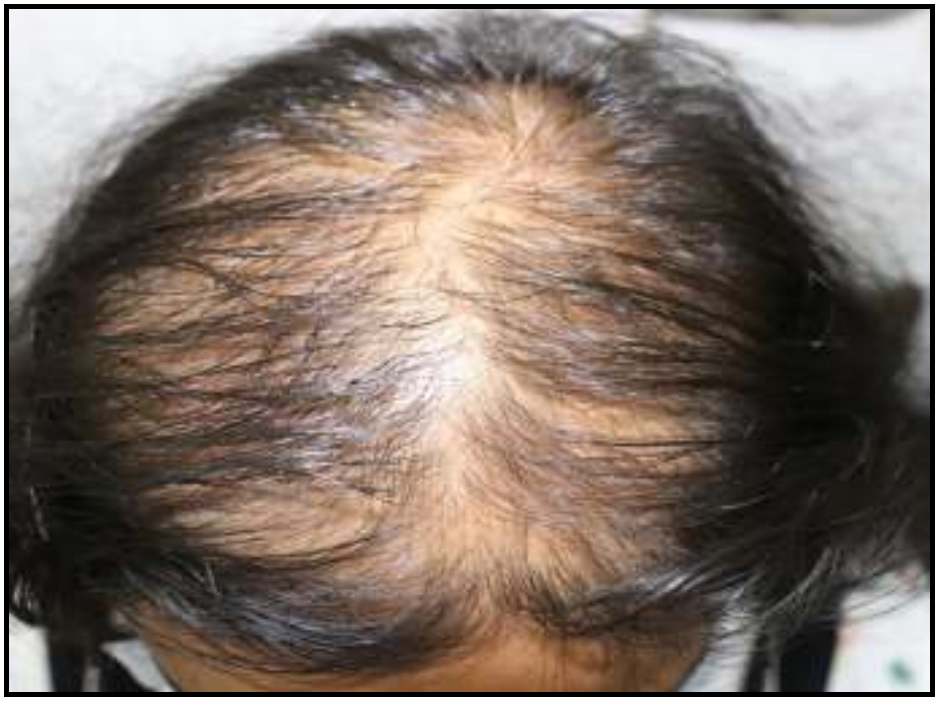
TRICHOSCOPY

NO	FEATURE	OCCIPITAL	PARIETAL	TEMPORAL	FRONTAL
1	**Hair Per Unit Follicle**				
2	**Follicular Patterns** Follicular openings Pinpoint white dots Yellow Dots White Dots Brown peripilar sign White Peripilar sign Others				
3	**Inter follicular Pattern** Scales Vessels Pigment Other				
3	**Hair Shafts** Hair density Hair diameter variability Others				
4	**Other features** Focal Atrichia				

ANNEXURE IV – PHOTOGRAPHS



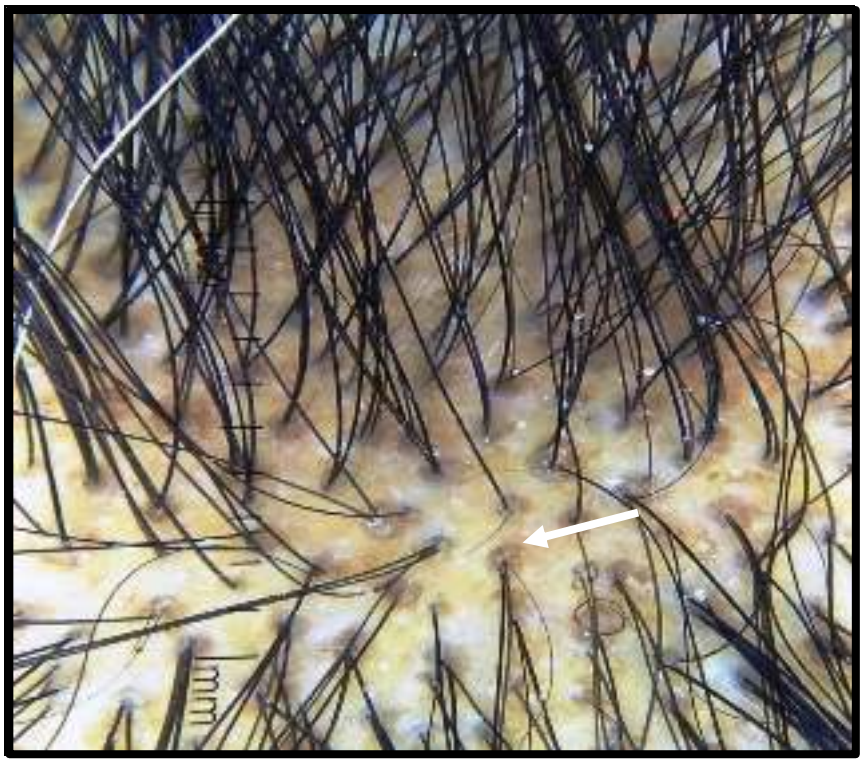
Photograph 1: Ludwig's stage I FPHL



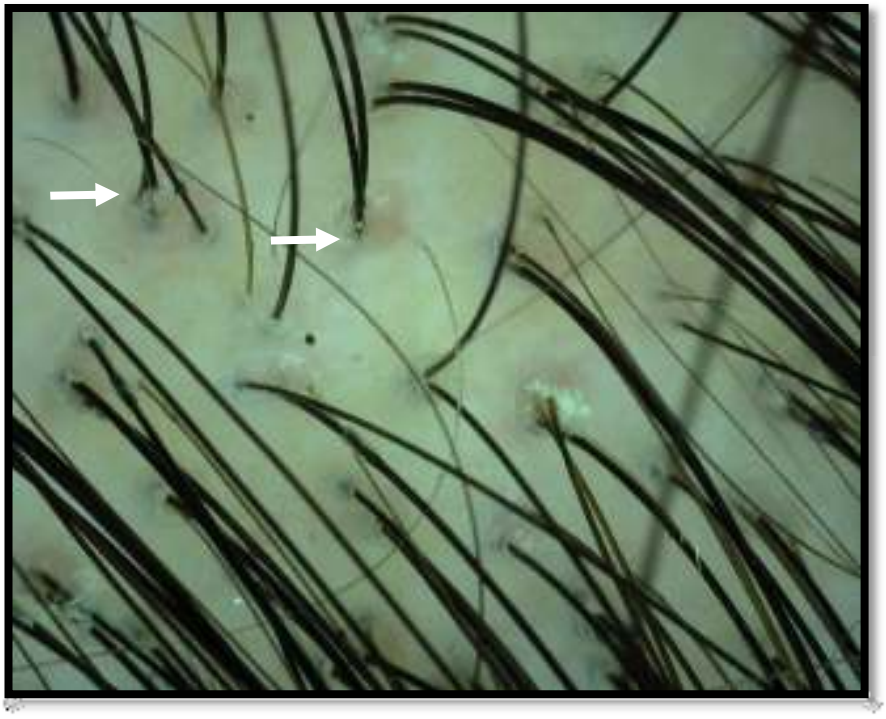
Photograph 2: Ludwig's stage II FPHL

Photograph 3: Ludwig's stage III FPHL

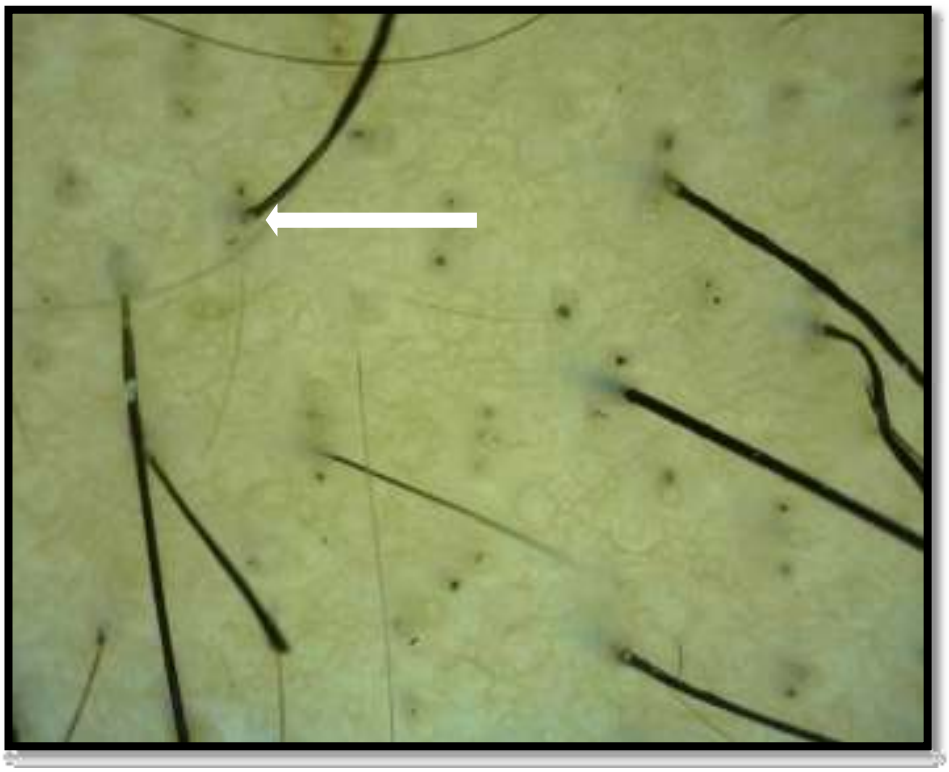
Photograph 4: Male temporal pattern hair loss



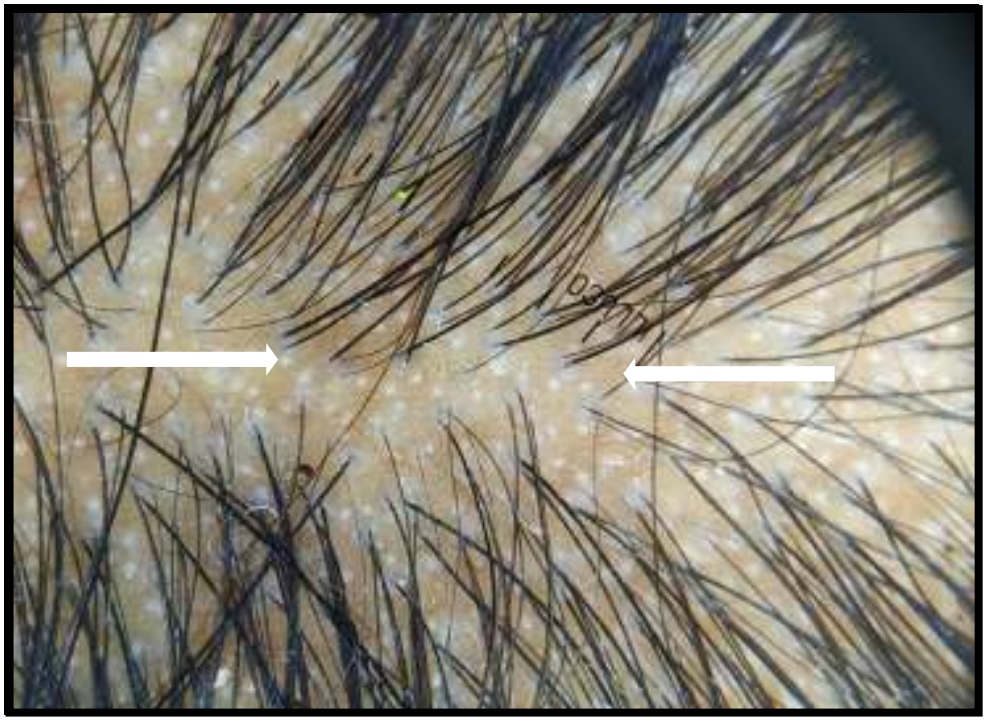
Photograph: 5 Trichoscopic polarised image at 10x magnification showing brown peripilar sign (marked by Arrow)



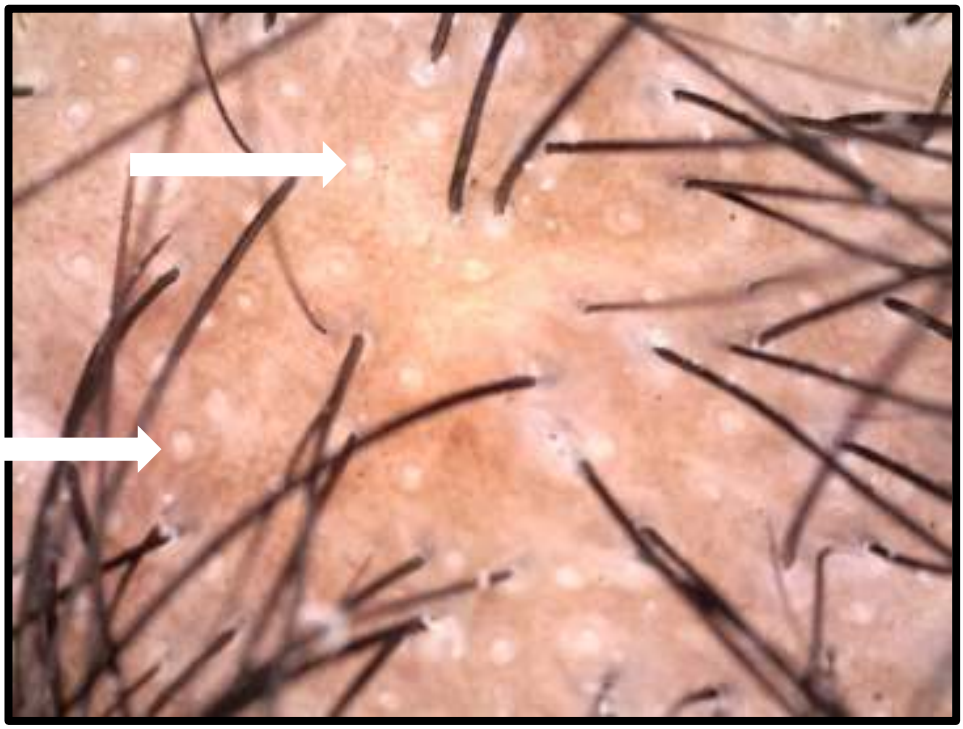
Photograph 6: Trichoscopic non-polarised image at 50x magnification showing White peripilar sign (marked by arrow)



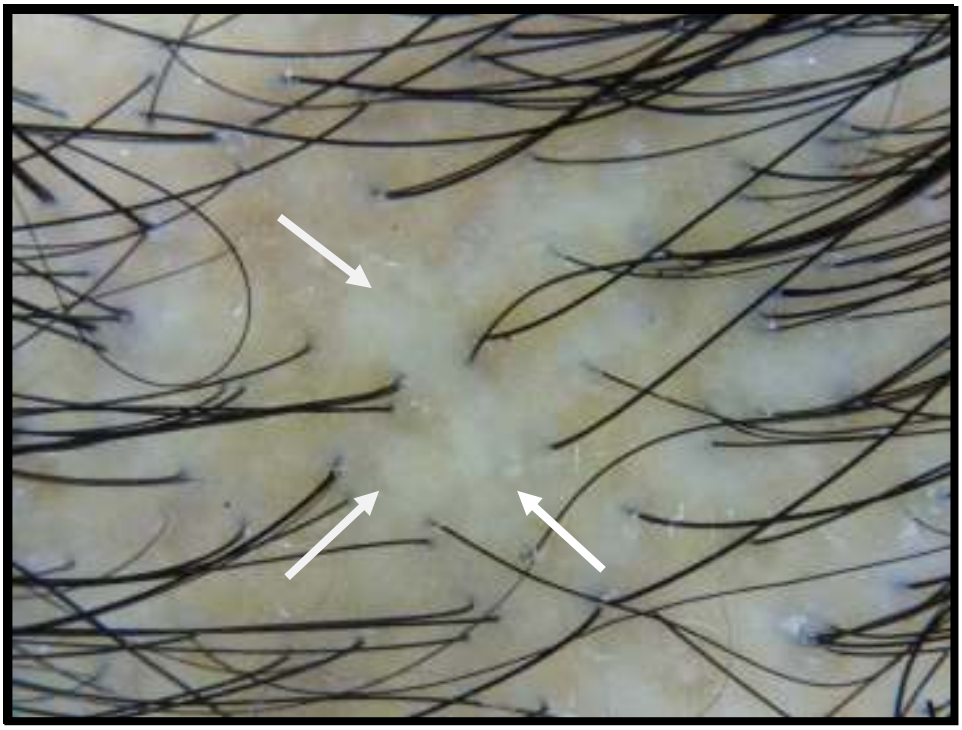
Photograph 7: Trichoscopic, polarized image, at 50x magnification showing single hair per follicular unit.



Photograph 8: Trichoscopic, polarized, at 10x magnification image showing multiple white dots. (Marked by white arrow)

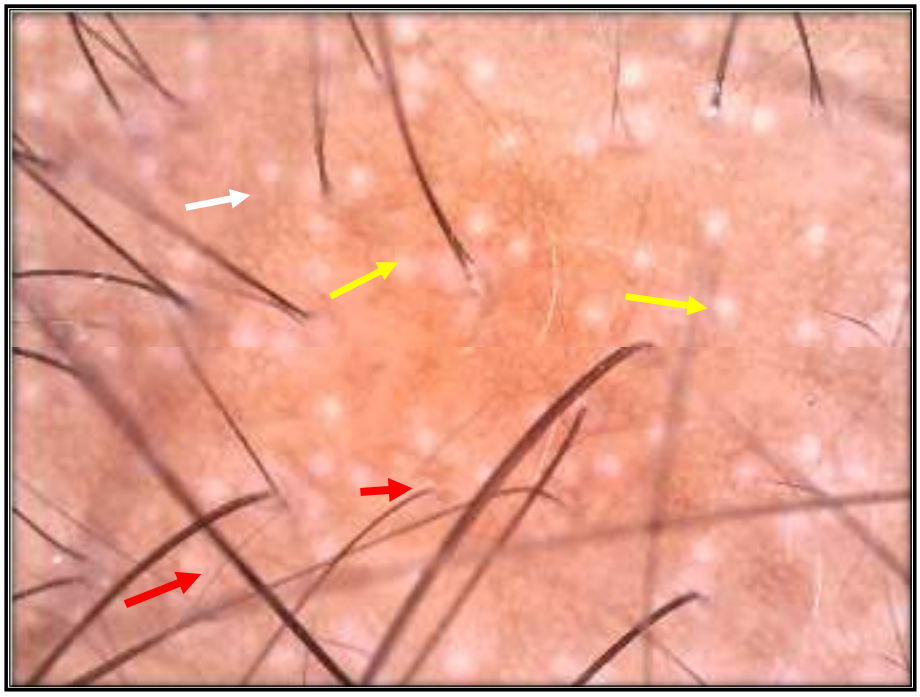


Photograph 9: Trichoscopic, polarized, at 50x magnification image showing multiple white dots. (Marked by arrow)



Photograph 10: Trichoscopic, polarized, at 10x magnification showing areas of Focal atrichia. (Marked by arrows)

Photograph 11: Trichoscopic image at 10x magnification showing honeycomb pigmentation (White arrow) and brown peripilar sign (Red arrow)



Photograph 12: Trichoscopic, polarized, at 50x magnification showing miniaturization (Red arrows) honey-comb pigmentation (White arrow) white dots (Yellow arrows)

ANNEXURE V MASTER CHART

SR.NO.	NAME	age	occupation	address	onset		duration		Hair falling		REGION INVOLVED	Exacerbating factors	cosmetic procedures	no. of hair			medical illness	drug history	family history	Menstrual	infertility	SD	ACNE	AN	HIRSUITISM	grade	HPT	BMI	FRONTAL										OCCIPITAL										
						0-3months	3-8month	>9MONTHS	from	of hairless				<100	100-300	>300				history									HDD	BPPS	WPPS	FA	I/HU	2-3/HU	WD	YD	SCALING	HCP	OTHERS	HDD	BPPS	WPPS	FA	I/HU	2-3/HU	WD	YD	SCALING	HCP	OTHERS
1	manika	40	HW	belgaum	G			2yrs	R	S	F/MF	N	N		Y		N	N	Y	R	N	N	N	N	N	L-I	NEG	19.14	Y	Y	N	N	Y	Y	Y	N	N	N	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
2	priyanka	29	O	belgaum	G		4m		R	S+T	F/P-T	n	N			Y	N	N	Y	R	N	N	N	N	N	L-II	NEG	20.37	Y	N	Y	Y	Y	N	Y	N	N	Y	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
3	gouravi	50	HW	chikkodi	G			1yr	R	S	MF	N	TEMP. Coloring		Y		N	N	Y	MP	N	N	N	N	N	L-I	NEG	22.2	Y	N	N	N	Y	N	Y	N	N	N	NAD	N	N	N	N	Y	Y	N	N	N	N	NAD
4	allabi	30	HW	athani	G			4 yrs	R	T	F/MF	N	N		Y		N	N	Y	R	N	N	N	N	N	L-II	NEG	24.15	Y	N	N	N	Y	Y	N	N	N	N	NAD	Y	N	N	N	N	Y	N	N	N	N	NAD
5	jyoti	50	O	khanapur	G		5m		R	T	F/MF	C	N		Y		N	N	Y	MP	N	N	N	N	N	MT	NEG	29.91	Y	Y	N	N	Y	Y	Y	N	Y	Y	NAD	N	Y	N	N	N	Y	N	N	Y	N	NAD
6	juhi	25	S	belgaum	G		6m		R	S+T	F/MF	N	TEMP. Coloring			Y	N	N	Y	R	NA	N	Y	N	Y	L-III	NEG	25.64	Y	N	N	N	Y	N	N	N	N	Y	NAD	Y	N	N	N	N	Y	N	N	N	N	NAD
7	farzana	20	HW	athani	G			1 yr	R	S	F	N	N		Y		N	N	Y	R	NA	N	N	N	N	L-I	NEG	21.7	Y	N	Y	N	Y	N	Y	N	Y	Y	PIGMENT	N	N	N	N	Y	Y	N	N	Y	N	NAD
8	bindiya	23	S	belgaum	G		3m		R	T	V	N	Hair straightening		y		N	N	y	IRREG. CYCLES	NA	N	Y	N	Y	L-I	NEG	28.9	Y	N	N	Y	Y	N	N	N	N	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
9	kavita	22	HW	belgaum	G			11 m	R	S+T	F/MF	N	TEMP. Coloring		Y		N	N	N	R	NA	N	N	N	Y	L-I	NEG	22.91	Y	N	N	Y	Y	N	Y	N	N	Y	NAD	N	N	N	N	Y	Y	Y	N	N	Y	NAD
10	farzana bi	36	HW	belgaum	G			2yrs	R	T	F/MF	C	N		Y		N	N	Y	R	N	N	N	Y	N	L-I	NEG	24.2	Y	N	N	N	Y	N	Y	N	N	N	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
11	laxmi	38	O	ramdurg	G			1.5YR	R	S	F/MF	N	N		Y		N	N	Y	R	N	Y	N	Y	N	L-II	POS	22.05	Y	N	N	Y	Y	N	Y	N	y	Y	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
12	shanti	24	S	athani	G			5YR	R/BIB	S+T	F/V/P-T	HB	N			Y	N	OCPs	n	R	NA	N	N	N	N	L-II	NEG	23.47	Y	Y	Y	Y	Y	N	Y	N	Y	Y	NAD	N	Y	N	N	N	Y	Y	N	N	N	NAD
13	disha	31	HW	belgaum	G			1YR	R	T	MF/P-T	N	N		Y		N	N	N	R	N	Y	N	N	N	L-I	NEG	25.51	Y	N	N	N	N	Y	N	N	Y	N	NAD	N	N	N	N	N	Y	N	N	Y	N	NAD
14	shanti	28	O	belgaum	G		5M		R	S+T	F/P-T	N	N		Y		N	N	Y	R	N	N	N	N	N	L-I	NEG	19.3	N	N	N	Y	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
15	kavita	46	O	belgaum	G		8M		R	T	F/MF	N	TEMP. Coloring		Y		N	N	N	R	N	N	N	N	N	L-I	POS	21.03	Y	Y	N	N	Y	N	N	N	N	Y	NAD	N	N	N	N	Y	Y	N	N	N	N	NAD
16	fatima	41	HW	athani	G			3Y	R	S	F/MF	C	TEMP. Coloring			Y	N	N	N	IRREG. CYCLES	N	N	N	N	N	L-I	NEG	23.43	Y	Y	N	N	Y	Y	Y	N	N	N	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
17	sarla	37	HW	belgaum	G			4YR	R	S+T	F/V/P-T	N	TEMP. Coloring			Y	N	N	Y	R	N	N	N	N	N	L-III	NEG	26.56	Y	N	N	Y	Y	N	Y	N	N	N	NAD	Y	N	N	N	Y	Y	N	N	N	N	NAD
18	nirmala	34	HW	belgaum	G		5M		R	T	F/MF	N	N		Y		N	N	N	IRREG. CYCLES	YES	N	N	N	N	L-I	POS	23.04	Y	N	N	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	Y	N	N	Y	N	NAD
19	sarita	37	HW	belgaum	G			4YR	R	S+T	F/MF	N	TEMP. Coloring			Y	N	N	Y	R	N	N	N	N	N	L-II	NEG	22.01	Y	N	Y	Y	Y	N	Y	N	N	Y	NAD	Y	N	N	N	N	Y	N	N	N	N	NAD
20	mayuri	21	S	belgaum	G			2.5Y	R	T	F/MF	HB	N			Y	N	N	Y	IRREG. CYCLES	NA	Y	Y	N	N	MT	NEG	21.09	Y	Y	N	N	Y	Y	N	N	y	Y	NAD	N	Y	N	N	N	Y	N	N	N	N	NAD
21	sangeeta	22	S	athani	G		5M		R	S+T	F/MF	HB	Hair straightening		Y		N	N	Y	R	NA	Y	N	N	N	MT	NEG	18.93	Y	N	N	N	Y	Y	Y	N	Y	N	NAD	N	N	N	N	N	Y	N	N	Y	N	NAD
22	madhavi	26	O	belgaum	G			2yrs	R	T	F/MF	C	Hair straightening		Y		N	N	N	R	NA	Y	N	N	N	L-I	NEG	21.23	Y	N	N	N	Y	Y	N	N	Y	N	NAD	N	N	N	N	N	Y	N	N	Y	N	NAD
23	madhuri	46	HW	belgaum	G		6M		R	T	F/MF	N	N		Y		N	N	N	MP	N	N	N	N	N	L-I	NEG	22.26	Y	Y	Y	N	Y	Y	Y	N	N	Y	NAD	N	Y	N	N	N	Y	Y	N	N	N	NAD
24	nandini	18	S	belgaum	G	2M			R	S+T	F/MF	N	Hair straightening		Y		N	N	Y	R	NA	N	N	N	N	L-I	NEG	20.16	Y	Y	Y	N	N	Y	N	N	Y	N	NAD	N	Y	N	N	N	Y	N	N	N	N	NAD
25	umadevi	45	HW	hukkeri	G			3YRS	R	T	F/MF	N	N		Y	Y	N	N	Y	MP	N	Y	N	N	N	L-II	NEG	25	Y	Y	Y	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	Y	N	N	N	Y	NAD
26	shailaja	32	O	belgaum	G		5M		R/BIB	S+T	F/MF	N	TEMP. Coloring		Y		N	N	N	R	N	N	N	N	N	L-I	NEG	28.57	Y	N	Y	N	Y	Y	Y	N	N	N	NAD	Y	N	N	N	N	Y	N	N	N	N	NAD
27	neerja	47	HW	athani	G		4M		R	S+T	F/MF	C/HB	TEMP. Coloring		Y		N	N	N	MP	N	N	N	N	N	L-I	NEG	24.62	Y	N	N	Y	Y	N	N	N	Y	N	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
28	rekha	49	HW	belgaum	G		5m		R	S	F/MF	N	TEMP. Coloring		Y		N	N	Y	MP	N	Y	N	Y	N	L-I	NEG	26.97	Y	N	Y	N	Y	N	N	N	Y	Y	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
29	ruksana	29	HW	belgaum	G			1YR	R	S+T	F	HB	TEMP. Coloring		Y		N	N	Y	R	NA	Y	N	N	N	L-I	NEG	20.14	N	N	Y	N	Y	N	N	N	y	N	NAD	N	N	N	N	N	Y	Y	N	N	N	NAD
30	prashila	38	HW	belgaum	G		5M		R/BIB	S	F	C	N		Y		N	N	Y	R	N	Y	N	N	N	L-I	POS	24.6	Y	N	N	N	Y	Y	Y	N	Y	Y	NAD	N	N	N	N	N	N	Y	N	N	N	NAD
31	rekha	43	HW	belgaum	G			2yrs	R	S+T	F/MF	N	N		Y		N	N	Y	IRREG. CYCLES	N	Y	N	N	N	MT	NEG	26.98	Y	Y	Y	Y	Y	Y	Y	N	y	Y	NAD	N	N	Y	N	N	Y	Y	N	N	N	NAD
32	bibihazra	31	HW	khanapur	G			1.5YR	R	S+T	F/V/P-T	N	TEMP. Coloring			Y	N	N	Y	R	N	N	N	N	N	L-III	NEG	25.86	Y	N	N	Y	N	Y	Y	N	N	N	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
33	lalita	23	S	athani	G		4M		R	T	RH	C/HB	TEMP. Coloring		Y		N	N	Y	R	NA	N	N	N	N	MT	NEG	20.88	Y	N	N	Y	Y	Y	N	N	N	N	NAD	Y	N	N	N	Y	Y	N	N	N	N	NAD
34	pranjal	29	HW	ramdurg	G		5M		R	T	F	N	TEMP. Coloring		Y		N	OCPs	Y	IRREG. CYCLES	N	N	N	Y	Y	L-I	NEG	16.21	Y	N	N	N	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
35	seema	41	HW	belgaum	S	1MN			R/BIB	S+T	MF	N	Hair straightening		Y		N	N	N	R	N	Y	N	N	N	L-I	NEG	25	Y	Y	N	N	Y	Y	Y	N	y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
36	anuja	22	O	belgaum	G		5M		R	S+T	F/MF	N	N		Y		N	N	N	R	N	Y	N	N	N	L-I	NEG	23	Y	N	Y	Y	N	N	N	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
37	pranali	27	O	belgaum	G			3YRS	R	S+T	F/MF	N	Hair straightening		Y		N	N	N	IRREG. CYCLES	NA	N	N	N	N	L-II	NEG	24.32	Y	N	Y	N	Y	N	Y	N	N	N	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
38	meghana	45	O	belgaum	G		4M		R	S+T	F/V/P-T	N	C+S			Y	N	N	Y	R	NA	N	N	N	N	L-II	NEG	20.95	Y	N	N	Y	Y	Y	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
39	gayatri	34	HW	belgaum	G		5M		R	S	F/V	N	TEMP. Coloring		Y		N	N	N	R	N	N	N	N	N	L-I	POS	20.37	Y	N	Y	N	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
40	megha	22	O	gokak	G			3Y	R/BIB	S+T	F/V/P-T	N	C+S			Y	N	N	N	R	NA	N	N	N	N	L-II	POS	21.13	Y	N	N	Y	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
41	parvati	45	HW	hukkeri	G			2yrs	R	S+T	MF	N	TEMP. Coloring			Y	N	N	Y	R	N	Y	N	N	Y	L-II	NEG	24.1	Y	N	Y	Y	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
42	rupali	31	HW	belgaum	G		5M		R	T	V	N	Hair straightening		Y		N	N	Y	R	N	N	Y	N	N	L-I	NEG	19.1	Y	N	N	N	Y	N	Y	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
43	pratima	42	HW	khanapur	G			1.5YR	R	S+T	MF	N	TEMP. Coloring		Y		N	N	N	IRREG. CYCLES	N	N	N	N	Y	L-I	NEG	18.6	Y	N	Y	N	Y	Y	N	Y	N	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
44	abhilasha	24	S	athani	G		6M		R	T	V	N	N				N	N	N	R	N	N	N	N	N	L-I	NEG	20.3	Y	Y	N	N	Y		Y	N	Y	N	PIGMENT	N	N	N	N	N	N	N	N	N	N	NAD
45	mansi	47	HW	gokak	G			2Y	R	S+T	F/V/P-T	N	TEMP.COLORING			Y	N	N	N	MP	N	N	N	N	N	L-II	NEG	24.62	Y	N	N	N	Y	Y	Y	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
46	shaila	48	HW	belgaum	S	2M			R	T	F/MF	HB	TEMP.COLORING		Y		N	N	Y	MP	NA	N	Y	N	N	L-I	POS	22.26	Y	Y	Y	N	Y	N	Y	N	N	Y	NAD	N	N	N	N	N	N	N	N	N	Y	NAD
47	aishwarya	21	S	belgaum	G			4Y	R	S+T	F/MF	HB	N		Y		N	N	Y	R	NA	Y	N	N	N	L-I	NEG	22.51	N	N	N	N	Y	Y	Y	N	Y	N	NAD	N	N	N	N	N	N	Y	N	N	N	NAD
48	dipti	31	HW	athani	G			1Y	R	T	F/MF	N	N		Y		N	N	N	R	N	Y	N	N	N	L-II	NEG	21.45	Y	N	N	Y	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	Y	Y	N	N	N	NAD
49	sonia	50	HW	ramdurg	G			1Y	R	T	F/MF	N	TEMP.COLORING			Y	N	N	N	MP	NA	Y	N	N	N	L-II	NEG	26.02	Y	N	Y	Y	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	Y	N	N	N	NAD
50	sushma	45	HW	belgaum	G			4Y	R	T	F/MF	C/HB	N		Y		N	N	Y	R	N	N	N	N	N	L-II	NEG	28.61	Y	N	N	Y	Y	N	N	N	N	Y	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
51	jayashri	23	S	gokak	S	2M			R	S+T	F/MF	N	N			Y	N	N	N	IRREG CYCLES	NA	Y	Y	N	N	L-I	POS	21.8	Y	N	Y	Y	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	Y	N	N	N	NAD
52	mayuri	30	HW	athani	G			1YR	R	T	F	N	N		Y		N	N	N	R	N	N	N	N	N	L-I	NEG	19.2	Y	N	N	N	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	Y	N	N	N	NAD
53	bharti	28	HW	ramdurg	S	3M			R	T	F	N	N		Y		N	N	Y	R	N	Y	N	N	N	L-I	POS	21.6	Y	N	N	N	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	N	Y	N	N	N	NAD

54	deeksha	51	O	khanapur	S	2M			R	T	MF	N	N		Y		N	N	N	R	N	Y	N	N	N	L-I	NEG	25	y	N	Y	N	N	Y	N	N	Y	N	NAD	N	N	Y	N	N	N	N	N	N	N	NAD
55	gouri	37	HW	belgaum	G	1MN			R	T	MF	N	TEMP.COLORING			Y	N	N	N	R	N	Y	N	N	N	L-II	NEG	22.5	Y	N	N	Y	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
56	sakira	44	HW	khanapur	G			1YR	R	S+T	MF/F	N	N		Y		N	N	N	MP	N	Y	N	N	N	L-I	NEG	21.2	Y	N	N	N	N	Y	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
57	parveen	36	HW	belgaum	G		5M		R	T	V	N	TEMP.COLORING		Y		N	N	Y	R	N	Y	N	Y	Y	L-I	NEG	21.8	Y	N	N	N	Y	N	N	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
58	naseema	42	HW	gokak	G		6M		R	T	MF	HB	N		Y		N	N	Y	R	N	N	N	N	N	L-I	NEG	20.7	Y	Y	N	N	N	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
59	AKSHADA	20	S	khanapur	G			5YR	R	T	MF/P-T	C/HB	N			Y	N	N	Y	R	NA	Y	N	N	N	L-II	NEG	20.7	Y	N	N	N	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	Y	N	N	N	N	NAD
60	MANESHA	26	O	athani	G	2M			R	T	MF	HB	N		Y		N	N	Y	R	N	N	N	N	N	L-I	NEG	21.3	Y	N	N	N	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
61	shila	20	S	belgaum	G			1YR	R	T	M-F/F	HB	N		Y		N	N	Y	R	NA	Y	N	N	N	L-I	NEG	20.4	Y	N	Y	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
62	sashikala	23	S	khanapur	G			2yrs	R	T	MF	C/HB	TEMP.COLORING		Y		N	N	N	R	NA	Y	Y	N	N	MP	NEG	19.5	Y	Y	N	N	N	N	N	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
63	vijaya	32	HW	bailhongal	G		6M		R	T	MF/V	N	N		Y		N	N	Y	R	N	Y	N	N	Y	L-II	NEG	19.2	Y	Y	N	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
64	madhavi	24	HW	belgaum	G		4M		R	T	MF	N	N		Y		N	N	N	R	NA	N	N	N	N	L-I	NEG	21.9	Y	N	N	N	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
65	shanti	52	HW	khanapur	G			1YR	R	T	MF	C/HB	TEMP.COLORING		Y		N	N	Y	MP	N	Y	N	N	N	L-I	NEG	20.6	Y	N	N	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
66	RINKI DEVI	45	HW	bailhongal	G			2YRS	R	T	MF/V	N AYURVEDIC SOLN	N		Y		N	N	Y	R	N	N	N	N	N	L-II	NEG	19.5	Y	Y	N	Y	Y	N	N	N	N	N	NAD	N	Y	N	N	N	N	N	N	Y	N	NAD
67	MEERA	43	O	belgaum	G			3YRS	R	RH+T	MF/F	C	N		Y		N	N	Y	R	Y	Y	N	N	N	MP	NEG	18.11	Y	Y	N	N	Y	N	N	N	Y	Y	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
68	aishwarya	45	HW	belgaum	G		6M		R	T	MF	C/HB	TEMP.COLORING			Y	N	N	Y	MP	N	Y	N	Y	N	L-II	NEG	21.7	Y	N	N	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
69	SAMIKSHA	21	S	gokak	G			5YRS	R	S+T	MF/P-T	N AYURVEDIC SOLN	N			Y	N	N	Y	R	NA	N	N	N	N	L-III	NEG	25.6	Y	Y	Y	Y	Y	N	N	N	N	N	NAD	N	N	N	N	Y	N	N	N	N	N	NAD
70	MEENAXI	33	HW	belgaum	G			1YR	R	S+T	MF/V	C	N			Y	N	N	Y	R	Y	N	N	N	N	L-II	NEG	19.6	Y	N	Y	Y	Y	N	N	N	Y	N	NAD	N	N	NN	N	N	N	N	N	N	N	NAD
71	SANGITA	29	HW	athani	G			9 M	R	S	MF/V	N	N		Y		N	N	Y	R	NA	Y	Y	N	N	L-II	NEG	20.6	Y	Y	N	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
72	SALONI	20	S	khanapur	G			2 YRS	R	T	MF/V/P-T	N	N			Y	N	N	Y	R	NA	N	N	N	N	L-III	NEG	22.1	Y	N	N	Y	Y	N	Y	N	N	Y	NAD	N	N	N	N	Y	N	N	N	Y	N	NAD
73	yallava	45	HW	belgaum	G		6M		BIB	S+T	MF/V	C	TEMP.COLORING			Y	N	N	N	MP	N	N	N	Y	N	L-II	NEG	19.5	Y	N	Y	N	Y	N	N	N	N	Y	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
74	RAJASHRI	48	HW	belgaum	G		5 M		R	S+T	MF/V	N	N		Y		N	N	Y	MP	N	Y	N	N	N	L-I	NEG	20.1	Y	N	N	N	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
75	SOUMYA	21	S	belgaum	G		5M		R	RH	FT	N	N		Y		N	N	N	R	NA	N	N	N	N	MP	NEG	20.1	Y	N	N	N	N	N	N	N	N	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
76	NIROOPA	31	HW	belgaum	G		6M		R	T	V	N	N		Y		N	N	N	R	N	N	N	N	N	L-I	NEG	21.9	Y	Y	N	N	N	N	Y	Y	N	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
77	RASHMI	20	S	athani	G		4M		R	T	V	N	N		Y		N	N	N	R	NA	Y	N	N	N	L-I	NEG	19.2	Y	N	N	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
78	LAXMI DEVI	41	HW	khanapur	G	3M			R	S+T	V/P-T	N	N			Y	N	N	N	R	N	Y	N	N	N	L-II	NEG	19.4	Y	N	N	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
79	JAYA	29	O	belgaum	G		4M		BIB	T	V	N	N		Y		N	N	Y	R	NA	Y	Y	N	N	L-I	NEG	21	Y	N	Y	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
80	JYOTI M	39	O	khanapur	G		6M		R	T	V/P-T	C/HB	N			Y	N	N	Y	R	N	Y	Y	N	N	L-II	NEG	23.6	Y	N	N	Y	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
81	ZEENA	22	S	khanapur	G		8M		R	T	MF/V	N	N		Y		N	N	N	R	NA	Y	N	N	N	L-I	NEG	19.6	Y	N	N	N	Y	N	Y	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
82	CHITRA	39	HW	athani	G			1YR	R/BIB	T	MF	C/HB	N		Y		N	N	Y	R	N	Y	N	N	N	L-I	NEG	21.6	Y	N	Y	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
83	TANVI	24	S	belgaum	G		6M		R	T	MF	N	Hair straightening		Y		N	N	N	IRREG CYCLES	NA	Y	N	N	Y	L-I	NEG	19.2	Y	N	Y	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
84	SHIVANI	31	HW	belgaum	G		4M		R	S+T	MF/P-T	HB	N		Y		N	N	Y	R	N	N	N	N	N	L-II	NEG	22.6	Y	N	N	N	Y	N	Y	N	N	Y	NAD	N	N	N	N	N	N	Y	N	Y	N	NAD
85	NISHA	29	HW	khanapur	G		6M		R	T	MF	N	N		Y		N	N	Y	R	N	Y	Y	N	N	L-I	NEG	20.1	Y	Y	N	N	Y	N	N	N	Y	N	NAD	Y	N	N	N	N	N	N	N	Y	N	NAD
86	madhavi	35	HW	belgaum	G			1YR	R/BIB	T	MF/P-T	N	N			Y	N	N	N	R	N	N	N	N	Y	MP	NEG	19	Y	N	N	Y	Y	N	Y	N	N	Y	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
87	JAYANTI	40	HW	athani	G			3YRS	R	T	MF/P-T	N	N			Y	N	N	Y	IRREG CYCLES	N	Y	N	N	N	L-III	NEG	23	Y	Y	N	Y	Y	N	Y	N	Y	N	NAD	N	Y	N	N	Y	N	N	N	N	N	NAD
88	preeti	27	S	belgaum	G		4M		R	T	MF	N	N		Y		N	N	N	R	NA	Y	N	N	N	L-I	NEG	21.4	Y	N	Y	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	Y	N	NAD
89	NATASHA	26	O	khanapur	G		6M		R	T	MF	C/HB	N		Y		N	N	N	R	NA	N	N	N	N	L-I	NEG	21.5	Y	N	Y	N	Y	N	N	Y	N	N	NAD	N	N	N	N	Y	N	N	N	Y	N	NAD
90	ADITI	32	HW	belgaum	G		4M		R	T	MF	N	N		Y		N	N	Y	R	N	Y	Y	N	N	L-I	NEG	19.27	Y	N	N	N	N	N	Y	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
91	ROSHNI	22	S	khanapur	G			1.5 YRS	R	S+T	MF/P-T	HB AYURVEDIC SOLN	Hair straightening			Y	N	N	N	R	NA	N	N	N	N	L-II	NEG	22.19	Y	N	Y	N	Y	N	Y	N	N	Y	NAD	N	N	N	N	N	N	Y	N	Y	N	NAD
92	URVASHI	39	HW	belgaum	G		6M		R	RH+T	MF	N	N		Y		N	N	Y	R	N	N	N	N	Y	MP	NEG	18.7	Y	N	N	N	Y	N	Y	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
93	APURVA	26	O	athani	G			1YR	R	T	MF	N	N		Y		N	N	Y	R	NA	N	N	N	N	L-I	NEG	22.5	Y	N	N	N	Y	N	N	N	N	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
94	MAYOORI	29	O	khanapur	G		4M		R	T	MF	N	N			Y	N	N	Y	R	NA	Y	N	N	N	L-I	NEG	20.21	Y	N	N	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
95	APURVA	35	O	belgaum	G			1YR	R	T	MF/V	C/HB	N		Y		N	N	N	R	N	N	N	N	Y	L-II	NEG	21.7	Y	N	Y	N	N	N	Y	N	N	Y	NAD	N	N	N	N	N	N	Y	N	Y	N	NAD
96	naina	30	O	belgaum	G		6M		R	T	MF	N	N		Y		N	OCPS	Y	R	N	N	N	N	N	L-I	NEG	19.26	Y	N	N	N	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
97	SUVARNA K	36	HW	athani	G			2 YRS	R	T	MF/P-T	N	TEMP.COLORING			Y	N	N	Y	R	N	Y	N	N	N	L-II	NEG	22.7	Y	Y	N	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	Y	N	N	N	Y	N	NAD
98	SEJAL	22	S	belgaum	G		7M		R	T	MF	C/HB AYURVEDIC SOLN	N		Y		N	N	N	R	NA	N	N	N	N	L-I	NEG	18.19	Y	N	N	N	Y	N	N	N	Y	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
99	KRITI K	25	S	khanapur	G		4M		R	T	MF	N	N		Y		N	N	N	R	NA	Y	N	N	Y	L-I	NEG	19	Y	N	Y	N	Y	N	N	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
100	WAHIDA	41	HW	khanapur	G			1.5 YRS	R	T	MF/V	N	N			Y	N	N	N	IRREG CYCLES	N	N	N	N	N	L-II	NEG	21.6	Y	Y	N	N	Y	N	Y	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
101	manali	35	O	belgaum	G	2m			R	T	MF	HB	N		Y		N	N	Y	R	N	N	N	N	N	L-I	NEG	20.37	Y	Y	N	N	Y	Y	Y	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
102	purva	30	O	belgaum	G			1yr	R	T	MF	HB	N		Y		N	N	Y	R	N	N	N	N	N	L-I	NEG	22.2	Y	N	Y	Y	Y	N	Y	N	N	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
103	pranjal	36	HW	belgaum	G			2 YRS	R	T	V	N	N		Y		N	N	Y	R	N	N	N	N	Y	L-I	NEG	24.15	Y	N	N	N	Y	N	Y	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
104	savitri	22	S	belgaum	G			2yrs	R	T	V	N	N			Y	N	N	Y	R	NA	Y	Y	N	N	L-I	NEG	29.91	Y	N	N	N	Y	Y	N	N	N	N	NAD	N	N	N	N	N	N	Y	N	Y	N	NAD
105	megha	25	S	belgaum	G			1yr	R	T	V	N	N		Y		N	N	Y	R	NA	N	Y	N	N	L-I	NEG	25.64	Y	Y	N	N	Y	Y	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
106	maria	41	HW	chikkodi	G			1yr	R	T	V	N	C+S		Y		N	N	Y	R	N	N	N	Y	N	L-I	NEG	21.7	Y	N	N	N	Y	N	N	N	N	Y	NAD	N	N	N	N	Y	N	N	N	Y	N	NAD
107	sejal	28	S	belgaum	G		5M		R	T	MF	N	C+S		Y		N	N	Y	R	N	Y	Y	N	N	L-II	NEG	19.26	Y	N	Y	N	Y	N	Y	N	Y	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
108	sulochana	22	S	belgaum	G			1Y	R	T	MF/P-T	N	C+S		Y		N	N	Y	R	NA	N	Y	N	N	L-I	NEG	22.7	Y	N	N	Y	Y	N	N	N	N	N	NAD	N	N	N	N	N	N	N	N	N	N	NAD
109	madhuri	32	O	belgaum	G			1.5YR	R	T	MF/P-T	N	C+S		Y		N	N	Y	R	N	N	N	N	N	L-I	NEG	18.19	Y	N	N	Y	Y	N	Y	N	N	Y	NAD	N	N	N	N	N	N	N	N	N	N	NAD
110	pratiksha	29	O	belgaum	G			2yrs	R	T	MF/P-T	N	C+S		Y		N	N	Y	R	N	N	N	N	N	L-II	NEG	19	Y	Y	N	N	Y	N	Y	Y	Y	N	PIGMENT	N	N	N	N	N	N	N	N	N	N	NAD

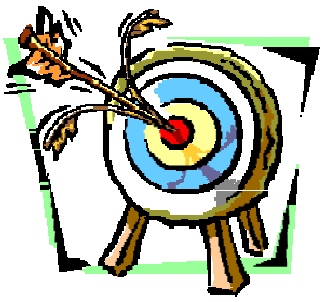
ANNEXURE-VI

KEY TO MASTER CHART

- Occupation: HW- Housewife, S- student, O- Others.
- Onset: G- gradual, S- sudden.
- Site of hair fall- R- roots, BIB- breaking in between.
- Characteristics of hair loss- RH- recession of hair line, S- shedding, T- thinning.
- Region involved: F- Frontal, MF- mid-frontal, V- vertex, P-T- parieto-temporal.
- Exacerbating factors: C- Combing, HB- head bath, Soln.- Solution, N- None.
- Cosmetic procedures: C+S- colouring + straightening, T.emp. Colouring- temporary colouring, N- none.
- Medical illness: DM- Diabetes Mellitus, HTN- Hypertension, BA- Bronchial Asthma, N- none.
- Drug history: AHTsive- Antihypertensive, OHAs- Oral hypoglycaemic agents, OCPs- oral contraceptive pills.
- Family history: Y- Yes, N-NO.
- Menstrual cycles: R- regular, MP- menopausal, Irreg. Cycles-irregular cycles.
- Infertility: Y- yes, N-No, NA- Not Applicable.
- SD: seborrheic dermatitis, Y-Yes, N- No
- Acne: Y-Yes, N- No.
- AN: Acanthosis Nigricans, Y-Yes, N- No
- Hirsuitism: Y-Yes, N- No
- Grade: L-I→ Ludwig's I, L-II→ Ludwig's II, L-III→ Ludwig's III, MT- Male type of fronto-temporal recession.
- Hair pull test: Pos- Positive, Neg- Negative

- BMI- body mass index
- Hb- haemoglobin
- TSH- thyroid stimulating hormone.
- FT3- Free T3
- FT4- free T4
- Anti-TPO- anti- thyroid peroxidase antibody
- PRL- Prolactin
- LH- luteinizing hormone
- FSH- follicle stimulating hormone
- DHEAS- Dehydroepiandrosterone sulphate.
- Pelvic USG: PCOD- polycystic ovarian disease, NAD- No abnormality detected.
- HDD- hair diameter diversity
- BPPS- brown peripilar sign
- WPPS- white peripilar sign
- FA- focal atrichia
- 1/HU-1 hair per unit
- 2-3/HU- 2-3 hairs per hair unit
- WD-white dots
- YD-Yellow dots
- HCP- Honey comb pigmentation
- NAD- no abnormality detected
- Y-Yes
- N-No

Introduction



Aims & Objectives

Review of Literature

Methodology

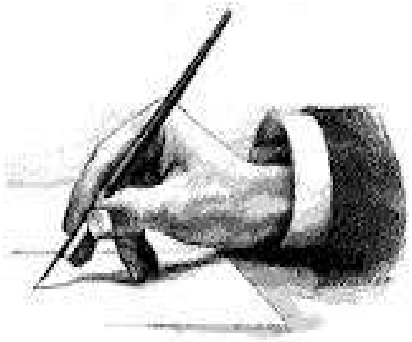
Results

Discussion

Conclusion

Summary

Bibliography



Annexure-I

Annexure-II

Annexure-IV

Annexure-V

Annexure-VI