"ASSOCIATION OF CLINICAL FEATURES, BIOLOGICAL PARAMETERS AND TRICHOSCOPIC PATTERNS IN FEMALE PATTERN BALDNESS-A CROSS SECTIONAL STUDY"

By

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DISSERTATION SUBMITTED TO

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DERMATOLOGY, VENEREOLOGY AND LEPROSY

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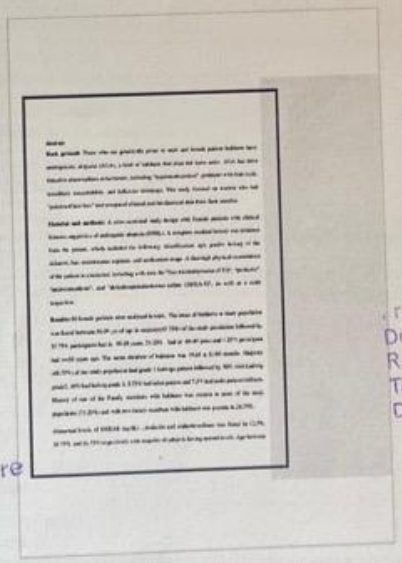

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LIST OF ABBREVIATIONS

Glossary	Abbreviations
5R	5'-reductase
AA	Alopecia areata
AAI	Alopecia areata incognita
AGA	Androgenetic alopecia
AR	Androgen receptor
BD	Black dot
BH	Broken hair
BMP	Bone morphogenetic protein
BPPS	Brown peripilar sign
CH	Coudability hair
DHT	Dihydrotestosterone
DP	Dermal papilla
Dsh	Dishevelled
FPHL	Female pattern hair loss
HDD	Hair shaft diameter diversity
HFs	Hair follicles
HSTH	Hair shaft thickness heterogeneity
ROS	Reactive oxygen species
Shh	Sonic hedgehog
SVH	Short vellus hairs
T	Testosterone
TC	Tinea capitis
TE	Telogen effluvium
TH	Tapering hair
TTA	Temporal triangular alopecia
TTM	Trichotillomania
WD	White dots
Wnt	Wingless type

WPPS	White peripilar sign
YD	Yellow dots

Abstract

Introduction:

Androgenetic alopecia (AGA) is a non-scarring alopecia that affects both men and women who are genetically predisposed to it. AGA has been linked to hormonal abnormalities, including hyperandrogenism, hair cycle issues, genetic predisposition, and follicular miniaturization. The purpose of this study was to examine women who experienced patterned hair loss and compare their clinical results to biochemical measurements.

Material and methods: A cross-sectional study design with Female patients with clinical features suggestive of androgenic alopecia (FPHL). A detailed history of the patient including name, age, sex, history of presenting illness, hair grooming pattern, habits & tics, nail changes, other skin changes, systemic disease, family history of similar complaints and drug intake were recorded.General physical examination of patient is done, scalp examination, hormone analysis of Dehydroepiandrosterone-sulfate (DHEA-S), prolactin, androstenedione, and free triiodothyronine (FT3), tests were done.

Results: a total of 80 women subjects were analaysed. The onset of baldness in study population was found between 30-39 yrs of age in majority(43.75%) of the study population followed by 33.75% participants had in 19-29 years, 21.25% had at 40-49 years and 1.25% participant had >=50 years age. The mean duration of baldness was 19.68 ± 21.90 months. Majority (48.75%) of the study population had grade 1 ludwigs pattern followed by 30% with Ludwig grade2, 10% had ludwig grade 3, 3.75% had oslen pattern and 7.5% had male pattern baldness. History of one of the Family members with baldness was present in most of the study population (71.25%) and with two family members with baldness was present in 28.75%.

Abnormal levels of DHEAS (ug/dL) , prolactin and androstenedione was found in 12.5%, 28.75% and 18.75% respectively with majority of subjects having normal levels. Age between 19-49 yrs had ludwigs grade 1 pattern of baldness while >50yrs of age had grade 2. The onset

of baldness in grade 1 was majorly found in between 19- 39yrs of age and >40 and >50 had grade 2.The proportion of pattern of baldness who had one family member history of baldness are as follows: Ludwig grade 1, Ludwig grade 2, Ludwig grade 3 and Oslen pattern the majority of 69.23%, 75%, 87.5% and 100.00% females had one family member. In Pattern of baldness with male pattern, the majority (66.67%) female had two family members. The mean difference of DHEAS (ug/dL) in pattern of baldness was statistically not significant (P value > 0.05).The mean difference of Prolactin(ng/mL) in pattern of baldness was statistically not significant (P value > 0.05).The mean difference of Androstenedione(ng/mL) in pattern of baldness was statistically not significant (P value > 0.05). The mean difference of DHEAS(ug/dL) in pattern of baldness was statistically significant (P value < 0.05).The mean difference of Prolactin(ng/mL) in pattern of baldness was statistically not significant (P value > 0.05).The mean difference of Androstenedione(ng/mL) in pattern of baldness was statistically significant (P value < 0.05). The difference in the proportion of prolactin(ng/mL) group between pattern of baldness was statistically not significant with P value 0.8698 with majority of 76.92% participants were in Ludwig grade 1 had normal prolactin(ng/mL) and 23.08% had abnormal prolactin(ng/mL).A weak positive correlation between Pattern of baldness and DHEAS (ug/dL), androstenedione and prolactin (r_s value: 0.02,0.09 and 0.17, P value: 0.8951).

Conclusions: All female patients with FPHL should be assessed for underlying hormonal imbalances because the biochemical results of our study support the role of hyperandrogenism as one of the main etiological factors in FPHL, with the role of adrenal androgens being central.

Keywords: Androgenetic alopecia, female pattern hair loss, hormone profile

INTRODUCTION

INTRODUCTION:

Hair is considered as one of the essential skin appendages in human. Thinning, damaging or abnormal shedding of hair can impair the physical protection of scalp and affects mental health. It can also lead to severe social dysfunction.

Worldwide, androgenic alopecia is the most common hair loss disease. It affects both the male and female. The process of hair loss in androgenic hair loss is associated to the gradual miniaturization of hair follicles and gradually leads to progressive, symmetric baldness.

Male pattern hair loss and female pattern hair loss are the two major hair loss patterns described in androgenic alopecia. Male pattern hair loss is also called as male pattern baldness and it manifests as a recession of the frontal hairline with or without baldness of the vertex area.

Female pattern hair loss is the nonscarring progressive thinning of hair with a gradual decline in the number of hair. It is mostly seen in the frontal, central and parietal scalp area. The loss of terminal hairs in the affected areas are mostly incomplete and the frontal hairline is usually spared. It is due to the progressive decline in the ratio of the terminal to vellus hair and is known as follicular miniaturization. [1]

Female pattern hair loss , diffuse hormonal alopecia and common baldness in women are the synonyms for the androgenetic alopecia in women. The term female pattern baldness was utilized for diffuse alopecia in females since it was thought to be a variant of androgenetic alopecia in women. The role of androgens in the development of female pattern baldness has not been fully identified, hence the term female pattern hair loss.

In women, FPHL typically presents as a diffuse reduction in hair density over the frontal and vertex areas, but parietal and occipital regions may be involved.

FPHL may have three different patterns.

(1) diffuse thinning of the crown region with preservation of the frontal hairline: two scales are used to describe this pattern: the commonly 3-point Ludwig scale and the 5-point Sinclair scale

(2) thinning and widening of the central part of the scalp with breach of frontal hairline, described by Olsen scale: Christmas tree pattern

(3) thinning associated with bitemporal recession; Hamilton-Norwood scale. [2,3]

It is proposed that the etiopathogenetic factors for androgenic alopecia are the genetic predisposition and hormonal abnormalities such as hyperandrogenism, hair cycle defects and follicular miniaturization.[4] Initial symptoms of female pattern hair loss develops during the teenage years and can lead to progressive hair loss with characteristic pattern distribution.[5]

The cause of alopecia in women is more difficult to assess clinically and the clinical diagnosis is also challenging. . Trichoscopy is a new method to diagnose hair loss using the dermoscopy of hair, scalp, eyebrows and eyelashes to see and count hair at high magnification. Trichoscopic evaluation of the scalp is based on the study of follicular, interfollicular and perifollicular hair shaft patterns and hair signs.[6]

NEED OF THE STUDY

The pathogenesis of patterned baldness are identified as different in men and women. Hence, female pattern hair loss is not the same as androgenetic alopecia found in women. The role of hyperandrogenemia in female pattern of hair loss is not very clear, but it has been observed that levels of androstenedione and dehydroepiandrosterone are increased in few cases. However, most women with FPHL have no other signs or symptoms of hyperandrogenism and have normal androgen levels, indicating that the pathogenesis of the disorder remains incomplete. Also the classification followed for the patterned baldness is different in males and females. Androgenetic alopecia in female with male pattern baldness is very uncommon and its pathogenesis remains unclear. The trichoscopy development has led to the establishment of diagnostic features of hair disorders. But trichoscopic features of all grades and different patterns of FPHL and its possible association with other clinic-biological parameters has not been specifically evaluated previously.

AIMS AND OBJECTIVES

Aims and Objectives of the Study

1.To determine the proportion of clinical features, biological parameters and trichoscopic findings in patients with female pattern baldness.

2.To assess the association of clinical features, biological parameters and trichoscopic findings in patients with female pattern baldness.

REVIEW OF LITERATURE

Review of literature:

Alopecia is the medical term for hair thinning or hair loss. The Greek word for fox was originally used to create the word "alopecia." It alludes to the constant hair loss experienced throughout these animals lifetime. Human hair is very important for social interaction as well as playing a crucial role in how we look physically. Losing scalp hair causes social inhibition, anxiety, and depression.[7]

"What (Time) hath scanted men in hair he hath given them in wit" by Shakespeare from "The comedy of errors".

Due to the lack of knowledge regarding the pathogenesis of androgenetic alopecia (AGA) in late 1500 AD, Shakespeare's time, people had two choices: they could either accept their condition or use wigs for cosmetic reasons.According to Greek historian Herodotus (490/480-424BC), the Egyptian "Physician of the Head," who treated ailments of the scalp, is one of the earliest medical specialties. In 350 BC, Aristotle had explained the cause of baldness in men as being due to natural humidity and heat as well as why eunuchs and women do not develop baldness. Later in 1942, Hamilton demonstrated the involvement of male hormones in development of pattern baldness in men; the term AGA was coined to highlight the associated genetic and hormonal factors with the development of the disease.[8]

Mammalian skin contains hair follicles (HFs), which produce filaments primarily made of the protein keratin. Hair can either be sebaceous, lanugo, short vellus, intermediate, or long terminal hair. In (Figure 1) The epidermis depends on hair to keep up the body's defenses against the outside world. However, in humans, the body hair has largely lost its significance as a barrier of physical protection. Many theories have been proposed to explain how humans evolved to have drastically different hair distribution patterns than the majority of other mammals. [9]

These include the process of adaptation to offer a more effective thermoregulatory system in the hot and dry climate conditions that first appeared around 3 million years ago when forests gave way to hot Savanna grasslands. Another theory is that men and women have different hair patterns due to sexual selection, which is supported by this dimorphism.

Hair still has a significant social impact on people, though. Healthy hair is a sign of vitality, youth, and health. Male pattern baldness, which can be hidden with a toupee, hats, or even just by shaving the head, is thought to be a sign of aging and vigor loss.[10]

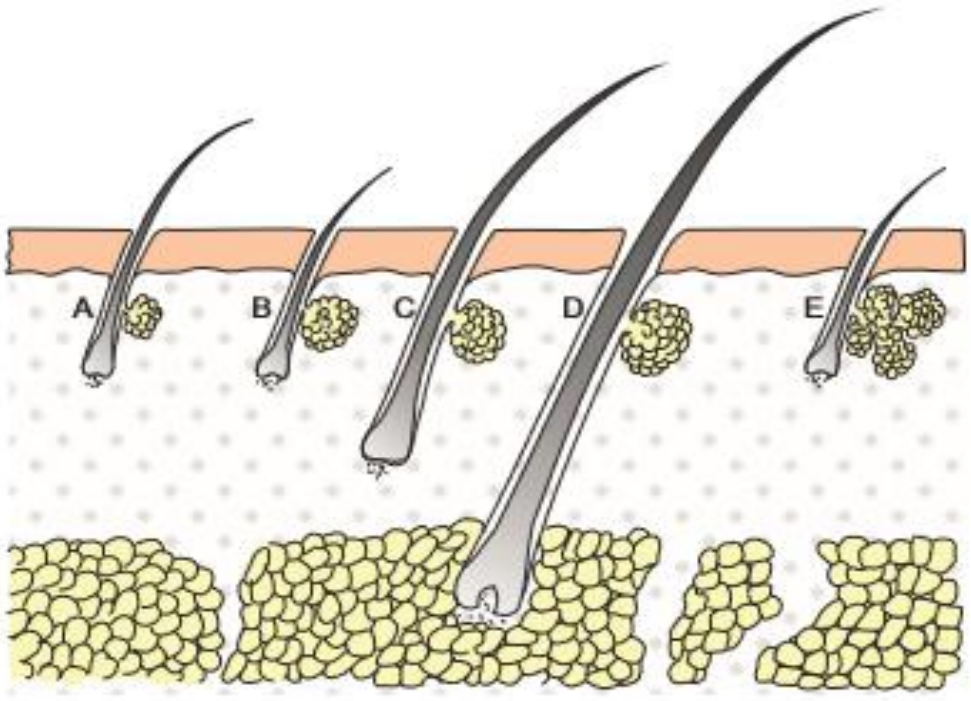


Figure 1: Types of hair follicles.[11]

A. Lanugo hair, B. Vellus hair, C. Intermediate hair D. Terminal hair, E. Sebaceous hair

HAIR FOLLICLE, HAIR CYCLE, STEM CELLS

HAIR FOLLICLE

The hair follicle penetrates every layer of skin. The medulla, cortex, and cuticle are three concentric regions that make up each hair strand (Figure 2). The function of the innermost medulla, a variable space that is sometimes present and other times absent, is still up for debate. The primary source of the hair's mechanical strength is its highly structured cortex. Melanin, which is found in the cortex, gives the fiber its color based on the quantity, distribution, and kinds of melanin granules. Hairs with a circular cross-section are straight, while those with an oval cross-section are curly because the shape of the hair follicle determines the shape of the cortex.[12]

The cuticle is indeed the outer covering of protein that is covered in a single lipid molecular layer to make the hair water-repellent. Human hair can range in diameter from 17 to 180 m.12 The hair follicle, which is embedded in the skin and made up of the papilla, matrix, root sheath, and bulge, is a complex mini-organ.[13]

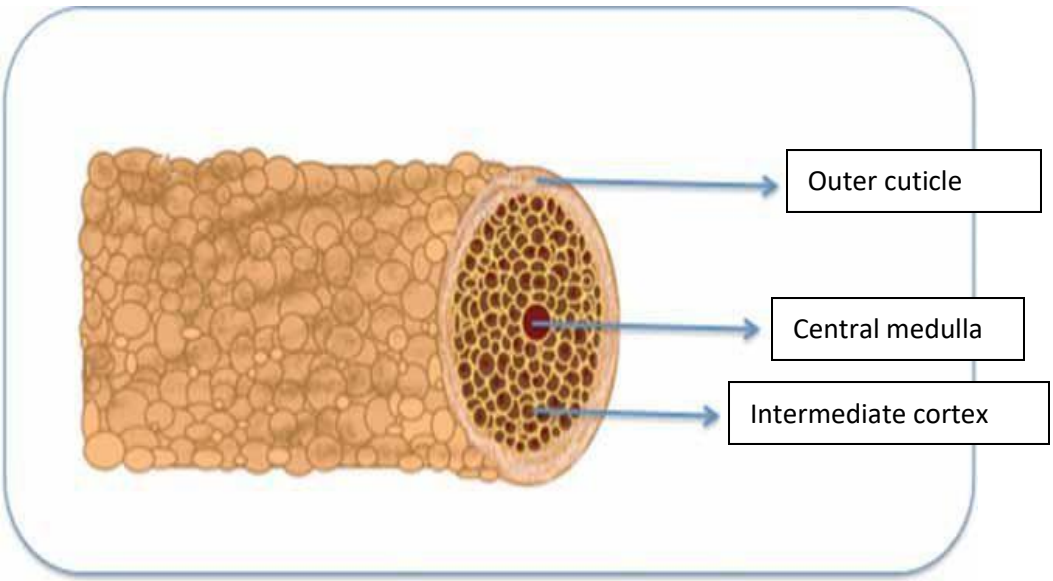


Figure 2. Cross-section of a normal hair showing central medulla, intermediate cortex, and outer cuticle.[14]

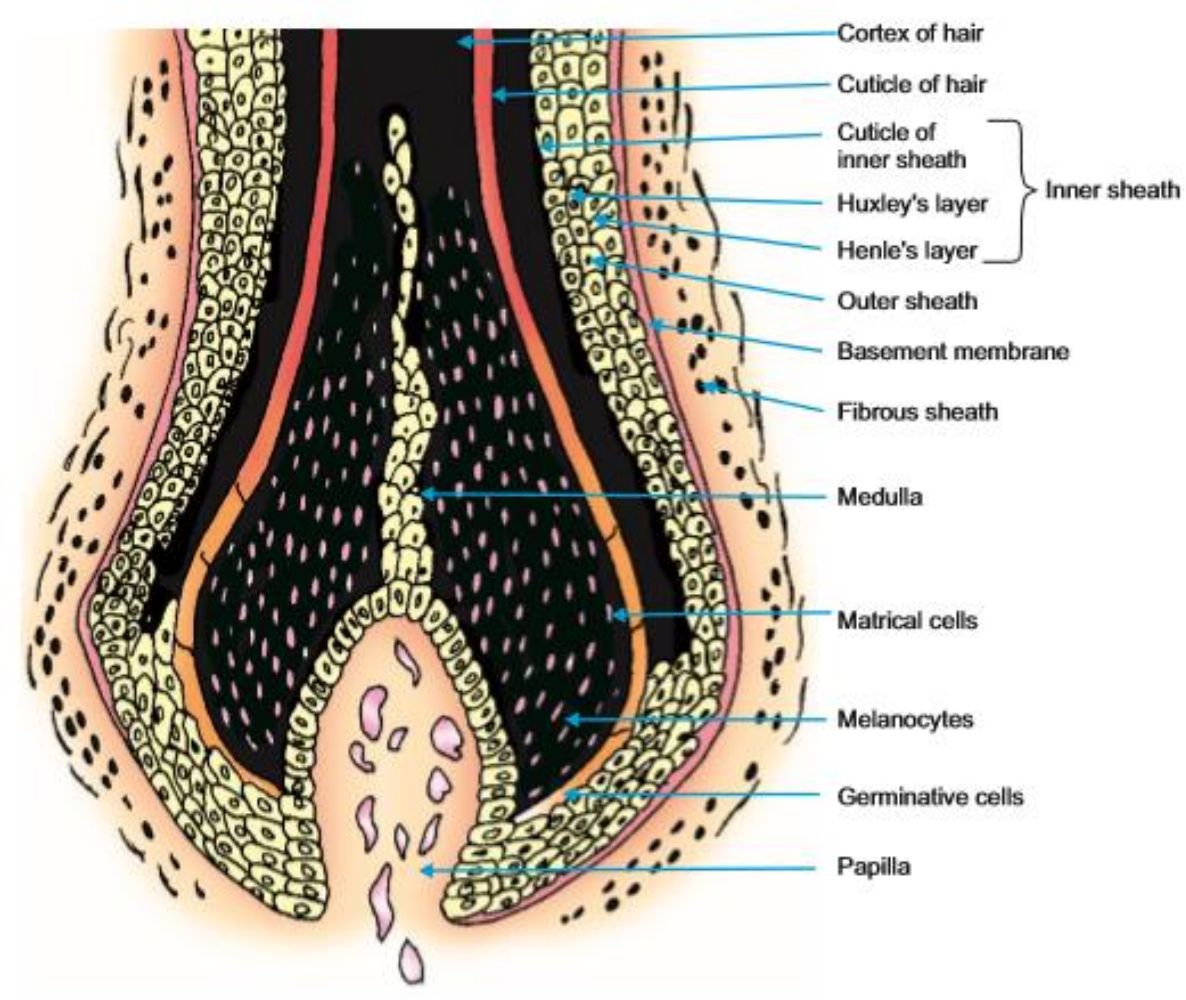


Figure 3: Differentiation of cells of follicular matrix along seven separate lines[11]

Structures closely associated with the Hair Follicle are sebaceous glands, apocrine glands, the arrector pili muscle and mechanoreceptors that respond to touch. **Figure 2** shows the anatomy of the Hair Follicle. There are between 250,000 and 500,000 Hair Follicle on the human scalp and as many as 5,000,000 on the whole body. [15,16]

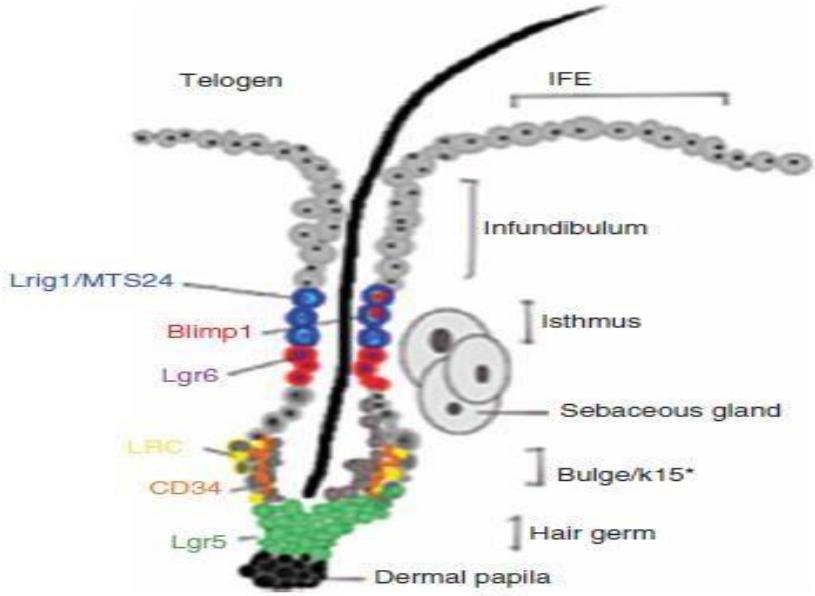


Figure 4. Organization of telogen-phase adult Hair Follicle showing location of the stem cells.

The stem cell populations are represented by their well- marked gene/ protein-expression or promoter-activity: Lgr5 (hair germ and bulge), CD34 (bulge), LRC (bulge), Lgr6 (lower isthmus), Lrig1/MTS24 (isthmus), Blimp1 (sebaceous gland) and K15* (K15 promoter, bulge area). HF: Hair follicle.[16]

The growth of hair occurs in a periodical manner and each follicle function as an independent unit. The stages of hair cycle are as follows:

Growth phase (Anagen)

Regression phase (Catagen)

Resting phase (Telogen)

Shedding phase (Exogen)

Lag phase (Kenogen)[14]

Anagen phase: The length of the anagen phase, which is the stage of follicle growth, varies depending on the body site and affects how long the hair will grow out in the end. It consists of seven stages (anagen I–VII). The anagen period for scalp hair is the longest, lasting between 2 and 8 years. According to reports, 90% to 93% of scalp follicles are typically in anagen, with the remaining follicles being in telogen.[17]

Catagen: The time when a follicle transitions from anagen to telogen. Two weeks pass during this phase. [11]

Telogen: Club hair is produced during the time between the end of the follicular regressive phase and the beginning of the subsequent anagen phase. 11 100–150 hairs are shed every day, and 5–10% of hair in each hair cycle is in the telogen phase. About two to three months pass during this phase.[18]

Exogen: An active procedure known as exogen causes the club hair to shed. The exogen hair shaft base is smaller, more elongated, and has a pitted margin than the telogen shaft base, which has a club-shaped smooth outline. [11]

The kenogen/lag phase, which occurs after hair shedding and lasts for an arbitrary amount of time, is a stage in the hair cycle where the follicle is empty. AGA is more common in both men and women. [11]

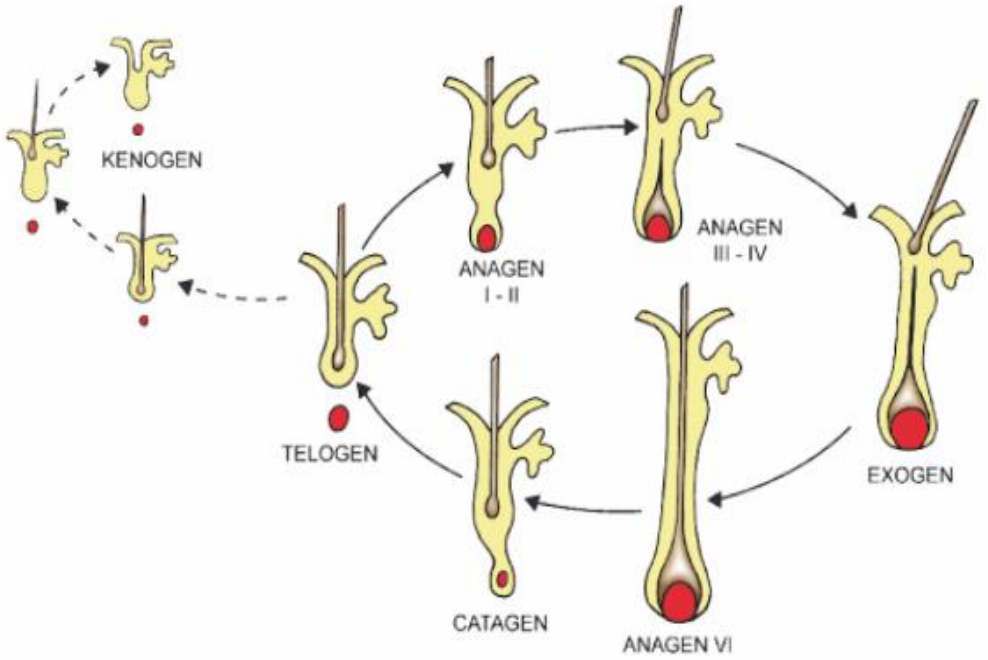


Figure 5: Demonstrates the different phases of hair cycle[19]

From deep to superficial, the anagen hair follicle is divided into 4 sections:

1. Hair follicle
2. The suprabulbar region, which stretches from the hair matrix to where the arrector pili muscle inserts
3. Isthmus: This is where the arrector pili muscle attaches and where the sebaceous gland duct enters.
4. The follicular orifice and the sebaceous gland are connected by the infundibulum.

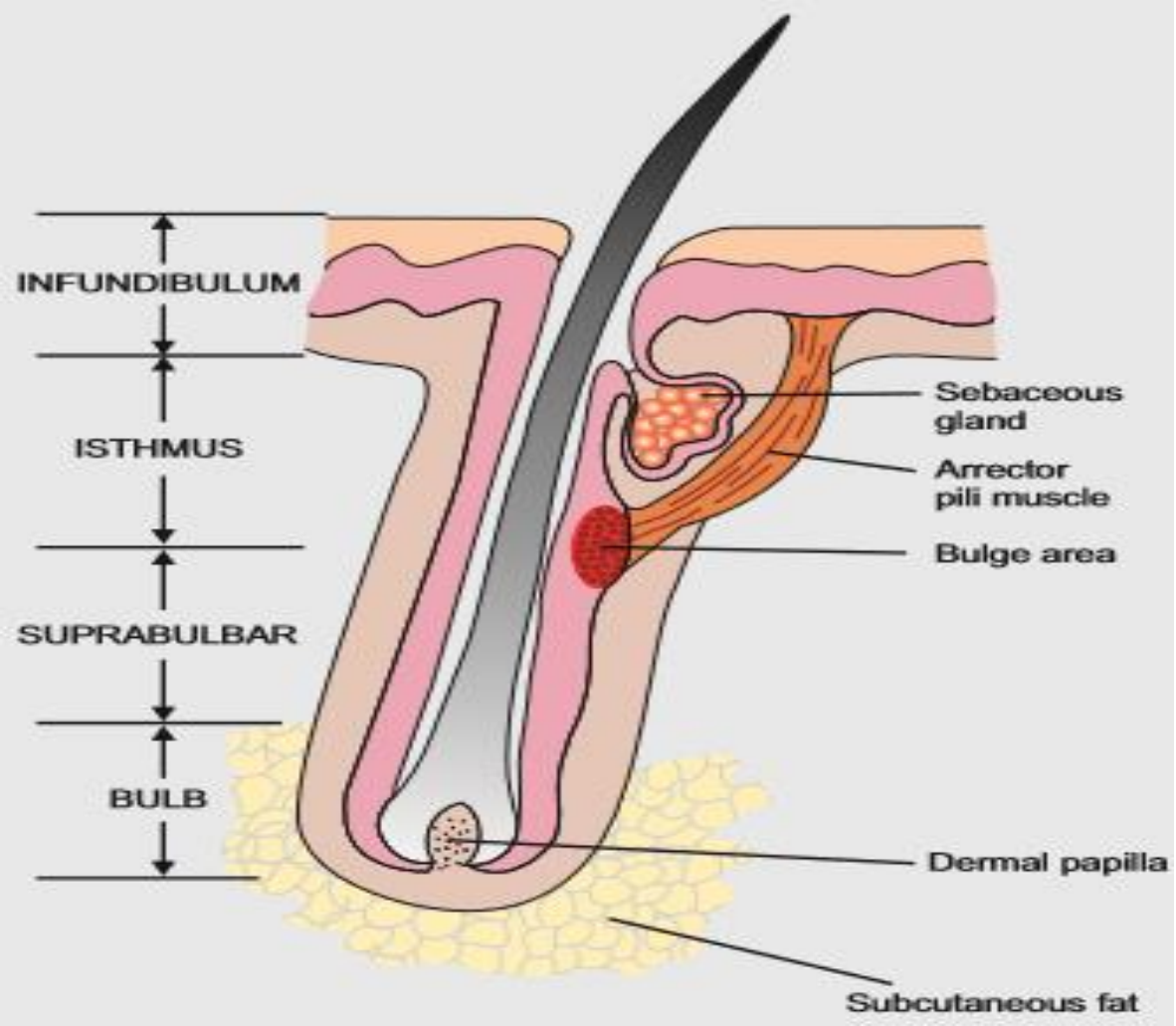


Figure 6: Principal subdivisions of a hair follicle, based on both morphologic and biological considerations.[11]

The complex and poorly understood signaling involved in the well-coordinated process of hair growth and HF cycling. The interaction between the mesenchymal and epithelial cell populations within the Hair Follicle unit is the fundamental driving force. A schematic diagram of various stem cell types and the specific differentiated skin structures to which they contribute can be seen in Figure 7. The dermal papilla houses the most significant mesenchymal cells (DP).[20]

The follicular epithelium's sequential cycling is regulated by the signals produced by these cells. Epithelial stem cells, which are found in the bulge region of the hair follicle, may be able to respond to signals coming from the dermal papilla. Progenitor cells are produced from the stem cells in the bulge area as a result of this activation, and these progenitor cells later differentiate into matrix cells with the capacity to produce the hair shaft and its sheath. Finally, these transiently amplifying cells expand downward into the deep dermis. However, the male and female sexes have very different hair phenotypes, which are controlled by the action of sex hormones, in both humans and especially in animals. [20]

Hair follicle cycling is influenced by a number of growth factor families, including the fibroblast growth factor, EGF, hepatocyte growth factor, IGF-I, and TGF-b families, among others. When stimulated with cytokines or growth factors, the latent cytoplasmic protein signal transducer and activator of transcription 3 (stat3) transmits signals to the nucleus, activating the transcription of downstream genes that contain the stat3 response element in their promoter region. Stat3 is essential for the cycling of hair follicles. [21,22]

Each stage of hair development and differentiation is regulated by a number of signaling molecules that have been identified through research on embryogenesis. Signaling of the Wingless type (Wnt) is essential for the beginning of hair follicle development. The ligand Wnt-protein binds to a member of the Frizzled family of cell-surface receptors and transmits a biological signal to the intracellular protein Dishevelled (Dsh). In order to function as a transcriptional co-activator of transcription factors that are a member of the TCF/LEF family, Dsh promotes the accumulation of b-catenin in the cytoplasm (by shielding it from degradation) and its eventual translocation into the nucleus.[23,24]

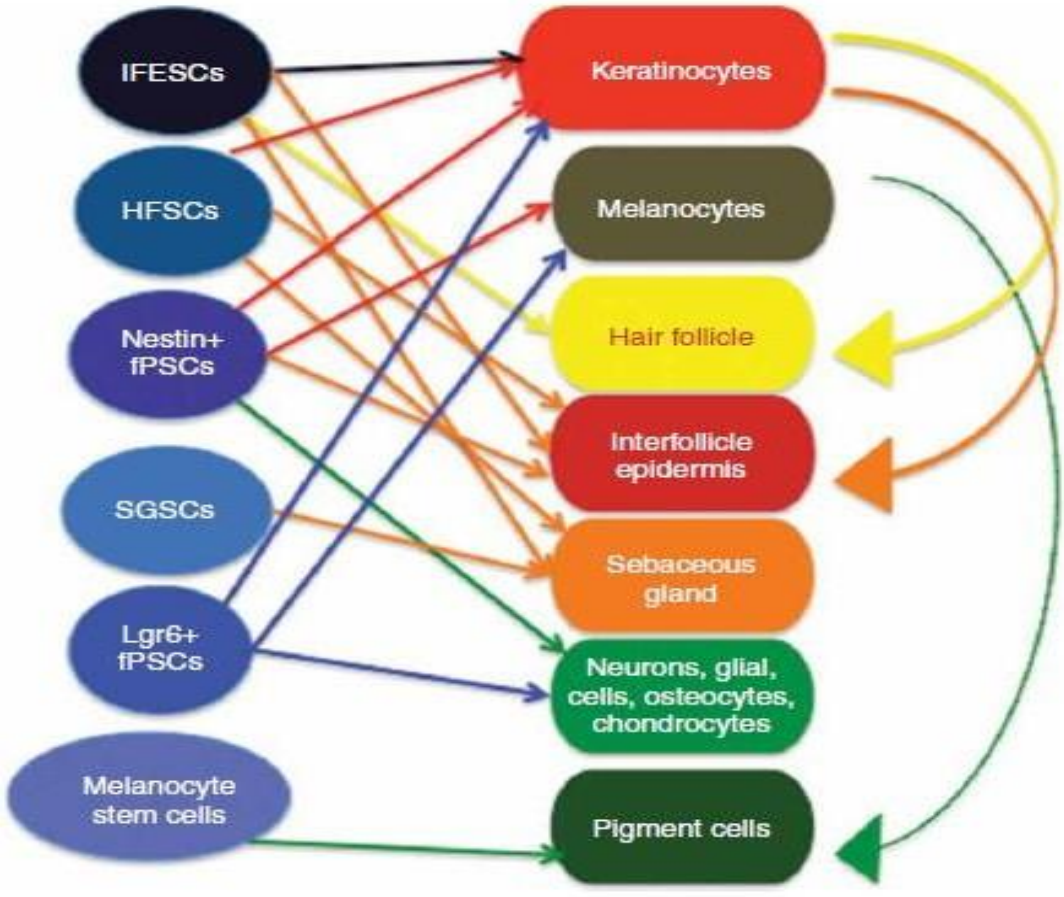


Figure 7. Interactions between stem cells, progenitor cells, and cells in and related to the skin.

IFESCs: Interfollicle epidermal stem cells; hair follicle SCs: Hair follicle stem cells; SGSCs: Sebaceous gland stem cells; fPSCs: follicle nestin + pluripotent stem cells; Lgr6 + fPSCs, could be identical to fPSCs.[24]

Sonic hedgehog‘ (Shh) signaling plays a role in both embryonic and adult hair follicle development. Shh binds to and inhibits the extracellular domain _Patched,‘ allowing the intracellular domain _Smoothened‘ to accumulate and inhibit the proteolytic cleavage of the Gli family of zinc-finger transcription factors.[25]Shh signaling (and in particular activation of Gli2[28] is obligatory for development of the epithelial hair germ, comprising epidermal placodes and associated dermal condensates. [26]Noggin, bone morphogenetic protein (BMP) and ectodysplasin signalling also play important roles at early stages of hair follicle placode

development. Dlx homeobox transcription factors regulate epidermal, neural and osteogenic cellular differentiation. It was found DLX3 played a central role as a transcriptional regulator of hair formation and regeneration. DLX3 co-localized with phosphorylated Smad1/5/8 complex, and regulated BMP signalling during hair morphogenesis in animal models.[27]

It was discovered that DLX3 was a crucial transcriptional regulator of hair growth and regeneration. In animal models, DLX3 co-localized with the phosphorylated Smad1/5/8 complex and controlled BMP signaling during hair morphogenesis. [28]

Reactive oxygen species (ROS), which are produced by mitochondria, were important mediators, according to an intriguing study by Hamanaka et al.[29] They produced mice lacking mitochondrial transcription factor A (TFAM), which is necessary for the transcription of mitochondrial genes encoding electron transport chain subunits, specifically in keratinocytes.

Hair follicle growth was hampered and death occurred two weeks after birth when TFAM in keratinocytes was removed. Keratinocytes lacking TFAM were unable to produce mitochondria-derived ROS, which interfered with the Notch and -catenin signals' ability to be transmitted. In vitro keratinocyte differentiation was inhibited by antioxidants, and exogenous H2O2 application partially reversed the decreased differentiation marker abundance in TFAM-deficient keratinocytes.[30]

The overlying epithelial matrix cells that differentiate to form the various hair follicle lineages, including cells that form the medulla, cortex, and cuticle of the hair shaft and IRS, are still connected to the dermal papilla. [31] Epithelial stem cells initiate in the bulge region of the hair follicle produce the matrix.[32] Gata3 and Cutl, which control IRS differentiation and BMP signaling[33], as well as transcription factors like Gdsma340, Msx241, Foxn142, and Hoxc1343 that are necessary for full hair follicle development and ideal hair shaft structure, are among the key pathways and transcription factors that initiate and promote differentiation of the matrix

cells have been identified.However, the hair follicle stands out as the best model system for studying adult stem cells. Cells with stem cell properties have recently been described in many integumental appendages, including feathers and teeth.[34,35] One of the most active areas of biological and biomedical research right now is the identification, characterization, and transplantation of adult stem cells. It is believed that TA cells and stem cells cannot be substituted for one another, and that TA cell differentiation is irreversible once it has begun.

FUNCTIONS OF HAIR

Hair aids in thermoregulation, which enables the species to endure extreme climatic variations by retaining heat in cold climates and losing it in hot climates. It shields the body from ultraviolet ray damage as well as trauma. Hair color can sometimes be used as a sexual attractant in addition to aiding in predator camouflage. It has a plentiful supply of nerves, which supports its tactile and communicative abilities. Each hair shaft's lower end is joined to the skin's surface by the arrector pili muscle. These tiny muscles constrict in response to emotional stimuli, making the hair stand up.

Specialized hair on humans, such as eyelashes, hair in the nostrils, and hair on the outside of the ears, provide some environmental protection. Sweat cannot enter the eyes thanks to the brows. The scalp hair helps to keep the brain's temperature stable. Hair is useful in forensic medicine because it can excrete toxic substances like arsenic. An individual experiences significant psychological distress and anxiety as a result of hair loss and excessive hair

growth. [367]

ALOPECIA

Alopecia is defined as hair thinning or hair loss.

Alopecia disorders can be divided into cicatricial (scarring) and non-cicatricial (nonscarring) forms, as well as into diffuse, patterned, and focal hair loss.

Scarring alopecia is characterized by the loss of the ability to produce new hair as a result of the permanent destruction of the hair follicular stem cell structure. The hair follicle is destroyed in non-scarring alopecia, and subsequent hair growth occurs after periods of hair shedding.47

Non scarring alopecia's include:

Alopecia areata (AA)

Alopecia areata incognita (AAI)

Androgenetic alopecia (AGA)

Female pattern hair loss (FPHL)

Telogen effluvium (TE)

Anagen Effluvium

Tinea capitis (TC)

Trichotillomania (TTM)

Traction alopecia

Temporal triangular alopecia (TTA)

Syphilitic alopecia.

Loose anagen syndrome.

Short anagen syndrome.[7]

Androgenetic alopecia (AGA)

Synonyms include female-pattern baldness and male-pattern baldness (FPHL), Hereditary thinning or balding, pattern baldness, or premature baldness.[37]

Definition: It is an androgen-dependent, hereditary condition that manifests as a distinctive pattern of scalp terminal hair turning into miniature vellus hair.

AGA is characterized by varying degrees of hair thinning or loss, mostly in the scalp's vertex and frontal regions. As individually follicle is in a diverse phase of the hair cycle, the residual hairs in men with AGA tend to be of different diameters and lengths. The presence of uneven hair extent and quality is a classic feature of this condition.[18]

- **Epidemiology,**

White patients are commonly affected followed by Asians and African Americans, Native Americans and Eskimos. Around 50% are affected by the age of 50 years old whereas, upto 80% by 70 years old. The disorder is very common in females with an increase in incidence after menopause. [38]

- **Brief description of androgenetic Alopecia in males vs females**

Pattern or androgenetic alopecia is a predetermined disorder occurs due to the excessive response to androgens. It affects up to 50% of males and females and is characterized by progressive loss of terminal hair of the scalp any time after puberty, in a characteristic distribution in both males and females. In males, hair loss is most prominent in the vertex and frontotemporal regions, whereas in females the frontal hairline is typically spared with diffuse apical hair loss noted as a wider anterior part of the hair. [39] [40] [41]

Pattern alopecia is a polygenic disorder with variable penetrance. It involves both the maternal and paternal genes. There is a familial predisposition to androgenetic alopecia with sons at 5-

6 time higher relative risk if their fathers had balding. It required androgen to occur and develops only after puberty. Males castrated before puberty and those with androgen insensitivity syndrome do not have pattern baldness. Hormone metabolism and androgen receptor play a major role in the pattern alopecia. [42]

Activation of the androgen receptor shortens the anagen or growth phase in the cycle of normal hair growth. In androgenetic alopecia, excessive activation can leads to follicular miniaturization through a progressively shorter anagen phase. It results in thinner and shorter hair follicles.[43] Androgenetic alopecia patients have increased production of dihydrotestosterone and increased levels of 5 alpha-reductase and androgen receptors in balding scalp.

1. **Female pattern hair loss.**

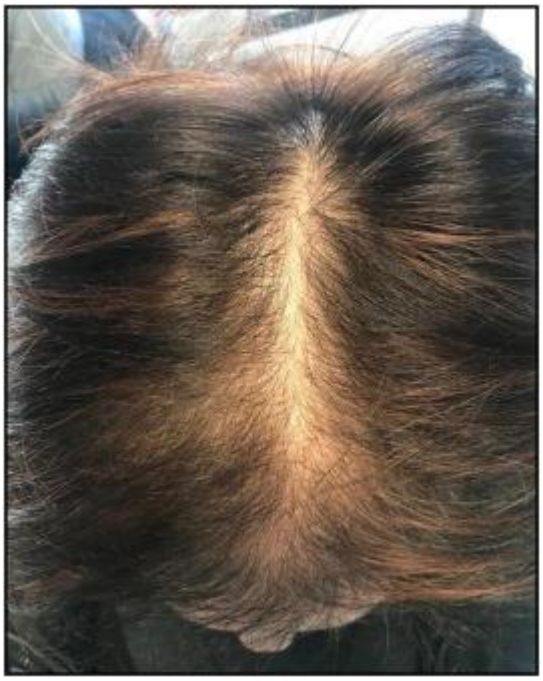
- Definition

The most common hair loss disorder in women is the female pattern hair loss. Initial symptoms develops during the teenage years and can lead to progressive hair loss with characteristic pattern distribution. [5]It is characterized as a nonscarring diffuse alopecia that can evolves from the progressive miniaturization of hair follicles and reduction in the quantity of hairs especially in the central, frontal and parietal scalp areas. [44]

Table 1: Silent features of female hair loss pattern

Distribution	Central portion of scalp and preserved frontal hairline
Onset	Gradual
Appearance	Hair thinning with wide midline part
Hair shedding	Minimal
Hair pull test	Usually negative
Other history	Positive family history
Scalp biopsy	T:V ≤4

Figure 8: female pattern hair loss [44]



- Epidemiology – global, Indian

Among healthy women, 6% - 38% are affected with some degree of frontal or frontal-parietal hair loss. The age of onset for FPHL is identified during the reproductive years and which is later than in men. Around 12% of women first develop clinically detectable FPHL by the age of 29 years whereas, 25% by 49 years, 41% by 69 years and more than 50% have some element of FPHL by 79 years.[2]

Risk factors and pathophysiology

In contrast to FPHL, where the role of androgens in the pathogenesis of male hair loss has been less well understood. It is still unclear how FPHL pathophysiology works. There is proof that environmental, hormonal, and possibly genetic factors play a role. The adjacent units in the scalp's mosaic pattern do not have synchronized hair follicle biological cycles. It is didactically divided into three phases: telogen, catagen, and anagen (growth phase) (resting

phase). The original hair is shed (exogenous phase) at the termination of the telogen phase and is replaced by a fresh hair at the early growth stage.[7]

The anagen phase typically lasts between two and eight years, the catagen phase between two and three weeks, and the telogen phase about three months. As a result, an adult's normal scalp has 80–90% of its hair in the anagen phase, 10–20% in the telogen phase, and 1-2% in the catagen phase. Trichogram or an anatomopathological examination can show this. The anagen phase's duration is shortened and the dermal papilla's size is reduced in FPHL (thinning of the hair). Miniaturized hairs gradually take the place of thick pigmented hairs. Additionally, there is a lag time between the conclusion of the telogen phase and the start of the brand-new anagen phase. [33]

The kenogen phase refers to this dormant stage, when the hair follicle is empty. 40 In the impacted areas, the capillary density gradually decreases. These changes take place in MPA as well, despite the fact that FPHL and MPA have clinical appearances with different forms. The hair follicle is a sophisticated structure that functions continuously. The onset of the catagen phase can be determined by an imbalance between various growth factors and cytokines that maintain the anagen phase and encourage apoptosis (Chart 1).[45] The follicular regression seen in the catagen phase is caused by the follicular keratinocytes' widespread apoptosis. An important development in the evolution of FPHL is the premature termination of the anagen phase. [33]

Apoptosis can be brought on in one of two ways: either through the extrinsic pathway, which is activated by specific binders to a subset of membrane necrosis factor receptors, or through the intrinsic pathway, which can be brought on by factors like a reduction in growth factors or a loss of keratinocyte adhesion, among other things. [46] Apoptosis begins in the melanogenic

region of the hair follicle (late anagen), spreads to the array (premature catagen), and finally affects the internal and external root sheath. [47]

Table 2: Anagen promoting factors and factors promoting follice apoptosis

Anagen promoting factors	Factors promoting follice apoptosis
Bfgf	FGF5
FGF7	IL-1a
HGF	PGD2
IGF	TGF-B1
PGE2	TNF-a
VEGF	
Wnt	

Although the miniature hair closely resembles vellus hair, it is not a real vellus hair. Contrary to vellus hair, which has no or very little piloerector muscle, the hair is thin but has a developed piloerector muscle. The loss of papilla cells due to apoptosis is one potential mechanism of follicle miniaturization.[48] The papilla is the lone part of the follicle that forever expresses greater levels of the antiapoptotic protein bcl-2, which in theory converses resistance against proapoptotic stimuli. Although apoptosis can be induced in papilla fi broblasts in experimental settings. [49]

An important aspect of the pathophysiology of FPHL that needs to be investigated is the malfunction of antiapoptotic mechanisms. Despite the significance of hormonal factors in the onset of baldness, the mechanisms by which they cause the anagen phase to shorten and the follicles to shrink have not yet been fully understood. HORMONE RESOURCES The relationship between androgens and the onset of male pattern baldness was first noted by Hippocrates in 400 BC and was later confirmed by Hamilton in 1942. It was discovered that

men who had been castrated before puberty and eunuchs did not go bald. However, testosterone (T) administration caused alopecia in people with a family history of baldness.[7]

All of these findings point to a role for T or one of its metabolites in the onset of MPA in people with a genetic predisposition. Androgens connect to particular intracellular receptors by crossing the cytoplasmic membrane. The transcription of genes that are primarily in charge of this complex hormone receptor's tissue actions is encouraged. Dihydrotestosterone (DHT) was the main androgen involved in the development of MPA, according to the observation that men with a genetic deficiency of type-2 5'-reductase (5R) did not become bald. An enzyme called 5R turns T into the DHT metabolite.[50]

There are two types of 5αR: type 1 and type 2. Type-1 5αR is present in sebaceous glands, and type-2 5αR is present in the genitourinary tract and in the hair follicles.The miniaturization of the hair follicle caused by androgens occurs primarily due to the action of DHT, which has fi ve times greater affinity for the androgen receptor (AR) than testosterone.The androgen linked to the AR leads to the transcription of the genes responsible for its biological action on the target cells. In addition, DHT may interfere with the follicular cycle by interacting with the Wnt signaling pathway. The Wnt pathway induces dermal papilla cells to maintain the anagen phase.[35]

The addition of DHT, which was mediated by Wnt3a, inhibited the growth of keratinocytes in cultures made up of cells from the bald patient's dermal papilla. The interaction of these pathways may be a key area for baldness treatment. Infusing a complex with Wnt activity improved the density and thickness of hair in men with MPA, according to a recent phase I clinical trial. Androgens were assumed to play a similar role in female baldness despite the fact that male and female baldness are thought to be the same condition despite the fact that the evidence supporting their involvement in baldness has come from studies in men.[51]

However, even at higher doses, finasteride and dutasteride produced inconsistent results in the treatment of FPHL.[52,53] The enzyme aromatase has an antiandrogenic effect by converting androstenedione to estrone and testosterone to estradiol. The aromatase levels of the follicles in the frontal region of women with FPHL were half as high as those in the occipital region in a 1997 study by Sawaya including 12 men and 12 women with baldness. These levels were six times higher than those found in the frontal follicles of males.[54] These findings imply that aromatase may prevent hair loss by converting androgens to estrogens.[55]

MICROINFLAMATION : A mild to moderate lymphohistiocytic infiltrate in the peri-infundibular region may coexist with baldness and the miniaturization process. To distinguish it from the inflammation that takes place in scarring alopecia, the term "microinflamation" has been used. [56] This process occurs on a sporadic basis. Whiting examined the presence of immune globulin in 106 men with MPA and 22 controls in 1993. (13 men and 9 women). In 30% of cases and controls, a mild immune inflammatory ltrate was discovered. However, compared to only 9.1% of controls, 36% of cases had a moderate immune infiltrate. [57,58]Since the inflamatory process occurs in the upper part of the follicle, it is possible that this is where the causal factor may have an effect. External factors, including ultraviolet rays, environmental pollutants, and members of the skin's microbiota and follicle (such as Propionibacterium sp., Staphylococcus sp., and Malassezia sp.), among others, may have an impact on the induction of the microinflammation process. As well as its potential relationship to the miniaturization process and the hormonal components involved in FPHL, the true significance of this inflammatory process for the development of FPHL is still unknown.[59]

GENETICS: FPHL patients frequently report family members who have the condition (40–54%), particularly in cases with an early clinical presentation (40 years). In a comparative

study evaluating male twin baldness, monozygotic twins demonstrated twice the level of agreement as dizygotic twins. Although the causes of family segregation are not fully understood, the high prevalence of FPHL and the fact that the disease manifests in varying degrees of severity and develops at various ages point to a polygenic pattern with partial penetrance. Additionally, family influences on baldness development can differ between men and women and depending on how it manifests (classical or diffuse).[60]

The SRD5A1 and SRD5A2 genes, which are in charge of producing type 1 and type 2 5'-reductase enzymes, were not associated with the onset of baldness, despite the fact that DHT is crucial for the development of MPA. [61]Studies involving the AR gene in men provide the clearest evidence of genetic involvement in the onset of baldness. In the first exon, a single nucleotide polymorphism called STUL was linked to baldness. Although 98% of men with premature baldness and 92% of men with late baldness had this change, 77% of men without baldness also had it. [62]

These factors imply that additional adjustments, such as hormonal adjustments, medication, and environmental stimuli, are required for the development of MPA, supporting the idea that this condition has a polygenic origin and is under the control of epigenetic factors. Individuals differ in the number of CAG repeats found in the first exon of the AR gene. The number of CAG repeats in its amino-terminal region and the activation of AR were found to be inversely correlated. This suggests that people with shorter repetitions have a higher risk of going bald. Although the AR gene is found on chromosome X, this does not necessarily explain why the phenotypes of fathers and sons are the same. Consequently, there is either a polygenic inheritance or a direct maternal transmission (autosomal).[33]

Another theory is that other environmental factors, in addition to this gene, play a role in the onset of baldness. Compared to MPA, there is less evidence that the AR gene is involved in

the pathophysiology of FPHL. However, just like in men, the number of CAG repeats in the first exon of the AR gene was inversely correlated with FPHL. This observation allowed the development of genetic testing, in which the detection of a small number of CAG repeats is associated with an increased risk of developing FPHL, whereas a larger number of repeats is associated with a lower risk. The presence of the StuI restriction fragment has not been linked to FPHL.[33,60]Nonfunctioning single nucleotide polymorphism (rs4646) in the aromatase gene was identified in women with FPHL, particularly in younger women (CYP19A1). [60]Six susceptibility loci for MPA were found by meta-analysis of seven genome-wide association studies: 1p36.22, 2q37.3, 7p21.1, 7q11.22, 17q21.31, and 18q21.1. 85 Redler's 2013 study, which included 469 controls and 405 balding women, found no correlation between these loci and FPHL. 86 Four new loci linked to MPA (2q35, 3q25.1, 5q33.3, and 12p12.1) in a later study were not linked to FPHL. 87,88 All of the information points to distinct etiopathogenic factors for MPA and FPHL.

External factors may also play a role in the development of FPHL in addition to genetics. A 2012 US study of 98 female identical twins raised several environmental factors that might be connected to FPHL. These included low levels of physical activity, psychological stress, high blood pressure, diabetes, and smoking. However, it is still unclear what exactly these factors contribute to the causal model of FPHL.[63]

FPHL can also happen when androgens aren't present. A genetic predisposition may be present in FPHL patients who do not have elevated androgen levels. It may enable the target follicular cells to be affected by average levels of circulating androgen. By attaching to specific intracellular androgen receptors, they become specifically sensitive. In other instances, the development of FPHL may be influenced by an androgen-independent mechanism. An increased number of gene loci (> 60) are associated with male AGA, according to two recent studies.[63]

Environmental factors play a role in the multifactorial and polygenic nature of hair loss in women. FPHL entails the gradual miniaturization of hair follicles as well as the transformation of terminal follicles into vellus-like follicles. Because the anagen phase is reduced in these vellus-like follicles, the hair cycle is shorter. It causes the growth of thin, short hair shafts. Compared to men, women's miniaturization differs from men's in both uniformity and intensity. As a result, there are no totally bald areas. Additionally, a mild-moderate lympho-histiocytic inflammatory infiltrate in the peri-infundibular region may coexist with the miniaturization process.[3]

Role of androgens in female pattern hair loss

The androgen receptor, 5α-reductase I, II and aromatase enzymes are located in the outer root sheath and dermal papillae of hair follicles of men and women with AGA. The androgen receptor content in female frontal hair follicles is 40% lower as compared to the male follicles. Frontal hair follicles in women have 3 and 3.5 times less 5α-reductase I and II levels than that in males. While, aromatase content in the frontal hair is 6 times higher in women. [64]

Conversion of testosterone to dihydrotestosterone requires free testosterone which is not bound to sex-hormone-binding globulin. Women with female pattern hair loss are more common to show signs of virilization and few reveal hyperandrogenemia. In a study performed in women with FPHL, 38.5% was identified to have clinical or biochemical evidence of hyperandrogenism. [65] Out of women with hair loss, 67% had hair loss alone whereas, 84% had hirsute with some degree of biochemical androgen access. [66]

Role of estrogens in female pattern hair loss

Enzymes aromatase and 17β-hydroxysteroid dehydrogenase expressed within dermal papilla cells and keratinocytes of the outer root sheath of hair follicle metabolises estrogens. Aromatase is the key enzyme responsible for the conversion of androgens to estrogens in tissue

sites. [67] Estrogens can modify androgen metabolism within dermal papilla cells by decreasing the DHT production from testosterone either as a result of direct inhibition of 5α-reductase or through estrogen-induced conversion of testosterone into weaker steroids. [12]

There is a prolongation of anagen phase during the pregnancy secondary to high systemic estrogen while the post-partum fall in estrogen levels can partially account for the simultaneous conversion of hair follicles into the telogen phase. It leads to telogen gravidarum. [12]Aromatase inhibitor therapies leading to lower estrogen levels have been identified to induce hair loss and topical estrogen applications have been mostly used as hair growth stimulants in female pattern hair loss.

- Diagnosis

Table 3: Classification of methods to evaluate hair loss

Category	Method
Non invasive	Questionnaire, daily and 60s hair counts. Standardized and modified wash test, global photographs, dermoscopy, phototrichogram. Trichoscan, polarizing and surface electron microscopy.
Semi invasive	Trichogram and unit area trichogram
Invasive	Scalp biopsy

Evaluation techniques for hair loss [11]

Daily hair counts: Patients are informed to collect the hairs shed in the shower, sink or on the brush in one day. Count them and place it in plastic bags for seven consecutive days. If the hair loss is more than 100 per day. Hair microscopy must be done in such patients.

60-s hair count: Patients collects the hair before shampooing by combing the hair starting with the back top of the scalp and moving forward to the front of the scalp. It is repeated before 3 consecutive shampooing.

Standardized and modified wash test: Patient refrains from shampooing for five days and then perform shampooing. Collects the hair shed in the basin and counted. It is divided into longer (5cm), intermediate length (>3-<5cm) and shorter (3cm).

Hair pull test: Stay away from shampooing for 2-5 days before the test. Around 50-60 hairs are collected between the thumb, index and middle fingers from the base of the hairs near the scalp and firmly pull away from the scalp. It is repeated in right+ left, parietal and frontal+occipital areas of the scalp. It is considered as positive if <10% of the grasped hairs or more than equal to 6 hairs are tugged away from the scalp.

Hair pluck test: Stay away from shampooing for five days before the test. Around 60-80 hairs are plucked at two specific scalp locations for AGA by using a rubber armed forceps. The hairs are firmly grasped at 0.5cm above the scalp and removed with a single sudden forceful pull perpendicular to the scalp along the direction of hair growth. The roots of the hair are examined under the microscope and is expressed in anagen and telogen ratio.

- Clinical features / presentation (in detail),

There are several hair loss scales used to categorize female pattern hair loss and each has its own advantages and disadvantages. [7 68] The three main clinical manifestations identified for the female pattern hair loss are the following

- Diffuse thinning of the upper biparietal and vertex regions and preservation of the anterior hair implantation line.

- Thinning of the upper bitemporal region and vertex with frontal accentuation that configures as a triangular or Christmas tree form with hair loss in a triangular shape in the frontal-vertical area,[69]
- Deep recession of the frontal-temporal hairline and true vertex balding. It is commonly identified in men and occasionally in women.[60]

Figure 9: Ludwig scale representation [3]

Grade	*Description*	*Clinical aspect*
Grade I:	Perceptible thinning of the hair on the crown, limited in the front by a line situated 1–3 cm behind the frontal hair line.	
Grade II:	Pronounced rarefaction of the hair on the crown within the area seen in Grade I.	
Grade III	Full baldness (total denudation) within the area seen in Grades I and II.	

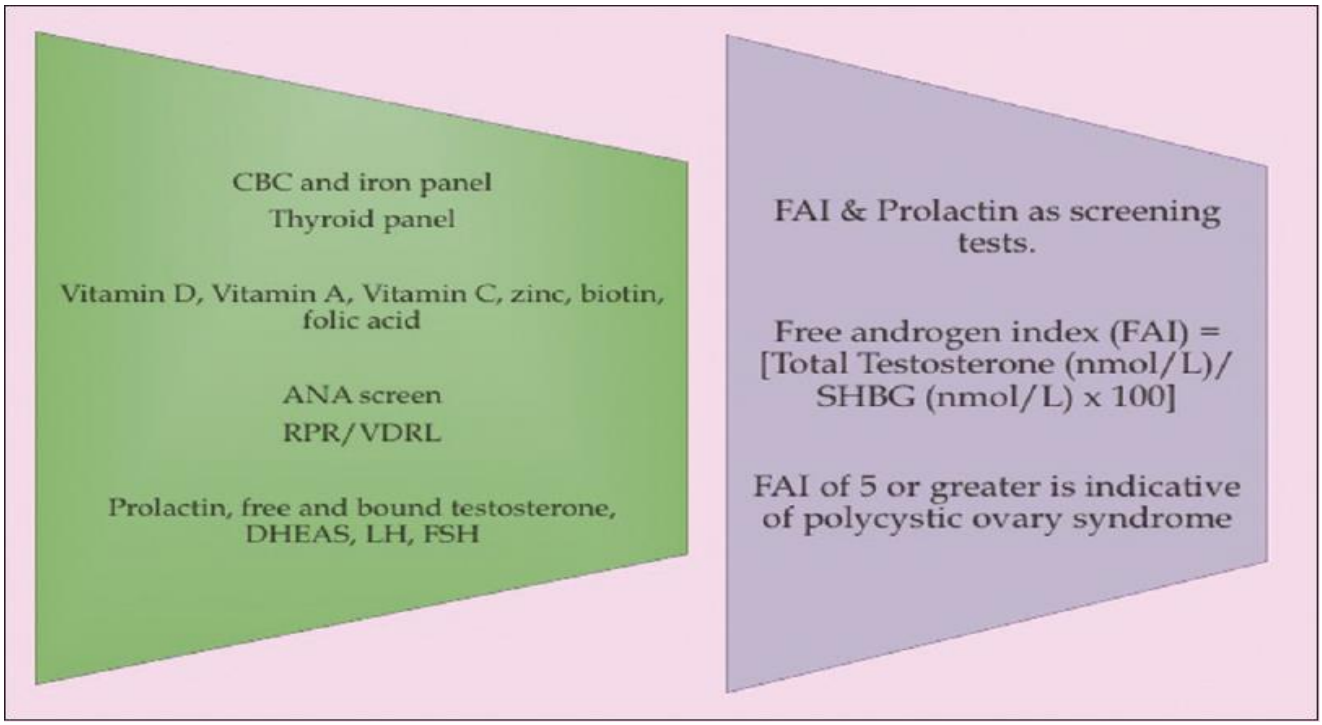
Figure 10: Sinclair Scale Sinclair's classification. [70]

Figure 11: Olseńs classification.[44]

- Biological parameters in female pattern baldness

The European Consensus recommends the free androgen index and prolactin levels as screening tests. The free androgen index is the association between the total testosterone and sex hormone-binding globulin [total testosterone (nmol/L)/SHBG (nmol/L) × 100]. Free androgen index of 5 or more is indicative of polycystic ovary syndrome. The use of hormonal contraceptives causes alterations in the SHBG levels. Hence, laboratory tests should only be performed after a hormonal contraceptive pause of at least two months. Thyroid function should be determined as the thyroid dysfunctions can contribute to the effluvium associated with female pattern hair loss.

Figure 12: Biochemical evaluation to be done in a patient of FPHL



- Trichoscopic pattern in female pattern baldness

Dermoscopy can help in the diagnosis of female pattern of hair loss, mostly in the early stages of the disease. The diversity in the thickness of the hairs with an increased number of miniaturized hairs, commonly in the frontoparietal region is the main dermoscopic finding. **Hair diameter density >20%** is considered as the diagnostic for female pattern hair loss. Short vellus hair (<0.03 mm) is a sign of severe miniaturization and their identification on the frontal scalp is very useful for identifying the female pattern hair loss. A decline in the number of hairs per follicular unit is also an important element.

The Brown peripilar sign: light brown area, slightly atrophic around the follicle, commonly seen in the early stages of female hair loss pattern and correlates with the inflammatory infiltrate. It can be brown sign which is identified in early grades of female pattern hair loss. **White peripilar sign** in later grades, reflects to perifollicular fibrosis in the late stage of AGA.

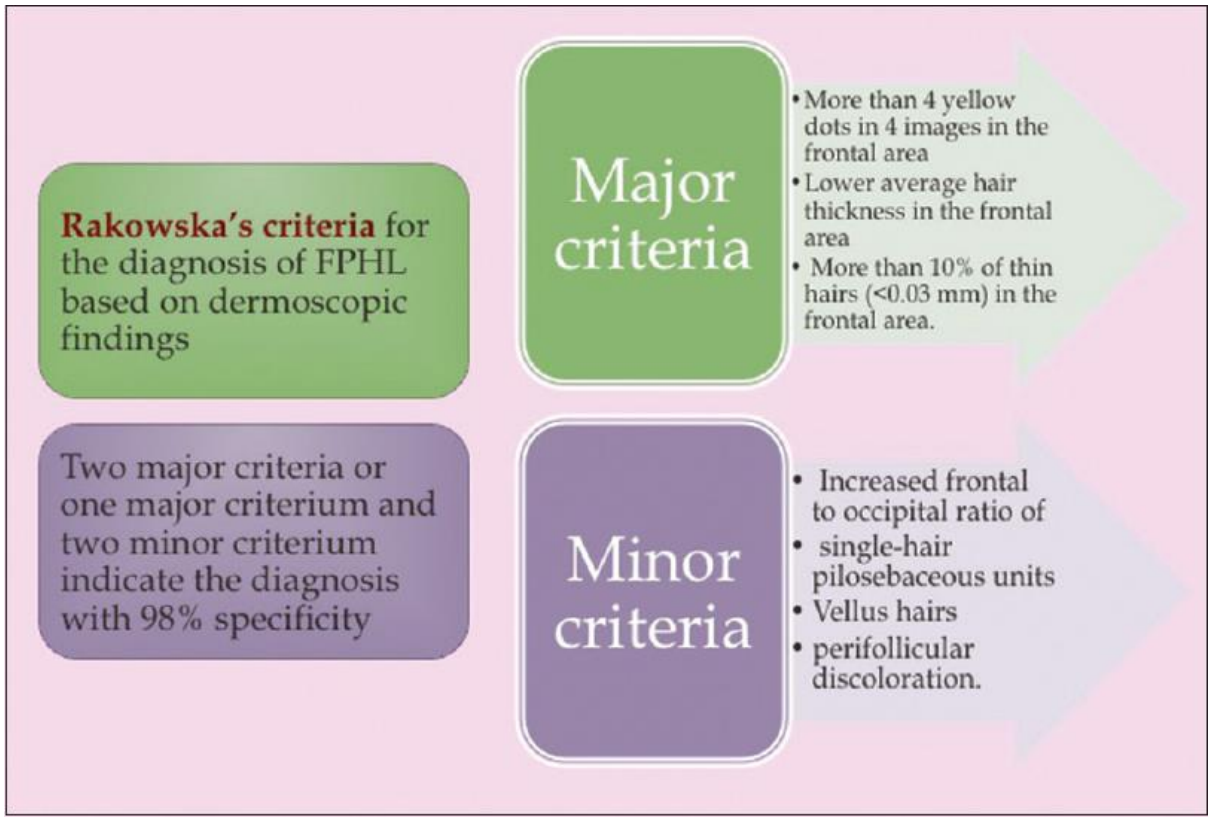
White dots : Reflect empty hair follicles

Yellow dots It is mostly as a result of sebum and keratin accumulation in dilated follicles.

Focal Atrichia resembles size of pencil eraser hair loss areas.

Scalp honeycomb pigmentation:With the enhanced thinning of the hairs, there is an increased penetration of ultraviolet radiation into the scalp and changes typical of photoaging like the honeycomb pigmented network. These signs are evaluated together, which helps in the early diagnosis of female pattern of hair loss. In 2009, Rakowska standardized the criteria for the diagnosis of female pattern hair loss as per the dermoscopic finding.[1]

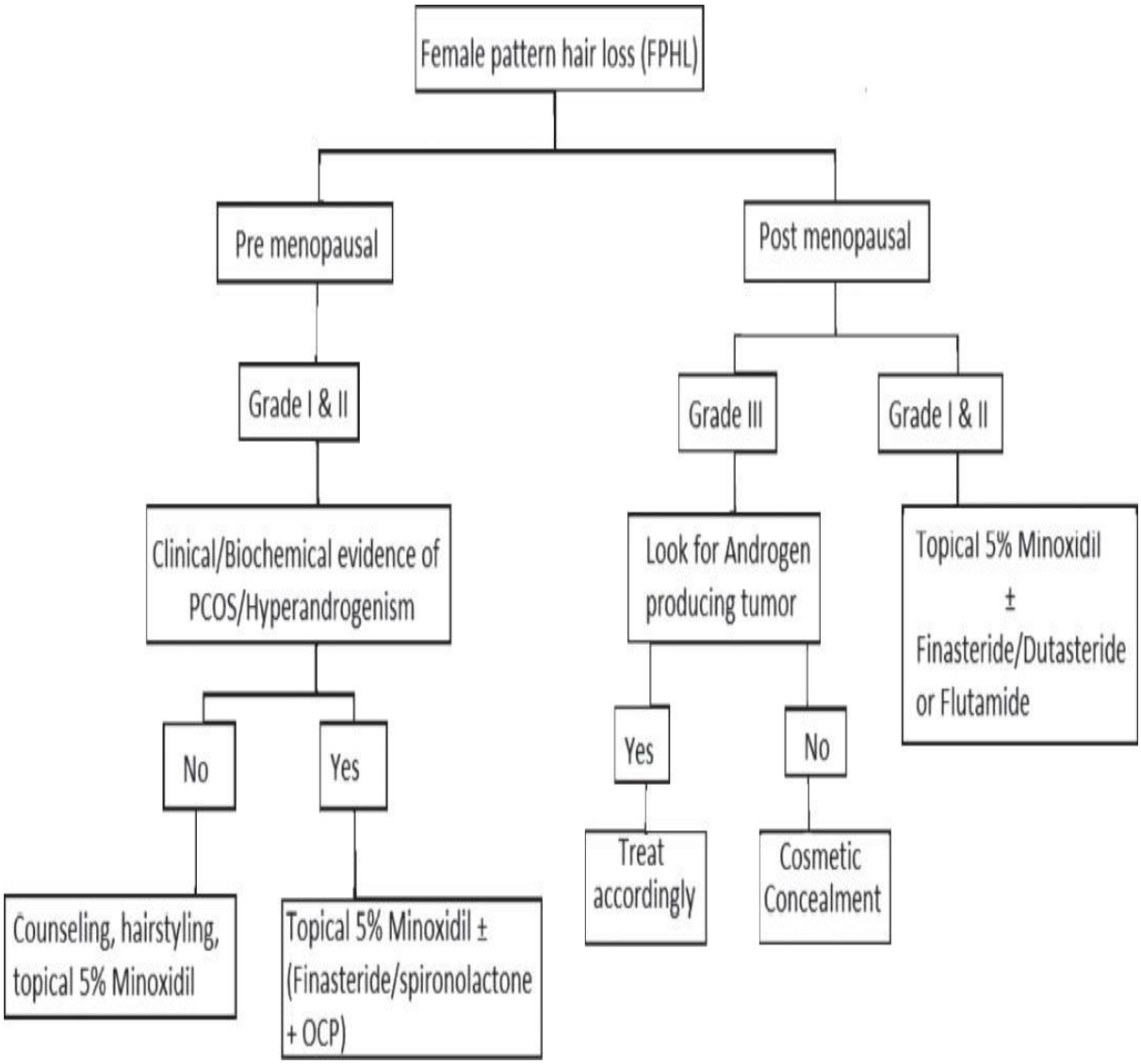
Figure 13: Rakowska's criteria for the diagnosis of FPHL on basis of dermoscopic findings.[1]



- Hormonal factors in detail,

Management in short

Figure 14: Algorithm of the management of female pattern hair loss



2. **Association of clinical features, biological parameters and trichoscopic findings in patients with female pattern baldness**

In Tandon S, et al.[44] study the mean values of dehydroepiandrosterone sulfate, prolactin, androstenedione and free triiodothyronine showed statistically significant difference between the cases and control. Also, the increase in the percentage of telogen hair was the most common histopathological finding. The peripilar signs, white dots, scalp pigmentation and focal atrichia were the characteristic trichoscopic features identified in X Z, et al., [50] study. There was a

positive correlation identified between the WPPS, scalp pigmentation focal atrichia, stage and duration of hair loss.

In an observational study[71] a positive correlation identified between the yellow dots, white dots, advanced Ludwig and Sinclair staging of hair loss. An association was also identified between the peripilar sign, honeycomb pigmentation, white dots and darker skin types. Siah T, et al., [72] conducted a study in which the normal Vitamin D level was identified in 38% of patients. While, the ferritin level above 30 μg/L and normal zinc level at the first consultation were identified in 71% and 85% of the participants.

Tandon S, et al.[44] conducted a study in 30 patients. The aim of the study was to determine the women with patterned hair loss and correlate their clinical findings with the histopathology and biochemical parameters. The mean values of dehydroepiandrosterone sulfate, prolactin, androstenedione and free triiodothyronine showed statistically significant difference between the cases and control. The increase in the percentage of telogen hair was the most common histopathological finding. This study concluded that all female patients with FPHL should be evaluated for underlying hormonal imbalances.

Hu, et al., [50] conducted a study in 60 patients. The purpose of the study was to determine the clinico-laboratory and trichoscopic features of female patients in the severity of hair loss in FPHL. The study results revealed 34.4±10.6 years as the mean age of the patients whereas, 4.49±3.76 years as the mean duration of hair loss. Family history of pattern hair loss was identified in 45% of patients. The peripilar signs, white dots, scalp pigmentation and focal atrichia were the characteristic trichoscopic features. There was a positive correlation identified between the WPPS, scalp pigmentation focal atrichia, stage and duration of hair loss. This study concluded that the family history of PHL causes an earlier onset of hair loss

SS, et al.[44] performed an observational study in 129 patients. The purpose of the study was to determine the association between trichoscopic signs and disease severity in dark-skinned females. Androgenetic alopecia was identified in 79 patients whereas 50 patients were diagnosed with telogen effluvium, alopecia areata, fibrosing alopecia in pattern distribution, folliculitis decalvans, discoid lupus erythematosus, lichen planopilaris, frontal fibrosing alopecia and end-stage cicatricial alopecia. The hair shaft diameter diversity was identified in all the patients. The predominance of one hair per follicle, peripilar brown halo, peripilar white halo, honeycomb-like scalp pigmentation, yellow dots, white dots and hidden hair were identified with 97.4%, 32.9%, 10.1%, 17.7%, 15.2%, 20.3% and 7.6% respectively. There was a positive correlation identified between the yellow dots, white dots, advanced Ludwig and Sinclair staging of hair loss. An association was identified between the peripilar sign, honeycomb pigmentation, white dots and darker skin types. This study concluded that the peripilar sign, honeycomb pigmentation, and white dots are characteristic signs of female androgenetic alopecia

Saqib NU, et al.[6] conducted a hospital-based observational, cross-sectional study. The purpose of the study was to determine the trichoscopic features of different types of alopecia in women. Female pattern of hair loss was identified with increased hair diameter diversity > 20%, single-hair follicular unit, vellus hair, peripilar sign, and focal atrichia. Loss of follicular ostia, erythema, white macules, blue-gray dots, white dots, tufted hair, and keratotic follicular plugging were noticed in cicatricial alopecias. This study concluded that the validity of trichoscopy in doubtful cases is evaluated using the validity parameters

R N, et al., [73] conducted a cross-sectional study in 460 participants. The aim of the study was to identify whether USB dermoscope can ascertain the hair-related changes in early FPHL. There was an association identified between dermoscopic hair changes and the frontal scalp

zone of cases. This study concluded that the microscopic changes recorded with the help of a simple USB dermoscope are helpful in establishing a diagnosis of FPHL

SA B, et al., [74] conducted a study in 114 patients. The purpose of the study was to identify whether trichoscopy can be used as a bedside tool to diagnose Early FPHL in women presenting without any visible thinning of hair. Anisotrichosis on trichoscopy was identified in 75% of patients. It was higher in FPHL Cases as compared to controls. Around 93% of Grade 2 FPHL controls were identified with the similar finding. The sensitivity and specificity of trichoscopy in diagnosis of early FPHL was 75% and 61.54% respectively. This study concluded that the negative result can be an indicative of absence of disease.

LH S, et al., [75] performed a study in 26,226 subjects. The aim of the study was to determine the factors associated with FPHL and to estimate its prevalence in women. The study results revealed 11.8% as the prevalence of FPHL for all ages. There was an association identified between FPHL, high fasting glucose, fewer childbirths, breast-feeding, oral contraceptive use and ultraviolet exposure of more than 16 hours per week. The study concluded the risk factors associated with FPHL.

EA O, et al., [76] performed a study in 381 women. The aim of the study was to identify if iron deficiency is more common in women with FPHL or chronic telogen effluvium than in control subjects without hair loss. The study results revealed that when ferritin </= 15 μg/L was used as definition, the iron deficiency was identified in 12.4%, 12.1, and 29.8% of premenopausal women with FPHL, CTE and control subjects respectively, Whereas, in 1.7%, 10.5% and 6.9% of postmenopausal women with FPHL, CTE and control subjects respectively. When ferritin </= 40 μg/L was used as the definition, ID occurred in 58.8%, 63.8%, and 72.3% of premenopausal women with FPHL, CTE, and control subjects, while in 26.1%, 36.8%, and

20.7% of postmenopausal women with FPHL, CTE and control subjects. This study concluded that the ID is common in women but not increased in patients with FPHL or CTE

Ramatulasi S, et al.[4] conducted a prospective case control study in 70 participants. The purpose of the study was to determine the clinico laboratory findings in women with pattern baldness and to correlate the trichoscopic parameters. The mean values of total testosterone, DHEAS and TSH showed statistically significant difference between the cases and controls. The serum ferritin level less than the cut off level of 40 mic.gram/L was identified in 52% of cases while the mean serum ferritin values of cases is less than that of controls. The most frequent trichoscopic findings were the hair diameter diversity and peripilar sign with 90% and 91.4% respectively. This study concluded that the trichoscopic findings did not differ significantly between the three forms of FPHL.

Siah T, et al., [72] conducted a study in 210 patients. The purpose of the study was to identify the clinical features, relevant medical and family history, laboratory evaluation, and treatment and compliance in patients with FPHL. The family history of androgenetic alopecia was identified in 85% of participants. The most common medical problems identified was the hypothyroidism and hypertension. While the most common concurrent hair loss condition was the telogen effluvium. Normal Vitamin D level was identified in 38% of patients. Ferritin level above 30 μg/L and normal zinc level at the first consultation were identified in 71% and 85% of the participants. This study concluded that the history of TE, hypothyroidism and hypertension, and low serum Vitamin D are common in our patients with FPHL**.**

Łukasik A, et al., [77] performed a study in 111 participants. The aim of the study was to identify the role of family history and its influence on the onsettime in female pattern hair loss. Family history of FPHL was identified in 62.2% of patients. While more than one person in the family suffered from hair loss in 28.8% of patients. Hair loss was identified in 18% of patients

grandparents. The study concluded that the the family history on the mother's side have a great significance for FPHL development.

Dhaher SA, et al., [78] performed a case control study. The purpose of the study was to determine the serum and hair zinc and iron levels in patients with FAGA. The study results revealed that the hair and serum zinc levels in FAGA group were identified as lower in the cases as compared to the control. Hair and serum iron level in FAGA was lower in the cases that than in control. This study concluded that the trace elements might play an important role in the etiopathogenesis of FAGA

Quinn M, et al.,[79] conducted a cross sectional study in 254 women. The aim of the study was to identify the prevalence of androgenic alopecia in patients with polycystic ovary syndrome and also to characterize associated clinical and biochemical features. The study results revealed that 22.0% of PCOS patients had AGA. Patients with PCOS and AGA were more likely to have acne or hirsutism as compared with the patients without AGA. Subjects with AGA showed hair loss in 70.4% of cases. This study concluded that the AGA is associated with other manifestations of clinical hyperandrogenism.

Rasheed H, et al., [80] conducted a study in 80 participants. The purpose of the study was to determine serum ferritin and vitamin D levels in females with chronic telogen effluvium or female pattern hair loss. Serum ferritin levels in the TE and FPHL were identified as 14.7 ± 22.1 µg/l and 23.9 ± 38.5 µg/l respectively. Whereas, the serum vitamin D_2 levels in females with TE and FPHL 28.8 ± 10.5 nmol/l and 29.1 ± 8.5 nmol/l respectively. Serum ferritin cut-off values for TE and FPHL were identified with 27.5 and 29.4 µg/l while, vitamin D cut off values for TE and FPHL with 40.9 and 67.9 nmol/l respectively. This study concluded that the low serum ferritin and vitamin D_2 are associated with hair loss in females with TE and FPHL.

B R-B, et al[81] conducted a study in 20 women. The aim of the study was to identify the ratio of estrogens to androgens in female pattern hair loss. The study results revealed that the absolute levels of androgens were normal in cases and control groups. The ratio of estradiol to free testosterone and the ratio of estradiol to DHEAS were identified lower in cases as compared to the control group. The study concluded that the ratio of estradiol to free testosterone can be responsible for the triggering of female pattern hair loss in women.

Chen X, et al.,[82] conducted a study in 73 patients. The aim of the study was to determine the trichoscopic features of female pattern hair loss. The hair density and hair shaft diameter of FPHL patients were reduced in the whole scalp. Vellus hair ratio and single hair follicle unit ratio were identified as increased in the cases as compared to the controls. The most affected area was the vertex while the most significant difference identified in the hair shaft diameter. The study concluded that the FPHL patients had decreased hair density and hair shaft diameter.

H K, et al., [83] performed a study in 258 women. The aim of the study was to determine the clinical office-based phototrichogram findings of multiple scalp areas in women with hair loss. Phototrichogram profiles of the women with hair loss was identified with decreased hair density and thickness as compared to the controls. . The telogen effluvium group was identified with the same order of distribution as the control group, while the density and thickness were lower than that of the controls. The values for the FPHL patient group was identified as decreased with the increasing Ludwig grade. This study concluded that the hair density and thickness vary over the entire scalp area in patients suffering from hair loss.

LACUNAE OF LITERATURES

The role of hyperandrogenemia is not clear in female pattern of hair loss. An association between iron deficiency and female pattern of hair loss is also limited in studies. Trichoscopy as a tool for evaluating hair loss disorders had gained momentum for the past one decade. But, the studies determining the features of patterned hair loss are less in females.

MATERIALS AND METHODS

Materials and Methods:

Source of data:

All female patients with non-scarring hair loss attending the outpatient clinic of Dermatology, Venereology and Leprosy in R L Jalappa Hospital and Research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar will be screened and those satisfying the inclusion criteria will be included in the study between January 2021 and July 2022.

Study Design:

Cross-sectional descriptive study.

Sample size calculation:

Sample size of the present study is calculated based on significant positive correlation (spearman's rank correlation) between focal atrichia and stage of hair loss 0.591 in a study done by Department of Dermatology at Sun Yat-sen University, Guangzhou, China assuming a population correlation coefficient 0.3, with power 90% and alpha error of 5% the estimated sample size for the study is 80.

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

Here

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

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

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

Statistical analysis

Categorical data will be represented in the form of Frequencies and proportions. Continuous data will be represented as mean and standard deviation. p value <0.05 will be considered as statistically significant .Chi-square/fisher's exact test will be used as test of significance. Data will be entered into Microsoft excel data sheet and will be analyzed using SPSS 22 version software.

Method of collection of data (including sampling procedure)

Sampling technique: Opd estimates 10 cases per month accounting for 180 cases for one and half years and required sample size being 80 , considering sampling interval of 2 , these 80 cases will be recruited by systematic random sampling technique.

SELECTION CRITERIA

Inclusion criteria

• Female patients with clinical features suggestive of androgenic alopecia (FPHL).

Exclusion criteria

- Female patients who are postmenopausal and those in postpartum (up to 1 year), pregnant, and lactation phase
- Female patients who have undergone hysterectomy
- Female patients with alopecia secondary to known endocrinologic disorder (Cushing's disease, Addison's disease, etc.)
- Female patients on hormonal treatment or oral and/or topical hair growth promoters
- Female patients on immunosuppressive drugs
- Female patients with other non scarring alopecias

Methods of data collection

All female with hair loss will be screened and those satisfying the inclusion criteria were

included in the study.

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

A written informed consent will be taken from the patients and in children from parents or guardian. General physical examination of patient is done, scalp examination, hormone analysis of Dehydroepiandrosterone-sulfate (DHEA-S), prolactin, androstenedione, and free triiodothyronine (FT3), tests are done ,under aseptic conditions 2ml of blood is collected from the vein of the hand and immediately sent to laboratory for respective hormone analysis and results are documented there are no side effects associated with blood drawing .All the guidelines of Central diagnostic laboratory services of R.L Jalappa Hospital are followed in discarding the extra samples if collected , clinical examination of scalp, tests for hair anchorage and fragility will be done in all cases.

Trichoscopy a target area of 2x2 cm is selected in the active alopecia site of the participants of the study ,structures are visualized by dermatoscope DL4N with 10x magnification using polarized and non polarized mode. The structures which are visualized include hair shafts, hair follicle openings, the perifollicular epidermis and cutaneous microvasculature and trichoscopic findings of that target area will be assessed, yellow dots (YD), tapering hair (TH), broken hair (BH), black dot (BD), Pig tail hair, coudability hair (CH) and short vellus hairs (SVH) will be documented in that particular target area. The number of hair in one pilosebaceous unit is assessed and documented. The trichoscopic patterns will be

analysed. The data thus collected will be entered in to a specially designed Case Record Form and subjected to statistical analysis like proportion and Chi-square test.

In case of any untoward event, it will be managed by Department of Dermatology R.L Jalappa Hospital.

Statistical methods:

Biological parameters, trichoscopic findings etc., were considered as outcome parameters. Pattern of baldness was considered as Primary explanatory variable. Age group, gender, etc., were considered as study relevant variables.

Descriptive analysis: Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram.

For normally distributed Quantitative parameters the mean values were compared between study groups using Independent sample t- test (2 groups) / ANOVA test (> 2 groups). For normally distributed Quantitative parameters, Medians and Interquartile range (IQR) were compared between study groups using Mann Whitney u test (2 groups) / Kruskal Wallis test (> 2 groups).

The association between explanatory variables and categorical outcomes was assessed by cross tabulation and comparison of percentages. Chi square test was used to test statistical significance. Association between quantitative explanatory and outcome variables was assessed by calculating spearman rank (r_s) correlation coefficient.

P value < 0.05 was considered statistically significant. Data was analysed by using coGuide software, V.1.01.[84]

RESULTS

RESULTS

A total 80 female participants were included in the final analysis.

Table 4: Descriptive analysis of Age group in the study population (N=80)

Age group	Frequency	Percentage
19-29 years	26	32.50%
30-39 years	32	40.00%
40-49 years	18	22.50%
>=50 years	4	5.00%

Among the study population, 26 (32.50%) participants were aged between 19-29 years, 32 (40%) participants were aged between 30-39 years, 18 (22.50%) participants were aged between 40-49 years and 4 (5%) participants were aged between >=50 years. (Table 1& Figure 15)

Figure 15: Pie chart of age group in the study population (N=80)

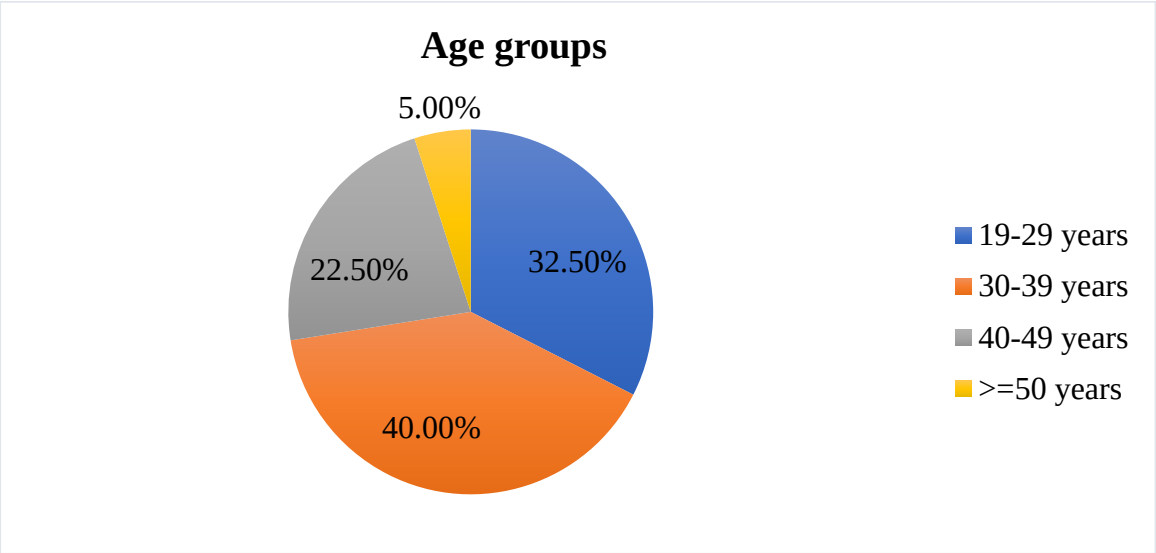


Table 5: Descriptive analysis of Age of onset group in the study population (N=80)

Age of onset group	Frequency	Percentage
19-29 years	42	52.5%
30-39 years	25	31.25%
40-49 years	11	12.5%
>=50 years	2	2.5%

Among the study population, 43 (52.5%) participants had 19-29 years age of onset group, 25 (31.25%) participants had 30-39 years age of onset group, 10 (12.25%) participants had 40-49 years age of onset group and 2 (2.5%) participant had >=50 years age of onset group. (Table 3 & Figure 16)

Figure 16: Pie chart of age of onset group in the study population (N=80)

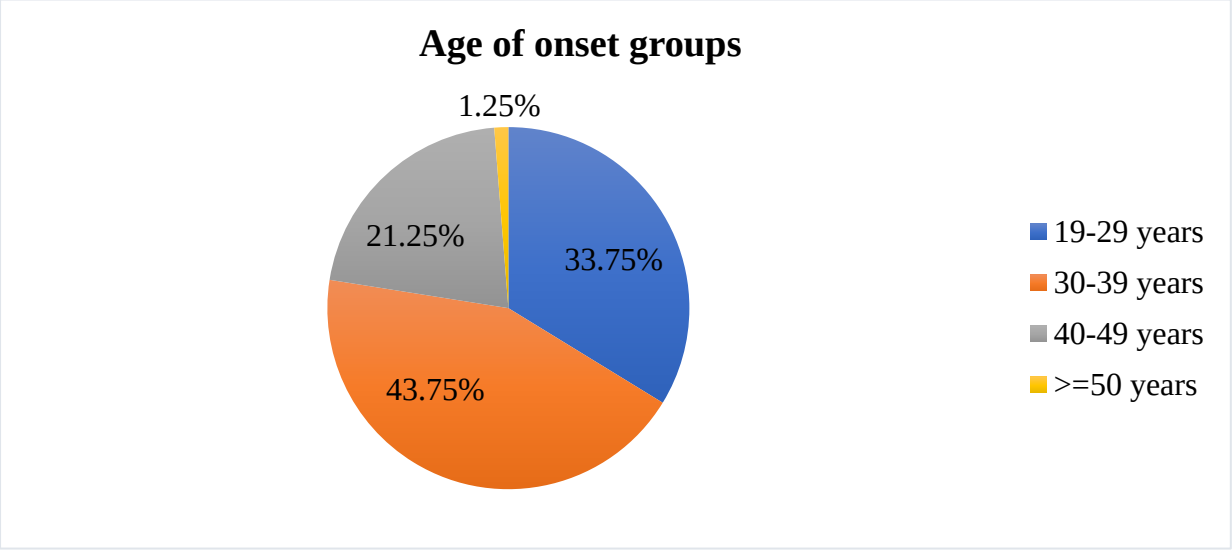


Table 6: Descriptive analysis of Duration in the study population (N=80)

Parameter	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Duration (years)	5.21±3.90	5	0.16	10.00	14.88	24.47

The mean duration(years) 5.21±3.90 was in the study population, ranged between 0.16 to 10 (95% CI 14.88 to 24.47). (Table 4)

Table 7: Descriptive analysis of Pattern of baldness in the study population (N=80)

Pattern of baldness	Frequency	Percentage
Ludwig grade 1	39	48.75%
Ludwig grade 2	24	30.00%
Ludwig grade 3	8	10.00%
Oslen Pattern	3	3.75%
Male Pattern baldness	6	7.50%

Among the study population, 39 (48.75%) participants had ludwig grade 1, 24 (30%) had ludwig grade 2, 8 (10%) had ludwig grade 3, 3 (3.75%) had oslen pattern and 6 (7.5%) had male pattern baldness (Table 5 & Figure 17)

Figure 17: Bar chart of Pattern of baldness in the study population (N=80)

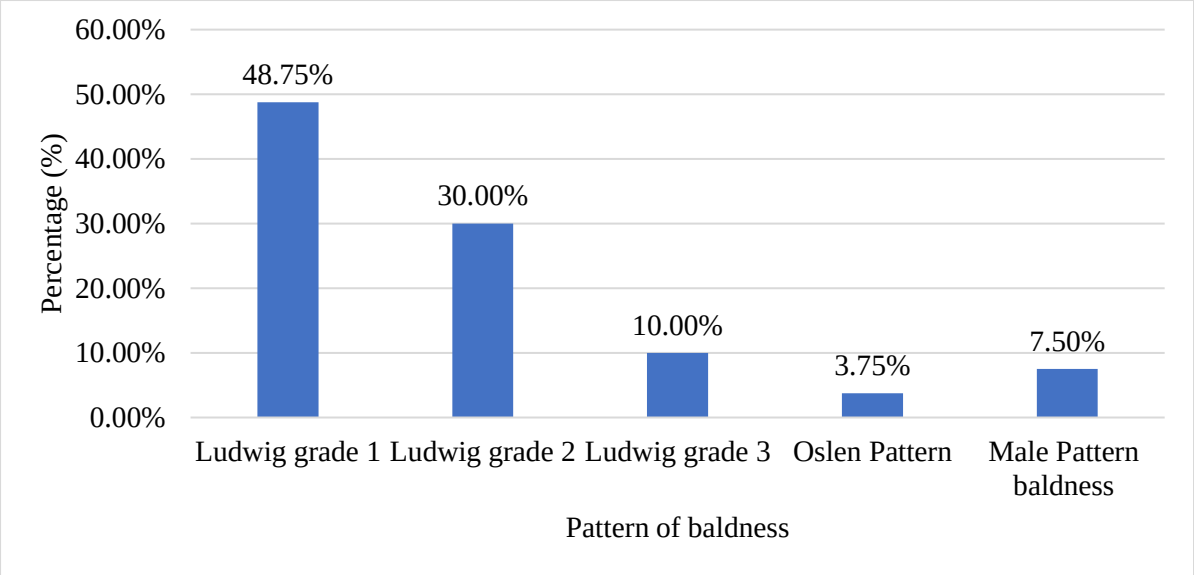


Table 8: Descriptive analysis of baldness in family members in the study population (N=80)

baldness in family members	Frequency	Percentage
YES	57	71.25%
NO	23	28.75%

Among the study population, 57 (71.25%) participants had one baldness in family members and 23 (28.75%) participants had two baldness in family members. (Table 6 & Figure 18)

Figure 18: Bar chart of baldness in family members in the study population (N=80)

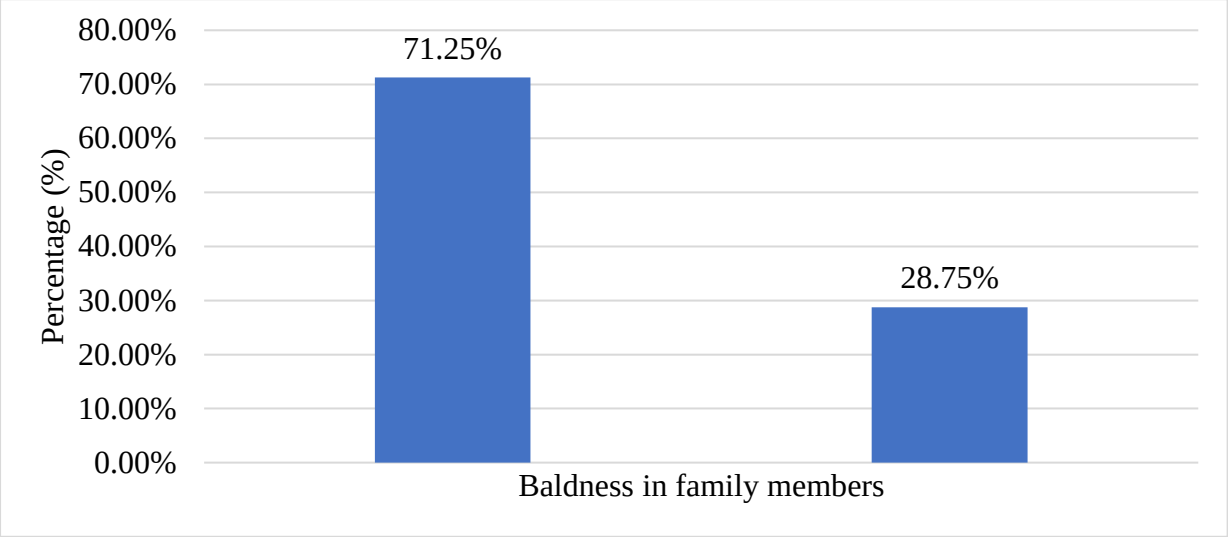


Table 9: Comparison of Duration with Pattern of baldness in the study population (N=80)

Pattern of baldness	Duration(Years)	Kruskal Wallis Test(P Value)
	Mean value	
Ludwig grade 1 (N=39)	3.8±1.72	0.00986
Ludwig grade 2 (N=24)	5.3±1.93	
Ludwig grade 3 (N=8)	9.5±4.5	
Oslen Pattern (N=3)	4.3±1.23	
Male Pattern baldness (N=6)	5.19 ±3.38	

The mean duration of baldness in Ludwig grade 1 is 3.8±1.72 in Ludwig grade 2, it is 5.3±1.93 in Ludwig grade 3 it is 9.5±4.5, in oslen pattern and it is 4.3±1.23 and in male pattern it i 5.19 ±3.38. The mean difference of duration in pattern of baldness is statistically significant (P <value 0.05). (Table 6 and Figure 19)

Figure 19: Line graph of comparison of Duration with Pattern of baldness in the study population (N=80)

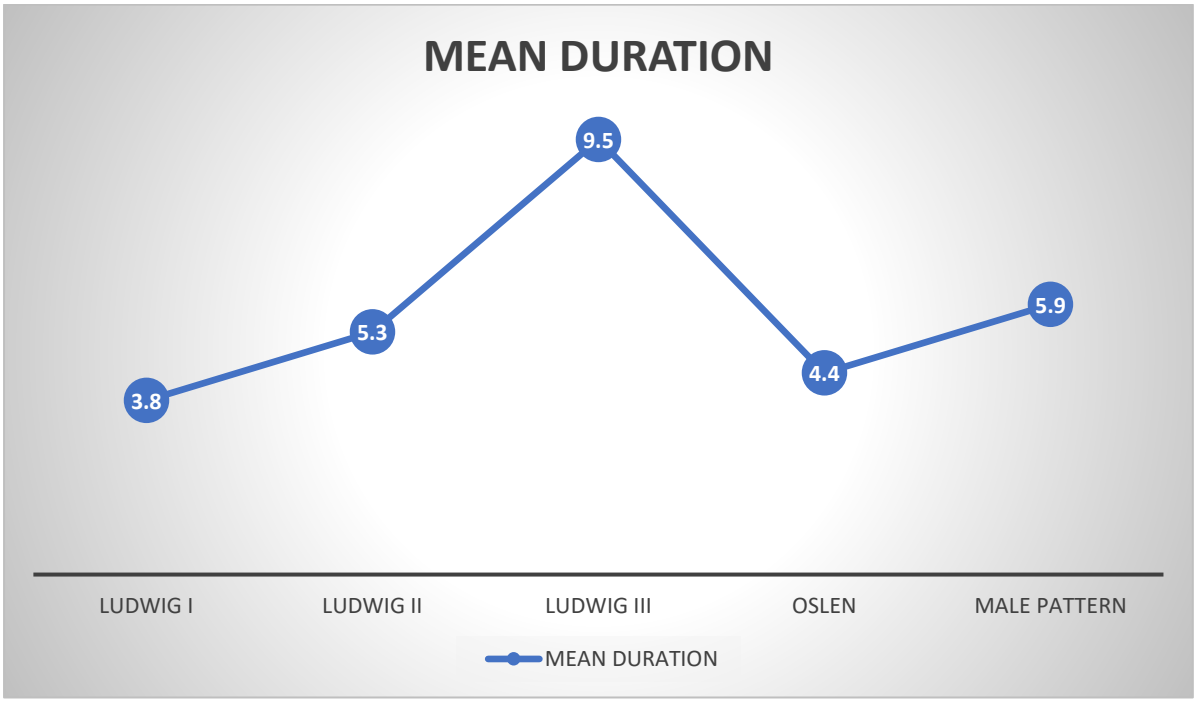


Table 10: Descriptive analysis of Biological Parameters in the study population (N=80)

Parameter	Mean ± S. D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
DHEAS (ug/dL)	232.24 ± 131.95	198.50	50.60	549.50	203.32	261.15
Prolactin(ng/mL)	23.21 ± 7.65	22.60	6.60	42.60	21.54	24.89
Androstenedione(ng/mL)	1.63 ± 0.83	1.63	0.42	4.04	1.45	1.81

The mean DHEAS (ug/dL) was 232.24 ± 131.95 in the study population. Ranged between 50.60 to 549.50 (95% CI 203.32 to 261.15). The mean prolactin(ng/mL) was 23.21 ± 7.65 in the study population. Ranged between 6.6 to 42.6 (95% CI 21.54 to 24.89). The mean androstenedione(ng/mL) was 1.63 ± 0.83 in the study population. Ranged between 0.42 to 4.04 (95% CI 1.45 to 1.81). (Table 7)

Table 11: Comparison of Biological Parameters with Pattern of baldness in the study population (N=80)

Parameter	Pattern of baldness					ANOVA P Value
	Ludwig I (N=39)	Ludwig II (N=24)	Ludwig III (N=8)	OSLEN (N=3)	Male pattern (N=6)	
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
DHEAS(ug/dL)	215.67± 127.15	220.49± 131.42	217.19± 128.12	223.50± 104.53	411.33± 60.78	0.0140
Prolactin(ng/mL)	21.66 ± 7.75	23.97 ± 7.29	24.85 ± 7.83	25.00 ± 5.85	27.17 ± 8.75	0.4169
Androstenedione (ng/mL)	1.46 ± 0.74	1.41 ± 0.64	1.90 ± 0.86	2.06 ± 0.63	3.05 ± 0.73	<0.001

The mean DHEAS(ug/dL) with in Ludwig I was 215.67 ± 127.15, it was 220.49 ± 131.42 Ludwig II, it was 217.19 ± 128.12 Ludwig III, it was 223.50± 104.53 in OSLEN and it was 411.33± 60.78 in male pattern. The mean difference of DHEAS(ug/dL) in pattern of baldness was statistically significant (P value < 0.05).

The mean Prolactin(ng/mL) with in Ludwig I was 21.66 ± 7.75, it was 23.97 ± 7.29 Ludwig II, it was 24.85 ± 7.83 Ludwig III, it was 25.00 ± 5.85 in OSLEN and it was 27.17 ± 8.75 in male pattern. The mean difference of Prolactin(ng/mL) in pattern of baldness was statistically not significant (P value > 0.05).

The mean Androstenedione(ng/mL) with in Ludwig I was 1.46 ± 0.74, it was 1.41 ± 0.64 Ludwig II, it was 1.90 ± 0.86 Ludwig III, it was 2.06 ± 0.63 in OSLEN and it was 3.05 ± 0.73 in male pattern. The mean difference of Androstenedione(ng/mL) in pattern of baldness was statistically significant (P value < 0.05). (Table 19)

Figure 20: Line chart of comparison of DHEAS(ug/dL) with Pattern of baldness in the study population (N=80)d

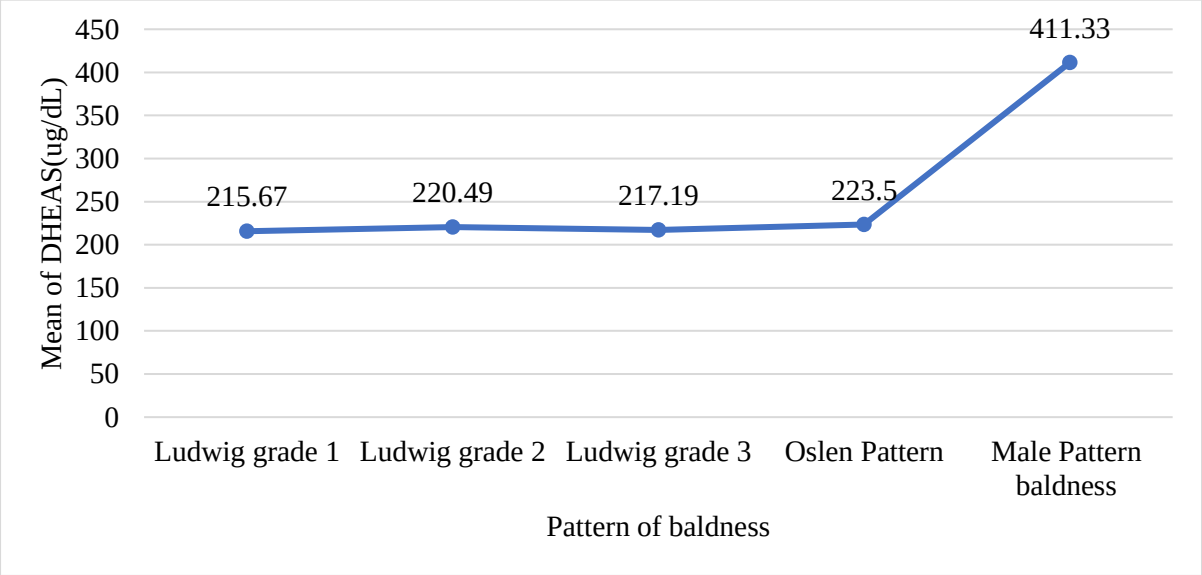


Figure 21: Line chart of comparison of Prolactin(ng/mL) with Pattern of baldness in the study population (N=80)

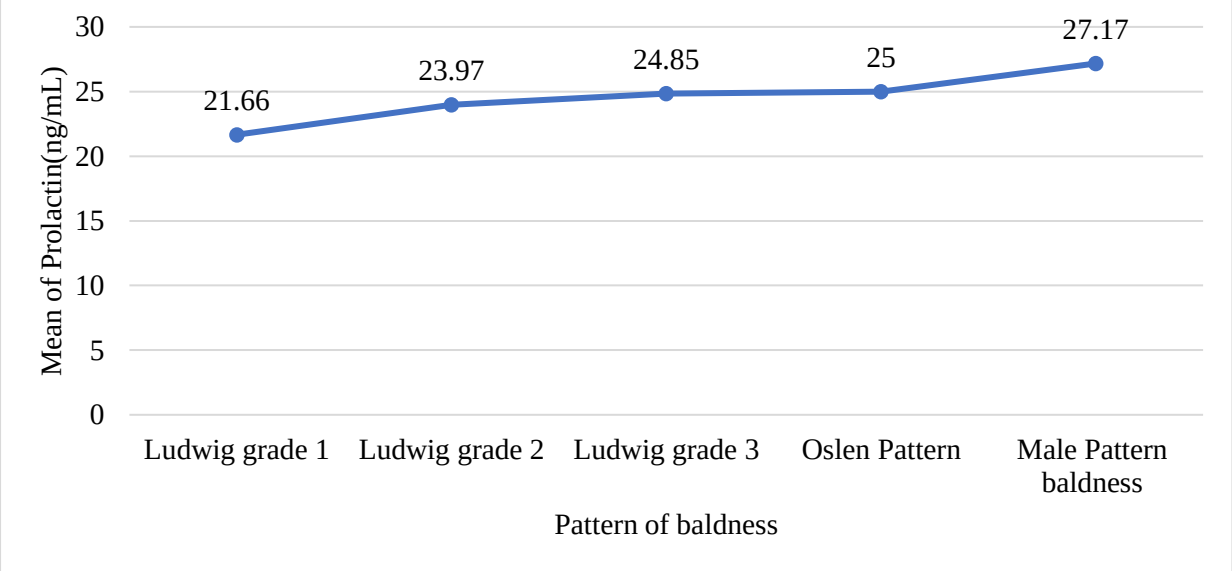


Figure 22: Line chart of comparison of Pattern of baldness with Androstenedione (ng/mL) group in the study population (N=80)

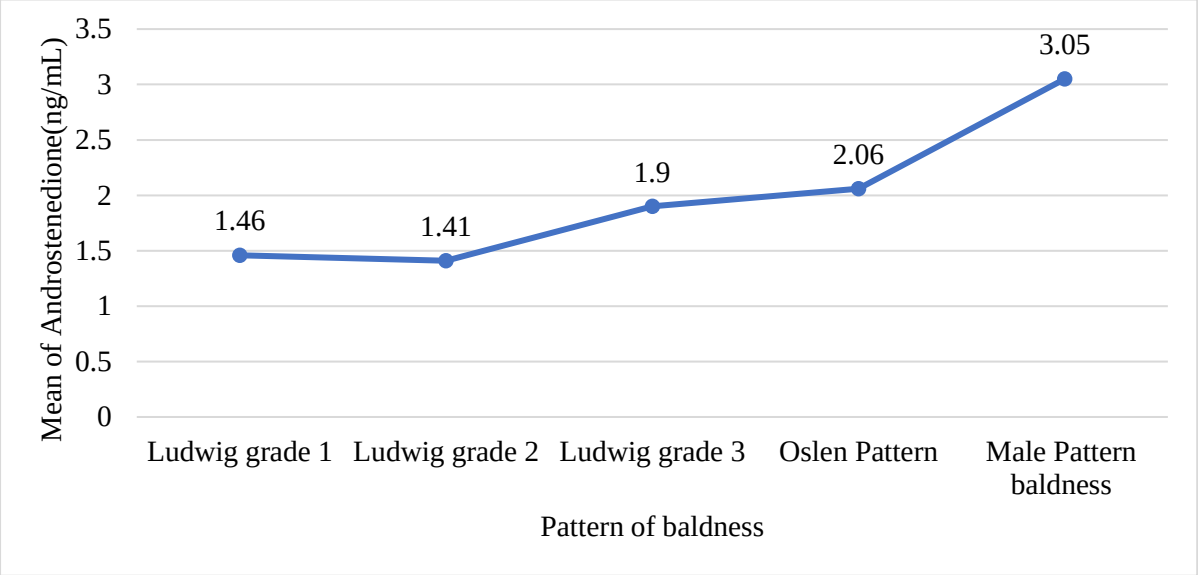


Table 12: Comparison of Free Triiodothyronine(pg/mL) with Pattern of baldness in the study population (N=80)

Parameter	Free Triiodothyronine(pg/mL) Median(IQR)	Kruskal Wallis Test(P Value)
Ludwig grade 1 (N=39)	2.80(2.5 to 3.4)	0.0685
Ludwig grade 2 (N=24)	2.75(1.87 to 3.52)	
Ludwig grade 3 (N=8)	2.40(2.18 to 2.9)	
Oslen Pattern (N=3)	2.70(2.55 to 2.95)	
Male Pattern baldness (N=6)	3.95(3.42 to 4.7)	

The median free triiodothyronine(pg/mL) with in Ludwig grade 1 was 2.80 (2.5 to 3.4), it was 2.75 (1.87 to 3.52) in Ludwig grade 2, it was 2.40 (2.18 to 2.9) in Ludwig grade 3, it was 2.70 (2.55 to 2.95) in OSLEN pattern and it was 3.95 (3.42 to 4.7) in male pattern. The median difference of free triiodothyronine(pg/mL) in pattern of baldness was not statistically significant (Table 23 & Figure 23)

Figure 23: Line chart of comparison of Free Triiodothyronine(pg/mL) with Pattern of baldness in the study population (N=80)

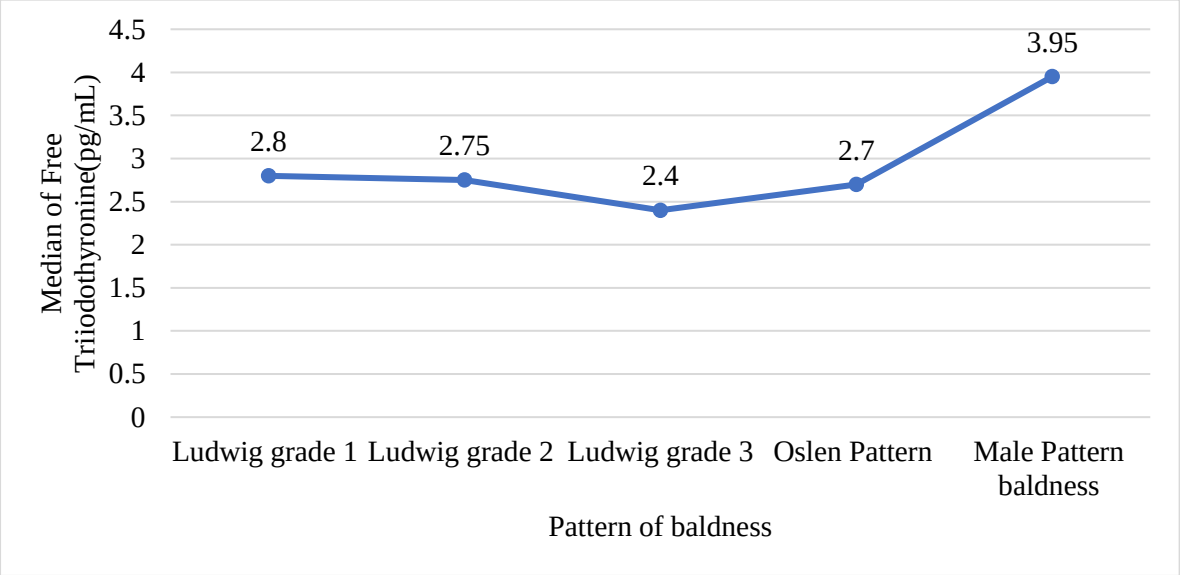


Table 13: Comparison of Pattern of baldness with DHEAS(ug/dL) group in the study population (N=80)

DHEAS(ug/dL) group	Pattern of baldness				
	Ludwig grade 1 (N=39)	**Ludwig grade 2 (N=24)**	**Ludwig grade 3 (N=8)**	**OSLEN Pattern (N=3)**	**Male Pattern baldness (N=6)**
Normal (35-430 ug/dL)	35 (89.74%)	21 (87.50%)	7 (87.50%)	3 (100.00%)	4 (66.67%)
Abnormal (>430)	4 (10.26%)	3 (12.50%)	1 (12.50%)	0 (0.00%)	2 (33.33%)

No statistical test was applied- due to 0 subjects in the cells

In Pattern of baldness with Ludwig grade 1, Ludwig grade 2, Ludwig grade 3, OSLEN pattern and male pattern the majority of 35 (89.74%), 21 (87.5%), 7 (87.5%), 3 (100.00%) and 4 (66.67%) females were in normal DHEAS(ug/dL) group. (Table 20 & Figure 24)

Figure 24: Stacked bar chart of comparison of Pattern of baldness with DHEAS(ug/dL) group in the study population (N=80)

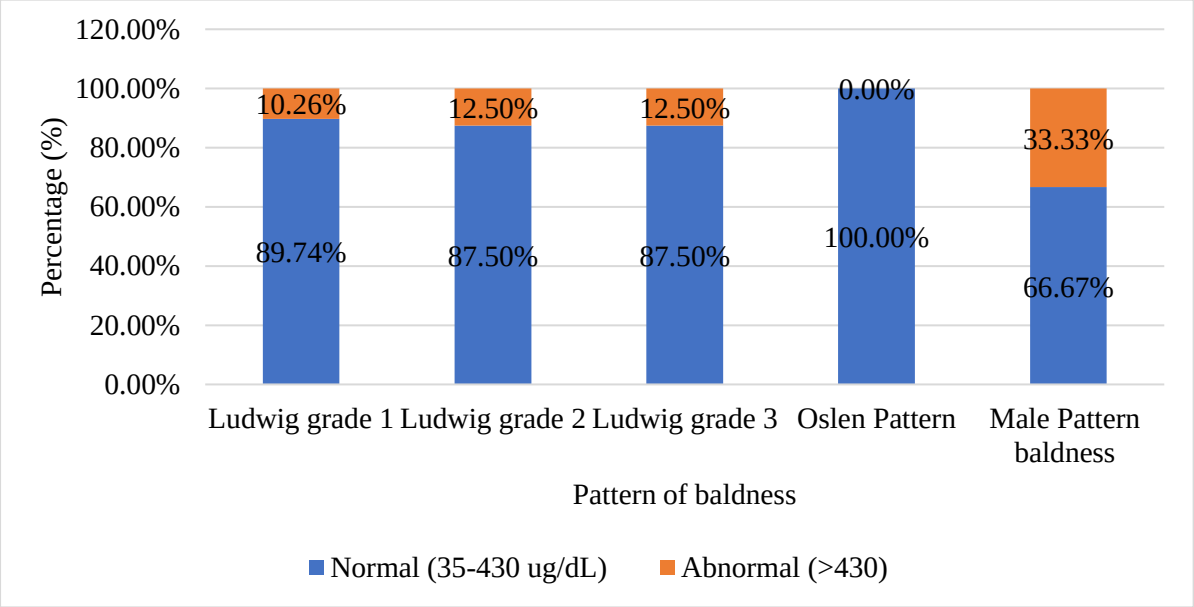


Table 14: Comparison of Pattern of baldness with Prolactin(ng/mL) group in the study population (N=80)

Prolactin(ng/mL) group	Pattern of baldness					Chi square value	P value
	Ludwig grade 1 (N=39)	Ludwig grade 2 (N=24)	Ludwig grade 3 (N=8)	OSLEN Pattern (N=3)	Male Pattern baldness (N=6)		
Normal (2.8-28.2 ng/dL)	30 (76.92%)	16 (66.67%)	5 (62.50%)	2 (66.67%)	4 (66.67%)	1.25	0.8698
Abnormal (>28.2 ng/dL)	9 (23.08%)	8 (33.33%)	3 (37.50%)	1 (33.33%)	2 (33.33%)		

The difference in the proportion of prolactin(ng/mL) group between pattern of baldness was statistically not significant with P value 0.8698 with majority of 30 (76.92%) participants were in Ludwig grade 1 had normal prolactin(ng/mL) group and 9 (23.08%) had abnormal prolactin(ng/mL) group. (Table 21 & Figure 25)

Figure 25: Stacked bar chart of comparison of Pattern of baldness with Prolactin(ng/mL) group in the study population (N=80)

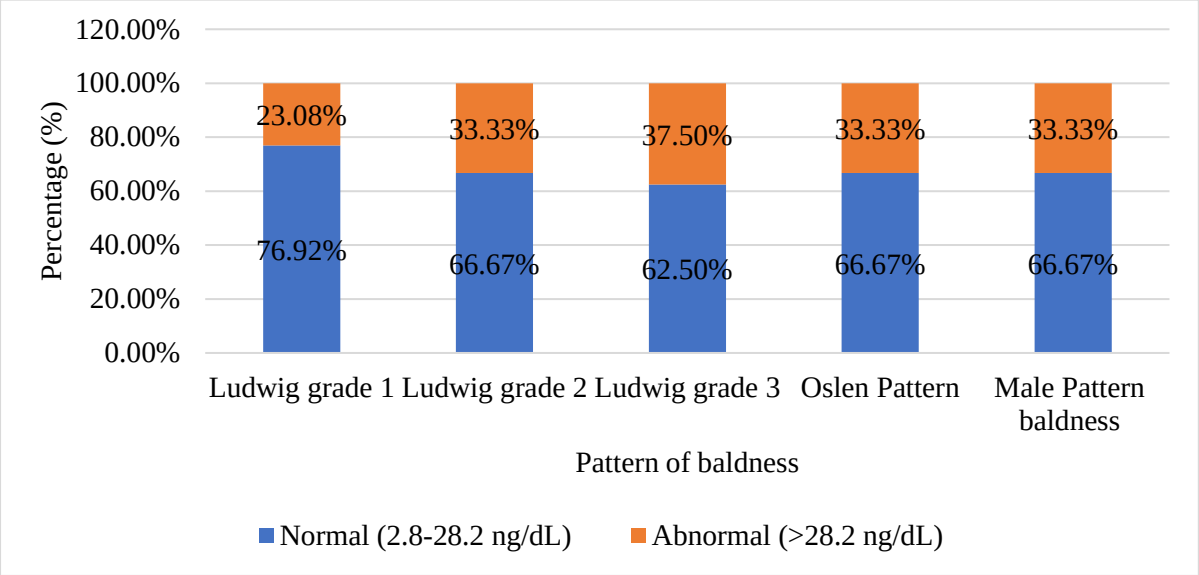


Table 15: Comparison of Pattern of baldness with Androstenedione(ng/mL) group in the study population (N=80)

Androstenedione (ng/mL) group	Pattern of baldness					Chi Square Value	P Value
	Ludwig grade 1 (N=39)	Ludwig grade 2 (N=24)	Ludwig grade 3 (N=8)	OSLEN Pattern (N=3)	Male Pattern baldness (N=6)		
Normal (0.3-2.4)	34 (87.18%)	22 (91.67%)	6 (75.00%)	2 (66.67%)	1 (16.67%)	19.66	0.781
Abnormal (>2.4)	5 (12.82%)	2 (8.33%)	2 (25.00%)	1 (33.33%)	5 (83.33%)		

The difference in the proportion of androstenedione(ng/mL) group between pattern of baldness was not statistically significant with P value >0.001 with majority of 34 (87.18%) participants were in Ludwig grade 1 had normal androstenedione(ng/mL) group and 5 (12.82%) had abnormal androstenedione(ng/mL) group. (Table 22 & Figure 15)

Figure 26: Stacked bar chart of comparison of pattern of baldness with androstenedione (ng/mL) group in the study population (N=80)

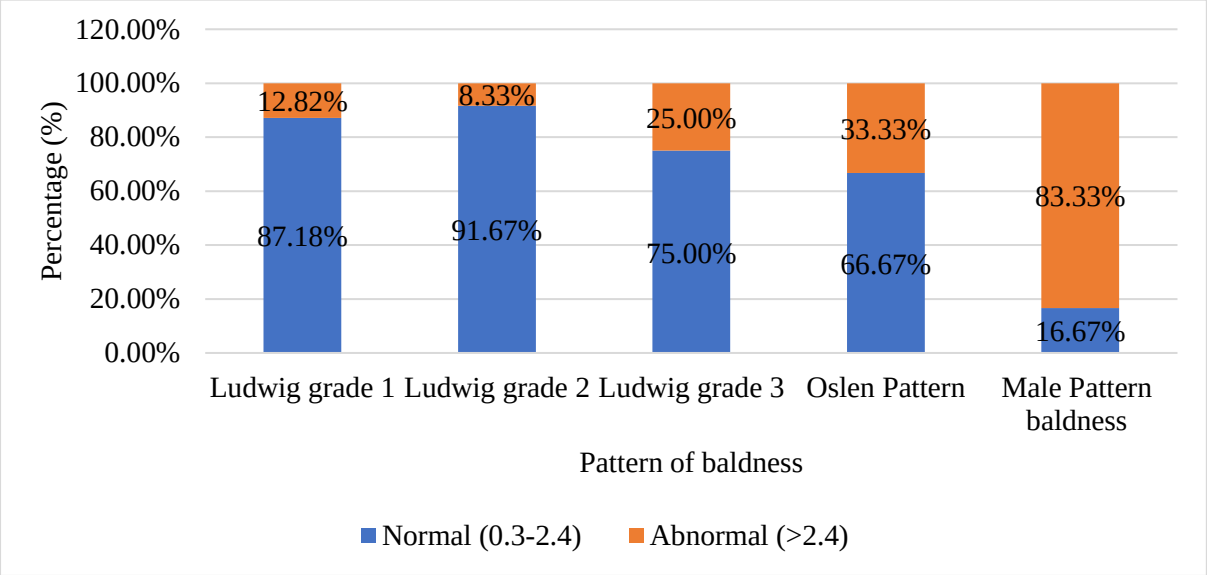


Table 16: Correlation of DHEAS(ug/dL) with Pattern of baldness (N=71)

Parameter	DHEAS(ug/dL)	P Value(spearman)
	rs Value(spearman)	
Pattern of baldness	0.02	0.8951

There was a weak positive correlation between Pattern of baldness and DHEAS(ug/dL) (r_s value: 0.02, P value: 0.8951). (Table 25 & Figure 27)

Figure 27: Scatter plot diagram of correlation of DHEAS(ug/dL) with Pattern of baldness (N=71)

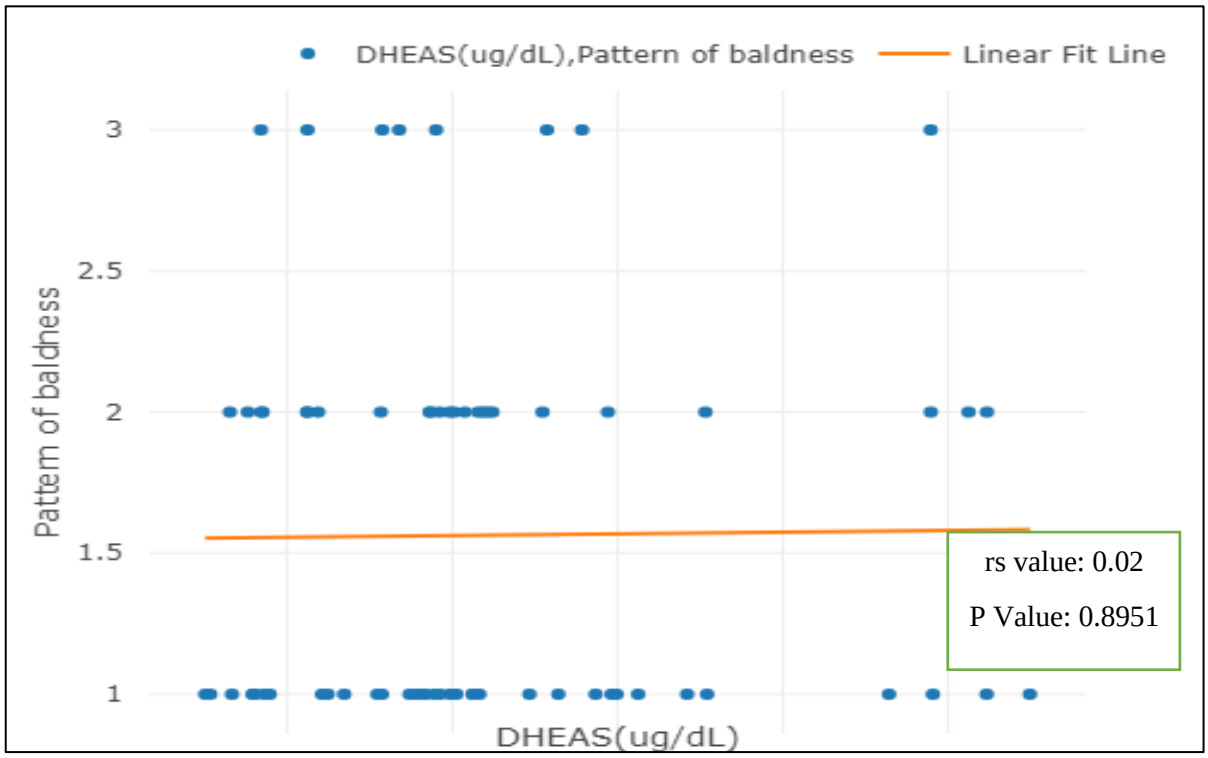


Table 17: Correlation of Prolactin(ng/mL) with Pattern of baldness (N=71)

Parameter	Prolactin(ng/mL)	P Value(spearman)
	rs Value (spearman)	
Pattern of baldness	0.17	0.1478

There was a weak positive correlation between Pattern of baldness and prolactin(ng/mL) (r_s value: 0.17, P value: 0.1478). (Table 26 & Figure 28)

Figure 28: Scatter plot diagram of correlation of Prolactin(ng/mL) with Pattern of baldness (N=71)

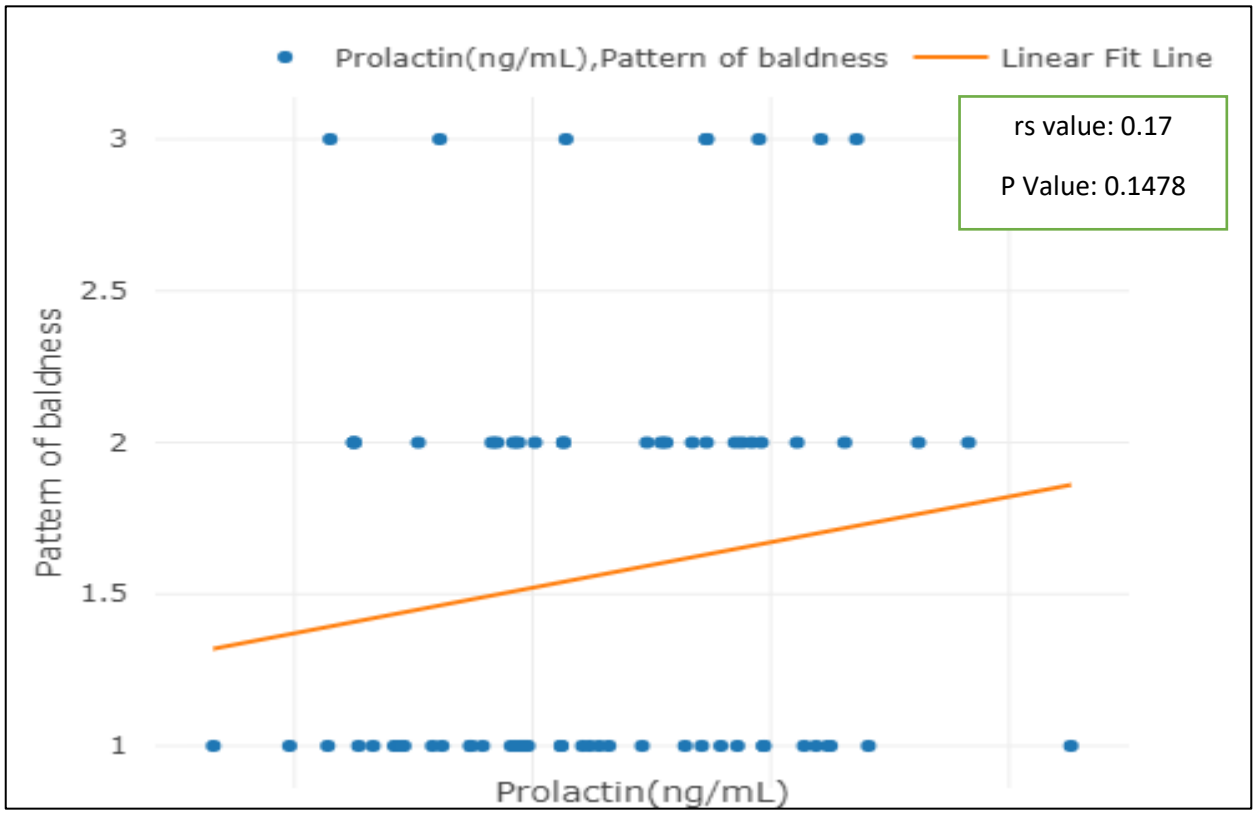
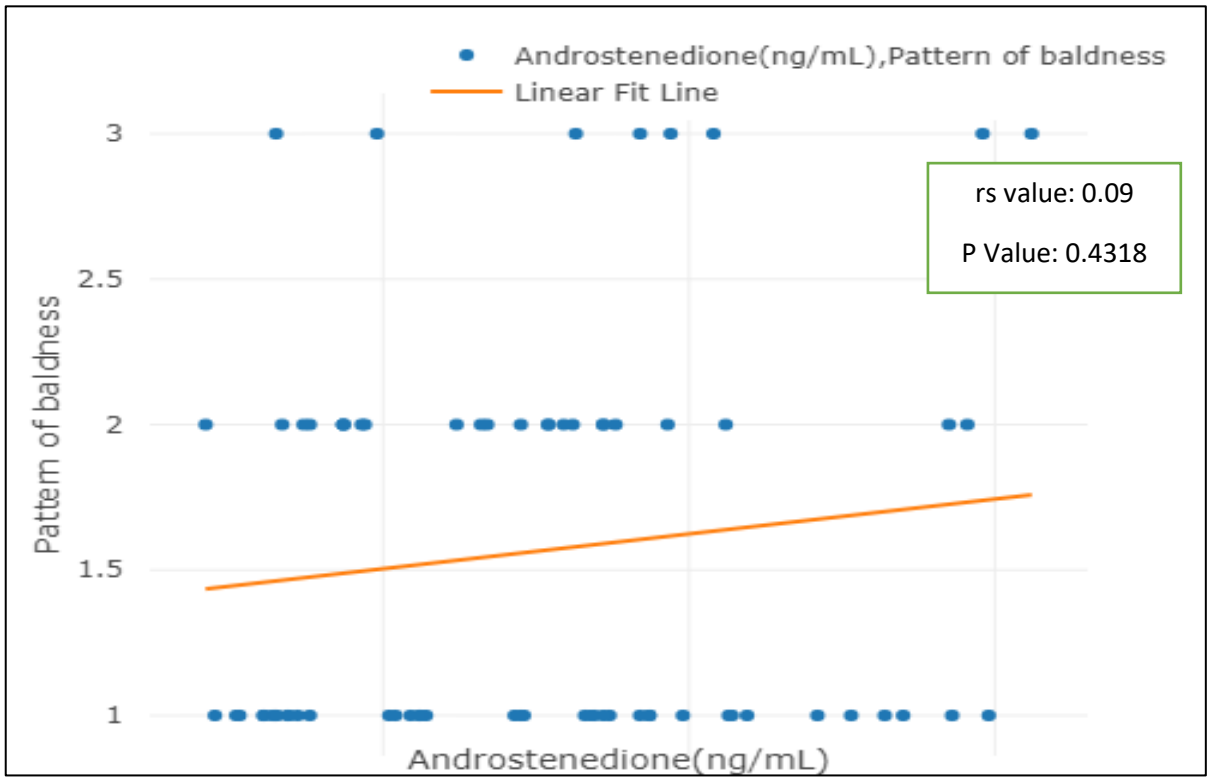


Table 18: Correlation of Androstenedione(ng/mL) with Pattern of baldness (N=71)

Parameter	Androstenedione(ng/mL)	P Value(spearman)
	rs Value(spearman)	
Pattern of baldness	0.09	0.4318

There was a weak positive correlation between Pattern of baldness and androstenedione (ng/mL) (r_s value: 0.09, P value: 0.4318). (Table 27 & Figure 29)

Figure 29: Scatter plot diagram of correlation of Androstenedione(ng/mL) with Pattern of baldness (N=71)



Trichoscopic findings

Table 19: Descriptive analysis of Trichoscopic findings in the study population (N=80)

Parameters	Frequency	Percentage
Brown Peri pilar signs (BPPS)	48	58.75%
White peri pilar signs (WPPS)	23	28.70%
White Dots	20	25.00%
Yellow Dots	46	57.5%
Focal Atrichia	30	37.55%
Hair shaft thickness heterogeneity (HSTH)	76	95.00%
Scalp Honeycomb pigmentation	47	58.70%

Among the study population, 48 (58.75%) participants had Brown Peri pilar signs (BPPS), 23 (28.7%) participants had White peri pilar signs (WPPS), 20 (25%) participants had white dots, 46 (57.5%) participants had yellow dots, 30 (37.5%) participants had focal atrichia, 76 (95%)

participants had hair shaft thickness heterogeneity (HSTH) and 47(58.5%) participants had Scalp Honeycomb pigmentation. (Table 16)

Table 20: Comparison of Pattern of baldness with Trichoscopic findings in the study population (N=80)

Parameter	Pattern of baldness					Chi square value	P value	Correlation Coefficient (Rs)
	Ludwig grade 1 (N=39)	Ludwig grade 2 (N=24)	Ludwig grade 3 (N=8)	Oslen Pattern (N=3)	Male Pattern baldness (N=6)			
Brown Peri pilar signs (BPPS)								
Present	30 (76.04%)	14 (58.3%)	1 (12.50%)	1 (33.33%)	2 (33.33%)	0.28	<0.001	-0.977
Absent	1 (2.56%)	7 (29.17%)	7 (87.50%)	2 (66.67%)	4 (66.67%)			
White peri pilar signs (WPPS)								
Present	3 (7.69%)	9 (37.50%)	7(87.5%)	2 (66.67%)	2 (33.33%)	0.23	<0.001	0.988
Absent	36 (92.31%)	15 (62.50%)	1 (1.25%)	1 (33.33%)	4 (66.28%)			
White Dots								
Present	8 (20.51%)	5 (20.83%)	5 (62.50%)	1 (33.33%)	1 (16.67%)	6.97	0.1372	
Absent	31 (79.49%)	19 (79.17%)	3 (37.50%)	2 (66.67%)	5 (83.33%)			
Yellow Dots								
Present	23(58.9%)	13(54.1%)	5 (62.5%)	2 (66.6%)	3 (50%)	*	*	
Absent	16 (41.02%)	11 (45.83%)	3 (37.5%)	1(33.3%)	3 (50.00%)			
Focal Atrichia								
Present	2 (5.13%)	19 (79.17%)	7 (87.50%)	2 (66.67%)	0 (0.00%)	*	*	+0.908
Absent	37 (94.87%)	5 (20.83%)	1 (1.25%)	1 (33.33%)	6 (100%)			
Hair diameter diversity (HDD) (Present)	35(89.70%)	24 (100.0%)	8 (100.0%)	3 (100.0%)	6 (100.0%)	0.00	1.000	

(Absent)	4(10.23%)	0	0	0	0			
Scalp Honeycomb pigmentation								
Present	18 (46.15%)	17 (70.83%)	7 (87.5%)	2 (66.67%)	3 (50.00%)	*	*	+0.998
Absent	21 (53.85%)	7 (29.17%)	1 (11.25%)	1 (33.33%)	3 (50.00%)			

No statistical test was applied- due to 0 subjects in the cells

The difference in the proportion of brown peri pilar signs (BPPS) between pattern of baldness was statistically significant with P value <0.001 with majority of 30 (76.04%) participants were in Ludwig grade 1 had brown peri pilar signs (BPPS). In Pattern of baldness with Ludwig grade I, Ludwig II , Ludwig grade III, Oslen and male pattern, 3 (7.69%), 9 (37.50%), 7 (87.5%), 2 (66.67%) and 2 (33.33%) females had white peri pilar signs (WPPS),with p value <0.05 and correlation coefficient(Rs=+0.98)

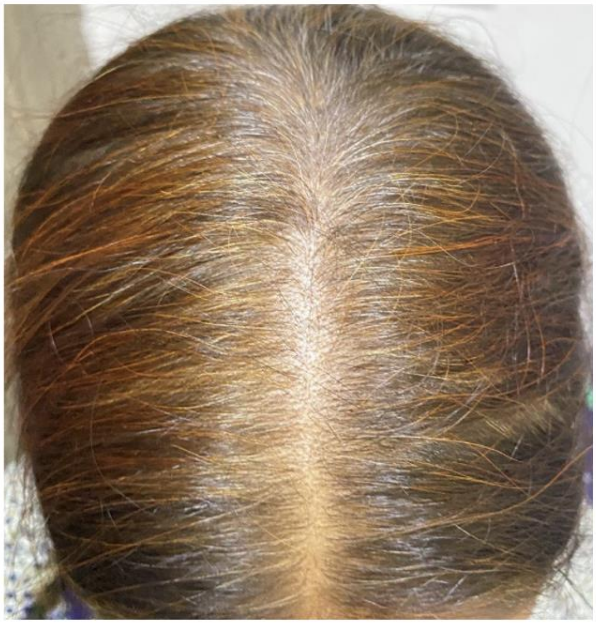
The difference in the proportion of white dots between pattern of baldness was statistically not significant with P value 0.1372 with majority of 8 (20.51%) participants were in Ludwig grade I had white dots.

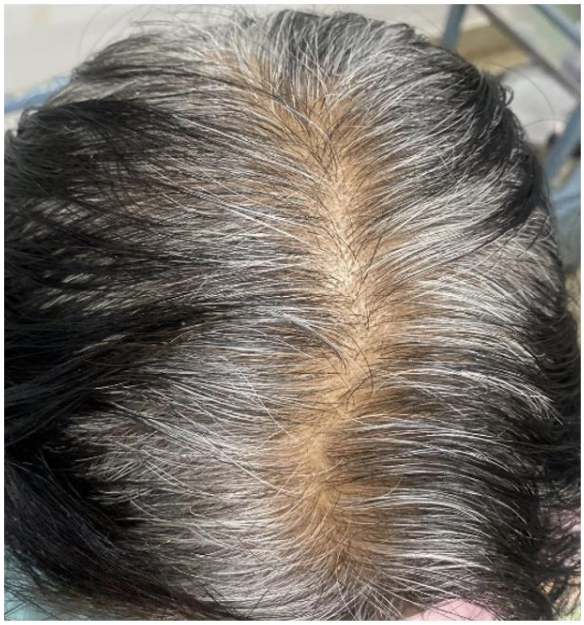
In Pattern of baldness with Ludwig grade I and Ludwig grade II, 6 (15.38%) and 1 (4.17%) female had yellow dots.

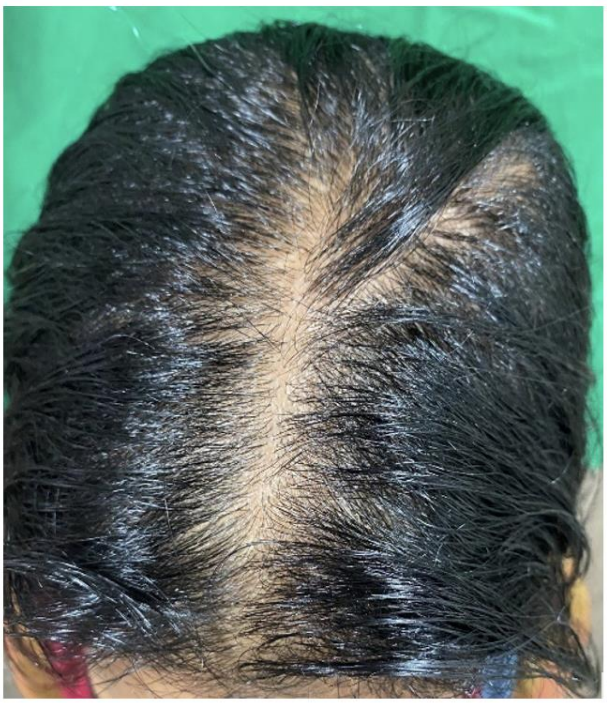
In Pattern of baldness with Ludwig grade I, Ludwig grade II, Ludwig grade III and Oslen pattern 2 (5.13%),19 (79.17%), 7 (87.5%) and 2 (66.67%) females had focal atrichia. None of the females with male pattern baldness had focal atrichia (0%).

In Pattern of baldness with Ludwig grade I, Ludwig grade II, Ludwig grade III, Oslen and male pattern, 18 (46.15%), 17 (70.83%), 7 (87.5%), 2 (66.67%) and 3 (50%) females had scalp honeycomb pigmentation. (Table 24)

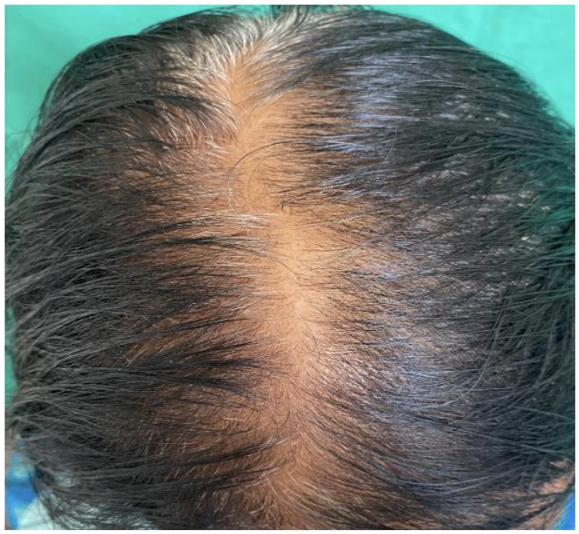
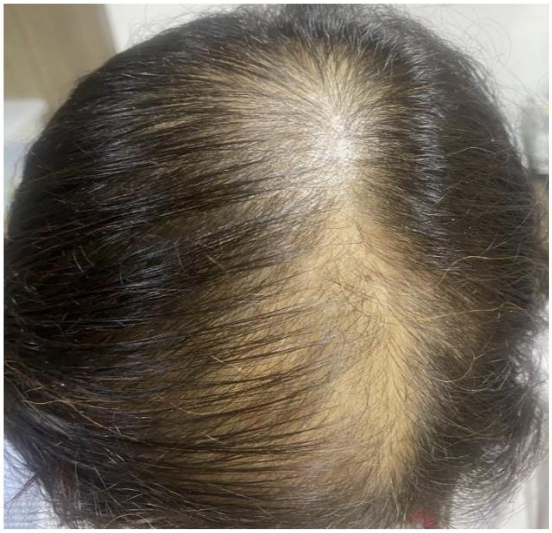
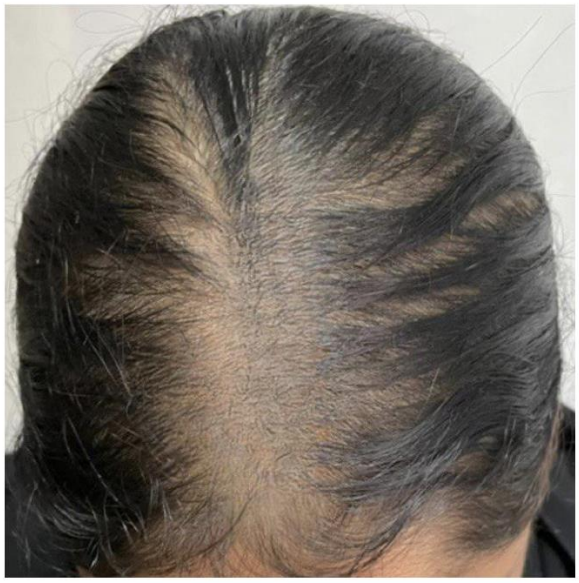
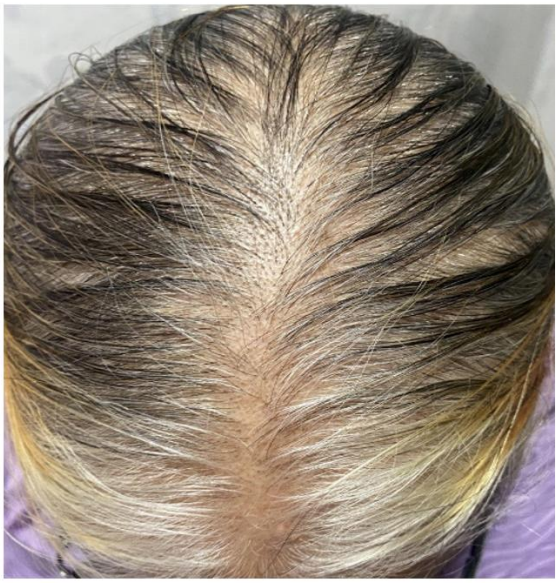
PHOTOGRAPHS: LUDWIG GRADE I



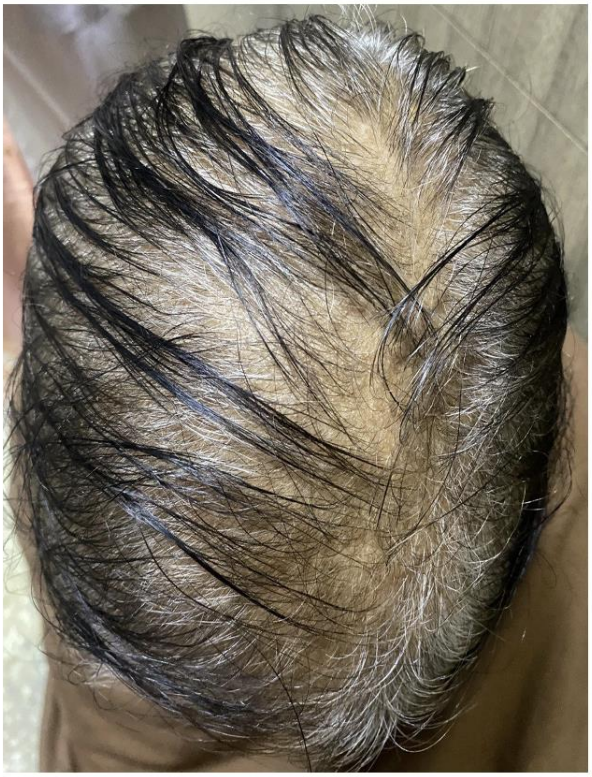


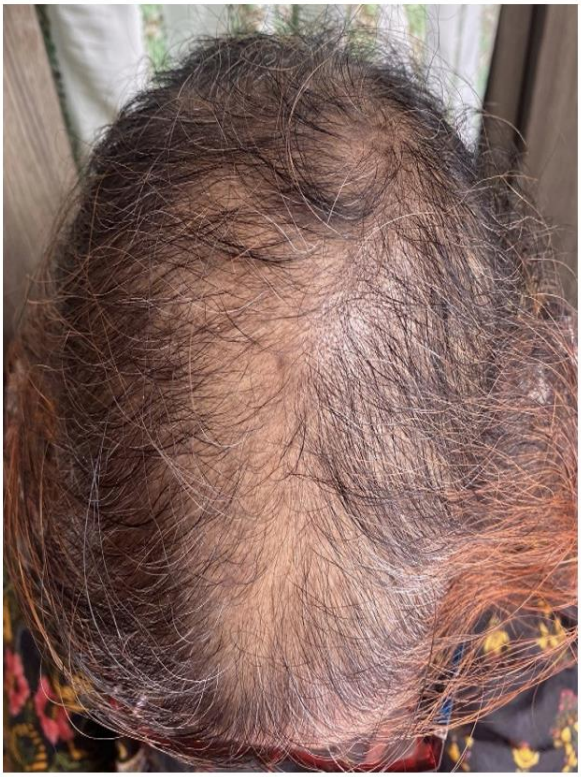


LUDWIG GRADE I

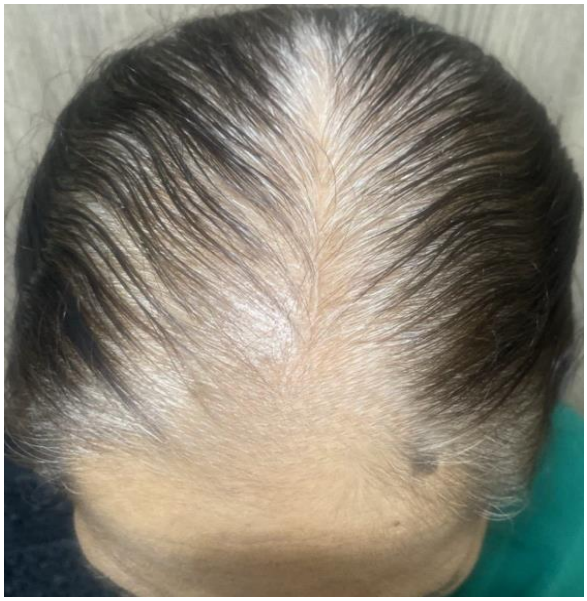





LUDWIG GRADE III

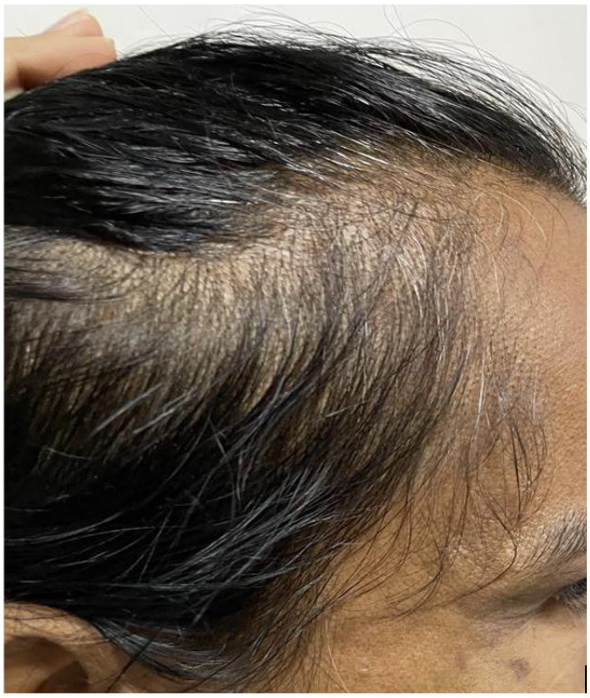




OSLEN TYPE OF BALDNESS

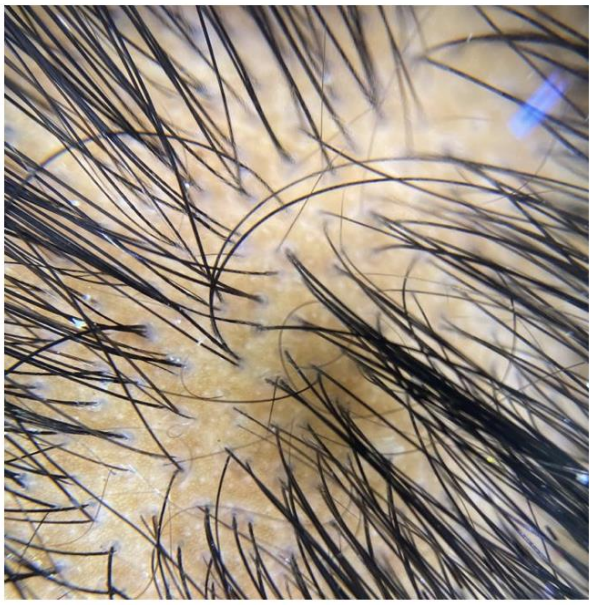


MALE PATTERN BALDNESS/NORWOOD HAMILTON PATTERN BALDNESS

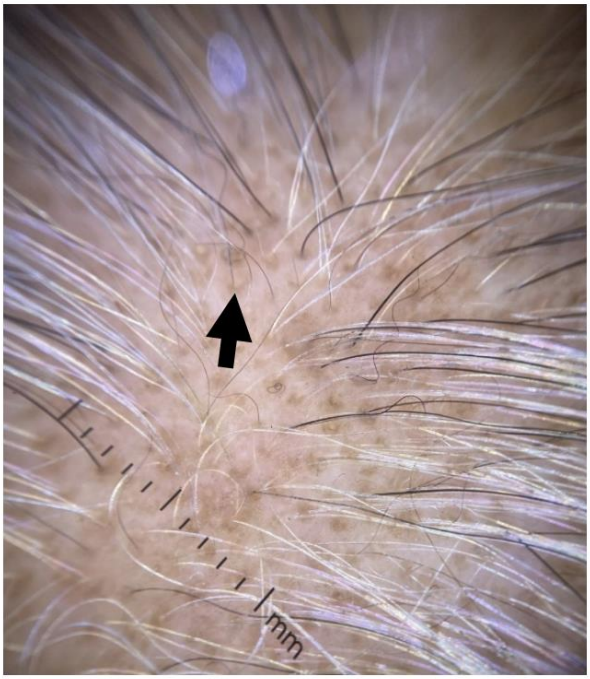


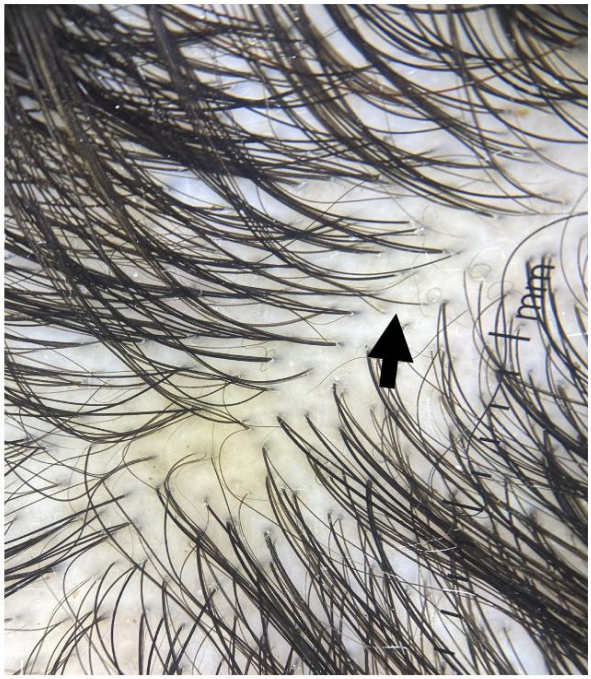
TRICHOSCOPY FINDINGS

HAIR DIAMETER DIVERSITY

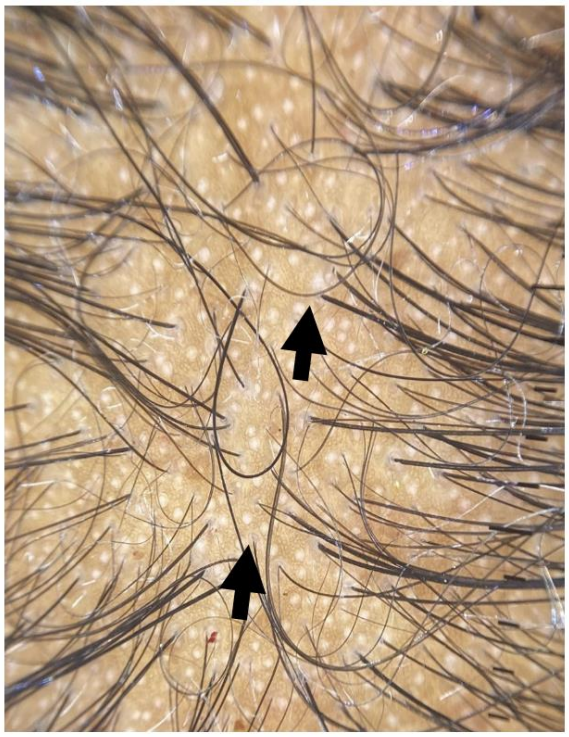


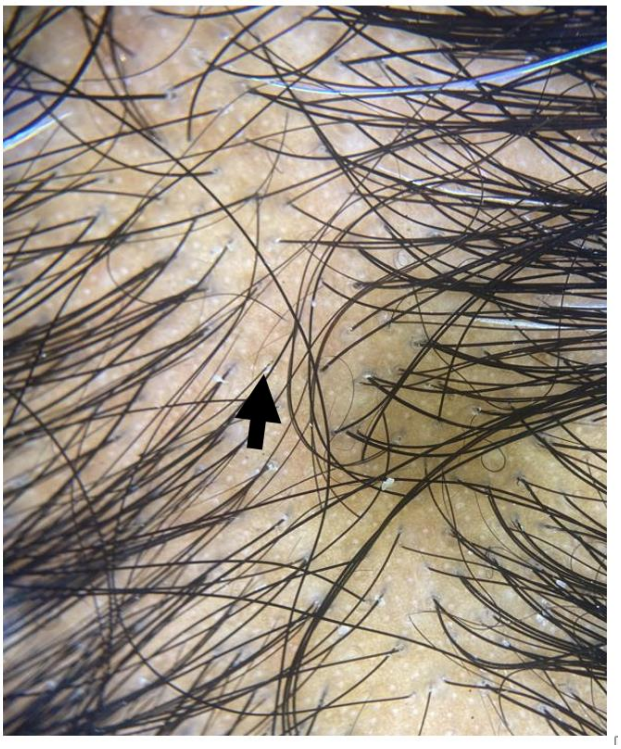
BROWN PERIPILAR SIGNS



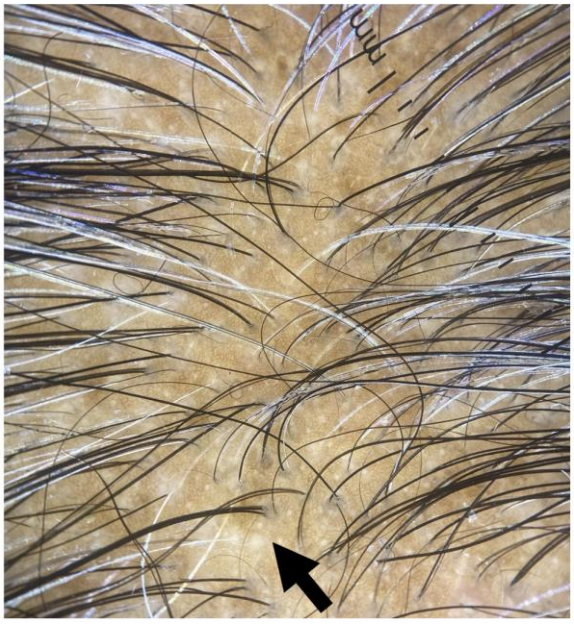


WHITE DOTS

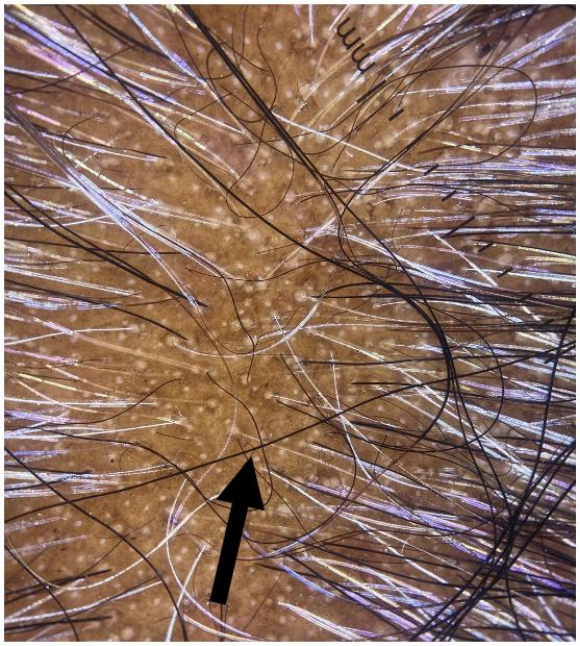


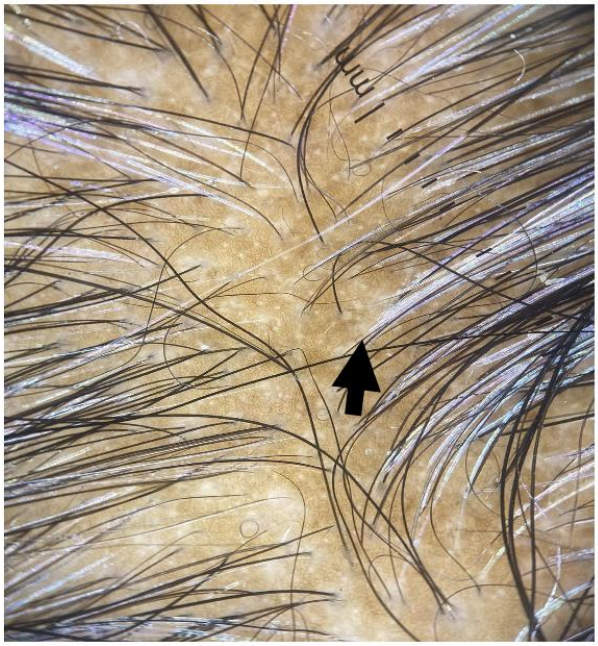


YELLOW DOTS

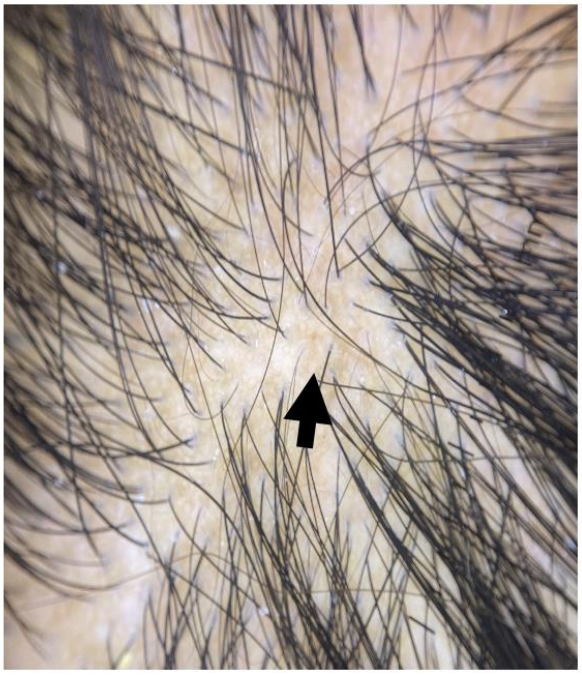


WHITE PERIPILAR SIGNS

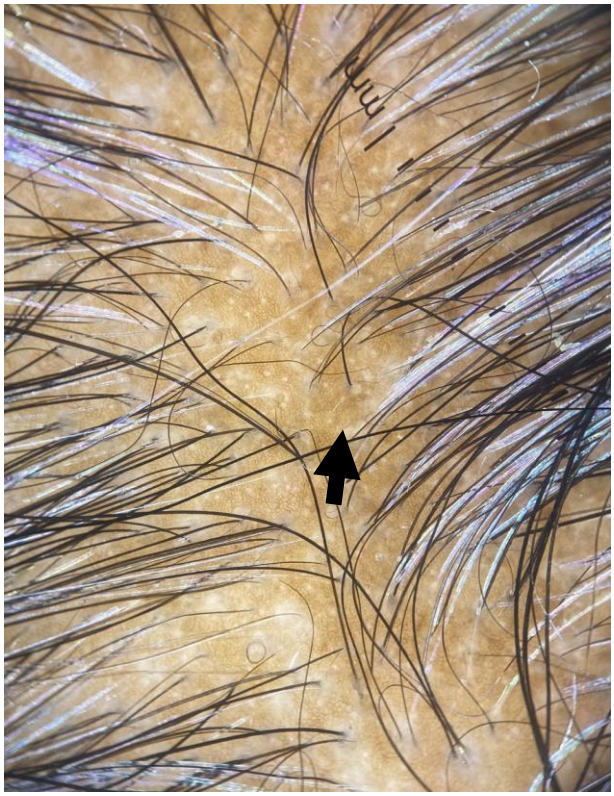




FOCAL ATRICHIA



SCALP HONEYCOMB PIGMENTATION



DISCUSSION

Discussion

Androgenic alopecia has been linked to genetic predisposition and hormonal abnormalities like hyperandrogenism, hair cycle disorders, and follicular miniaturization. The pathogenesis of patterned baldness differs between men and women. In FPHL, the function of hyper androgenemia is not entirely clear.[85] The past ten years have seen a rise in the use of trichoscopy as a diagnostic tool for hair loss disorders. However, studies investigating the features of patterned hair loss in females are few.[4] Hence this study aimed to assess and associate the proportion of clinical features, biological parameters and trichoscopy findings in patients with female pattern baldness.

Age distribution:

The present study is a cross-sectional study involving 80 women subjects where majority of them aged between 30-39yrs (40%), followed by 32.50% participants aged between 19-29 least percentage of participants are aged between >=50 years. The mean age of presentation in the cases is 33 years ± 4.5. This is in agreement with a case control study by Ramatulasi, S et al.[4] involved 70 female subjects with most of them (60%) were in the age group of 29 to 38 years, and Tandon S et al.[85] also found more than half of the patients (56.68%) were in the 28-37 year age group, and in Lee et al[86] majority of patients(40%) were in this age range , this might be because patients in the younger age group are more concerned about their appearance and mostly report for the treatment early.

Age of Onset :

In our study the majority of the patients, 42(52.5%) participants noticed the onset of hair fall before the age of 30 years and the mean age of onset was 29.65 years, which is nearly in agreement with previous study by by Zhang et al [50] among Chinese women, the mean age of onset was 29.8 years. According to previous studies Ramatulasi, S et al.[4] and Tandon, S et al.[85] the median age of onset of hair loss in females was 26.6 years old and 26.1 years respectively.According to Norwood et al.[49] the average age of onset for Caucasian women was 28.4 years. Onset of Female pattern hair loss in such younger age groups might be attributed to stress factors like the need to be competitive in the present society and lifestyle modifications.

Duration of Hair Loss

The mean duration of baldness was 5.21±3.90 years in the study population. Similar observations were made in Ramatulasi, S et al.[4] study found the mean duration of hair fall to be 4.8 ±1.8yrs. In Zhang et al.[50] and Tandon S et al.[85] the mean duration was 5.1 years and 4.49 years respectively.

Family History

Many studies have reported positive family history of baldness in patients with female androgenetic alopecia. In previous studies, Ramatulasi, S et al.[4] found family history of baldness in 56% and in Shilpashree et al.[90] it was 51% of the study population. A study by Łukasik, A et al.[77] who studied the association of family history to baldness found that 69 (62.2%) patients with FPHL had a positive family history and by Tee Wei Siah et al[72].reported 85% of patients had family history of baldness, even in our study the history of the Family

members with baldness is present in most of the study population 71.25% and hence we infer that expression of female pattern hair loss can be influenced by familial prevalence.

Clinical grading / pattern of hair loss

Majority 48.75% of the study population in our study had grade I ludwig pattern followed by 30% with Ludwig grade II, 10% had ludwig grade III, 3.75% had oslen pattern and 7.5% had male pattern baldness. This is in agreement with previous study by Zhang, et al[50] where 40%, 23.3%, 26.7%, and 10% of patients had hair loss as per Ludwig type I, II, II and male-type frontotemporal recession, respectively. Tandon, S et al.[85] study also found majority 76.6% with Ludwig pattern, 20% had Olsen pattern, and 3.3% patient had Hamilton and Norwood pattern. Ludwig pattern being the most common pattern of baldness seen in females because androgen receptor content in female frontal hair follicles is 40% lower than that in male follicles and they have 3 and 3.5 times less 5a reductase I and II levels, respectively and aromatase content in frontal hair is six times higher in women.

Mean Duration of baldness in various patterns of baldness.

The mean duration of baldness in Ludwig grade I is 3.8±1.72 years, in Ludwig grade II, it is 5.3±1.93 in Ludwig grade III it is 9.5±4.5 in oslen pattern and it is 4.3±1.23 and in male pattern it i 5.19 ±3.38. The mean difference of duration in pattern of baldness is statistically significant (P <value 0.05) and duration of baldness is positively associated with severity of baldness, which is in agreement with Zhang et al.[50] in which duration of hair loss condition among the four groups were different and was positively associated with severity of baldness we might infer that as the severity of baldness progresses with the duration increases.

Biochemical parameters

The mean DHEAS (ug/dL) is 232.24 ± 131.95 in the study population. Ranged between 50.60 to 549.50 (95% CI 203.32 to 261.15) {In ug/mL it is 2.32 ± 1.3, ranged between 0.56 to 5.49)}. The mean prolactin(ng/mL) is 23.21 ± 7.65 in our study population (Ranged between 6.6 to 42.6). The mean androstenedione (ng/mL) is 1.63 ± 0.83 in the study population. Ranged between 0.42 to 4.04 (95% CI 1.45 to 1.81). {In pmol/L it is 3.66 ± 0.8, ranged between 0.93 to 9.07). The mean free triiodothyronine fT3(pg/mL) is 3.62 ± 0.85 in the study population (Ranged between 3.4 to 13.8),all the mean values of laboratory investigations are higher when compared with the findings of Tandon, S et al.[85], in which the mean DHEAS (range in μg/mL) was 1.7 ± 0.57 (0.84–2.89), mean Androstenedione (range in pmol/L) 1.7 ± 0.99 (0.18–4.5), Prolactin (range in ng/mL) was 14.09 ± 6.79 (1.4–29.7), and mean free T3(pg/ml) was 3.70 ± 0.66 (2.64–5.37) and in Ramatulasi et al.[4] the mean DHEAS (range in μg/mL was 1.79 ± 0.6 and Prolactin (range in ng/mL) was 16.95 ± 6.22 ,however in above mentioned studies the difference between the mean values of DHEAS ,Prolactin, Androstenidione and fT3 of cases were statistically significant and higher when compared with that of controls; these findings can be attributed to the following factors DHEAS, apart from conversion to potent androgens, has a direct action on hair follicle as inhibitor of G6PD (glucose 6 phosphate dehydrogenase) there by inhibiting nucleic acid synthesis, raised prolactin levels inhibit estrogen, increasing the potency of androgen, which in turn causes FPHL and fT3 can interact with androgen metabolism at various levels affecting the hair growth and Androstenedione, which is mostly produced in the ovary and adrenal glands, is converted to testosterone by 17β-hydroxysteroid dehydrogenase this testosterone either binds to intracellular androgen receptors in the hair bulb and dermal papilla, which facilitates miniaturization of the follicle, This suggests that the se low potent adrenal androgens are sufficient to cause FPHL.

Correlation of biochemical parameters with clinical pattern of baldness

In our study 12.5% had increased DHEAS (ug/dL) 18.75% of population had increased androstenedione,8.75% population had increased fT3 and highest being 28.75 % population had increased prolactin, but no correlation was noted between the severity of FPHL and the levels of DHEAS, Prolactin, Androstenidione and fT3. All the correlation values obtained were closer to zero which indicated a weak association between the ranks. This is in agreement with previous studies Venkatta RB et al. and Zhang, et al.[50] in which correlation tests did not reveal any association between the severity of FPHL and the changes in laboratory results

However, mean DHEAS is significantly higher in the male pattern of baldness in female (P value < 0.05), which is higher level of Dihydrotestosterone (DHT) can also be found in the frontal compared with the occipital lobe, which is important proof of the participation of this enzyme in the pathogenesis of Male pattern baldness.

TRICHOSCOPY FINDINGS:

Hair shaft diameter diversity (HDD) was established by Rakowska et al [87]as one of the major criteria of FPHL. It was observed in 95% of patients with female pattern baldness; 89.7% of Ludwig I and 100% of Ludwig II and Ludwig III grade patients which is in agreement to the study done Galliker et al.[88]where HDD was seen in 72% of early and 100% of advanced female androgenetic alopecia patients and Ramatulasi, S et al.[4] It was observed in 93% of patients with FPHL had HDD and Hu R et al.[89] reported 100% of Female androgenetic Alopecia patients had HDD .Hence we can infer that hair shaft thickness heterogeneity also named “hair diameter diversity” is observed in the affected scalp region of all AGA patients, which represents progressive and unsynchronized miniaturization of hair follicles which is the main pathogenesis involved in female androgenic alopecia. Hence the diversity in hair diameter is

considered as the main and most accurate clinical parameter in diagnosing androgentic alopecia

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Brown peripilar sign (BPPS) is seen in 58.75% of our patients

Which is comparable to Ramatulasi, S et al.[4] 45% and Ummiti A et al.[90] 40% and Hu et al.[89] 44.5% patients presented BPPS. In our study the difference in the proportion of brown peri pilar signs (BPPS) between pattern of baldness is statistically significant with P value <0.001 with majority of (30/39)(76.44%) participants with Ludwig grade 1 had brown peri pilar signs (BPPS).Which is in agreement with by Zhang et al[50] and Ramatulasi, S et al.[4] which observed BPPS in early AGA grades. The presence of BPPS in the early stages of baldness is linked to superficial perifollicular infiltration which is more common at the early stage of alopecia. The higher percentage of cases with BPPS in our study compared to other studies can be attributed to the higher percentage of cases presenting with Ludwig I in our study. In contrast, Deloche et al.[56] reported BPPS was found in 86% of caucasian women with FPHL This may be owing to the concealment of brown halo over the scalp by the skin color of Asian subjects .

White peripilar sign (WPPS) is noticed in 28.7% of patients in the present study. In Pattern of baldness with Ludwig grade I, Ludwig grade II, Ludwig grade III, OSLEN and male pattern, 7.69%, 37.50%, 87.5%, 66.67% and 33.33% females had white peri pilar signs (WPPS) . with majority of (7/8)(87.5%) participants with Ludwig grade IIII had white peri pilar signs (BPPS). Which is in near agreement with by Zhang et al.[50] in which 26.7% of FAGA patients with advanced stages had WPPS. We suppose that sign is related to perifollicular fibrosis in the later stage of FHPL, hence it is commonly found in the later stages of baldness, Hence we can consider WPPS to be a poor prognostic factor.

In our study 25.00% of patients had **White dots (WD)** which is in agreement with Hu et al.[89] in which 22.0% of female AGA patients, Zhang et al.[50] in which 21.7% of patients had White dots. The difference in the proportion of white dots between pattern of baldness was statistically not significant with P value 0.1372. It has been revealed to represent the empty hair follicle ostia or to the epidermal portion of eccrine sweat ducts. But it has also been discovered that white dots can also found in healthy patients of Fitzpatrick skin phototypes IV– VI as they are visible on the contrasting background of the pigmented network, hence we can infer that it cannot be considered to be a specific sign for severe grade of baldness.

Yellow dots (YD) were seen in 46/80 (56.7%) of patients present in all grades of baldness similar to Ramatulasi, S et al.[4] where it was seen in 40 out of 70 pts (57%) . In Contrast its incidence was high in Ummiti A et al.[90]with 88% of patients having yellow dots and very low in Zhang et al.[50] study(1.67%). In Hu et al study this finding was seen in 24% of FAGA patients. This disparity of findings can be because of different skin phenotypes with variation in sebaceous gland activity.

Focal atrichia referred to as pencil erased focal loss of hair is found in 37.55% in patients in our study and showed a positive correlation with disease severity, similar to the study by Ummiti A et al.[90] (24%) with positive correlation with severity of baldness Hu *et al*.[89] who observed it in 30.5% of FAGA patients with late stages and also in a study by Zhang et al [50]who observed, 56.7% in FAGA patients which correlated with advancing stage of AGA.

In agreement with Zhang et al[50] a similar observation was made regarding a difference in the miniaturization process in Ludwig type hair loss and male-type frontotemporal recession. In advanced stages of FPHL, even though some pilosebaceous unit ended up in complete atrophy manifesting as atrichia, most of them had at least one residual terminal hair fiber; in contrast,

in male type frontotemporal recession, most of hair fibers had vellus transformation and no focal atrichia was noted.[50]

Scalp honeycomb pigmentation is found in 58.7% female AGA in our study and was positively correlated to severity of baldness similar to Zhang et al.[50] (61.7%) where it was positively correlated to severity of baldness whereas in a Chinese study by Hu *et al*.[89] SHCP was seen in 30.5% of female patients in late stages of AGA. This is may be because scalp honeycomb pigmentation is due to sun exposure, formed by hypomelanotic areas (less in overlying dermal papillae) bordered by hyperchromic lines (melanin of rete ridges), usually seen in thinning or completely balding areas .[89]

Conclusion:

- Female Androgenetic alopecia is a multifactorial disease which occurs due to interactions between various genetic and environmental factors.
- The present study shows a weak positive correlation between severity of baldness and DHEAS, androstenedione and prolactin and fT3 hormones but abnormal values of hormones were seen in some patients hence patients who present with FPHL should be advised a hormonal evaluation as adrenal androgens can be raised in some patients
- But it shows positive correlation of presence of BPPS with early stages of AGA and WPPS and Focal atrichia with late stages, which may aid in diagnosing AGA at early stages and starting of treatment early prevents the poor prognosis of the condition in patients.

Summary

- Female pattern hair loss (FPHL) is a type of diffuse, non-scarring hair loss and pattern hair thinning. It is unclear how hyperandrogenemia plays a part in FPHL. It is also disputed whether iron deficiency and FPHL are related. There isn't much literature examining the hormonal and biochemical features of this condition. Hence the study aimed to assess the biochemical parameters in the three types of FPHL, namely Ludwig, Olsen, Hamilton, and Norwood, and to correlate the clinico-laboratory findings in women with pattern baldness.
- Female AGA cases attending the Department of Dermatology at R.L Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar from January 2021 to July 2022 were identified and a total of 80 cases satisfying the inclusion criteria were included in the study.
- A detailed history was taken, thorough examination was done and all the findings were documented as per proforma.
- A cross-sectional study involving 80 women subjects, majority 40% participants were aged between 30-39 years.
- 52.5% participants had 19-29 years age of onset group.
- The mean duration(years) is 5.21±3.90 in the study population ranged between 0.16 to 10 .
- Majority 48.75% participants had Ludwig I, 30% had ludwig II, 10% had ludwig III, 3.75% had oslen pattern and 7.5% had male pattern baldness .
- History of one of the Family members with baldness was present in 71.25population.
- Among the study population 58.75% participants had Brown Peri pilar signs (BPPS).
- 28.7% participants had White peri pilar signs (WPPS).

- 25% participants had white dots, 57.5% participants had yellow dots, 37.5% participants had focal atrichia, 95% participants had hair shaft thickness heterogeneity (HSTH) and 57.5% participants had Scalp Honeycomb pigmentation.
- Abnormal levels of DHEAS (ug/dL) , prolactin and androstenedione was found in 12.5%, 28.75% and 18.75% respectively with majority of subjects having normal levels.
- In Pattern of baldness with Ludwig grade 1, Ludwig grade 2, Ludwig grade 3, OSLEN pattern and male pattern the majority of 89.74%, 87.5%, 87.5%, 100.00% and 66.67% females had in normal DHEAS(ug/dL).The difference in the proportion of prolactin(ng/mL) group between pattern of baldness was statistically not significant with P value 0.8698 with majority of 76.92% participants were in Ludwig grade 1 had normal prolactin(ng/mL) and 23.08% had abnormal prolactin(ng/mL).A weak positive correlation between Pattern of baldness and DHEAS (ug/dL), androstenedione and prolactin (r_s value: 0.02,0.09 and 0.17, P value: 0.8951).

Limitations and recommendation

- The gap in the current study is the absence of a case control design and an institution-based cross- sectional study.
- A population-based descriptive study aids in pinpointing the precise prevalence and other FPHL risk factors.

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ANNEXURES

ASSOCIATION OF CLINICAL FEATURES, BIOLOGICAL PARAMETERS AND TRICHOSCOPIC PATTERNS IN FEMALE PATTERN BALDNESS-A CROSS SECTIONAL STUDY

PROFORMA

Name:	**Op/Ip Number:**
Age& Gender:	**Date:**
Address:	**Occupation:**

CHIEF COMPLAINTS:

Duration:

Baldness History in Family:

Type Of Hair Loss Pattern:

TRICHOSCOPY FINDINGS:

1.Increased frontal to occipital single hair ratio: PRESENT/ABSENT

2.Hair shaft thickness: P / A

3.Brown Peripilar signs : P / A

4.White Dots: P/ A

5.White Peripilar signs :P / A

6.Yellow Dots: P / A

7.Focal Atrichia: P/ A

8.Honeycomb Pigmentation: P / A

LABORATORY INVESTIGATIONS:

1.DHEAS (ug/dL):

2.PROLACTIN (ng/ml):

3.ANDROSTENEDIONE (ng/ml):

4. FREE TRIIODOTHYRONINE (pg/ml):

CONSENT FORM

Study title: ASSOCIATION OF CLINICAL FEATURES, BIOLOGICAL PARAMETERS AND TRICHOSCOPIC PATTERNS IN FEMALE PATTERN BALDNESS-A CROSS SECTIONAL STUDY.

Chief researcher/ PG guide's name: DR.E.MEGHANA REDDY

Under the guidance of: DR. RAJASHEKAR T.S

Name of the subject:

Age :

Address :

I have been informed in my own vernacular language the purpose of the study, the necessity of relevant investigations to be carried out and photographs to be taken.

a. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.

b. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.

c. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

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

Participant's signature

Signature of the witness: Date:

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

Chief Researcher/ Guide signature Date:

PATIENT INFORMATION SHEET

Study title: ASSOCIATION OF CLINICAL FEATURES, BIOLOGICAL PARAMETERS AND TRICHOSCOPIC PATTERNS IN FEMALE PATTERN BALDNESS -A CROSS SECTIONAL STUDY.

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

Aim: To correlate the clinical features, biological parameters and trichoscopic findings in patients with female pattern baldness

Androgenetic alopecia (AGA) is a nodon scarring alopecia and is the most common cause of hair loss in females, known as female pattern hair loss (FPHL), diffuse, which affects around 40% of women in the world. FPHL is largely attributed to hormonal imbalance and studies showed that hyperprolactinemia along with other hormonal imbalances like increased levels of DHEA-S, epiandrostenedione and FT3H levels are seen in FPHL. Scalp dermoscopy or trichoscopy is one of the noninvasive techniques for the evaluation of patients with hair loss that allows for magnified visualization of the hair and scalp skin, and helps in early diagnosis of the condition.So, in this study we aim to correlate the clinical features, biological parameters and trichoscopic patterns which aids in better understanding and diagnosis, treatment and follow up of females with FPHL.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study A detailed history of the patient including name, age, sex, history of presenting illness, hair grooming pattern, habits & tics, nail changes, other skin changes, systemic disease, family history of similar complaints and drug intake will be taken. We will collect information (as per proforma) from you. A written informed consent will be taken from the patients and in children from parents or guardian. General physical examination of patient is done, clinical examination of scalp, tests for hair anchorage and fragility will be done in all cases. Hormone analysis of Dehydroepiandrosterone-sulfate (DHEA-S), prolactin, androstenedione, and free triiodothyronine (FT3) are done through blood tests .Under aseptic conditions 2ml of blood is collected from the vein of the hand and immediately sent to laboratory for analysis and there are no side effects associated with blood drawing , all the guidelines of Central diagnostic laboratory services (CDLS) of R.L Jalappa Hospital are followed in discarding the extra samples if collected. The results thus obtained are documented. Trichoscopy is an non invasive procedure, a target area of 2x2 cm is selected in the active alopecia site of the participants of the study ,structures are visualized by dermatoscope DL4N with 10x magnification using polarized and non polarized mode. The structures which are visualized include hair shafts, hair follicle openings, the perifollicular epidermis and cutaneous microvasculature , trichoscopic pictures of your scalp will be taken and trichoscopic findings of that target area will be assessed and there are no adverse effects associated with trichoscopy procedure as it is non-invasive procedure.This information collected will be used for dissertation and publication only. In case of any untoward event, it will be managed by Department of Dermatology R.L Jalappa Hospital.

The cost for the required blood investigations and any side effects will be borne by the primary investigator (concerned post graduate).

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

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

For any further clarification you can contact the study investigator:

Dr. E.Meghana Reddy

Mobile no: 9663479180

E-mail id: send2meghana05.mr@gmail.com

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ:

ಕ್ಲಿನಿಕಲ್ ವೈಶಿಷ್ಟ್ಯಗಳು, ಜೈವಿಕ ಪ್ಯಾರಾಮೀಟರ್‌ಗಳು ಮತ್ತು ಟ್ರೈಕೊಸ್ಕೋಪಿಕ್ ಪ್ಯಾಟರ್ನ್‌ಗಳ ಸಂಯೋಜನೆ ಸ್ತ್ರೀ ಪ್ಯಾಟರ್ನ್ ಬಾಲ್ಡ್ನೆಸ್-ಎ ಕ್ರಾಸ್ ಸೆಕ್ಷನಲ್ ಸ್ಟಡಿ.

ಮುಖ್ಯ ಸಂಶೋಧಕ / ಪಿಜಿ ಮಾರ್ಗದರ್ಶಿ ಹೆಸರು: ಡಿ.ಆರ್.ಇ.ಮೆಘಾನಾ ರೆಡ್ಡಿ

ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ: ಡಿ.ಆರ್. ರಾಜಶೇಕರ್ ಟಿ.ಎಸ್

ವಿಷಯದ ಹೆಸರು:

ವಯಸ್ಸು:

ವಿಳಾಸ:

ಎ. ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಸಂಬಂಧಿತ ತನಿಖೆಗಳ ಅವಶ್ಯಕತೆ ಮತ್ತು ತೆಗೆದುಕೊಳ್ಳಬೇಕಾದ s ೀಯಾಚಿತ್ರಗಳನ್ನು ನನ್ನ ಸ್ವಂತ ಭಾಷೆಯಲ್ಲಿ ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ.

ಬೌ. ಈ ಅಧ್ಯಯನದಿಂದ ಉತ್ಪತ್ತಿಯಾಗುವ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯು ಸಾಂಸ್ಥಿಕ ದಾಖಲೆಯ ಭಾಗವಾಗಲಿದೆ ಮತ್ತು ಈ ಸಂಸ್ಥೆಯು ಗೌಪ್ಯವಾಗಿಡುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಸಿ. ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ ಮತ್ತು ಭಾಗವಹಿಸಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು ಮತ್ತು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ನನ್ನ ಪುಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಗೆ ಯಾವುದೇ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಭಾಗವಹಿಸುವುದನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಡಿ. ಈ ಅಧ್ಯಯನವು ಉದ್ಭವಿಸುವ ಯಾವುದೇ ಡೇಟಾ ಅಥವಾ ಫಲಿತಾಂಶಗಳ ಬಳಕೆಯನ್ನು ನಿರ್ಬಂಧಿಸದಿರಲು ನಾನು ಒಪ್ಪುತ್ತೇನೆ, ಅಂತಹ ಬಳಕೆಯು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ.

ಇ. _____ (ಪಿಜಿ ಮಾರ್ಗದರ್ಶಿಯ ಮುಖ್ಯ ಸಂಶೋಧಕ / ಹೆಸರು) ನನಗೆ ಸಂಶೋಧನೆಯ ಉದ್ದೇಶ ಮತ್ತು ನಾನು ಅನುಭವಿಸಲಿರುವ ಅಧ್ಯಯನ ವಿಧಾನ ಮತ್ತು ನನ್ನ ಸ್ವಂತ ಭಾಷೆಯಲ್ಲಿ ನಾನು ಅನುಭವಿಸಬಹುದಾದ ಸಂಭವನೀಯ ಅಪಾಯಗಳು ಮತ್ತು ಅಸ್ವಸ್ಥತೆಗಳನ್ನು ವಿವರಿಸಿದ್ದೇನೆ ಎಂದು ನಾನು ಖಚಿತಪಡಿಸುತ್ತೇನೆ. ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯಲ್ಲಿ ವಿಷಯವಾಗಿ ಭಾಗವಹಿಸಲು ಮಾನ್ಯ ಒಪ್ಪಿಗೆ ನೀಡಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಸಹಿ

ಸಾಕ್ಷಿಯ ಸಹಿ: ದಿನಾಂಕ:

ನಾನು _____ (ವಿಷಯ) ಗೆ ಸಂಶೋಧನೆಯ ಉದ್ದೇಶ, ಸಂಭವನೀಯ ಅಪಾಯ ಮತ್ತು ನನ್ನ ಸಾಮರ್ಥ್ಯಕ್ಕೆ ಉತ್ತಮವಾದ ಪ್ರಯೋಜನಗಳನ್ನು ವಿವರಿಸಿದ್ದೇನೆ.

ಮುಖ್ಯ ಸಂಶೋಧಕ / ಮಾರ್ಗದರ್ಶಿ ಸಹಿ ದಿನಾಂಕ:

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಕ್ಲಿನಿಕಲ್ ವೈಶಿಷ್ಟ್ಯಗಳ ಸಂಘ, ಜೈವಿಕ ಪ್ಯಾರಾಮೀಟರ್‌ಗಳು ಮತ್ತು ಟ್ರೈಕೊಸ್ಕೋಪಿಕ್ ಪ್ಯಾಟರ್ನ್‌ಗಳು ಸ್ತ್ರೀ ಪ್ಯಾಟರ್ನ್ ಬಾಲ್ಡ್‌ನೆಸ್ -ಒ ಕ್ರಾಸ್ ಸೆಕ್ಷನಲ್ ಸ್ಟಡಿ.

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್.ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆ, ತಮಾಕಾ, ಕೋಲಾರ.

ಗುರಿ: ಸ್ತ್ರೀ ಮಾದರಿಯ ಬೋಳು ರೋಗಿಗಳಲ್ಲಿ ಕ್ಲಿನಿಕಲ್ ಲಕ್ಷಣಗಳು, ಜೈವಿಕ ನಿಯತಾಂಕಗಳು ಮತ್ತು ಟ್ರೈಕೊಸ್ಕೋಪಿಕ್ ಸಂಶೋಧನೆಗಳನ್ನು ಪರಸ್ಪರ ಸಂಬಂಧಿಸುವುದು

ಆಂಡ್ರೊಜೆನೆಟಿಕ್ ಅಲೋಪೆಸಿಯಾ (ಎಜಿಎ) ಒಂದು ನೋಡಾನ್ ಗುರುತು ಅಲೋಪೆಸಿಯಾ ಮತ್ತು ಸ್ತ್ರೀಯರಲ್ಲಿ ಕೂದಲು ಉದುರುವಿಕೆಗೆ ಸಾಮಾನ್ಯ ಕಾರಣವಾಗಿದೆ, ಇದನ್ನು ಸ್ತ್ರೀ ಮಾದರಿಯ ಕೂದಲು ಉದುರುವಿಕೆ (ಎಫ್‌ಪಿಹೆಚ್‌ಎಲ್), ಪ್ರಸರಣ ಎಂದು ಕರೆಯಲಾಗುತ್ತದೆ, ಇದು ವಿಶ್ವದ ಸುಮಾರು 40% ಮಹಿಳೆಯರ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುತ್ತದೆ. ಎಫ್‌ಪಿಹೆಚ್‌ಎಲ್ ಹೆಚ್ಚಾಗಿ ಹಾರ್ಮೋನುಗಳ ಅಸಮತೋಲನಕ್ಕೆ ಕಾರಣವಾಗಿದೆ ಮತ್ತು ಅಧ್ಯಯನಗಳು ಹೈಪರ್‌ಪ್ರೊಲ್ಯಾಕ್ಟಿನೆಮಿಯಾ ಜೊತೆಗೆ ಇತರ ಹಾರ್ಮೋನುಗಳ ಅಸಮತೋಲನಗಳಂತಹ ಹೆಚ್ಚಿದ ಡಿಹೆಚ್‌ಇಎ-ಎಸ್, ಎಪಿಯಾಂಡ್ರೊಸ್ಟೆಡಿನಿಯೋನ್ ಮತ್ತು ಎಫ್‌ಟಿ 3 ಎಚ್ ಮಟ್ಟಗಳು ಎಫ್‌ಪಿಹೆಚ್‌ಎಲ್‌ನಲ್ಲಿ ಕಂಡುಬರುತ್ತವೆ ಎಂದು ತೋರಿಸಿದೆ. ಕೂದಲು ಉದುರುವಿಕೆ ರೋಗಿಗಳ ಮೌಲ್ಯಮಾಪನಕ್ಕಾಗಿ ನೆತ್ತಿಯ ಡರ್ಮೋಸ್ಕೋಪಿ ಅಥವಾ ಟ್ರೈಕೊಸ್ಕೋಪಿ ಒಂದು ಅನಾನುಕೂಲ ತಂತ್ರಗಳಲ್ಲಿ ಒಂದಾಗಿದೆ, ಇದು ಕೂದಲು ಮತ್ತು ನೆತ್ತಿಯ ಚರ್ಮದ ವರ್ಧಿತ ದೃಶ್ಯೀಕರಣಕ್ಕೆ ಅನುವು ಮಾಡಿಕೊಡುತ್ತದೆ, ಮತ್ತು ಸ್ಥಿತಿಯ ಆರಂಭಿಕ ರೋಗನಿರ್ಣಯಕ್ಕೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ.ಆದ್ದರಿಂದ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಾವು ಕ್ಲಿನಿಕಲ್ ಅನ್ನು ಪರಸ್ಪರ ಸಂಬಂಧಿಸುವ ಗುರಿ ಹೊಂದಿದ್ದೇವೆ ವೈಶಿಷ್ಟ್ಯಗಳು, ಜೈವಿಕ ನಿಯತಾಂಕಗಳು ಮತ್ತು ಟ್ರೈಕೊಸ್ಕೋಪಿಕ್ ಮಾದರಿಗಳು ಎಫ್‌ಪಿಎಚ್‌ಎಲ್‌ನೊಂದಿಗೆ ಉತ್ತಮ ತಿಳುವಳಿಕೆ ಮತ್ತು ರೋಗನಿರ್ಣಯ, ಚಿಕಿತ್ಸೆ ಮತ್ತು ಹೆಣ್ಣುಮಕ್ಕಳ ಅನುಸರಣೆಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ.

ದಯವಿಟ್ಟು ಈ ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ರೋಗಿಯ ಹೆಸರು, ವಯಸ್ಸು, ಲಿಂಗ, ಅನಾರೋಗ್ಯವನ್ನು ಪ್ರಸ್ತುತಪಡಿಸಿದ ಇತಿಹಾಸ, ಕೂದಲಿನ ಅಂದಗೊಳಿಸುವ ಮಾದರಿ, ಅಭ್ಯಾಸಗಳು ಮತ್ತು ಸಂಕೋಚನಗಳು, ಉಗುರು ಬದಲಾವಣೆಗಳು, ಇತರ ಚರ್ಮದ ಬದಲಾವಣೆಗಳು, ವ್ಯವಸ್ಥಿತ ಕಾಯಿಲೆ, ಇದೇ ರೀತಿಯ ದೂರುಗಳ ಕುಟುಂಬದ ಇತಿಹಾಸ ಮತ್ತು drug ಷಧಿ ಸೇವನೆಯನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ನಾವು ನಿಮ್ಮಿಂದ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ (ಪ್ರೊಫಾರ್ಮಾದ ಪ್ರಕಾರ). ರೋಗಿಗಳಿಂದ ಮತ್ತು ಮಕ್ಕಳಲ್ಲಿ ಪೋಷಕರು ಅಥವಾ ಪೋಷಕರಿಂದ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಸಮ್ಮತಿಯನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ. ರೋಗಿಯ ಸಾಮಾನ್ಯ ದೈಹಿಕ ಪರೀಕ್ಷೆಯನ್ನು ಮಾಡಲಾಗುತ್ತದೆ, ನೆತ್ತಿಯ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ, ಕೂದಲು ಆಂಕಾರೇಜ್ ಮತ್ತು ಸೂಕ್ಷ್ಮತೆಯ ಪರೀಕ್ಷೆಗಳನ್ನು ಎಲ್ಲಾ ಸಂದರ್ಭಗಳಲ್ಲಿ ಮಾಡಲಾಗುತ್ತದೆ. ರಕ್ತ ಪರೀಕ್ಷೆಗಳ ಮೂಲಕ ಡಿಹೈಡ್ರೊಪಿಯಾಂಡ್ರೊಸ್ಟರಾನ್-ಸಲ್ಫೇಟ್ (ಡಿಹೆಚ್‌ಇಎ-ಎಸ್), ಪ್ರೊಲ್ಯಾಕ್ಟಿನ್, ಆಂಡ್ರೊಸ್ಟೆಡಿನಿಯೋನ್ ಮತ್ತು ಉಚಿತ ಟ್ರೈಯೋಡೋಥೈರೋನೈನ್ (ಎಫ್‌ಟಿ 3) ಗಳ ಹಾರ್ಮೋನ್ ವಿಶ್ಲೇಷಣೆಯನ್ನು ಮಾಡಲಾಗುತ್ತದೆ .ಅಂಡೆಪ್ಟಿಕ್ ಪರಿಸ್ಥಿತಿಗಳ ನಂತರ 2 ಮಿಲಿ ರಕ್ತವನ್ನು ಕೈಯ ರಕ್ತನಾಳದಿಂದ ಸಂಗ್ರಹಿಸಿ ತಕ್ಷಣವೇ ವಿಶ್ಲೇಷಣೆಗಾಗಿ ಪ್ರಯೋಗಾಲಯಕ್ಕೆ ಕಳುಹಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ರಕ್ತದ ರೇಖಾಚಿತ್ರಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಯಾವುದೇ ಅಡ್ಡಪರಿಣಾಮಗಳಿಲ್ಲ, ಆರ್ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆಯ ಕೇಂದ್ರ ರೋಗನಿರ್ಣಯ ಪ್ರಯೋಗಾಲಯ ಸೇವೆಗಳ (ಸಿಡಿಎಲ್‌ಎಸ್) ಎಲ್ಲಾ ಮಾರ್ಗಸೂಚಿಗಳನ್ನು ಸಂಗ್ರಹಿಸಿದರೆ ಹೆಚ್ಚುವರಿ ಮಾದರಿಗಳನ್ನು ತ್ಯಜಿಸುವಲ್ಲಿ ಅನುಸರಿಸಲಾಗುತ್ತದೆ. ಹೀಗೆ ಪಡೆದ ಫಲಿತಾಂಶಗಳನ್ನು ದಾಖಲಿಸಲಾಗಿದೆ. ಟ್ರೈಕೊಸ್ಕೋಪಿ ಆಕ್ರಮಣಶೀಲವಲ್ಲದ ಕಾರ್ಯವಿಧಾನವಾಗಿದೆ, ಅಧ್ಯಯನದ ಭಾಗವಹಿಸುವವರ ಸಕ್ರಿಯ ಅಲೋಪೆಸಿಯಾ ತಾಣದಲ್ಲಿ 2x2 ಸೆಂ.ಮೀ.ನ ಗುರಿ ಪ್ರದೇಶವನ್ನು ಆಯ್ಕೆ ಮಾಡಲಾಗಿದೆ,

ರಚನೆಗಳನ್ನು ಡರ್ಮಟೊಸ್ಕೋಪ್ ಡಿಎಲ್ 4 ಎನ್ ಮೂಲಕ 10x ವರ್ಧನೆಯೊಂದಿಗೆ ಧ್ರುವೀಕರಿಸಿದ ಮತ್ತು ಧ್ರುವೀಕರಿಸದ ಮೋಡ್ ಬಳಸಿ ದೃಶ್ಯೀಕರಿಸಲಾಗುತ್ತದೆ. ದೃಶ್ಯೀಕರಿಸಿದ ರಚನೆಗಳಲ್ಲಿ ಹೇರ್ ಶಾಫ್ಟ್‌ಗಳು, ಕೂದಲಿನ ಕೋಶಕ ತೆರೆಯುವಿಕೆಗಳು, ಪೆರಿಫೋಲಿಕ್ಯುಲರ್ ಎಪಿಡರ್ಮಿಸ್ ಮತ್ತು ಕಟಾನಿಯಸ್ ಮೈಕ್ರೊವಾಸ್ಕುಲೇಚರ್, ನಿಮ್ಮ ನೆತ್ತಿಯ ಟ್ರೈಕೊಸ್ಕೋಪಿಕ್ ಚಿತ್ರಗಳನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ ಮತ್ತು ಆ ಗುರಿ ಪ್ರದೇಶದ ಟ್ರೈಕೊಸ್ಕೋಪಿಕ್ ಆವಿಷ್ಕಾರಗಳನ್ನು ನಿರ್ಣಯಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಟ್ರೈಕೊಸ್ಕೋಪಿ ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಯಾವುದೇ ಪ್ರತಿಕೂಲ ಪರಿಣಾಮಗಳಿಲ್ಲ ಆಕ್ರಮಣಶೀಲವಲ್ಲದ ಕಾರ್ಯವಿಧಾನವಾಗಿದೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಯಾವುದೇ ಅಹಿತಕರ ಘಟನೆಗಳಿದ್ದಲ್ಲಿ, ಇದನ್ನು ಚರ್ಮರೋಗ ವಿಭಾಗದ ಆರ್.ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆಯು ನಿರ್ವಹಿಸುತ್ತದೆ.

ಅಗತ್ಯವಾದ ರಕ್ತ ತನಿಖೆ ಮತ್ತು ಯಾವುದೇ ಅಡ್ಡಪರಿಣಾಮಗಳ ವೆಚ್ಚವನ್ನು ಪ್ರಾಥಮಿಕ ತನಿಖಾಧಿಕಾರಿ (ಸಂಬಂಧಪಟ್ಟ ಸ್ನಾತಕೋತ್ತರ) ಭರಿಸುತ್ತಾರೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತು ಬಹಿರಂಗಗೊಳ್ಳುವುದಿಲ್ಲ. ಮೇಲಿನ ತನಿಖೆಗೆ ಅಗತ್ಯವಾದ ಖರ್ಚುಗಳನ್ನು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಗಳು ನೀಡುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿ ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ ಸಹಿ / ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

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