MEASURING THE EPILATION FORCE OVER THE FRONTAL AND OCCIPITAL REGION IN CASES OF FEMALE PATTERN HAIR LOSS USING HAND HELD TRICHOTILOMETER

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In fulfilment of the regulations for the award of the degree

M.D.

DERMATOLOGY, VENEREOLOGY AND LEPROLOGY

DEPARTMENT OF DERMATOLOGY, VENEROLOGY AND LEPROLOGY

PSG INSTITUTE OF MEDICAL SCIENCE AND RESEARCH
THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

APRIL 2017

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CERTIFICATE

This is certify that the thesis entitled **"MEASURING THE EPILATION FORCE OVER THE FRONTAL AND OCCIPITAL REGION IN CASES OF FEMALE PATTERN HAIR LOSS USING HAND HELD TRICHOTILOMETER"** is a bonafide work of **Dr. Subha Ranjani K.** done under the direct guidance and supervision of **Dr. C.R. SRINIVAS, MD,** in the department of Dermatology, Venereology and Leprology, PSG Institute of Medical Sciences and Research, Coimbatore in fulfillment of the regulations of Dr.MGR Medical University for the award of MD degree in Dermatology, Venereology and Leprology.

Dr. C. R. SRINIVAS
Professor & Head of Dept.
Dept. of DVL

Dr.RAMALINGAM
DEAN

DECLARATION

I hereby declare that this dissertation entitled **"MEASURING THE EPILATION FORCE OVER THE FRONTAL AND OCCIPITAL REGION IN CASES OF FEMALE PATTERN HAIR LOSS USING HAND HELD TRICHOTILOMETER"** was prepared by me under the direct guidance and supervision of **Dr. C.R. SRINIVAS, MD,** PSG Institute of Medical Sciences and Research, Coimbatore.

The dissertation is submitted to the Tamilnadu Dr.MGR Medical University in fulfillment of the University regulation for the award of MD degree in Dermatology, Venereology and Leprology. This dissertation has not been submitted for the award of any other Degree or Diploma.

Dr. SUBHA RANJANI K.

CERTIFICATE BY THE GUIDE

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Dr. C.R. SRINIVAS
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Institutional Human Ethics Committee

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To
Dr K Subha Ranjani
Postgraduate
Department of Dermatology
PSG IMS & R
Coimbatore

Ref: Project No. 14/415

Date: April 9, 2015

Dear Dr Subha Ranjani,

Institutional Human Ethics Committee, PSG IMS&R reviewed and discussed your application dated 06.12.2014 to conduct the research study entitled "*Measuring the epilation force over the frontal and occipital region in cases of female pattern hair loss using hand held trichotillometer*" during the IHEC review held on 23.01.2015.

The following documents were reviewed and approved:

1. Project Submission form
2. Study protocol
3. Informed consent form
4. Data collection tool
5. Current CVs of Principal investigator, Co-investigator
6. Budget

The following members of the Institutional Human Ethics Committee (IHEC) were present at the meeting held on 23.01.2015 at College Council Room, PSG IMS & R between 2.00 pm am and 4.30 pm:

Sl. No.	Name of the Member of IHEC	Qualification	Area of Expertise	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
1	Mrs Y Ashraf	MPT	Physiotherapy	Female	Yes	Yes
2	Dr. S. Bhuvaneshwari (Member-Secretary, IHEC)	MD	Clinical Pharmacology	Female	Yes	Yes
3	Mr Gowpathy Velappan	BA., BL	Legal Advisor	Male	No	Yes
4	Mr P Karuppuchamy	M Phil in PSW	Social Scientist	Male	Yes	Yes
5	Mrs G Malarvizhi	M Sc	Nursing	Female	Yes	No
6	Mr. R. Nandakumar (Vice-Chairperson, IHEC)	BA., BL	Legal Expert	Male	No	Yes
7	Dr. G. Rajendiran	DM	Clinician (Cardiology)	Male	Yes	Yes

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8	Dr. V. Ramamurthy	Ph D	Biotechnology	Male	Yes	Yes
9	Mrs P Rama	M Pharm	Non-Medical (Pharmacy)	Female	Yes	No
10	Dr. P. Sathyan (Chairperson, IHEC)	DO, DNB	Clinician (Ophthalmology)	Male	No	Yes
11	Dr. Seetha Panicker	MD	Clinician (Obstetrics & Gyanaecology)	Female	Yes	No
12	Dr. S. Shanthakumari	MD	Pathology, Ethicist	Female	Yes	Yes
13	Dr. Sudha Ramalingam (Alternate Member-Secretary, IHEC)	MD	Public Health, Epidemiology, Genetics, Ethicist	Female	Yes	No
14	Mrs. Swasthika Soundararaj	MBA	Lay person	Female	No	Yes
15	Dr. D. Vijaya	M Sc, Ph D	Basic Medical Sciences (Biochemistry)	Female	Yes	Yes

The study is approved in its presented form. The decision was arrived at through consensus. Neither PI nor any of proposed study team members were present during the decision making of the IHEC. The IHEC functions in accordance with the ICH-GCP/ICMR/Schedule Y guidelines. The approval is valid until one year from the date of sanction. You may make a written request for renewal / extension of the validity, along with the submission of status report as decided by the IHEC.

Following points must be noted:

1. IHEC should be informed of the date of initiation of the study
2. Status report of the study should be submitted to the IHEC every 12 months
3. PI and other investigators should co-operate fully with IHEC, who will monitor the trial from time to time
4. At the time of PI's retirement/intention to leave the institute, study responsibility should be transferred to a colleague after obtaining clearance from HOD, Status report, including accounts details should be submitted to IHEC and extramural sponsors
5. In case of any new information or any SAE, which could affect any study, must be informed to IHEC and sponsors. The PI should report SAEs occurred for IHEC approved studies within 7 days of the occurrence of the SAE. If the SAE is 'Death', the IHEC Secretariat will receive the SAE reporting form within 24 hours of the occurrence
6. In the event of any protocol amendments, IHEC must be informed and the amendments should be highlighted in clear terms as follows:
    a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
    b. Alteration in the budgetary status should be clearly indicated and the revised budget form should be submitted
    c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval
    d. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented
    e. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IHEC and only then can they be implemented

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f. Any deviation-Violation/waiver in the protocol must be informed to the IHEC within the stipulated period for review

7. Final report along with summary of findings and presentations/publications if any on closure of the study should be submitted to IHEC

Kindly note this approval is subject to ratification in the forthcoming full board review meeting of the IHEC.

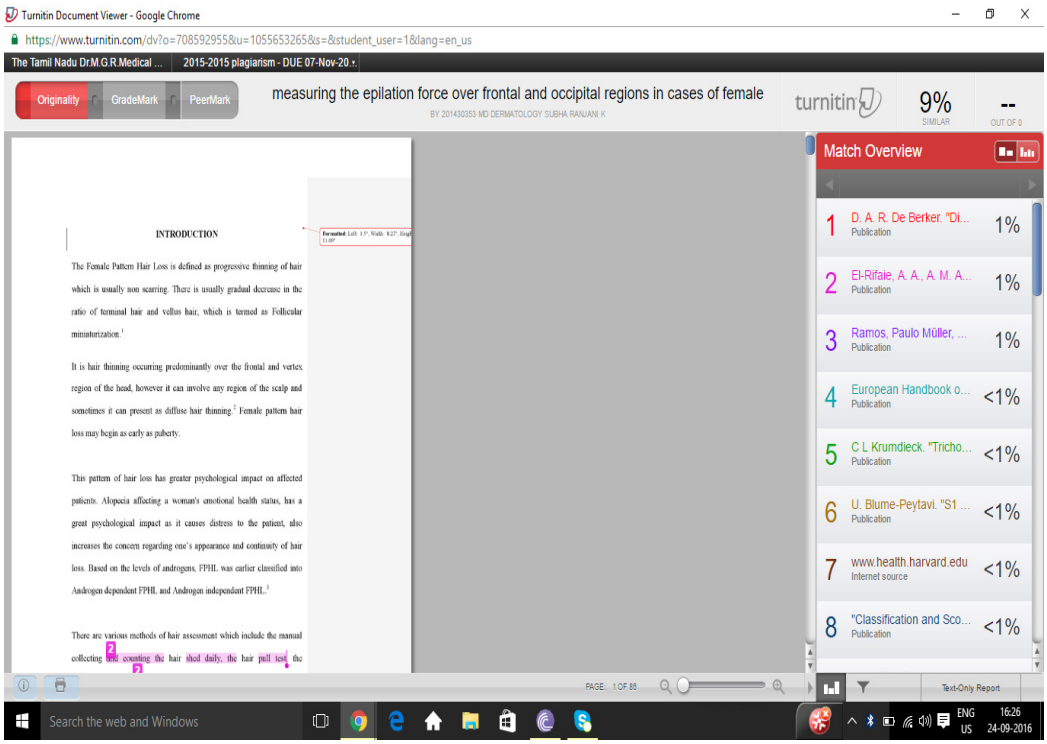
Thanking You,

Yours Sincerely,

SECRETARY
PSG IMS&R
COIMBATORE-641004.
INSTITUTIONAL HUMAN ETHICS COMMITTEE

Dr S Bhuvaneshwari
Member-Secretary
Institutional Human Ethics Committee

PLAGIARISM TOOL



ACKNOWLEDGEMENT

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Finally, I pay my obeisance to GOD, the almighty to have bestowed upon me good health, courage, inspiration, zeal and the light.

Dr SUBHA RANJANI K

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INTRODUCTION

The Female Pattern Hair Loss is defined as progressive thinning of hair which is usually non scarring. There is usually gradual decrease in the ratio of terminal hair and vellus hair, which is termed as Folliculminiaturization.[1]

It is hair thinning occurring predominantly over the frontal and vertex region of the head, however it can involve any region of the scalp and sometimes it can present as diffuse hair thinning.[2] Female pattern hair loss may begin as early as puberty.

This pattern of hair loss has greater psychological impact on affected patients. Alopecia affecting a woman's emotional health status, has a great psychological impact as it causes distress to the patient, also increases the concern regarding one's appearance and continuity of hair loss. Based on the levels of androgens, FPHL was earlier classified into Androgen dependent FPHL and Androgen independent FPHL.[3]

There are various methods of hair assessment which include the manual collecting and counting the hair shed daily, the hair pull test, the trichogram and its evolution the unit area trichogram, the PhotoTrichoGram and histopathological examination of scalp punch biopsies sectioned vertically along the length of the hair follicle and known as folliculogram.[4-8]

Trichotillometer was first designed and constructed by C L Krumdieck in the year 1981. It is a hand held device, which helps in measuring the force, necessary to epilate the individual hair.[9] It can be used in an outpatient basis, to determine the force required to pluck the individual hair, in patients with alopecia.

AIM

To determine the epilation force (EF) required to pluck the anagen hair and telogen hair from frontal and occipital region of the scalp in female pattern hair loss.

REVIEW OF LITERATURE

Anatomy of hair follicle

Hair, a keratinized product of hair follicle, is a tube like structure that extends into dermis and subcutaneous layer and continues with the epidermis at its upper end. From the mid wall of the hair follicle emerges the arrector pili muscle, which is an oblique muscle, extends to a point in the papillary dermis, at the dermal epidermal junction. The sebaceous glands, to some extent, the apocrine glands opens into the hair follicle, which is situated above the arrector pili muscle.

Hair bulb at the base of the follicle gives rise to outer cuticle, the cortex and central medulla, which constitutes the hair fibre. The inner root sheath emerging from the hair bulb winds the hair fibre and disintegrates. The inner and outer root sheath arising from the hair bulb extends to the epidermis. The other components of hair follicle include the connective tissue sheath and dermal papilla, which invaginates the hair bulb.

Conventionally the hair follicle is divided into two regions:

1. Upper part – infundibulum and the isthumus
2. Lower part – hair bulb and suprabulbar matrix

The lower part plays a major role, by undergoing constant regression and regeneration during the hair cycle.

The *infundibulum* emerges with the epidermis, extending from the skin surface and joins the junction of the isthumus, at the level of opening of the sebaceous gland.

The component of upper part is the *isthmus*, which starts from the opening of the sebaceous gland duct to the insertion of arrector pili muscle. It is composed of multilayered outer root sheath and well keratinized inner root sheath, ends at the level of sebaceous duct. Each follicle is surrounded by the arrector pili muscle like a sling.[10]

Usually the hair follicles are arranged in a group consisting of three or more follicles, termed as Follicular unit. Single arrector pili muscle slings around each follicular unit.[11]

The stem cells reside in the hair follicle, at the level of the isthmus.[12]

The stem cells have varied biochemical properties. During the onset of anagen phase they tend to proliferate. The transient amplifying cells, also called as daughter cells, extend from the lower end of outer root sheath

and enters into the hair bulb. They undergo terminal differentiation at this level and give rise to hair shaft and the so called inner root sheath.[13]

Between the isthmus and the hair bulb is the *suprabulbar region*. It is composed of three layers, the outer root sheath, inner root sheath and the hair shaft. The three layers are arranged in such a way that the outer root sheath lies outer to the inner root sheath and the hair shaft lies at the center. This region is a well keratinized region, where the hair shaft at the center, terminates within this keratogenous zone. The inner root sheath keratinizes much earlier than the hair shaft, and has a major role in moulding of hair fibre.

The *hair bulb* is the deeper most part of the hair follicle. It lies in the subcutaneous fat. The dermal papilla innervates the hair bulb at its base, which is surrounded by epithelial cells.

Hair bulb matrix or germinal epithelium, comprises of the inner root sheath and hair shaft derived from these epilthelial cells of dermal papilla. The hair bulb is also surrounded by melanocytes and occasional langerhan cells.

A flask shaped structure, *the dermal papilla*, in the anagen follicle, consists of specialized fibroblasts cells, proteoglycans and basement membrane proteins and blood vessels. Dermal papilla plays an important role in follicular epithelial differentiation.[14-17]

The follicle type can be determined by analyzing the dermal papilla. Androgens target mainly the dermal papilla, and this region also controls the hair follicle size and hair fibre.[18]

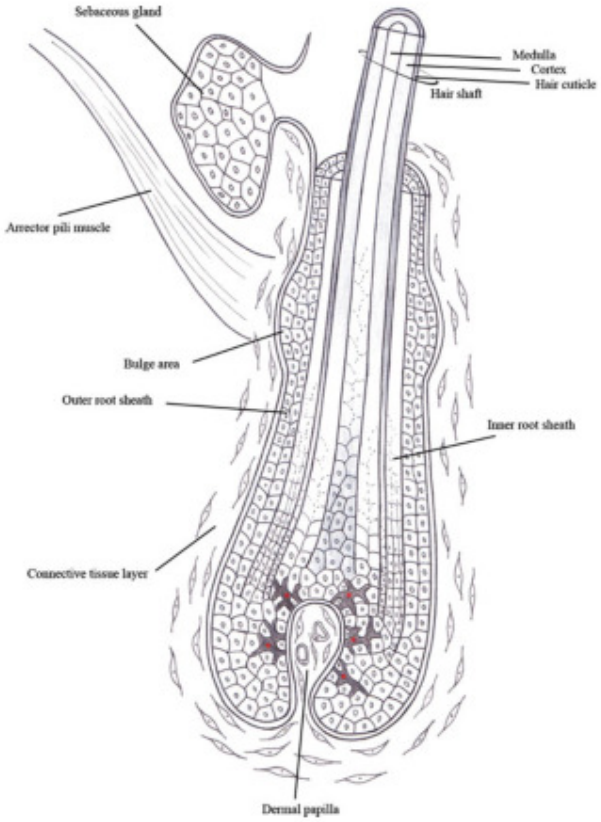
The collagenous layer that surrounds the hair follicle is also called as *dermal sheath*. It is also composed of specialized fibroblasts cells. It contributes in the formation of hair follicle.[19]

The thickness of the dermal sheath decreases, above the level of arrector pili muscle.

The *inner root sheath* is composed of three layers. The outermost layer is Henle's layer, the innermost layer is the inner root sheath cuticle and the middle layer is the Huxley's layer. Keratinization occurs in all the three layers at various levels.

The *outer root sheath* encloses the inner root sheath. It is multilayered in the upper hair bulb region.

The *cuticle* withstands the physical and chemical insults and act as a protective barrier and also provides stability to the hair shaft.



The Hair cycle:

Hair is a unique structure with special and cherished features, which provides protection of skin from mechanical insults. Its psychological functions are extremely important, and this can be vouched for, by any

consulting dermatologists or cosmetician, from their routine daily practice.

Mechanism of hair cycle is of importance to understand hair growth disorders. Hair is a keratinized product of hair follicle, which undergoes sequential phases of growth and rest, in a monotonous pattern, which is termed as *hair cycle*. It is a potent, alternating process, regulated by hormones and cytokines. Factors like age, environmental factors, nutritional habits also contribute in hair growth cycle maintenance.[20] The three phases of hair cycle are

1. Anagen phase

2. Catagen phase

3. Telogen phase

The duration of each phase varies between species, between each follicle, between each region of scalp.

Anagen is the active phase of hair follicle, which lasts for several years[21,22] and the duration of this phase determines the final length of hair. 80-90% of hair follicles are in anagen phase at any one time. It is during this phase, that the hair shaft begins to develop, melanocytes shows pigment producing activity and ends with the formation of epithelial hair bulb and new hair shaft begins to develop. The perifollicular vascularization is increased, and there is increase in vascular endothelial growth factor expression by keratinocytes of ORS.[21]

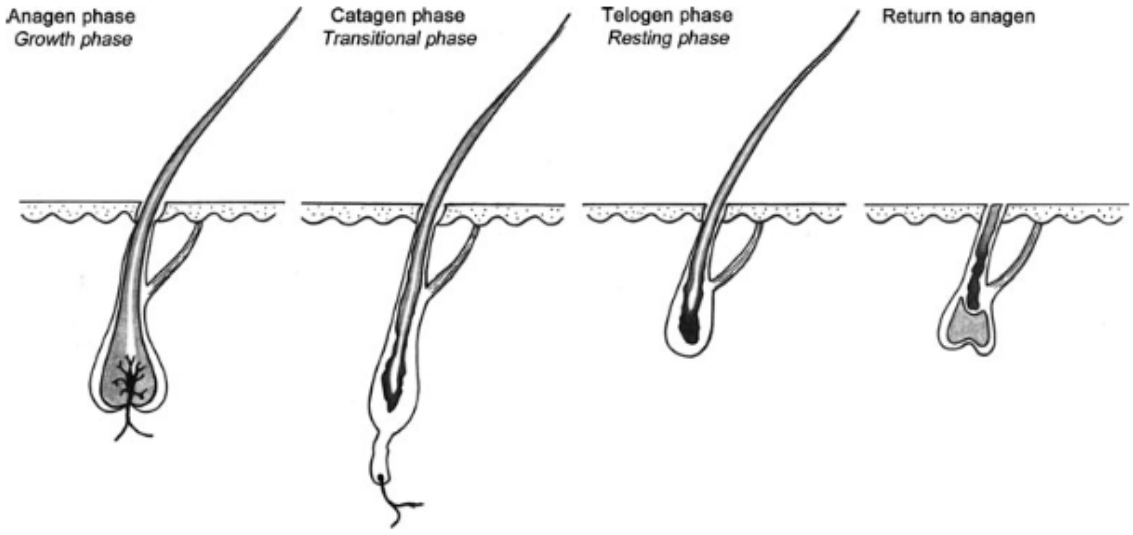
When anagen phase comes to an end, the hair follicle enters into *catagen* phase, which is otherwise called as involutionary phase, lasting for few weeks. During this phase, there is a significant reduction in the differentiation and proliferation of hair matrix keratinocytes and the melanocytes stops its pigment producing activity. At this stage, the hair shaft production is completed.

Resting phase is also called as telogen phase, which lasts for few weeks to eight months.[21] The hair follicle lacks the property of pigment producing melanocytes in this stage. 10-15 % of hair follicles are in telogen phase at one point of time.

In a normal individual, the anagen phase lasts for many years. The telogen lasts for few months and catagen lasts only for few weeks. It is estimated that, anagen lasts for 2 to 8 years. This is followed by catagen phase, and this phase lasts only for about two to three weeks. The last phase, the resting phase, lasts for about three months[23].

Thus, anagen phase comprises of around 80-90% of hair follicles, 10-20% of hair follicles will usually be in the telogen phase and 1-2% in the catagen phase in a normal adult scalp, which can be demonstrated by using trichogram.[24]

HAIR CYCLE:[108]



Causes of hair loss can be categorized into localized and diffuse forms[25]

The localized form appears in two forms

- *Scarring alopecia*
- *Non-scarring alopecia*

Conditions like androgenetic alopecia, alopecia areata, traumatic alopecia, telogen effluvium, fungal infection like tinea capitis are the common conditions that causes *non scarring alopecia.*

Discoid lupus erythematosus, viral and fungal infections (herpes zoster, tenia capitis or kerion), lichen planus, injuries are the important causes of *localized scarring alopecia*.

-

Causes of *diffuse hair loss*:

The etiology of diffuse hair loss is vast, that includes, androgenic alopecia, telogen effluvium, metabolic causes, hypo and hyperthyroidism, hypopituitarism, infections like HIV and syphilis, nutritional deficiencies, any chronic illness involving liver and kidney, alopecia areata and radiotherapy.

Androgenetic alopecia:

Common baldness presents with patterned hair loss, with genetic predisposition and hormonal influence. The pattern of hair loss is different in males and females. Hence they are termed individually as male pattern hair loss and female pattern hair loss.[26]

Hair loss in genetically susceptible[27] men, along with the contribution of androgens, causes androgenetic alopecia.

Androgens play an important role in hair growth. Androgens are responsible for inducing the hair follicle growth, only in few areas of skin, that usually occurs after puberty.

Whereas, in humans and other mammals, this hormone is responsible for balding over the scalp. Eunuchs, pseudohermaphrodites and patients with androgen insensitivity syndrome do not develop baldness. Hence, this provides details that androgens are a major contributing factor for patterned hair loss. James Hamilton, an American Anatomist, was the first person to identify the role of testosterone in male pattern baldness.[28]

The incidence of male pattern hair loss increases with age, and these patients usually have a strong family history of hair loss. The role of genetics in male androgenetic alopecia, has been proved in a twin study,

that, the concordance rate is 80-90% in case of identical twins.[29, 30] Age, pattern, gradual progression of AGA depends on genetic polymorphism.[31]

Androgenetic alopecia results in progressive decrease in the duration of anagen phase and increase in the latent phase.[32]

The another important component of this male pattern hair loss, is the follicular miniaturization.[33]

Hair loss can occur due to changes in hair cycle, hair shaft disorders, or it can occur with loss of hair follicles. Hence, while evaluating a case of alopecia, it is important to rule out the various causes associated with hair loss.

The patho-mechanism of common baldness involves transformation of terminal hair into vellus hair, which is intervened by androgens.

Genetic susceptibility to AGA, the age at which it starts, the rapid or gradual progression, and finally the pattern of hair loss, interact with each other and helps to find out the degree to which a patient will become bald.

Causes of diffuse hair loss in women:

The causes of diffuse hair loss in women can be categorized as follows[25]

1. Congenital causes – congenital atrichia, congenital hypotrichosis
2. Hair shaft disorders – unruly hair, hair breakage
3. Disturbances in hair cycle – telogen effluvium, anagen effluvium, loose anagen hair syndrome
4. Autoimmune – Alopecia Areata
5. Hormonal – Androgenetic alopecia – Female Pattern Hair Loss

   Of these, the most important causes of hair loss in women are telogen effluvium, female pattern hair loss and chronic telogen effluvium in decreasing order of frequency. Hair loss in women is a common entity and has become a challenging problem for the dermatologists.

Telogen effluvium

Klingman, in the year 1961, [34] proposed the condition, Telogen effluvium (TE). The onset of TE is abrupt, rapid, diffuse and is usually self-limited, causing excessive shedding of normal club hair, often triggered by stress, trauma, chronic illness like Systemic Lupus Erythematosus, syphilis, immediate and delayed post-partum period, infections like malaria and

typhoid, nutritional deficiencies, crohn's disease, endocrine abnormalities like hypo and hyperthyroidism.[35-37]

Sudden onset, rapid diffuse generalized shedding of hair, positive hair pull test, trichogram showing significant reduction in anagen:telogen ratio, biopsy features describing the rise in telogen follicles[35], usually 15 % increase in telogen follicles are the important diagnostic features of telogen effluvium.

Chronic telogen effluvium

An idiopathic condition, affecting female patients age ranging from 30-60 years, with prolonged fluctuating course of diffuse hair loss.[33]

If the triggering factor of classical telogen effluvium persists beyond six months' duration, it leads to chronicity. There is usually alteration in the hair growth cycle. While FPHL is associated with miniaturization of the hair follicles and reduction in anagen growth phase, chronic telogen effluvium is only associated with reduction in anagen growth phase.[25]

History of sudden onset of hair loss, persisting for more than 6 months, with positive hair pull test during active phase, slightly increased telogen follicles in histopathology are the important features of chronic telogen effluvium (CTE). While FPHL present as thinning of hair along with

central widening and miniaturization of hair follicles, CTE presents with diffuse shedding of hair.

Congenital atrichia:

The disease is characterised by diffuse hair loss, also involves the body hair. The disease is inherited as autosomal recessive disease. The patient usually present with skin coloured papules over the scalp, associated with hair loss.[107]

Trichotillomania:

The disease starts at a younger age, affecting both sexes equally.[109] In children, trichotillomania forms the most important cause of hair loss. The most important characteristic feature of trichotillomania, is that, it can involve the body hair also. Children will have the habit of eating the hair, which is termed as trichophagy. It is because of this habit, children usually present with features of bowel disturbances.[110]

There is no patterned distribution of hair loss in trichotillomania. On examination, there will be erythematous papules and pustules over the affected regions. Biopsy plays a major role in diagnosing the condition, also helps to rule out other causes of hair loss.

The psychological impact plays a major role in this condition, hence proper councelling in the form of behaviour therapy is important as a part of treatment strategy.

Traction alopecia:

One of the many reasons for traction alopecia also includes various hairstyles, were the hair are pulled and tied in different styles. It is usually seen in people from Africa. It is also seen in athletes, specially women, who pull their hair and tie it as a pony.

As people use different hair styles, the hair loss occurs in the region where there is traction, for example, women of Africa who braid their hair tightly, either manually or by the use of braiding machines, which create more intense tractional force, lose hair in the particular region, that has been graded. Diffuse hair loss occurs over the frontal and temporal areas in case of traction alopecia.[109]

Endocrinological causes:

The exact mechanism of endocrinological condition leading to hair loss is not clear.[113]

Conditions like hypothyroidism, hyperthyroidism, hypopituitarism, hyperpituitarism can cause hair loss.

Hypothyroidism:

Hypothyroidism can occur spontaneously or it can be iatrogenic in nature. There is usually diffuse hair loss associated with hypothyroidism. Clinically, there is diffuse hair loss, increased hair shedding. The ratio of telogen and anagen hair is decreased.[114]

Usually there is abberant retention of club hair in normally growing hair follicles, delay in growth of anagen hair follicle, cessation of growth of large number of hair in the anagen phase. There will be loss body hair. When a patient complaints of hair

loss, the drug history also plays a major role. There are many drugs responsible for hair loss.

One such group is, the drugs that are used to treat hypothyroidism. These drugs stimulate the follicles which are resting in the anagen phase and induce the displacement of the club shaped hair out.

There is late resumption or failure of anagen hair in hypothyroidism because there is no replacement for the lost hair. This effect leads to increase in the number of hair that are in the telogen phase, because there are increased number of club hair in the dominant follicles and they gradually fall off.[114]

When the patients who are suffering from hypothyroidism are treated with hormonal replacement therapy the normal anagen-telogen ratio is restored. In these patients there is also inhibition of mitosis due to which there is reduced cell division in the appendages and the layers of the skin, this promotes the

formation of catagen and this intern increases the time taken for the hair in telogen phase to go to the anagen phase.[115]

Hyperthyroidism:

The exact mechanism of hair loss induced by hyperthyroidism is not known. There is usually diffuse pattern of hair loss with increase in the number of telogen hair follicles. There is an increase in telogen and anagen ratio. The condition when treated will restore the anagen telogen ratio, but the chances of hair loss complaints can recur.[114, 115]

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Hypopituitarism:

There are many conditions associated with hypopituitarism, which can be classified into primary and secondary. Malignancies, infections, inflammation of the pituitary gland are the secondary causes. It can also occur following surgical ressection of the gland.

Massive bleeding occurring during delivery can cause severehypotension which inturn causes necrosis of the gland. This is called as Sheehan syndrome.[113,116] Patients affected by this syndrome usually complaints of

diffuse hair loss following delivery. Sometimes the body hair is also lost following delivery.[113,116]

Hypoparathyroidism:

Hair loss in hypoparathyroidism can occur because of the fact that the hair in this condition is rough and dry and trivial injuries can cause hair loss. There is usually uneven pattern of hair loss in this condition.[113,116]

Infections:

Syphilis:

Primary syphilis does not cause hair loss, but when the primary lesion appears over scalp, the propability of hair loss increases. Hair loss usually occurs in 2^{o} and 3^{o} syphilis.Either the hair loss is diffuse or it can appear in a characteristic pattern termed as "the moth eaten appearance".[113]

Sometime hair loss will be the only complaint or it can occur along with other manifestations over the scalp.[113]In few patients both moth eaten pattern and diffuse hair loss can occur together.

The hair loss due to syphilis will reduce with treatment.Hair loss occurs following 1^{o} syphilis with increase in number of telogen hair follicles.[117]

Similarly, following treatment with penicillin, as a part of Jarisch Herzheimer reaction, hair loss can occur, which is usually associated with reversal of anagen telogen ratio. Patients with hair loss secondary to syphilis will have a positive hair pull test.

Tineacapitis:

Tinea capitis, a very well known dermatophytic infection of the scalp, which is caused by trichophyton and microsporum species.[112] It usually occurs before puberty.

The dreadful form of tinea capitis is called kerion. Clinically it is characterised by appearance of boggy swelling associated with hair loss. There is no patterned distribution of hair loss. The hair breaks often and the hair loss is diffuse in tinea capitis.

Cicatricial alopecia:

The hair loss is permanent in cicatricial alopecia.[118] There is no patterned distribution of hair loss. Clinically, there is inflammation over the affected areas, with uneven pattern of hair loss.

Chronic cutaneous lupus erythematosus, lichen plano pilaris, pseudopelade of brocq are few important causes of cicatricial alopecia. The inflammatory process is continuous, as it destroys the hair follicles completely leading to permanent hair loss. Apart from cicatricial alopecia, antinuclear antibodies will be positive.

Alopecia induced by chemotherapeutic agents:

Anti-cancer agents cause anagen effluvium, and it is characterised by sudden permanent hair loss. Hair loss occurs only with few anti-cancer agents, and sometimes the condition is reversible. The psychological impact is more in case of female patients.[120, 121]

Alopecia areata:

Inflammatory condition, with an autoimmune etiology, having an uneven distribution of hair loss. it can affect the body hair also. When alopecia affects the whole body, it is termed as alopecia universalis. When alopecia affects the scalp region alone, it is termed as alopecia totalis.[119]

Nutritional causes:

The nutritional deficiency of zinc, iron, vitamin D is the major cause of hair loss in premenopausal women.[122, 123]Reduced haemoglobin is one of the most common causes of hair loss[122, 123]. Deficiency of Vitamin H, also known as biotin, also causes hair loss. However, nutitional deficiencies can affect women of younger age group also. The pattern of hair loss is diffuse. Hence it is important to investigate a patient with hair loss, to rule out the above mentioned nutritional causes.

Stress:

Stress can affect the hair growth cycle and can induce hair loss. They also contribute as one of the major causes of hair loss.

Postpartuminduced hair loss:

A common cause of hair loss, occurring few months after delivery, lasts upto 1 year. It is usually accompanied by nutitional insufficiencies which also contributes to the cause.

The level of androgens, sensititivity towards the androgen follicles also increase during this period.[124, 125]

Loose anagen syndrome:

In this disease, the pathology lies with the anagen hair follicle attachment. It runs in families and has an autosomal dominant pattern of inheritance[108]. There is improper keratinization in the inner root sheath. There is change in the anatomy of the anagen hair follicle, that on clinical examination, the hair gets pulled off from the scalp, without any pain over the affected region.

Thallium:

It is a very well known fact that thallium leads to loss of hair. Thallium has been used uin patients with tuberculosis to treat night sweats and also for epilation in cases of ring worm[116]. Such patients experienced hair loss which was attributed to the use of thallium. Nowadays, thallium salts are used in a preparation of pesticides and rat poisons.

Thallium poisoning is caused due to the intake of grains and food stuff contaminated with it. Such patients present with neurological symptoms,

only after a long period of thallium poisoning does hair loss occur, as the element gets deposited in the hair follicles of anagen phase and causes impaired keratinization.

This causes the detachment of hair while still in the follicle. They are irregular in structure in the keratogenic zone and might show air bubbles near the pointed end in the hair shaft. Similar to anagen effluvium hair loss begins after about 7 days. For the diagnosis, urine and stool should be tested for thallium.

Female pattern hair loss

Female Pattern Hair Loss leads to non-scarring thinning of hair, characterized by follicular miniaturization[1,38].

Alopecia can severely affect a woman's emotional well-being and also the quality of life. The psychological impact is more in case of females as it causes distress

to the patient, increases the concern regarding one's appearance and continuity of hair loss[39]

In men, hair loss usually begins above the temples, and the receding top of the head also thins, often progressing to baldness. In women, it begins with gradual thinning at the part line, followed by increasing diffuse hair loss radiating from the top of the head.

FPHL results from a progressive decrease in the ratio of terminal hair to shorter, thinner vellus hair, a process known as follicular miniaturization[1]. This miniaturization follows usually a pattern distribution. 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[2]. Androgens are mainly involved in the pathophysiology of FPHL. In some patients, follicular miniaturization along with diffuse hair loss can also occur without the involvement of androgens[40].

In **female pattern baldness**:

There is thinning of hair involving the top and crown portion of the scalp which gradually widens from the ventral part line. Usually the frontal hair line is preserved in case of FPHL.

There are three forms of FPHL[41]

1. Increased hair loss involving the crown region with frontal line preservation[42,43]
2. Frontal line preservation is lost. The central parting line gets widened[38,44]
3. Vertex of scalp is involved. There is usually frontotemporal recession or bitemporal recession[45]

Epidemiology

In men, Androgenetic alopecia (AGA) is characterized by progressive hair loss and its prevalence increases in Caucasian population. It occurs more frequently when compared to other causes of hair loss. The prevalence increases with age. It increases from 30% to 50% in patients age ranging from 30 -50 years[46].

In females, the FPHL is the most common cause of hair loss which characterized by miniaturization of hair follicles. Various studies have showed that the prevalence increases with age and it is more common among Caucasian populations[47].

It can occur at two peaks. One immediately after puberty and the other peak can occur after menopause[38,48,49].

The prevalence was 3% in women age ranging from 20 to 29 years, whereas, it was 29% in women age ranging from 70 and 89 years[47].

The prevalence was 38% in women age over 70 years, in a British study conducted amongst 377 women[50]. The disease is more prevalent in USA, that more than 21 million female are affected by this condition[51].

When compared with other data, the prevalence of FPHL was much lower in Asian population[52].

Patients with FPHL, sometimes possess a hyperandrogenic state. Polycystic ovarian syndrome is found to be the most common hyperandrogenic condition[53].

Though it is said that FPHL is common in adults, studies have proved the prevalence in childhood. 12 female children between the age group of 10-16 years had FPHL, in a study conducted by Tosti, in the year 2004. All the children had a positive family history of FPHL[54].

Patients with a hyperandrogenic state associated with FPHL, also develop insulin resistance. Insulin resistance in turn causes promotion of

dyslipidemia, and other metabolic abnormalities, thereby forming a proartherogenic mileu.[55-57].

Etiopathogenesis

Basically, FPHL leads to conversion of terminal hair into vellus hair. Hence the ratio of terminal hair to vellus hair will get reversed. This process is also termed as Follicular miniaturization[1]. Normally, the anagen phase lasts for many years and the telogen phase lasts for few months[23]. But in case of FPHL, the duration of anagan
phase is decreased.

The pathogenesis of female pattern hair loss depends upon various factors.

This includes

1. Hormonal influence
2. Changes related to the hair cycle
3. Growth factors
4. Micro inflammation
5. Cytokines[58]
6. Genetic predisposition

7. Other factors

Each factor contributes to the etiopathogenesis of FPHL, however, hormonal factors, genetic suseptibilty, inflammatory cascade plays a major role, when compared to other factors.

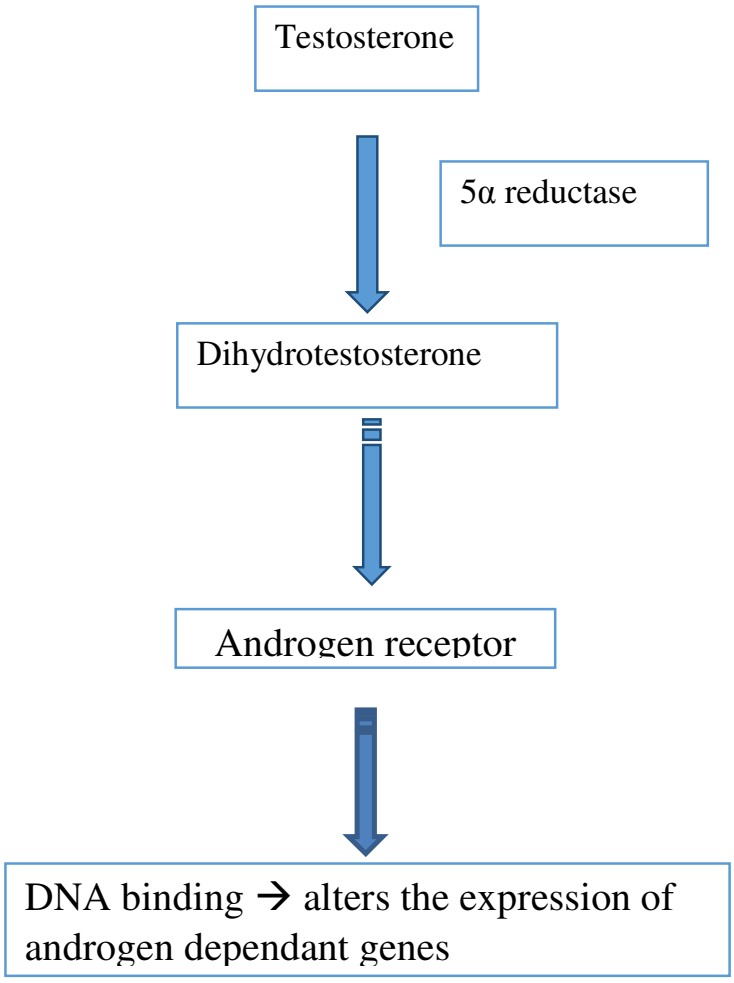
Hormonal changes:

Androgens, play a major role, both in inducing hair growth in body hair and also cause reduction in scalp hair growth. Hippocrates (400 BC) was the first person, to observe the correlation between androgen and hair loss, and he found that baldness is mainly influenced by androgens.

Dihydrotestosterone, a hormone derived from testosterone, is an important androgen playing major role in the pathogenesis of FPHL. The hormone has a directly affects the dermal papilla[59].

Testosterone, is converted into dihydrotestosterone with the help of the enzyme 5α reductase.

Mechanism:



The testosterone diffuses into the cell membrane and gets converted to DHT. The derived DHT, then binds to the androgen receptors in the hair follicle. It has a greater affinity to the androgen receptors, when compared to testosterone.

Once the DHT binds to the androgen receptors, then it enhances the genes that causes transformation of terminal hair follicles to vellus hair[60]. The vellus hair is otherwise called as miniaturized hair, which

is the most important change that occurs in FPHL.[38,59,61] But there is no direct effect on the number of follicles per unit area.[59] The occipital region is typically spared in case of FPHL, which could be due to decreased sensitivity towards androgens by the hair follicles in this region.

Hyperandrogenism:

Women with features of hyperandrogenism also suffer from FPHL, with increased risk of developing insulin resistance and atherosclerosis.

5 α reductase enzyme is of two types. Type 1 and type 2. Type 1 enzyme is more concentrated in the sebaceous glands, whereas type 2 enzyme is more concentrated in the genitourinary tract and hair follicles.[62]

Most of the patients with FPHL do not possess features of hyperandrogenism. Additionally, their androgen levels were also normal. This implies that the pathogenesis of FPHL is not totally dependent on androgens as other factors also have some role in the pathomechanism.

Estrogen also has a role in preventing early onset of FPHL. This is evidenced by the fact that, the prevalence of FPHL increases as the age increases.

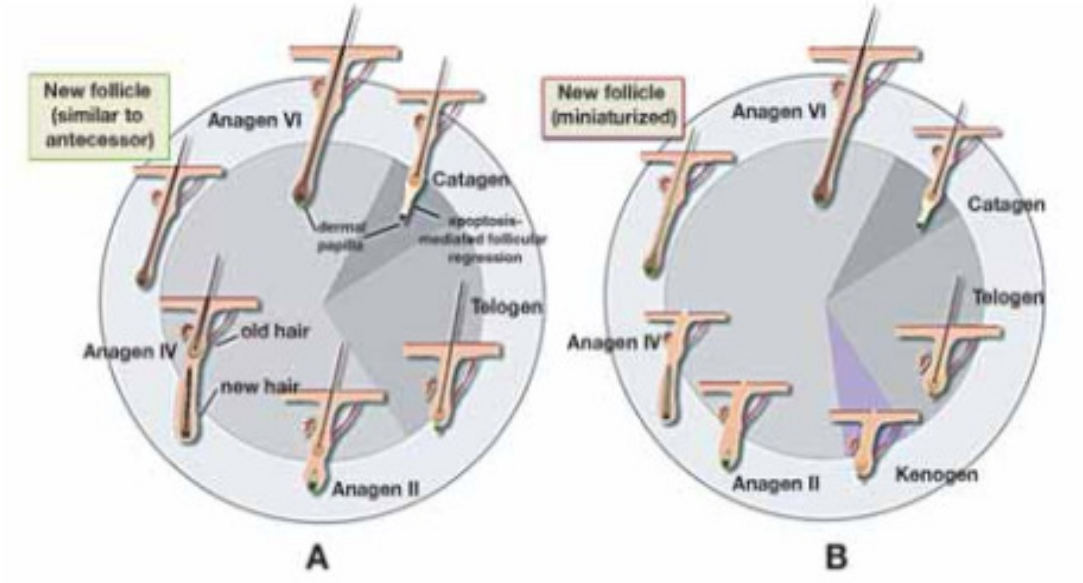
To prove this hormonal pathology, a study was conducted by Sawaya et al, to determine the level of androgens, androgen receptors, levels of 5α reductase and levels of cytochrome p-450 aromatase enzyme genes in the hair follicles. The study group involved 2 groups with 12 women in one group and 12 men in another group. Both the study groups showed increased 5 α reductase enzyme levels. The androgen receptors were increased in the frontal region, and there was a concurrent decrease in the level of androgen receptors in the occipital region.[63]

Apart from estrogen, enzyme aromatase also has a protective role in FPHL. Aromatase, an enzyme required for the conversion of testosterone and androstenedione into estradiol and estrone respectively.

This enzyme has an antiandrogenic mechanism, hence it protects against hair loss in females.[63]

Changes related to growth cycle:

Growth cycle is altered, which leads to progressive reduction in the period of anagen phase.[40]



Growth factors:

The imbalance between anagen promoting factors and follicular apoptosis promoting factors also gives rise to FPHL. The anagen promoting factors are basic fibroblast growth factor, fibroblast growth factor 7, hepatocyte growth factor, insulin-like growth factor-1, interleukin-1 alpha, prostaglandin E and vascular endothelial growth factors.

On the other side, the follicular apoptosis inducing factors includes fibroblast growth factor 5, interleukin 1α, prostaglandin D2, transforming growth factor β1 and tumour necrosis factor α.

Folloicular apoptosis can also be promoted by intrinsic pathway. There is usually reduction in the growth factors, along with absence of keratinocyte adhesion.[64]

The follicular miniaturization is also explained by means of apoptosis induced reduction in the dermal papilla cells.[65]

Role of micro inflammation:

Usually inflammatory process is more common in case of cicatritial alopecia, whereas in FPHL, there is only minimal amount of lymphohistiocytic infiltrates seen.[66,67]

Other factors:

Ultraviolet radiation and sometimes the normal flora of the skin which includes staphylococcus species, propionibacterium bacterium, malassezia also contribute the inflammatory process.[66]

Genetics:

The role of genetics and the inheritance pattern was studied by various authours.[68] The chance of acquiring FPHL is high in first degree relatives. The same pattern is observed in case of AGA. There are several genes involved in the pathogenesis of AGA[69]. Though AGA and FPHL differ in clinical aspects, they have a common genetic background.[70]

Early onset FPHL will usually have AR gene and ectodysplastin A2 receptor gene involvement in their pathomechanism.[71]The prevalence of FPHL was found to be more in monozygotic twins, when compared with dizygotic twins.[27] Apart from AR gene, insulin gene is also a candidate gene in FPHL.[72]

Although prevalence of FPHL is more in first degree relatives, the age at which the family members develop the illness varies. This proves that the disease has a polygenic pattern and the level of penetrance is also incomplete.[73,74] The pattern of baldness also differs between men and women of the affected family members.[75]

The genes that produce the 5α reductase enzyme are SRD5A1 and SRD5A2. Studies found that there was no significant association between these two genes. The results did not correlate with the degree of development of baldness.[76]

Studies have proved a significant correlation between the AR gene and the evolution of baldness. In these patients, a single nucleotide polymorphism was responsible for the development of baldness. There was a concordant increase in the value of this polymorphism in both premature baldness patients and late baldness patients. But the polymorphism was also found in 77% of non-bald men.[77-79]

The importance of aromatase enzyme was mentioned earlier. Study data showed that the gene that codes this enzyme, CYP19A1, following SNP was found nonfunctioning[80]. The genome and the associated loci found in MPA were not identical to that of FPHL.[81,82]

However, additional new loci[83,84], which were responsible for the development of baldness was found in MPA. These additional new loci were not found in patients having FPHL.[83,84] Hence these data show that AGA in male and females had varied pathogenic mechanism.

Other factors responsible for FPHL[85]

- Systemic hypertension
- Type 2 diabetes mellitus
- Photosensitivity

- Stress

Clinical diagnosis:

It is the chronicity of hair loss that brings women to the dermatologist. There are various factors contributing the psychological impact of FPHL. Since the condition is progressive, patients show more distress towards this illness due to cosmetological issues. Although FPHL is a common entity, there are various types of presentations associated with this condition. Pattern, duration of illness, other associated features should be addressed before starting the treatment.

The following parameters should be included while examining a case of FPHL.

- History
- Clinical examination – pattern of baldness, skin examination, total body examination
- Investigations
- Assessment of hair loss

History taking:

Recording the age of onset, duration of illness and progression of illness is very important in a case of FPHL. The patient should be asked about thinning and shedding of hair. Usually there will be accentuation of the frontal, parietal, or vertex region, however, FPHL can be associated with diffuse thinning. Most of the patients will have a positive family history of similar illness. Most of the patients will give a negative family history, which does not exclude the diagnosis. Familial hair disorders like alopecia areata or hirsutism, may influence the further investigations. The other causes of hair loss also can affect the efficacy of the FPHL treatment.

Iron deficiency anemia, infections, malabsorption, thyroid dysfunction or chronic dietary deficiencies are the other causes of diffuse effluvium, which are supposed to be excluded before diagnosing a case of FPHL.

Drugs that may influence the hair loss includes

- Chemotherapeutic agents
- Androgen inducing hormones
- Antithyroid agents
- Radiotherapy / UV radiation exposure[86]

- Smoking

  In a study, smoking was also found to be one of the etiological factors, contributing to the illness[87]

  Eating behavior of the patients (chronic deficient diet or rapid weight loss) can trigger diffuse effluvium.

Gynecological History:

Increased menstrual bleeding, amenorrhoea, late menarche, regularity of menstrual cycles, age at which menopause was attained, history of any surgeries increases the risk of FPHL.

Intake of oral contraceptives, treatment regarding infertility, signs suggestive of hyperandrogenism should be ruled out while evaluating a case of FPHL. Hyperandrogenism is accompanied by increased facial hair growth or body hair growth along with acne vulgaris and features of hypersebborhoea. Androgens, the main hormone involved in the pathogenesis of hair loss, increases the body hair growth as explained earlier.

Hyperandrogenism, gives a clue to the diagnosis of polycystic ovarian syndrome. Patients with PCOS, usually present with history of irregular periods.

Stress in the form of surgeries induces the body to produce more androgens, and plays a part in the role of pathogenesis.

Hence detailed history about duration, progression of illness, family history, drug history, menstrural history, treatment history should be taken for evaluating a case of FPHL.

Physical examination

It includes examination of scalp hair, body hair and total body examination. Each has its own importance in the diagnosis of FPHL.

Examination of scalp hair:

Clinician must look for the pattern of hair loss, the thickness and density of hair. Sometimes features of hypersebborhoea also must be looked for.

Female pattern hair loss presents in three different patterns. The patterns are described in terms of scales by various authors.

Among them, Ludwig's classification or grading of hair loss is universally accepted.

Ludwig, in the year 1977, classified FPHL into three grades or stages, based upon the severity of hair loss.[42] This classification helps us to understand three important factors

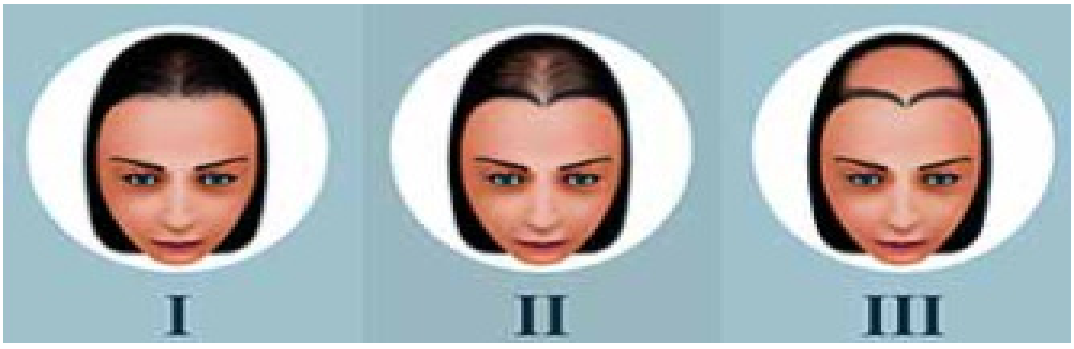
1. The extent of hair loss that has already occurred

2. To assess the potential for further hair loss in future

3. To determine the outcome of treatment

The grades are as follows[42]

Ludwig's Classification[42]



Grade 1:

This stage is characterized by mild hair loss. In this stage the frontal hair line is preserved. Hence women will not notice the hair loss. The area of hair loss over the scalp involves mainly the front and top. But when the hair is parted in the midline, the hair loss will be visible.

Grade 2:

This stage is characterized by moderate amount of hair loss. This stage is considered to be a progressive stage, where patients will feel that there is shedding of hair, the thickness of hair is reduced, reduction in the volume of hair. The center part widens more in this stage, which is presumed by patients to be an ongoing process and increases the anxiety and worries about the hair loss.In this stage, hair transplantation can be planned as a treatment option of hair loss.

Grade 3:

The stage is characterized by severe hair loss. The psychological impact is more in this stage. The hair loss is more visible, that can be seen by naked eye. The thinning of hair follicles is more severe

in this stage, which is also associated with follicular miniaturization..

Limitation of Ludwig's classification:

The limitation of using this scale is that it does not explain the details of intermediate stages clearly. Ludwig's scale is otherwise known as three-point grading scale.

However, Sinclair's classification[43] is divided into four grades, using normal scalp as a parameter, which aids in comparing the other four grades. It is also known as 5-point grading system.

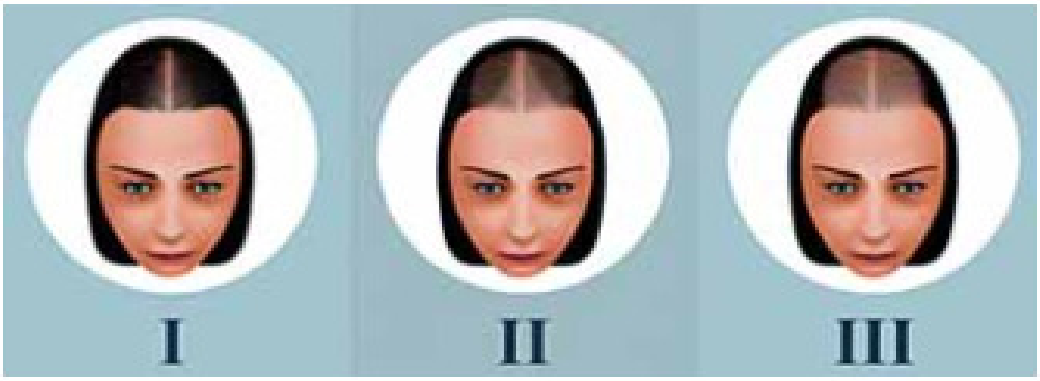
Sinclair's classification[43]

Both Ludwig and Sinclair's classification – both the grading systems explains that the frontal line is preserved.

Second pattern:

Olsen, in the year 1999, described this pattern.[44] It involves the bitemporal region and also the vertex. There is thinning in both the above mentioned areas with frontal line acceleration. It is so called Christmas tree pattern, since there is involvement of central line along with frontal line.

Olsen's classification[44]



Third pattern: the classical pattern, described by Hamilton and redefined by Norwood in the year 1975.[45] This pattern actually explains about the male baldness, however women can rarely present this pattern.

Hamilton- Norwood classification:

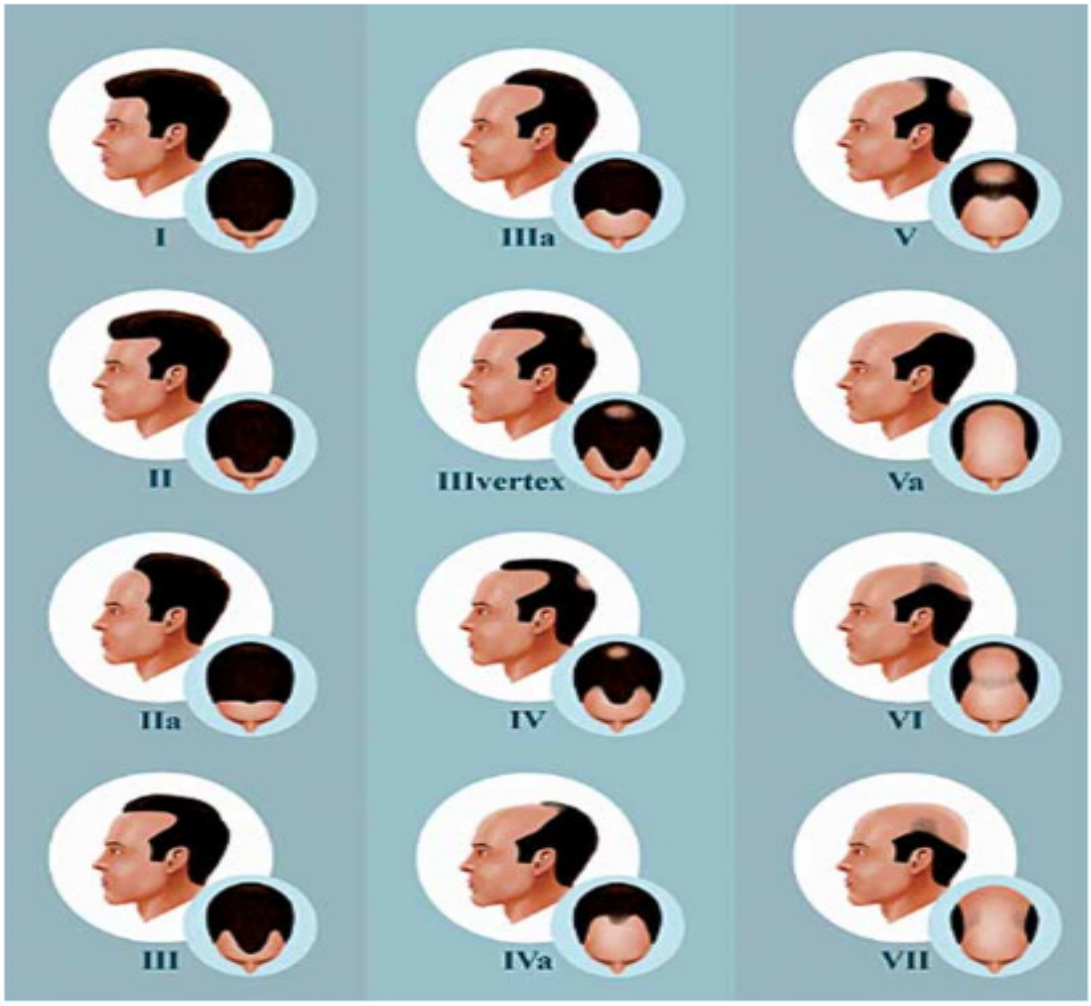


Table 1: Modified Norwood-Hamilton classification

Type	Clinical definition
I	Minimal recession of the hairline along the anterior border in the frontotemporal (FT) region[1,46,47]
II	The anterior border of the hair in the FT region has triangular areas of recession that tend to be symmetrical. These areas extend no further posterior than approximately 2 cm anterior to a line drawn in a coronal plane between the external auditory meatus on both sides. Hair is either lost or sparse along the mid-frontal border of the scalp
III	Characterized by deep FT hair recession, usually symmetrical and either bald or sparsely covered with hair. These areas of hair recession extend further posterior than a point that lies approximately 2 cm anterior to a line drawn in a coronal plane between the external auditory meatus on either side
IIIv (vertex)	Hair is mainly lost in the vertex. There may be some frontal recession but it does not exceed than that seen in type III
IV	The frontal and FT recession is more severe than type III. There is also sparseness or absence of hair in the vertex area. These bald areas are extensive, but separated from each other by a band of moderately dense hair that joins the fully haired fringe on each side of the head.
V	The hair loss over the vertex and FT areas is larger than in type IV and the band of hair between them is narrower and sparser
VI	The hair loss over the FT and vertex regions is confluent and the bridge of hair that crosses the crown is absent
VII	There is only a narrow horseshoe-shaped band of hair that begins laterally just anterior to the ear and extends posteriorly on the sides and fairly low on the occipital area
Variants (Type variants-'a')	Constitutes 3% of all cases of AGA: (i) the entire anterior border of the hairline progresses posteriorly without the normal island of hair in the mid-frontal region and (ii) there is no simultaneous development of a bald area on the vertex. Instead, the anterior recession just advances posterior to the vertex
IIa	The entire anterior border of the hairline lies high on the forehead. The usual mid-frontal island of hair is represented by only a few sparse hairs. The area of denudation extends no farther than 2 cm from the frontal line
IIIa	The area of denudation reaches the mid-coronal line
IVa	The area of denudation extends beyond the mid-coronal line and there may be considerable thinning of hair posterior to the actual hair line
Va	Most advanced degree of alopecia; however, the bald area does not reach the vertex

AGA: Androgenetic alopecia, FT: Frontotemporal

Recently, Lee et al, classified a new pattern BASP – basic and specific, which is a simple scale, and it can be used both for male and female pattern hair loss[88-90]

A routine physical examination including skin and nail is important to rule out the other causes of hair loss. Hair loss due to alopecia areata, iron deficiencies, lichen planus will be usually associated with nail changes. Features of hirsutism or hyperandrogenism includes, increased facial hair and body hair.

It is important to identify the pattern of hair loss, distribution of hair and caliber of hair. In active cases of FPHL, hair pull test will be positive. Clinical features such as inflammation, scarring, or hyperseborrhea potentially aggravates the FPHL.

As mentioned earlier, the most important causes of hair loss in females includes FPHL, telogen effluvium and chronic telogen effluvium.
These causes are difficult to assess immediately by a physician. The important differentiating features between them are:

Table 1: Differences between FPHL, CTE, TE[25]

FPHL	TE	CTE
Multiple etiologies – hormonal, genetics, inflammatory cascade, growth factor involvement.	Positive triggering factors-infections, chronic illness, stress, surgeries etc.	Idiopathic
Gradual and progressive onset	Sudden onset	Sudden onset
Thinning involves the central region of scalp along with central widening	Thinning of hair is usually diffuse	Usually absent.
Seen only in male type of FPHL, and is usually mild to moderate.	Bitemporal recession is not a feature	Moderate to severe Bitemporal recession is a common form.
Follicular miniaturization is the key feature	Absent follicular miniaturization	Absent follicular miniaturization
Hair pull test is absent, if present, it is limited to the central region alone	Hair pull test is strongly positive	Hair pull test is positive only during active phase.

Investigations required:

As mentioned earlier, there are many systemic factors responsible for hair loss. Serum levels of FT3, FT4, serum TSH levels, serum level of iron ferritin, TBIC(total iron binding capacity), vitamin d levels are important investigation necessary to rule out the secondary causes of hair loss.

Patients presenting with increased facial and body hair growth, who complaints of irregularity in menses, weight gain and high body mass index should be investigated for polycystic ovarian disease. Hence serum levels of 5α dehydrotestosterone, 17 β hydroxyprogesterone, ratio of total testosterone and sex hormone binding protein should be explored to assess the androgen state of a body. If patient shows features of PCOS, then ideally endocrinologist opinion should be sought to treat the primary cause.

Hair assessment:

There are various methods of hair assessment which includes

- Non invasive
- Semi – invasive
- Invasive

Non–invasive and semi invasive method

1. Manual collecting and counting the hair shed daily[4]

2. Hair pull test[5]

3. The trichogram[6] and the unit area trichogram[7]

4. The PhotoTrichoGram – targeted areas of the scalp will be photographed and magnified[91,92]

5. The TrichoScan: it is the combination of PhotoTrichoGram and epiluminescence microscopy. It involves analysis by digital method[93]

Invasive methods:

1. Folliculogram: vertical sectioning of scalp biopsies and its histopathological examination[8]

2. Follicular matrix examination – analyzing the actively dividing cells and the determining the number of cells entering mitosis at a given point of time[94]

3. Hair bulb – volume measurement[95]

4. Sectioning the scalp biopsy horizontally.[33]

Trichoscopy

A non-invasive technique, Trichoscopy or scalp dermatoscopy, is an important diagnostic tool, very helpful for diagnosing early stage of FPHL.[96] It helps in follow-up of patients with hair and scalp disorders.[97]

Hair diameter is measured with this technique, and it helps to assess FPHL in children or young females[98]. The hair diameter variability which is usually greater than 20%[99] is considered significant. Conditions like

alopecia areata also present with hair diameter variability, however, it shows uniform miniaturization instead of hair shafts with different degree of thinning.

Hair pull test:

A simple office procedure, which belongs to non-invasive category, can be used to analyze the progressive disease activity and helps in follow-up of patients. It is a rapid procedure and it does not require any special requirements.

In case of FPHL, the pull test becomes positive in the central region. In case of telogen effluvium or chronic telogen effluvium, the pull test becomes positive in all the regions of the scalp. From the base of the scalp, some hairs are grasped using thumb, middle and index fingers and the hair are pulled from the scalp.[100]

If 10% or more hair can be pulled, then the test is significant[101]. The main disadvantage of this technique is that the results vary between observers. This method of investigation requires standardization.

Scalp biopsy:

There are many conditions associated with cicatricial alopecia. DLE, SLE, scleroderma, lichen planus ate usually associated with cicatricial alopecia. Sometimes FPHL can present with some features of cicatricial alopecia, making the diagnosis difficult. Hence scalp biopsy plays a major role in exploring the causes of cicatricial alopecia.[102]

Females usually have miniaturized hair follicles in the temporal region, [41] hence scalp biopsy should best be avoided in this site. For doing a scalp biopsy, 4mm punch biopsy should be used. Horizontal sectioning is done which usually provides details about the number of hair follicles and its diameter.

The morphology of the hair follicle can also be determined[103]. For a normal individual, the proportion of terminal hair and vellus hair is >7:1. In FPHL, as mentioned earlier, miniaturized hair follicles will be increased (vellus like). The proportion of terminal to miniaturized follicle is >3:1. in FPHL.[96]

Trichogram:

Conditions like loose anagen syndrome often, present with features that are similar to FPHL. This technique helps to determine the number of anagen and telogen hair, helps to evaluate the hair follicle diameter and analyze the hair growth cycle[104]. Phototrichogram, a variant of trichogram, utilizes the digital images to evaluate the hair follicles and its phase.

Trichotillometer:

A non-invasive technique used to determine the force required to epilate the hair, was first described by C. L. Krumdieck in the year 1981.[9] Using this technique, the force required to pluck the anagen hair and telogen hair, measured in grams can be determined.

This instrument was previously used to evaluate the nutrional status of patients affected by protein energy malnutrition.[9] This simple, cost effective technique can be used to compare the epilation force with various parameters like age, sex and diameter of hair follicle. It can also be used for follow ups of patients affected with hair loss.

MATERIALS AND METHODS

This was a hospital based observational study carried out on female patients attending the outpatient department of Dermatology Venerology and Leprology, PSGIMSR, Coimbatore.

The sample size was 30. All the volunteers in our study population experienced noticeable increased hair shedding or changes in hair quality or quantity. None of the patients in the study group had suffered from serious illness or general health disturbance. The menstruating female volunteers had no menstrual cycle irregularities, gynecological disturbance, or been taking contraceptive preparations. Detailed history was taken regarding the above mentioned parameters and the patients were enrolled for the study.

The study was conducted in the department of Dermatology, PSG IMS& R, Coimbatore from 2014 – 2016.

The institute ethics committee clearance was obtained on 2014 before commencement of data collection.

An informed consent was obtained from all the volunteers prior to the procedure.

PATIENT SELECTION

INCLUSION CRITERIA:

1. Age more than 20 years
2. Patients diagnosed to have female pattern hair loss clinically using LUDWIG's classification (only class 2)
3. Only female patients were included in the study

EXCLUSION CRITERIA:

1. Pregnancy
2. Lactation
3. Endocrinological disorders: hypothyroidism, hyperthyroidism, Polycystic ovarian syndrome, diabetes mellitus
4. Systemic Hypertension
5. Chronic illness – liver and kidney disease
6. Psychological stress
7. Currently on treatment for hair loss
8. On treatment for any systemic or endocrinological illness

METHODOLOGY

- Participants visiting the Dermatology OPD, fitting the inclusion criteria were recruited by convenient sampling
- All the recruited patients were explained about the procedure. The patients were assured that their participation was voluntary. Informed and written consent were obtained from all patients prior to the procedure. Ethics committee clearance was obtained.
- Patients refrained from hair wash for three days before the procedure.
- The patients were subjected to test by using trichotillometer and the hair was plucked from two sites, frontal and occipital.
- Patients were made to sit on the stool comfortably.
- For all the patients, the frontal region and occipital region were located using a thread. A thread of 20cm length was taken and it was marked using a marker at 5cm and 10cm from one end of the thread.
- One end of the calibrated thread was placed over the center of glabella and the site when measured 10cm in the upward direction from the glabella was selected as the frontal region.
- Similarly, one end of the calibrated thread was placed over the center of mastoid prominence and the site when measured 5cm in the diagonal direction from the glabella was selected as the occipital region.

- The gripper at the lower end of the trichotillometer was attached to the distal end of a single hair shaft . Spring balance is pulled upwards gently by the hand at the top end with the help of a thumb ring attached to its top till the hair is released from thescalp.
- Force indicator slides down the inner wall of the spring balance and stops at the point when the hair is detached from the scalp.
- The force was noted and taken as the epilation force.
- The hair was plucked one by one from both the sites. Force was noted while plucking each hair.
- Each hair was examined under microscope to determine whether the hair was in anagen or telogen phase.
- The details were recorded in excel sheet for each patient. The force required to pluck each hair was recorded and the mean was calculated for each patient.
- The ultimate mean of all 30 patients was calculated.
- The mean of anagen hair and telogen hair was calculated for both the regions individually.
- The results were statistically analyzed.
- The results were calculated by *paired t test* and the cut-off for significance was defined as <0.05.

HAND HELD TRICHOTILLOMETER:

The instrument was constructed by adapting or modifying the laboratory spring balance.

Parts of trichotillometer:

1. Extension rod
2. Force indicator with hold facility
3. Gripper
4. Cords to reset the indicator
5. Calibrated scale (from 0.1 to 1N)

Principle of hand held trichotillometer:

To apply a force on a hair follicle, by means of a spring balance which can be stretched gradually to increase the force until the hair gets released from the scalp.

Working of trichotillometer:

The gripper at the lower end of the trichotillometer was attached to the distal end of a single hair shaft. The upper end of the spring was fixed, whereas the lower end was attached to the extension rod along with the gripper. Spring balance is pulled upwards gently by the hand at the top

end with the help of a thumb ring attached to its top untill the hair is released from the scalp. Force indicator slides down the inner wall of the spring balance and stops at the point when the hair is detached from the scalp. The force will be noted and taken as the epilation force.

Calibration:

The instrument has a calibrated scale with markings from 0.1 to 1N. The instrument we devised was calibrated to correct the following errors.

1. Zero error
2. Sensitivity

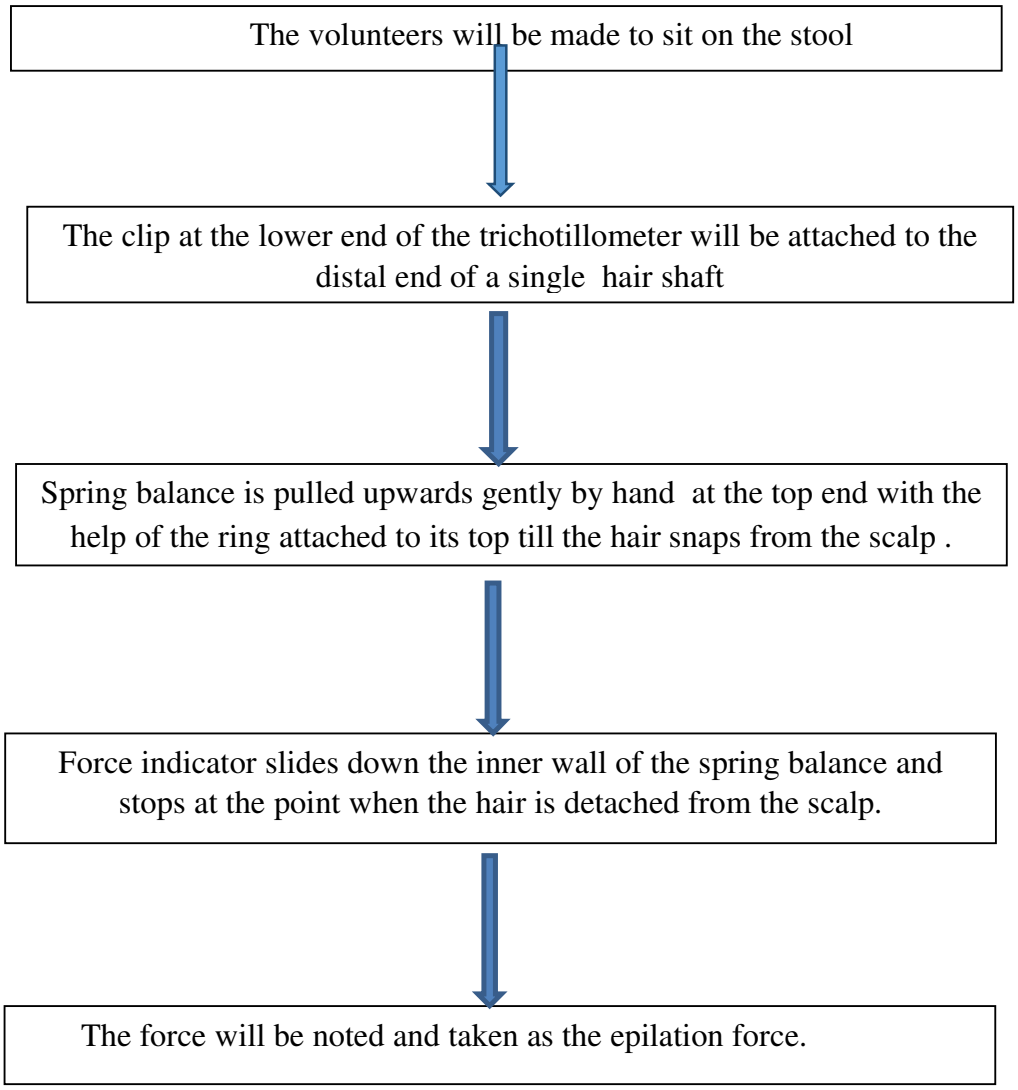
The formula used to determine the corrected force was

Actual value = Zero error + K (measured value)

where **K** is constant (1)

The corrected calibration was entered into Microsoft excel sheet.

FLOWCHART


The volunteers will be made to sit on the stool
The clip at the lower end of the trichotillometer will be attached to the distal end of a single hair shaft
Spring balance is pulled upwards gently by hand at the top end with the help of the ring attached to its top till the hair snaps from the scalp .
Force indicator slides down the inner wall of the spring balance and stops at the point when the hair is detached from the scalp.
The force will be noted and taken as the epilation force.

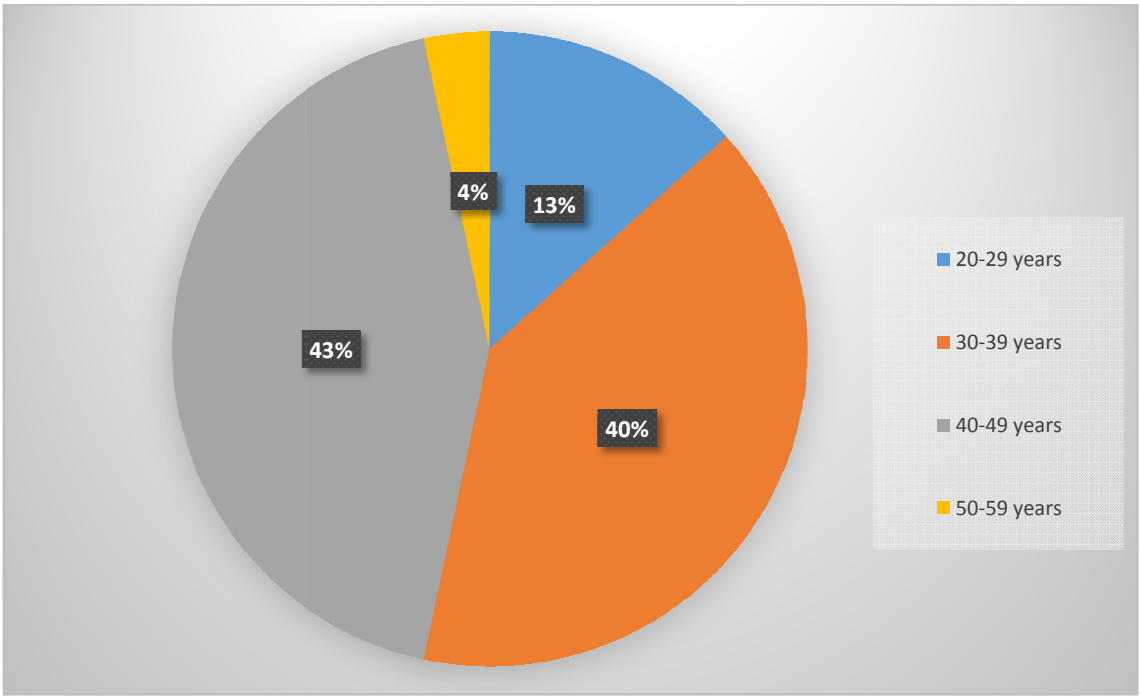
Results

A total of 30 patients were enrolled in our study. The age group ranged from 21 to 60 years. 43.34% (13 patients) of the patients were between the age group of 40-49 years. 40% (12 patients) of the patients were between the age group of 30-39 years. 13.34% (4 patients) of the patients were between 20-29 years and only 3.34%(1 patient) was between the age group of 50-59 years.

Table 2: Age Distribution

Age	**20-29 years**	**30-39 years**	**40-49 years**	**50-59 Years**
No. of patients	4	12	13	1
Percentage	13.34%	40%	43.34%	3.34%

AGE DISTRIBUTION

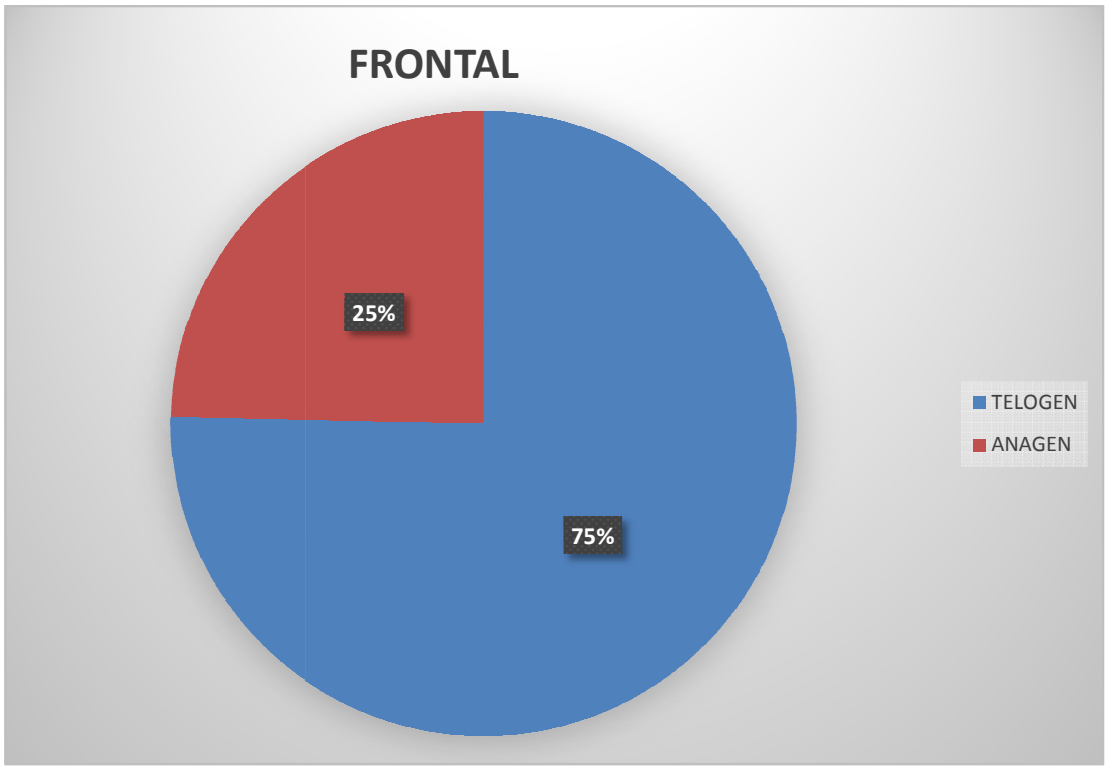


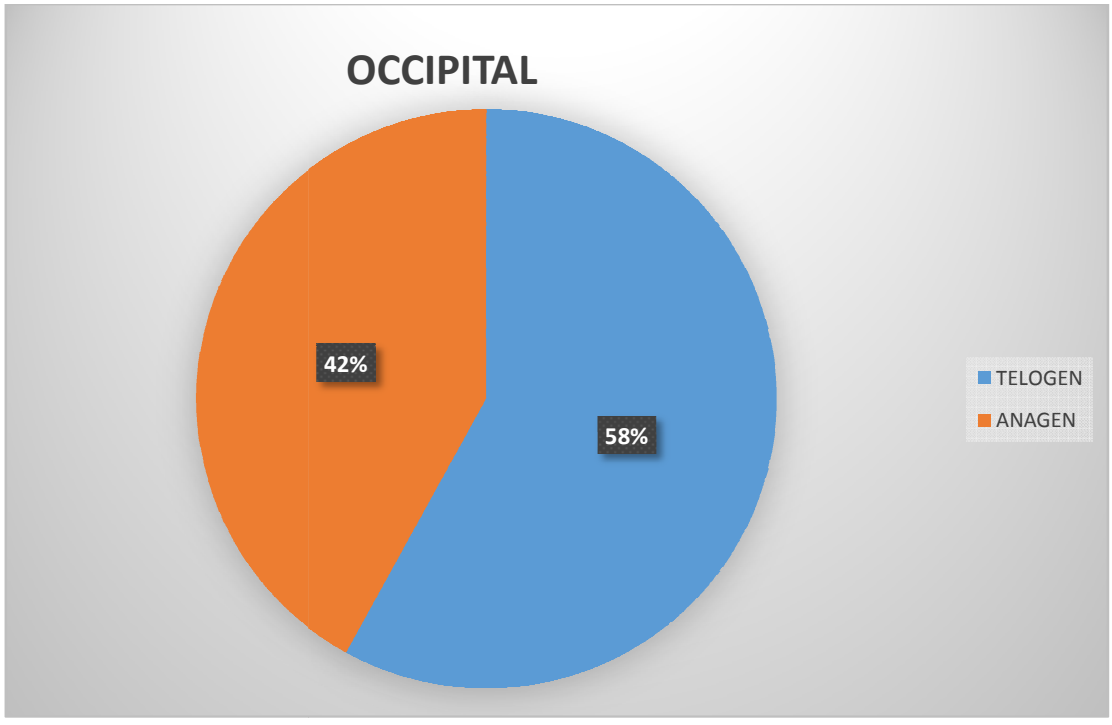
None of the patients in our population, had any systemic or endocrinological illness. Hair was epilated from frontal and occipital region using the hand held trichotillometer. A total of 5 hair was epilated from each region. Each epilated hair from both the regions was examined under microscope, individually, to determine whether the hair was in anagen or telogen phase.

ESTIMATION OF TELOGEN AND ANAGEN HAIR :

The total number of telogen hair was 200 (200/300) and anagen hair was 100 (100/300). The ratio of telogen to anagen hair in the frontal region was 75:25. The ratio of telogen and anagen hair over occipital region was 58:42. The percentage of telogen and anagen hair was calculated.

PERCENTAGE OF TELOGEN AND ANAGEN HAIR OVER FRONTAL REGION





Determination of epilation force required to pluck the telogen hair and anagen hair, in both frontal and occipital region, using hand held trichotillometer

TABLE: EPILATION FORCE OF TELOGEN HAIR OVER FRONTAL REGION

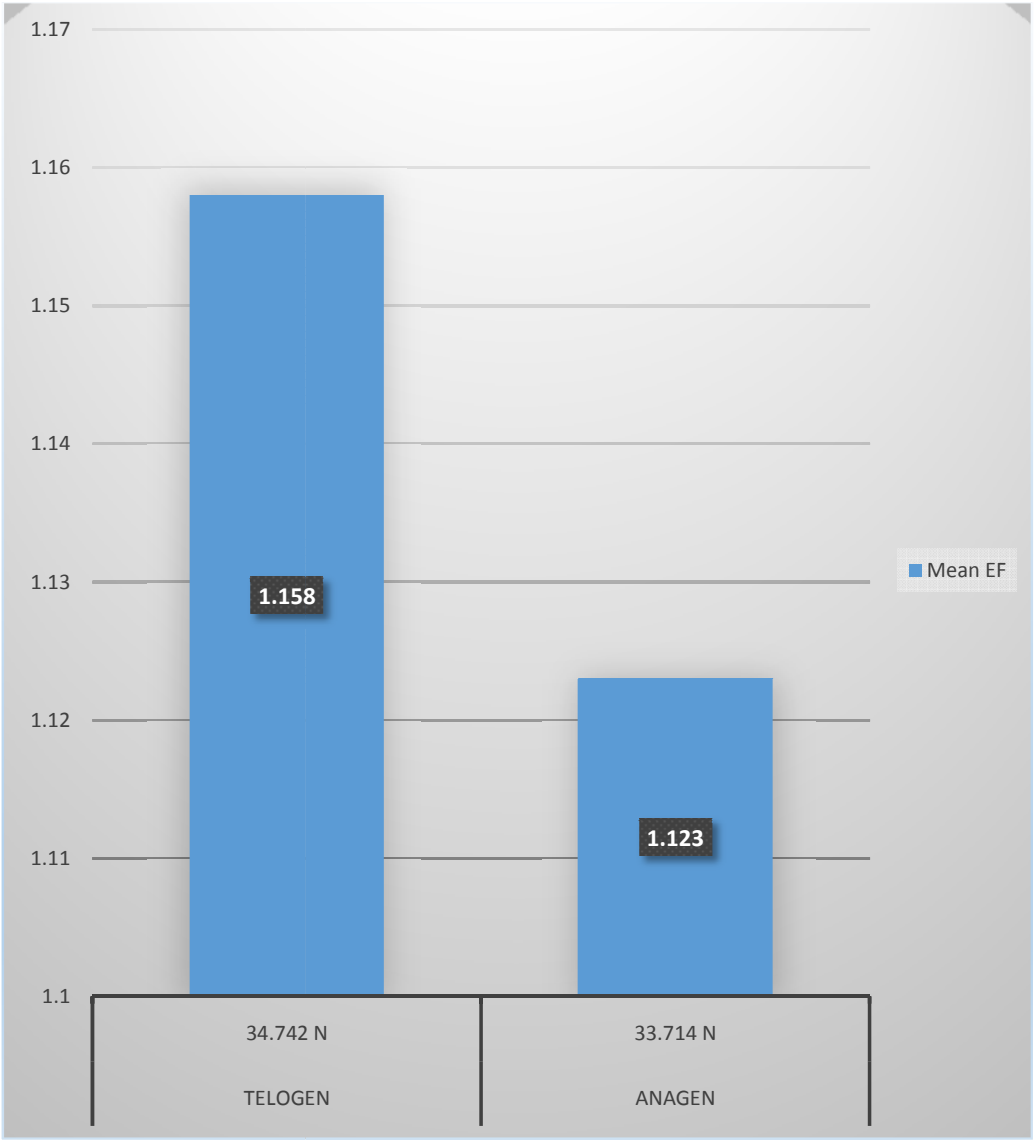
Parameters	Telogen Hair
Total Epilation Force	34.742N
Mean Epilation Force	1.158

TABLE: EPILATION FORCE OF TELOGEN HAIR OVER OCCIPITAL REGION

Parameters	Anagen Hair
Total Epilation Force	33.714N
Mean Epilation Force	1.123

Comparison of total EF and Mean EF required to pluck anagen hair and telogen hair, in both frontal and occipital region		
Parameters	**Telogen**	**Anagen**
Total EF	**34.742 N**	**33.714 N**
Mean EF	**1.158**	**1.123**

MEAN EPILATION FORCE:



Total and Mean epilation force required to pluck telogen hair and anagen hair in the frontal region

TABLE: EPILATION FORCE OF ANAGEN HAIR OVER FRONTAL REGION

Parameters	Anagen Hair
Total Epilation Force	15.386N
Mean Epilation Force	0.51

TABLE: EPILATION FORCE OF TELOGEN HAIR OVER FRONTAL REGION

Parameters	**Telogen Hair**
Total epilation force	21.502
Mean epilation force	0.71

Total and Mean epilation force required to pluck telogen hair and anagen hair in the occipital region

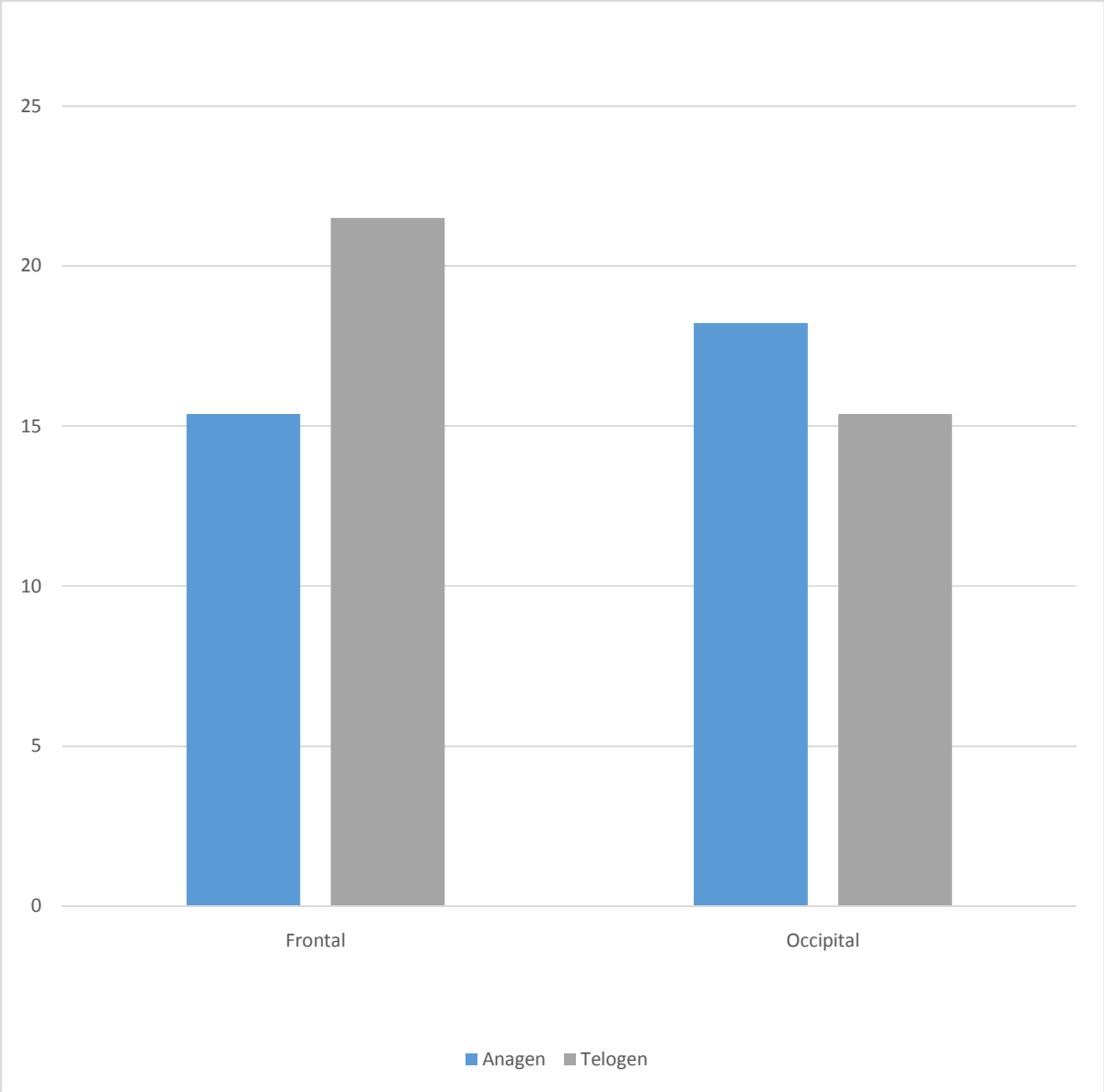
TABLE: EPILATION FORCE OF ANAGEN HAIR OVER OCCIPITAL REGION

Parameters	Anagen Hair
Total Epilation Force	18.228N
Mean Epilation Force	0.61

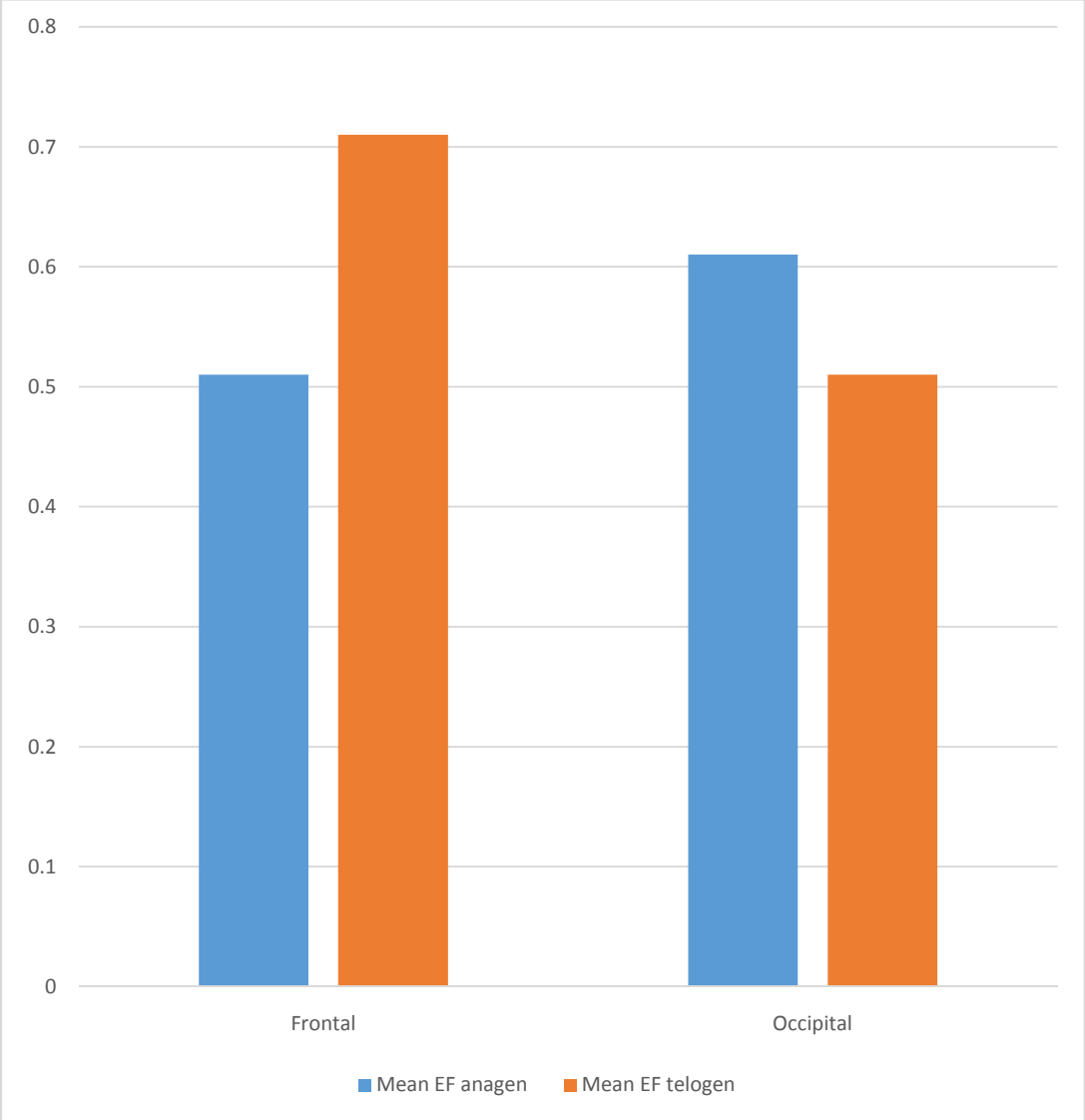
TABLE: EPILATION FORCE OF TELOGEN HAIR OVER OCCIPITAL REGION

Parameters	**Telogen Hair**
Total Epilation Force (occipital)	15.386
Mean Epilation Force (occipital)	0.51

Total EF of Frontal and Occipital region:



Mean EF of frontal and occipital region



Statistical Analysis:

TABLE: The Mean±SD EF over frontal region

FRONTAL	MEAN ±SD	P Value
ANAGEN	.51±.49	.152
TELOGEN	.73±.39	

TABLE: The Mean±SD EF over occipital region

Occipital	MEAN ±SD	P Value
ANAGEN	.61±.34	.333
TELOGEN	.51±.31	

TABLE: The Mean±SD EF of anagen and telogen hair

Total	MEAN ±SD	P Value
ANAGEN	1.12±.56	.659
TELOGEN	1.19±.47	

Discussion

Our aim of the study was to measure the epilation force required to pluck the anagen and telogen hair in frontal and occipital region in cases of female pattern hair loss.

Female pattern hair loss is usually associated with decrease in anagen and telogen ratio[1], which is more marked over the frontal region. Gradually the number of terminal hair decreases along with significant increase in the number of vellus hairs.

The hair follicles in the occipital region are less sensitive to the androgen receptors, when compared to frontal region. This could possibly explain the theory of occipital region being spared in case of female pattern hair loss.

There are various invasive and non-invasive techniques available for evaluating hair loss. Although the non-invasive techniques are uncomplicated and easy to perform, the question of inter observer variations, limits their use in the field of hair assessment.

The invasive techniques, though preferred for a precise and error free diagnosis, are laborious and inconvenient for field purpose.

In an attempt to overcome these barriers, we constructed a trichotillometer,based on Instron Universal Tensile Strength tester, in the year 2009, in order to measure the diameter of hair and to determine the force required to pluck anagen and telogen hair, however the equipment used was large and expensive. (unpublished observation)

The study was conducted on 10 healthy volunteers (5 males and 5 females), and the hair was plucked from all four regions of the scalp. The mean force required to pluck anagen hair and telogen hair over frontal region was 0.63N and 0.45N respectively (the force in grams was converted to N using the formula 1grm = 0.0098N). The mean SD was significant (p value – 0.000). Over the occipital region, the mean force of anagen and telogen hair was 0.44N and 0.26N respectively. The mean SD was significant (p value was 0.002).

However our study was conducted on patients with female pattern hair loss, and it was found that, the mean force required to pluck anagen and telogen hair over frontal region was 0.51N and 0.71N respectively, which wasnot statistically significant (p value – 0.152). Similarly, the mean

force required to pluck anagen and telogen hair over occipital region was 0.61N and 0.51N respectively, however the results did not reach its statistical significance.

In our previous study, the mean force required to pluck anagen hair and telogen hair in females was 0.51N and 0.32N respectively. The mean SD was statistically significant in the previous study. Whereas, in the present study, the mean EF required to pluck telogen hair (frontal and occipital) was 1.15N, while that for anagen hair was 1.12N. The mean SD required to epilate telogen hair and anagen hair was 1.19±.47, and 1.12±.56 respectively (p value - 0.659, was not significant). The mean EF required to epilate the telogen hair (mean SD- .51±.31) in occipital region was low when compared to anagen hair (mean SD- .61±.34). The results, however, did not reach the statistical significance (p value - 0.333).

In our previous study, we had a large equipment, which was not mobile and patient friendly, but had significant data. But our current equipment, though the results were not statistically significant, the instrument was portable, user friendly, and can also be used for outpatient purposes for a rapid analysis.

With this hand held trichotillometer, similar study was conducted in our department,on 30 healthy volunteers (15 males and 15 females). The aim of the study was to evaluate the epilation force in normal individuals. The hair was plucked from all four regions of the scalp.According to this study, the mean epilation force was high in occipital region, when compared with other regions. The mean EF required to epilate the anagen hair was 0.82N and for telogen hair it was 0.48N, however the results were not statiscally significant (awaited publication).

Varied amount of force is required to the epilate hair in different areas of scalp, in different stages of hair cycle and in different ethnic groups. Because of this biologically varying properties, the EF in each person is different. This is further responsible for different EF in our study.

A.A. El-Rifaie et al., conducted a study on 622 healthy volunteers, to acquire a normal standard value of the shear strains of different follicles by determining the follicular surface area and force required to epilate each hair follicle over various regions of the scalp. In their study, the mean shear strain for anagen hair and telogen hair in frontal region was 60.47±.21.47 and 36.79±20.40 respectively (measured in g/mm^3). Similarly the mean shear strain for anagen and telogen hair in occipital region was 61.52±22.46 and 38.44±23.43 respectively.[105]

Similar studies were conducted using the trichotillometer, to evaluate the nourishment status in adults.[9,106] Chase et al., conducted a study on 17 adults patients who had features of protein energy malnutrition. The patients were selected based on clinical and laboratory criteria. The study population included adult patients affected with marasmus, kwashiorkor and combination of both forms of protein energy malnutrition.

The aim of their study was to assess the force required to pluck the hair amongst the study population and it was compared with healthy individuals. The plucking force had significant correlation with that of serum albumin, hair shaft diameter, triceps skin fold, arm muscle circumference, weight, hematocrit and beta carotene.[9]There was no significant correlation between the epilation force and vitamin status[9.]

Similar study using trichotillometer, was performed on South American and African children affected with protein energy malnutrition. The aim of the study was to evaluate the relationship between the hair shaft diameter and the epilation force. It was found that, there was a considerable decrease in the hair shaft diameter and the epilation force in the study population.[108,109]

Conclusion:

We conclude that, Trichotillometer is a simple, portable and easy to use device which can be used in the OPD for the diagnosis of pattern hair loss. We can also compare the epilation force of anagen hair, before and after the treatment in diseased patients with the help of this equipment to find the effectiveness of the given hair loss treatment.

Limitations

Although the research has reached its aims, there were some unavoidable limitations.The study was conducted on a small size of population, because of the time limit.Therefore, to generalize the results for larger groups, the study should have involved more participants at different parameters like age, sex and hair follicle diameter.

FPHL may begin as early as puberty, but in this study, the age group was restricted and did not include adolescent patients.

FPHL usually affects the frontal region, however diffuse hair shedding is also possible, but the measured epilation force was compared only over two regions, frontal and occipital.

Since it was a hospital based study, control group were not included, as it was difficult to recruit healthy female volunteers for the study.The measured epilation force of anagen and telogen hair,could have been compared with other parameters of female pattern hair loss.

BIBLIOGRAPHY

1. A. G. Messenger and R. Sinclair, "Follicular miniaturization in female pattern hair loss: clinicopathological correlations," British Journal of Dermatology, Vol.155, No.5, pp.926–930,2006.

2. V. H. Price, "Androgenetic alopecia in women," Journal of Investigative Dermatology Symposium Proceedings, vol.8,no.1,pp.24–27,2003.

3. N.Atanaskova Mesinkovska and W.F.Bergfeld, "Hair: what is new in diagnosis and management? Female pattern hair loss update: diagnosis and treatment," Dermatologic Clinics, vol.31, no.1, pp.119–127, 2013

4. Orentreich N. Scalp hair replacement in man. In: Montagna W and Dobson RL, eds. Advances in biology of the skin, Vol. IX. Oxford: Pergamon Press; 1969: 99–108. 2. Guarrerra M, Semino MT, Rebora A. Quantitating hair loss in women: a critical approach. Dermatology 1997; 194:12–16.

5. Guarrerra M, Semino MT, Rebora A. Quantitating hair loss in women: a critical approach. Dermatology 1997;194:12–16.

6. Barman JM, Astore I, Pecoraro V. The normal trichogram of the adult. J Invest Dermatol 1965;44:233–236.

7. Rushton H, James KC, Mortimer CH. The unit area trichogram in the assessment of androgen-dependent alopecia. Br J Dermatol 1983;109: 429–437.

8. Uno H, Adachi K, Montagna W. Morphological and biochemical studies of hair follicle in common baldness of stumptailed macaque (Macacaspeciosa). In: Montagna W and Dobson RL, eds. Advances in 1biology of the skin, Vol. IX. Oxford: Pergamon Press;1969:221–241.

9. Emily S. Chase, Roland L. Weinsier, George T. Lavenetal. Trichotillometry: the quantitation of hair pluckability as a method of nutritional assessment. The American Journal of Clinical Nutrition 1981;34:2280-2286.

10. Narisawa Y, Kohda H. arrecctorpilimusclessurround human facial vellus hair follicles. Br J Dermatol 1993;129:138-9.

11. Poblet E, Ortega F, Jimenez F. The arrectorpili muscle and the follicular unit of the scalp: a microscopic anatomy study. *DermatolSurg* 2002; 28: 800-3.

12. Cotsarelis G, Sun TT, Lavker RM. Label-retaining cells reside in the bulge area of pilosebaceous unit: implications for follicular stem cells, hair cycle, and skin carcinogenesis. Cell 1990;61:1329-37

13. Oshima H, Rochat A, Kedzia C et al. Morphogenesis and renewal of hair follicles from adult mutipotent stem cells. Cell 2001;104:233-45

14. Oliver RF. Whisker growth after removal of the dermal papilla and lengths of follicle in the hooded rat. *J EmbryolExpMorphol*1967;16: 231-44

15. Oliver RF. The experimental induction of whisker growth in the hooded rat by implantation of dermal pappilae. *J EmbryolExpMorphol* 1967;18 43-51.

16. Oliver RF. The induction of hair follicle formation in the adult hooded rat by vibrissa dermal papilla. *J EmbryolExpMorphol* 1970;23:219-36.

17. Jahoda CA, Horne KA, Oliver RF. Induction of hair growth by implantation of cultured dermal papilla cells. *Nature* 1984;311:560-2.

18. Van Scott EJ, Ekel TM. Geometric Relationships between the matrix of the hair bulb and its dermal papilla in normal and alopecic scalp. *J Invest Dermol*1958;31:281-7.

19. Reynolds AJ, Lawrence C, Cserhalmi-Friedman PB et al. Trans-gender induction of hair follicles. Nature 1999; 402: 33-4.

20. Wolfram LJ. Huma hair: a unique physicochemical composite. J Am Acad Dermatol 2003; 48: S106-S114.

21. Randall VA, Botchkareva NV. The biology of hair growth. In: Ahluwalia GS, ed. Cosmetic Application of Laser and Light-Based System. Norwich, NY: William Andrew Inc., 2009: 3–35.

22. Dhurat RP, Deshpande DJ. Loose anagen hair syndrome. Int J Trichology 2010;2:96-100.

23. Paus R, Cotsarelis G. The biology of hair follicles. N Engl J Med. 1999;341:491-7.

24. Pereira JM. Alopecia Androgenética (Calvície) naMulher. 2. São Paulo: Di Livros; 2007.

25. Shrivastava SB. Diffuse hair loss in an adult female: Approach to diagnosis and management. Indian J Dermatol Venereol Leprol 2009;75:20-8.

26. Sinclair R.Male pattern androgenetic alopecia. BMJ 1998;317:865-9

27. Nyholt DR, Gillespie NA, Health AC et al. genetic basics of male pattern balding. *J Invest Dermatol*2003;121:1561-4.

28. Hamilton JB. Male hormone stimulation is responsible and an incident in common baldness. *Am J Anat* 1942;71:451-80.

29. Hayakawa K, Shimizu T, Ohba Y et al. Interpair differences of physical aging and longevity in identical twins. *Acta Genet Med Gemellol (Roma)* 1998;41:177-85.

30. Rexbye H, Peterson I, Iachina M et al. Hair loss among elderly men: etiology and impact on perceived age. *J Gerontol A BiolSci* 2005;60: 1077-82.

31. Heading pattern hair loss in a randomized study in identical twins, Eur J Dermatol 2002; 12: 32-37.

32. Courtis M, Loussouaran G, Hourseau C. Aging and hair cycles. *Br J Dermatol* 1995;132:86-93

33. Whiting DA. Diagnostic and predictive value of horizontal sections of scalp biopsy specimens in male pattern androgenetic alopecia. *J Am Acad Dermatol* 1993; 28: 755-63.

[34.] Kligman AM. Pathologic dynamics of hair loss.1. Telogen effluvium. Arch Dermatol.-1961;83:175-98.

35. Headington JT. Telogen effluvium: New concepts and views. Arch Dermatol 1993;129:356-63.

36. Feidler VC, Hafeez A. Diffuse alopecia: telogen hair loss. In: Olsen EA, editor.

37. Disorders of hairs growth. Diagnosis and treatment. 2nd ed. New york: McGraw- Hill;2003.p.301-21.

38. Olsen EA. Female pattern hair loss. J Am Acad Dermatol.2002;45:70-80

39. Girman CJ, Hartmaier S, Roberts J, Bergfeld W, Waldstreicher J. Patient-perceived importance of negative effects of androgenetic alopecia in women. J Women's Health Gend Based Med 1999;8:1091-5.

40. Orme S, Cullen DR, Messenger AG. Diffuse female hair loss: are androgens necessary? Br J Dermatol 1999;141:521-23.

41. U. Blume-Peytavi, A. Blumeyer, A. Tosti et al., "S1 guideline for diagnostic evaluation in androgenetic alopecia in men, women and adolescents," *British Journal of Dermatology*, vol. 164, no. 1, pp. 5–15, 2011.

42. E. Ludwig, "Classification of the types of androgenetic alopecia (common baldness) occurring in the female sex" British Journal of Dermatology, vol. 97 no. 3, pp. 247-254, 1977.

43. R.Sinclair, D.Jolley, R.Mallari, and J.Magee, "There liability of horizontally sectioned scalp biopsies in the diagnosis of chronic diffuse telogen hair loss in women," Journal of the American Academy of Dermatology,vol.51,no.2,pp.189–199,2004.

44. E.A. Olsen, "Current and novel methods for assessing efficacy of hair growth promoters in pattern hair loss," Journal of the AmericanAcademyofDermatology,vol.48,no.2,pp.253–262, 2003

45. J. B. Hamilton, “Patterned loss of hair in man; types and incidence,” Annals of the NewYork Academy of Sciences, vol.53, no.3,pp.708–728,1951.

46. Wang TL, Zhou C, Shen YW, Wang XY, Ding XL, Tian S, et al. Prevalence of androgenetic alopecia in China: A community-based study in six cities. Br J Dermatol 2010;162:843-7.

47. O. T. Norwood, “Incidence of female androgenetic alopecia(female pattern alopecia),” *Dermatologic Surgery,* vol. 27, no. 1, pp.53–54,2001

48. Tosti A, Piraccini BM. Androgenetic alopecia. In: Tosti A, Piraccini BM, editors. Diagnosis and treatment of hair disorders: An evidence based atlas London: Taylor and Francis; 2006. p.45-56.

49. Pereira JM. Propedêutica das doenças dos cabelose do courocabeludo. Rio de Janeiro: Atheneu;2001.

50. M.P.Birch, J.F.Messenger, and A.G.Messenger, “Hairdensity, hair diameter and the prevalence of female pattern hair loss,” British Journal of Dermatology,vol.144,no.2,pp.297–304,2001.
51. Leavitt M. Understanding and management of female pattern alopecia. Facial Plast Surg. 2008;24(4):414-27.

52. BirchJ. H.Paik, J.-B.Yoon, W.-Y.Sim, B.-S.Kim, and N.-I.Kim, “The prevalence and types of androgenetic alopecia in Korean men and women,”BritishJournalofDermatology,vol.145,no.1,pp. 95–99,2001.

53. Lee AT, Zane LT. Dermatologic manifestations of polycystic ovary syndrome. Am J ClinDermatol. 2007; 8: 201-19.

54. Kim BJ, Kim JY, Eun HC, Kwon OS, Kim MN, Ro BI. Androgenetic alopecia in adolescents: a report of 43 cases. J Dermatol. 2006;33:696-9.

55. Madnani N, Khan K, Chauhan P, Parmar G. Polycystic ovarian syndrome. Indian J DermatolVenereolLeprol. 2013;79:310-21.

56. Dalamaga M, Papadavid E, Basios G, Vaggopoulos V, Rigopoulos D, Kassanos D, et al. Ovarian SAHA syndrome is associated with a more insulin-resistant profile and represents an independent risk factor for glucose abnormalities in women with polycystic ovary syndrome: A prospective controlled study. J Am Acad Dermatol. 2013;69:922-30.

57. Ding EL, Song Y, Manson JE, Hunter DJ, Lee CC, Rifai N, et al. Sex hormone binding globulin and risk of type 2 diabetes in women and men. N Engl J Med. 2009;361:1152-63

58. Kishimoto J, Burgeson RE, Morgan BA. Wntsignaling maintains the hair-inducing activity of the dermal papilla. Genes Dev. 2000;14:1181-5.

59. D.A.Whiting, "Possible mechanisms of miniaturization during androgenetic alopecia or pattern hair loss," Journal of the American Academy of Dermatology, vol. 45, supplement 3, pp. S81–S86,2001.

60. P. Vexiau, C. Chaspoux, P. Boudou et al., "Role of androgens in female-pattern androgenetic alopecia, either alone or associated with other symptoms of hyper androgenism," Archives of Dermatological Research,vol.292,no.12,pp.598–604,2000.

61. E.A.Olsen, Disorders of Hair Growth: Diagnosis and Treatment, vol.12,McGraw-Hill,NewYork,NY,USA,1994

62. Imperato-McGinley J, Guerrero L, Gautier T, Peterson RE. Steroid 5α-reductase deficiency in man: an inherited form of male pseudohermaphroditism. Science. 1974;186:1213-5.

63. M.E.Sawayaand V.H. Price, "Different levels of 5α-reductase type I and II, aromatase, and androgen receptor in hair follicles of women and men with androgenetic alopecia," Journal of Investigative Dermatology,vol.109,no.3,pp.296–300,1997.

64. Sawaya ME, Blume-Peytavi U, Mullins DL, Nusbaum BP, Whiting D, Nicholson DW, et al. Effects of finasteride on apoptosis and regulation of the human hair cycle. J Cutan Med Surg. 2002;6:1-9.

65. Jahoda CA. Cellular and developmental aspects of androgenetic alopecia. Exp Dermatol. 1998;7:235-48

66. Gupta AK, Charrette A. The efficacy and safety of 5alpha-reductase inhibitors in androgenetic alopecia: a network meta-analysis and benefit-risk assessment of finasteride and dutasteride. J Dermatolog Treat. 2014;25:156-61

67. Eun HC, Kwon OS, Yeon JH, Shin HS, Kim BY, Ro BI,, et al. Efficacy, safety, and tolerability of dutasteride 0.5 mg once daily in male patients with male pattern hair loss: a randomized, double-blind, placebo-controlled, phase III study. J Am Acad Dermatol. 2010;63:252-8

68. L. Yip, N. Rufaut, and R. Sinclair, "Role of genetics and sex steroid hormones in male androgenetic alopecia and female pattern hair loss: an update of what we now know," Australasian Journal of Dermatology,vol.52,no.2,pp.81–88,2011

69. :Kaliyadan F, Nambiar A, Vijayaraghavan S. Androgenetic alopecia: An update. Indian J Dermatol Venereol Leprol 2013;79:613-25.

70. K¨uster and R. Happle, "The inheritance of common baldness: two Bornottwo B?" Journal of the American Academy of Dermatology,vol.11,no.5,part1,pp.921–926,1984.

71. S. Redler, F. F. Brockschmidt, R. Tazi-Ahnini et al., "Investigation of the male pattern baldness major genetic susceptibilityloci AR/EDA2R and 20p11 in female pattern hair loss," British JournalofDermatology,vol.166,no.6,pp.1314–1318,2012.

72. Ellis JA, Stebbing M, Harrap SB. Insulin gene polymorphism and premature male pattern baldness in the general population. ClinSci 1999; 96: 659-62.

73. Bergfeld WF. Androgenetic alopecia: an autosomal dominant disorder. Am J Med. 1995;98:95S-98S.

74. Yazdan P. Update on the genetics of androgenetic alopecia, female pattern hair loss, and alopecia areata: implications for molecular diagnostic testing. SeminCutan Med Surg. 2012;31:258-66.

75. Lee R, Chang SY, Trinh H, Tu Y, White AC, Davies BS, et al. Genetic studies on the functional relevance of the protein prenyltransferases in skin keratinocytes. Hum Mol Genet. 2010;19:1603-17.

76. Ellis JA, Stebbing M, Harrap SB. Genetic analysis of male pattern baldness and the 5alpha-reductase genes. J Invest Dermatol. 1998;110:849-53.

77. Birch MP, Messenger AG. Genetic factors predispose to balding and non-balding in men. Eur J Dermatol. 2001;11:309-14

78. Ellis JA, Harrap SB. The genetics of androgenetic alopecia. Clin Dermatol Clin Dermatol. 2001;19:149-54

79. Ellis JA, Stebbing M, Harrap SB. Polymorphism of the androgen receptor gene is associated with male pattern baldness. J Invest Dermatol. 2001;116:452-5.

80. Yip L, Zaloumis S, Irwin D, Severi G, Hopper J, Giles G, et al. Gene-wide association study between the aromatase gene (CYP19A1) and female pattern hair loss. Br J Dermatol. 2009;161:289-94.

81. Li R, Brockschmidt FF, Kiefer AK, Stefansson H, Nyholt DR, Song K, et al. Six novel susceptibility Loci for early-onset androgenetic alopecia and their unexpected association with common diseases. PLoS Genet. 2012;8:1002746.

82. Redler S, Dobson K, Drichel D, Heilmann S, Wolf S, Brockschmidt FF, et al. Investigation of six novel susceptibility loci for male androgenetic alopecia in women with female pattern hair loss. J Dermatol Sci. 2013;72:186-8.

83. Heilmann S, Kiefer AK, Fricker N, Drichel D, Hillmer AM, Herold C, et al. Androgenetic alopecia: identification of four genetic risk loci and evidence for the contribution of WNT signaling to its etiology. J InvestDermatol. 2013;133:1489-96.

84. Nuwaihyd R, Redler S, Heilmann S, Drichel D, Wolf S, Birch P, et al. Investigation of four novel male androgenetic alopecia susceptibility loci: no association with female pattern hair loss. Arch Dermatol Res. 2014;306:413-8.

85. Gatherwright J, Liu MT, Gliniak C, Totonchi A, Guyuron B. The contribution of endogenous and exogenous factors to female alopecia: a study of identical twins. Plast Reconstr Surg. 2012;130:1219-26.

86. R. M. Tr¨ueb, "Is androgenetic alopecia a photo aggravated dermatosis?" Dermatology,vol.207,no.4,pp.343–348,2003

87. L. H. Su and T. H. Chen, "Association of androgenetic alopecia with smoking and its prevalence among Asian men: a community-based survey," Archives of Dermatology,vol.143,no.11,pp.1401–406,2007.

88. Lee WS, Oh Y, Ji JH, Park JK, Kim do W, Sim WY, et al. Analysis of familial factors using the basic and specific (BASP) classification in Korean patients with androgenetic alopecia. J Am Acad Dermatol. 2011;65:40-7

89. Lee WS, Lee HJ, Choi GS, Cheong WK, Chow SK, Gabriel MT, et al. Guidelines for management of androgenetic alopecia based on BASP classification--the Asian Consensus Committee guideline. J Eur Acad Dermatol Venereol. 2013;27:1026-3

90. Lee WS, Ro BI, Hong SP, Bak H, Sim WY, Kim do W, et al. A new classification of pattern hair loss that is universal for men and women: basic and specific (BASP) classification. J Am Acad Dermatol. 2007;57:37-46.

91. Pelfini C, Calligaro A. Some notes on the evaluation of hair growth by means of morphometric computerized analysis. J Appl Cosmetol 1986; 4: 67–76.

92. Van Neste DJJ, Dumortier M, De Brouwer B, De Coster W. Scalp immersion proxigraphy (SIP): an improved imaging technique for phototrichogram analysis. J Eur Acad Dermatol Venereol 1992; 1: 187–191.

93. Hoffman R. TrichoScan: combining epiluminescence microscopy with digital image analysis for the measurement of hair growthin vivo. Eur J Dermatol 2001; 11: 362–368.

94. Epstein WL, Maibach HI. Cell proliferation and movement in human hair bulbs. In: Montagna W and Dobson RL, eds. Advances in biology of the skin, Vol. IX. Oxford: Pergamon Press; 1969: 83–97.

95. Alcaraz MV, Villena A. Perezde Vargas I. Quantitative study of the human hair follicle in normal and androgenetic alopecia. J CutanPathol 1993; 20: 344–349.

96. Ekmekci TR, Koslu A. Phototrichogram findings in women with androgenetic alopecia. Skin Res Technol. 2006;12:309-12.

97. A. F. Pedrosa, P. Morais, C. Lisboa, and F. Azevedo, "The importance of trichoscopy in clinical practice," Dermatology Research and Practice, vol. 2013, Article ID 986970, 3 pages, 2013.

98. A.Tosti, M.Iorizzo, and B.M.Piraccini, "Androgenetic alopecia in children: report of 20 cases," British Journal of Dermatology, vol.152,no.3,pp.556–559,2005.

99. O.deLacharri`ere, C.Deloche, C.Miscialietal., "Hairdiameter diversity: a clinical sign reflecting the follicle miniaturization," Archives of Dermatology,vol.137,no.5,pp.641–646,2001.

100. M.Guarrera, M.T.Semino, and A.Rebora, "Quantitating hair lossinwomen:acriticalapproach,"Dermatology,vol.194,no.1, pp.12–16,1997.

101. K. Hillmann and U. Blume-Peytavi, "Diagnosis of hair disorders,"Seminars in Cutaneous Medicine and Surgery,vol.28,no. 1,pp.33–38,2009.

102. R.Sinclair, D.Jolley, R.Mallarietal., "Morphological approach to hair disorders," Journal of Investigative Dermatology Symposium Proceeding,vol.8,no.1,pp.56–64,2003.

103. D.M. Elston, M.L. Mc Collough, and V.L.Angeloni, "Vertical and transverse sections of alopecia biopsy specimens: combining the two to maximize diagnostic yield," Journal of the American Academy of Dermatology,vol.32,no.3,pp.454–457, 1995.

104. V. Pecoraro, I. Astore, J. Barman, and C. Ignacio Araujo, "The normal trichogram in the child before the age of puberty," The Journalofinvestigativedermatology,vol.42,pp.427–430,1964

105. A. A. El. Rifaie et al., "The trichotillometry: a technique for hair assessment", Skin and Research and Technology, 2016; 22: 15-19.

106. Smelser DN, Smelser NB, Krumdieck CL, Schreeder MT, Laven GT: Field use of hair epilation force in nutrition status assessment. *Am J ClinNutr*1982, 35:342-346

107. BarbarraBuffoli et al., "The human hair: from anatomy to physiology", International Journal of Dermatology, 2013

108. DawberRP,deBeker D, Wojnarowaska F. Disorders of hair: loose anagen syndrome. In: Champion RH, Burton JL, Ebling FJ ed. Textbook of Dermatology .BlackwellScientific Publication, 1998;66:2955.'

109. Sperling LC, Mezebish DS. Hair diseases. Med Clin North Am 1998;82:1155-69.

110. Messinger ML, Cheng TL. Trichotillomania. Pediatr Rev 1999;20:249-50.

111. Alsafwah S, Alzein M. Small bowel obstruction due to trichobezoar: role of upper endoscopy in diagnosis. GastrointestEndosc 2000;52:784-6.

112. Kemna ME, Elewski BE. A U.S. epidemiologic survey of superficial fungal diseases. J AmAcadDermatol 1996;35:539-42.

113. Linda V Spencer, Jefferey P Callen. Hair loss in systemic diseases. DermatolClin 1987; 5: 565-70.

114. Ruth Freinkel, Norbert Freinkel. Hair growth and alopecia in hyperthyroidism. Arch Derm 1972; 349-352.

115. Rodney Sinclair. Diffuse hair loss. Int J of Deramtol 1999; 38(suppl):8-18

116. Bortosova L, Stava Z, Jorda V. clinical trichology. In: Mail JWH, editor.Current problems in dermatology. Diseases of hair and the scalp. Sydney: Karger, 1984; 12: 224-238.

117. Lee J Y-Y, HSU M-L. Alopecia syphylitica, a simulator of alopecia areata: histopathology and differential diagnosis. J CutanPathol 1991; 18: 87-92.

118. Headington JT. Cicatricial alopecia. DeramtolClin 1996; 14: 773-82.

119. D. Wasserman, D. A. Guzman-Sanchez, K. Scott and A. mCmICHAEL, "Alopecia Areata", Int J of Dermatol, vol. 46, No. 2, 2007, pp. 121-131.

120. H. Roe, "Chemotherapy-induced alopecia: Advice and Support for hair loss," British Journal of Nursing, Vol.20,No.10, 2011,pp. S4-S11.

121. N. Shoefeld, O. Rosenberg, M. Kotler and P. N. Dannon, "Trichotillomania: Pathophysiology Theories and Treatment Possibilities," The Istrael Medical Association Journal, Vol ., No. 2, 2012, PP. 125-129.

122. W. F. Bergfeld and F. Mulinari-Brenner, "Shedding: how to manage a common Cause of hair loss," Clevland Clinic Journal of Medicine, Vol. 68, No. 3, 2001, pp. 256-261.

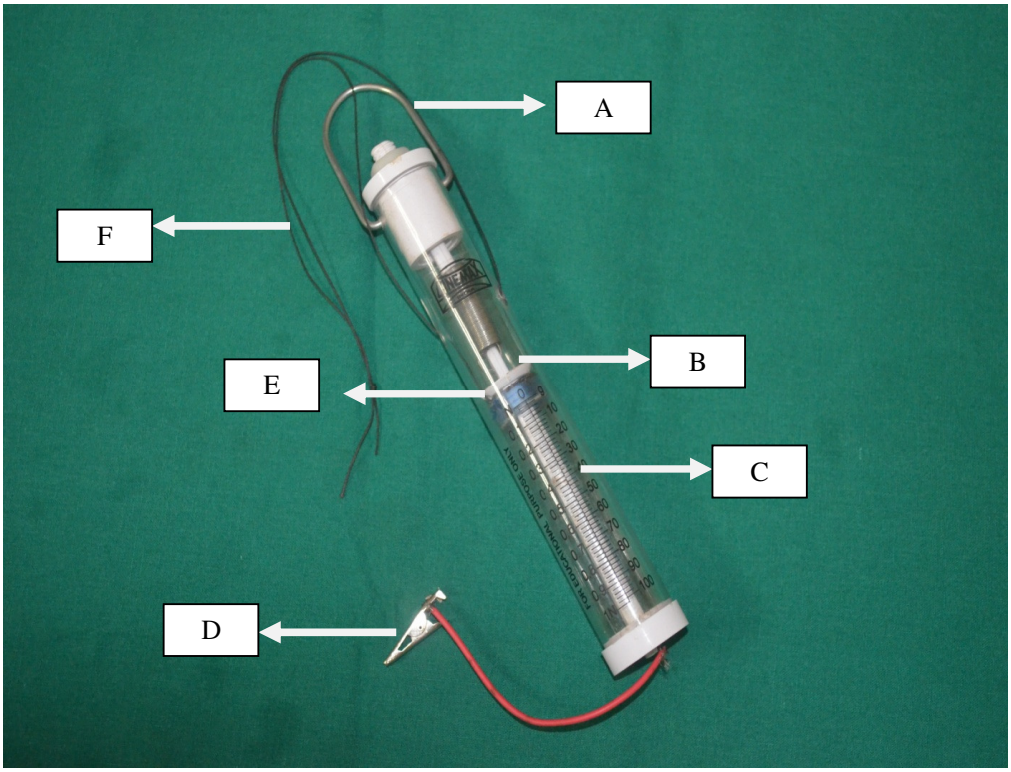
123. R. Paus, V. A. Botchkarev, N. V. Botchkareva, L. Merklenburg, T. Leger and A. Solminski, The skin POMC System (SPS). Leads and Lessons from the Hair Follicle," Annals of the New York Academy of Sciences, Vol. 885, 1999, pp. 350-363.

124. J. H. Eastham, "Postpartum Alopecia," The Annals of Pharmacotherapy, Vol. 35, No. 2, 2001, pp. 255-258.

125. L. Millikan, “Hirsutism, Postpartum Telogen Effluvium, and Male Pattern Alopecia,” Journal of Cosmetic Dermatol, Vol. 5, No. 1,2006, pp. 81-86.

CLINICAL PHOTOGRAPHS

HAND HELD TRICHOTILLOMETER



A – Thumb Ring

B – Extension Rod

C – Calibrated scale

D – Gripper

E – Force indicator

F – Cords (for adjustment)

GRIPPER ATTACHED TO THE DISTAL END OF A SINGLE HAIR SHAFT

SPRING BALANCE PULLED UPWARDS GENTLY AT THE TOP END WITH THE HELP OF A THUMB RING

CONSENT FORMS

PSG Institute of Medical Science and Research, Coimbatore

INFORMED CONSENT

I , **SUBHA RANJANI K**, am carrying out a study on the topic:

MEASURING THE EPILATION FORCE OVER THE FRONTALAND OCCIPITAL REGION IN CASES OF FEMALE PATTERN HAIR LOSS USING HAND HELD TRICHOTILLOMETER as part of my research project being carried out under the aegis of the Department of : DERMATOLOGY VENEROLOGY & LEPROLOGY.

My Research Guide is: Dr. C.R.SRINIVAS.

The justification for this study is:

Trichotillometer, a simple non invasive technique, is a hand held device, used to determine the epilation force of hair. Previous data showed that this procedure can be used to assess the nutritional status of patients. Androgenetic alopecia is a common problem faced by women. Patients usually present with hair loss predominantly involving the crown, frontal region. Trichotillometer can be used to determine the epilation force of frontal hair and it can be compared with other sites.

The objectives of this study are:

To determine the epilation force (EF) required to pluck the anagen hair and telogen hair from frontal and occipital region of the scalp in female pattern hair loss.

Study participants:

Female patients, age more than 20 years, who are diagnosed to have female pattern hair loss (based on Ludwig classification- only class 2).

Location: PSGIMS & R

We request you to kindly cooperate with us in this study. We propose to collect background information and other relevant details related to this study. We will be carrying out:

Initial interview (specify approximate duration): 15 minutes.

Data collected will be stored for a period of 10 years. We will not use the data as part of another study.

Clinical examination will be done to assess if any active lesions are present in the body.

Procedure:

Hair will be plucked from frontal and occipital region using hand held 114elogen114lometer.

Benefits from this study: Epilation force of anagen and telogen hair can be compared in both the regions

Risks involved by participating in the study: very rarely redness, irritation can occur at the epilated site.

How the **results** will be used:
We can assess the effectiveness of new upcoming hair loss treatment kits.

If you are uncomfortable in answering any of our questions during the course of the interview / biological sample collection, **you have the right**

to withdraw from the interview / study at anytime. You have the freedom to withdraw from the study at any point of time. Kindly be assured that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to a patient. You will **NOT** be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, – whether directly related to you or to other participants of this study, developed during the course of this research which may relate to your willingness to continue participation.

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the PI with date: Witness:

Contact number of PI: 9003934051

Contact number of Ethics Committee Office: 0422 2570170 Extn.: 5818

ஒப்பதல் படிவம்

தேதி

கா. சுபரஞ்சனி ஆகிய நான் PSG மருத்துவக்கல்லூரியின் **தோல் பால்வினை மற்றும் தொழுநோய்** துறையின் கீழ் **தலையில் இருந்து கூந்தலை பிடுங்குவதற்கு தேவையான திறனை பற்றிய ஆய்வு (பெண்கள் தலையில் முடி உதிர்தல்-வழுக்கை)** என்ற தலைப்பில் ஆய்வு மேற்கொள்ள உள்ளேன்

என் ஆய்வு வழிகாட்டி : **சி.ஆர். ஸ்ரீநிவாஸ்**

ஆய்வு மேற்கொள்வதற்கான அடிப்படை:

டிரைகோடீலோமீட்டர் எனும் கருவி தலையில் இருந்து கூந்தலை பிடுங்குவதற்கு தேவையான திறனை அளவிடும். இதை வியாதி இல்லாத நிலையிலும் வியாதியின் போதும் உபயோகிக்கலாம். ஓய்வு நிலையில் (Telogen Hair) உள்ள கூந்தலை விட வளரும் நிலையில் (Anagen Hair) உள்ள கூந்தலை தலையில் இருந்து பிடுங்குவதற்கு அதிக பழு தேவைப்படும். தலையில் இருந்து கூந்தலை பிடுங்குவதற்கு தேவையான திறனை அளவிடும் கருவியின் மூலம் உடல் பரிசோதனை இல்லாமல் மருத்துவத்தின் பயன்பாட்டினை அளவிடமுடியும்.

ஆய்வின் நோக்கம் :

தலையில் இருந்து கூந்தலை பிடுங்குவதற்கு தேவையான திறனை பற்றிய ஆய்வு.

ஆய்வு மேற்கொள்ளும் இடம் : **பி.எஸ்.ஜி மருத்துவமனை, தோல் பால்வினை மற்றும் தொழுநோய் துறை**

பரிசோதனைக்கு உட்படுத்தப்பட்டவர்களின் எண்ணிக்கை : 30

வயது வரம்பு : 20 வயதிற்கு மேல்

இடம் : தோல், பால்வினை மற்றும் தொழுநோய்துறை
பி.எஸ்.ஜி மருத்துவமனை, கோயம்புத்தூர்

இந்த ஆய்வுக்கு எங்களுக்கு ஒத்துழைப்பைத் தருமாறு தங்களைக் கேட்டுக் கொள்கிறோம். மேலும் இந்த ஆய்வுக்குத் தேவையான தகவல்கள் உங்களிடமிருந்து பெற்றுக் கொள்ளப்படும்.

ஆய்வுபற்றிய விளக்கப்படம் : -

உடல் பரிசோதனை : இல்லை.

பரிசோதனையின் போது ஏற்படும் பக்கவிளைவுகள் தோல் : சிவந்துபோதல், எரிச்சல்

கொடுக்கப்பட்ட எடுத்துக் கொள்ளப்பட்ட மருந்துகள், அதன் பயன்கள் மற்றும் பக்கவிளைவுகள்

- ஸ்டீராய்டு களிம்புகள் : தோல் சிவந்துபோதலை குறைக்கும்
- ஆன்டிபயாட்டிக் களிம்புகள் : தேவைப்பட்டால்

புகைப்படம் எடுக்கப்பட்டதா? அதன் நோக்கம் :

பரிசோதனைக்கு முன்பும், பின்பும் உள்ள மாற்றத்தை ஒப்பிட

இந்த ஆய்வில் பங்கேற்க ஒப்புக் கொள்ளுவதால் எந்தவிதமான பலனும் உங்களுக்கு கிடைக்காது. எந்த நேரத்தில் வேண்டுமானாலும் ஆய்விலிருந்து விலகிக் கொள்ளும் உரிமை உங்களுக்கு உண்டு.

ஆய்விலிருந்து விலகிக் கொள்வதால் உங்களுக்கு அளிக்கப்படும் சிகிச்சையில் எந்த வித மாற்றமும் இருக்காது.

மேலும் இந்த ஆய்வில் பங்கு கொள்வது உங்கள் சொந்த விருப்பம். இதில் எந்த விதக் கட்டாயமும் இல்லை. நீங்கள் விருப்பப்பட்டால் இந்த ஆய்வின் முடிவுகள் உங்களுக்குத் தெரியப் படுத்தப்படும்.

ஆய்வாளரின் கையொப்பம் :

தேதி :

ஆய்வுக்குட்படுபவரின் ஒப்புதல்

நான் இந்த ஆராய்ச்சியின் நோக்கம் மற்றும் அதன் பயன் பாட்டினைப் பற்றி தெளிவாகவும் விளக்கமாகவும் தெரியப்படுத்தப்பட்டுள்ளேன். இந்த ஆராய்ச்சியல் பங்கு கொள்ளவும் இந்த ஆராய்ச்சியின் மருத்துவ ரீதியான குறிப்புகளை வரும் காலத்திலும் உபயோகப்படுத்திக் கொள்ளவும் முழு மனதுடன் சம்மதிக்கிறேன்.

ஆய்வுக்குட்படுபவரின் பெயர், முகவரி :

கையொப்பம் :

தேதி :

PROFORMA

Name :

Age :

Occupation :

Duration of hair loss :

Medication history :

Treatment history :

Pattern : Ludwig classification type 2

FRONTAL:

EF					
Phase					

Occipital

EF					
Phase					

MASTER CHARTS

TABLE - 1

EF OF ANAGEN AND TELOGEN OVER FRONTAL AND OCCIPITAL REGION

S. No	ANAGEN (N)	TELOGEN (N)
1	0.98	0.784
2	1.176	0.931
3	2.156	0.686
4	0.882	1.274
5	1.274	1.47
6	1.568	0.882
7	2.352	0.98
8	2.156	1.764
9	1.274	1.078
10	1.372	1.96
11	0.686	1.176
12	2.45	0.98
13	1.274	1.47
14	1.274	1.764
15	0.392	2.352
16	0.98	0.98
17	1.274	0.686
18	1.176	0.683
19	0.588	1.078
20	0.49	0.882
21	0.882	0.784
22	0.784	0.88
23	1.47	0.294
24	0.686	1.274
25	0.49	1.274
26	0.49	1.96
27	0.686	0.98
28	0.49	1.966
29	0.98	1.274
30	0.882	1.078

TABLE – 2
EF OVER FRONTAL REGION

S. NO	ANAGEN	TELOGEN
1	0.686	0.294
2	0.294	0.539
3	1.666	0.196
4	0	0.882
5	0.686	0.784
6	0.49	0.686
7	0.784	0.686
8	1.764	0.196
9	0	1.666
10	0.588	1.274
11	0.49	0.588
12	1.568	0.49
13	0.392	0.784
14	0.588	0.98
15	0	1.467
16	0	0.784
17	0.98	0.294
18	0.882	0.196
19	0.294	0.588
20	0	0.49
21	0.294	0.49
22	0.294	0.588
23	0.98	0.098
24	0.686	0.882
25	0	0.784
26	0.49	1.274
27	0.294	0.686
28	0	1.274
29	0.196	0.98
30	0	0.882

Table – 3
EF OVER OCCIPITAL REGION

S. No	ANAGEN	TELOGEN
1	0.294	0.49
2	0.882	0.392
3	0.49	1.372
4	0.882	0.392
5	0.588	0.686
6	1.078	0.196
7	1.568	0.294
8	0.392	1.568
9	1.274	0.294
10	0.784	0.686
11	0.196	0.588
12	0.882	0.49
13	0.882	0.686
14	0.686	0.784
15	0.392	0.882
16	0.98	0.294
17	0.294	0.392
18	0.294	0.49
19	0.294	0.49
20	0.49	0.392
21	0.588	0.294
22	0.49	0.294
23	0.49	0.196
24	0.294	0.392
25	0.49	0.49
26	0	0.686
27	0.392	0.294
28	0.49	0.392
29	0.784	0.294
30	0.588	0.196

ABBREVIATIONS

FPHL	**Female Pattern Hair Loss**
EF	**Epilation Force**
CTE	**Chronic Telogen Effluvium**
TE	**Telogen Effluvium**
DHT	**Dihydrotestosterone**
PCOS	**Polycystic ovarian syndrome**
AR	**Androegen receptor**
OS	**Outer root sheath**
AGA	**Androgenetic Alopecia**