"A CROSS SECTIONAL STUDY TO DETERMINE VARIOUS CAUSES OF DIFFUSE HAIR LOSS IN WOMEN"

Submitted by

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LIST OF ABBREVIATIONS

AE - Anagen effluvium

CTE - Chronic telogen effluvium

FPHL - Female pattern hair loss

TE - Telogen effluvium

TSH - Thyroid stimulating hormone

WHO - World Health Organization

ABSTRACT

Background: Diffuse hair loss is a common complaint encountered by dermatologists in their daily clinical practice. Hair loss in women is a distressing condition. Various underlying factors individually or in combination contribute to the pathogenesis.

Objectives: Objective of the study was to determine various causes of diffuse hair loss in women and to find the correlation between causes of diffuse hair loss and various laboratory parameters.

Method: It is a hospital-based, cross-sectional study. One hundred eighty adolescent girls and women with diffuse hair loss attending the Dermatology, Venereology and Leprosy out patient department of a tertiary care hospital were included in this study. Detailed history was taken from all study subjects with special emphasis upon the major febrile illness, psychological stress or any surgery or child birth 3 months prior to the onset of hair loss. History of chronic blood loss, recent past history of crash diet, detailed history of medication was also recorded.

Specific signs of anemia, jaundice, and thyroid swelling were noted in general clinical examination. Type of hair loss, hair thinning and temporal recession was noted on scalp examination. Hair pull test was performed. Specific laboratory investigations for determining iron deficiency anemia, thyroid dysfunction and parasitic infestations were done.

Results: A total of 180 patients with diffuse hair loss were examined during the study period. Out of 180 patients, 130 (66.6%) had telogen effluvium, 28 (14.3%) had chronic telogen effluvium, 35 (19.4%) had female pattern hair loss and 1 (0.5%) had anagen effluvium. The probable causes of telogen effluvium based on history was

found to be psychological stress in 48 cases, fever in 16, intake of medication in 12, topical application of native medications in 8, crash diet in 8, child birth (postpartum TE) in 7, chronic blood loss in 6, preceding surgery in 3 and worm infestation in 1 case. More than one factor was recorded in 10 cases. In 32 cases probable etiological factors could not be correlated. Out of 130 patients, low hemoglobin level was observedin 50/88 (47.6%), low serum ferritin level in 69/74 (93.24%), hypothyroidism in 2/74 (2.70%) and hyperthyroidism in 3/74 (4.05%). Psychological stress and hemoglobin level were found to be significantly associated with telogen effluvium. Out of 35 patients with FPHL, low hemoglobin level was observed in 6/20 (30%) and low serum ferritin level in 14//17 (82.35%). Relationship between hemoglobin level and FPHL was statistically significant. Out of 28 patients with CTE, low hemoglobin was observed in 10/20 (50%) and low serum ferritin level in 12/17 (70.58%). No significant relationship was observed between CTE, hemoglobin level and serum ferritin level.

Conclusion: Diffuse hair loss is a multifactorial condition. A detailed history, thorough clinical examination and appropriate investigations will help to identify the causative factors and treat them accordingly.

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INTRODUCTION

Hair is an ectodermal structure with great cosmetic importance. It helps an individual to maintain self-image and carry on healthy and fruitful social interactions.[1] Loss of hair becomes a matter of concern in all individuals irrespective of age and sex, especially in females.[2] Normal hair cycle results in replacement of every hair on the scalp by 3-5 years.[3]

Diffuse hair loss is a common complaint encountered by dermatologists in their daily clinical practice.[4] Women present more frequently with this complaint.[5] Diffuse hair loss usually occurs without any inflammation or scarring.[3] There are various causes of diffuse hair loss, which include telogen effluvium, female pattern hair loss, chronic telogen effluvium, anagen effluvium, loose anagen hair syndrome, diffuse type of alopecia areata, congenital atrichia, congenital hypotrichosis and hair shaft abnormalities (hair breakage, unruly hairs).[6]

Hair is essential in identity of many women. Femininity, sexuality, attractiveness, and personality are symbolically linked to woman's hair rather than in men. Women are more likely to have lowered quality of life and restrict social contacts as compared to men as a result of hair loss.[2] Psychiatric disorders are more prevalent in patients with alopecia than in general population, suggesting that those with alopecia may be at higher risk for developing a serious depressive episodes, anxiety disorders, social phobia, or paranoid disorder.[1] Medical treatment for the disorder has limited effectiveness, and a failure to find a cure may leave the patient greatly distressed.[1]

However, hair loss should not be looked upon as merely a complaint of cosmetic concern. This may be a pointer to various systemic illnesses like anemia,

hypothyroidism, hyperthyroidism, chronic infectious diseases, etc. Hence determining the causes of diffuse hair loss is mandatory in all cases.[4] Hair loss in otherwise healthy females presents several challenges like, to identify the cause which may be complicated by more than one factor or to find out the effective treatment and to establish requirements for long term management.[7]

Although diffuse hair loss is a commonly encountered problem, there are only few studies conducted in India and other countries exploring the causes of diffuse hair loss.[7] Various studies have stressed the importance of a detailed history, a thorough examination and appropriate laboratory investigations in a patient of diffuse hair loss to find out all the probable causative factors and treat each of them in an appropriate manner. The diagnosis can usually be established with a detailed history, particularly focusing on the chronology of events, examination of the bulbs of the shed hairs, and a few simple screening blood tests. In chronic cases a scalp biopsy may be required.[7]

This cross sectional study has been conducted to find out causes of diffuse hair loss, so that common etiologies for this condition in this region of India are established. This will be helpful to clinicians, as based on this knowledge patients can be treated empirically in future, if cost and availability of the investigations become a constraint. This will be beneficial for patients as well, as underlying disorders, if any, can be treated.

OBJECTIVES OF STUDY

1. To determine various causes of diffuse hair loss in women.
2. To find the correlation between causes of diffuse hair loss and various laboratory parameters.

REVIEW OF LITERATURE

Scalp hair grows in cycles, with each hair follicle undergoing 10 to 30 cycles in its lifetime. Diffuse hair fall is the result of a disruption of one phase of the hair cycle, i.e., anagen phase (active hair growth), catagen phase (involution), or telogen phase (resting). The anagen phase may last for about 2 to 8 years, the catagen phase lasts 4 to 6 weeks, and the telogen phase lasts for 2 to 3 months. The exogen phase of hair follicle (the release of dead hair) coincides with the end of telogen phase.[8]

Normally, each hair follicle has an independent cycle, so that while some hairs are growing, others are resting and some others are shedding. Thus, the scalp hair density and the total number of scalp hairs remain stable. Most people have about 100,000 scalp hairs, and normally of about 10% to 15% of these are in the telogen phase. Shedding of 100 to 150 telogen hairs/day is normal.[8] Normal hair cycle results in replacement of every hair on the scalp every 3-5 years. In patients with diffuse hair loss symptoms are usually out of proportion to the clinical signs.[4] This is because at least 25% of the 10,000 scalp hairs should be lost to produce noticeable thinning of hair.[4] Of the three major categories of diffuse hair loss, telogen effluvium (TE) is the most common variety followed by female pattern hair loss (FPHL) and chronic telogen effluvium (CTE).[6]

Telogen effluvium

By definition it is a nonscarring, diffuse, hair loss from the scalp that occurs around 3 months after a triggering event and is usually self-limiting, lasting for about 6 months (figure 2). In telogen effluvium hair loss is usually less than 50% of the scalp hair.[9] This condition was first described by Kligman in 1961, as a disease state of hair follicle, where diffuse shedding of telogen hairs is seen.[6] Kligman hypothesized that whatever may be the cause of the hair loss, the follicle tends to

behave in a similar way, in the form of premature termination of anagen. The follicle is precipitated into catagen and transforms into resting stage that mimics telogen.[10] The observation of increased telogen hair shedding does not infer a cause. Establishing etiology of telogen effluvium requires elicitation of relevant history and appropriate laboratory investigations to exclude endocrine, nutritional and autoimmune disorders.[10] A wide variety of potential triggers have been implicated in the pathogenesis of telogen effluvium.[11]

Epidemiology

True incidence of telogen effluvium is not well determined due to lack of data, especially of subclinical cases. One hospital-based study from north India had reported the incidence of telogen effluvium as 92% among female patients presented with diffuse hair loss (n=100).[7]

Etiopathogenesis

Telogen effluvium occurs if a significant number of anagen hairs are triggered to stop growing prematurely by any stimulus and subsequently enter catagen phase, followed by telogen phase. After about 2-3 months of initial insult there is excessive hair shedding. The causes of TE have been presented in table 1.

Table 1: Causes of telogen effluvium

Physiological causes	Postpartum effluvium (telogen gravidarum)
	Physiological effluvium of newborn
Febrile states	Typhoid
	Malaria
	Tuberculosis
	HIV infection
Stress	Severe febrile illness
	Emotional stress
	Serious injuries
	Major surgery
	Difficult labor
	Hemorrhage
	Starvation
	Crash diet
Drugs	Oral retinoids (etretinate and acitretin)
	Oral contraceptives
	Antithyroid drugs
	Anticonvulsants
	Hypolipidemic drugs
	Heavy metals
	Beta blockers[11]
	Captopril[11]
	Amphetamines[11]
Endocrine	Hyperthyroidism
	Hypothyroidism
Organ dysfunction	Renal failure
	Hepatic failure
Disorder of hair cycle	Short anagen syndrome
Nutritional	Iron deficiency anemia
	Acrodermatitis enteropathica
	Acquired zinc deficiency
	Malnutrition
Local cause	Hair dye application
Others	Syphilis
	Systemic lupus erythematosus

The physiological daily shedding of 100-150 telogen club hairs from the scalp is a natural consequence of the hair cycle. Follicles normally retain telogen hair until they have re-entered anagen phase. Eventually the old telogen hair is pushed out by

new anagen hair. This shedding does not produce visible alopecia and does not alter the trichogram.[11]

A temporary alopecia develops as the long telogen hairs are replaced by the shorter new anagen hairs, provided the insult is not repetitive. Alopecia resolves as the new anagen hairs grow.[11] There is no genetic cause for telogen effluvium.[12]

In post-partum TE, follicles remain in prolonged anagen phase rather than cycling into telogen phase. When finally released from anagen, the clinical sign of increased shedding of telogen hair will be found.[12] Headington has described five functional types of telogen effluvium as follows:[12]

1. ***Immediate anagen release:***

It is a common form of telogen effluvium, typically occurring after a period of physiological stress including episodes of high fever. During fever, cytokines drive the hair follicle keratinocytes into apoptosis initiating catagen followed by telogen.[9]

2. ***Delayed anagen release:***

This type of telogen effluvium typically occurs in post-partum hair loss. It is also termed as telogen gravidarum. It occurs due to the high level of circulating placental estrogen which prolongs anagen phase and lead to a full head of hair during pregnancy. The withdrawal of these trophic hormones at delivery causes all the overdue anagen hairs to enter into catagen phase simultaneously. This leads to increased shedding of telogen hair, seen after few months of delivery.[11]

3. ***Immediate telogen release:***

Hair follicles are normally programmed for release of the club hair after an usual interval of 100 days. This results from a shortening of the normal telogen cycle. This type of hair shedding usually occurs 2-8 weeks after initiation of therapy with

topical minoxidil.[4,9] This paradoxical phenomenon occurs because, with the anagen phase being stimulated, there is release of the exogen hairs which were resting.[4]

4. ***Delayed telogen release:***

In this type hair follicles remain in prolonged telogen rather than being shed and recycling into anagen. Clinical sign of increased shedding of club hair is observed when finally teloptosis (termination of telogen phase with hair shedding) sets in. This process underlies mottling in mammals and probably also seasonal shedding of hair in human or mild telogen effluvia which occurs following travel from low-daylight to high-daylight environment.[9]

5. ***Short anagen phase***:

It is characterized by the inability to grow long hair because of an idiopathic short anagen phase. The condition is not associated with hair shaft fragility or hair unruliness. This results in resistant TE. It occurs in hereditary hypotrichosis, ectodermal dysplasia and as an isolated disorder in otherwise healthy children.[9]

Ribonuclease reductase requires iron as an essential cofactor, which is involved in DNA synthesis. It has been proposed that iron deficiency reduces the proliferation of matrix cells. The arrest of matrix proliferation results in telogen effluvium. Iron deficiency without anemia is seen in 20% of cases and manifests solely with a serum ferritin below 20 µg/l.[11]

Decreased thyroid hormone level in blood inhibits cell division both in the epidermis and in the cutaneous appendages. In some patients this inhibition of mitosis induces catagen and delays re-entry of telogen hair into anagen. The pathogenesis of hair loss in hyperthyroidism is unknown.[11]

Clinical features

The period of dramatic hair loss occurs diffusely from the scalp approximately 2-3 months after the triggering event.[11] The diffuse loss may produce thinning of hair all over the scalp, but frequently manifest with bitemporal recession. Loss is normally not more than 50% of the scalp hair.[9] Usually patients do not relate these events to their recent illness and are anxious that they may go bald.[11] Scarring and inflammation are absent.[3] Clumps of telogen hair can be extracted with ease from both the vertex and the margins of the scalp.[11] Chronic starvation, in particular marasmus may result in dry, lusterless, fine, straight hair that are sparse and easily pluckable. Kwashiorkor results in periods of interrupted hair growth that either sends the hair into telogen phase, or, if less severe, it affects the hair caliber more than its linear growth, hence producing multiple Pohl Pinkus lines. Hair color change is a prominent feature in this situation. Dark hair becomes brown or red, while brown hair changes to blond. This color change along with periodic constrictions produces the so called “flag sign” of Kwashiorkor. Essential fatty acid deficiency produces lightening of hair color and also marked telogen hair loss.[11]

The hair pull test is strongly positive in telogen effluvium. It is done by grasping 40-60 closely grouped scalp hair with thumb and index finger and gentle traction is applied as the hairs are pulled firmly and slowly from the scalp. Normally only 2-3 hairs are pulled out by this method. In excessive shedding, more than 10% hairs are easily pulled out from any part of the scalp provided that the patient has not shampooed for more than 24 hours.[6,13] The trichogram (hair pluck test) from a hair pluck sample is abnormal and shows greater than 25% telogen hair.[11] The hair collected during pull test and pluck test on light microscopic examination shows club hair (figure 3).[14] Hair pull test has been found to be a poorly sensitive method as

telogen percentage in trichogram does not correlate with severity of hair loss. While daily hair count is a cumbersome method, it has been proposed that the wash test is probably the best method to adopt. In wash test, the patient is instructed to wash hair after 5 days of last shampoo, in a sink with its drain covered by gauze. The hair entrapped in the gauze are then counted.[9]

Jain et al recorded the following probable etiological causes of diffuse hair loss in their study (n=100); fever (33%), psychological stress (30%) and systemic illness (23%).[7] Detailed history and clinical examination help to detect the cause of TE. If not, a minimum battery of laboratory tests should be performed, which includes complete blood count, urine analysis, serum ferritin, and T3, T4, and thyroid stimulating hormone (TSH).[6] Syphilis serology, antinuclear antibody titer and serum zinc level should be done if there are other features on history or on examination to suggest these conditions.[11] Two common conditions associated with TE are iron deficiency anemia and thyroid disorders and many a times there are no apparent clinical features to suggest these conditions. Hence, these are included in the minimum battery of tests in cases with TE/diffuse alopecia with no apparent cause.[6]

TE must be differentiated by psychogenic pseudoeffluvium in which the patient seeking advice for hair loss is not necessarily balding. Normal dense scalp hair, and absence of any clinically convincing evidence of hair loss is regarded as the features of imaginary hair loss or psychogenic pseudoeffluvium. In these cases patients might need psychological consultation.[9]

Investigations

Scalp biopsy is usually not needed but is the most definitive way to diagnose TE. Biopsy of telogen effluvium shows normal histopathology except for an increase in the telogen follicles. The proportion of normal telogen follicles of more than 15% is considered suggestive of TE. While the proportion of telogen follicles of 25% or more is considered as definitive (normal telogen counts 6-13%) feature of TE. Though scalp biopsy may not be necessary for the diagnosis, it helps to rule out FPHL and alopecia areata.[6]

On videodermoscopy, there are large number of short-tip pointed regrowing hair in the absence of hair diameter variability.[6]

Treatment

Reassuring the patients is of utmost importance. They should be explained that TE represents excessive hair shedding rather than actual hair loss and it is a temporary phenomenon; sufficient hair regrowth would be observed in time.[6,11] As the condition is self-limiting, it does not require any specific drug and it usually resolves in 3-6 months if the trigger is eliminated. Identification of the cause and its treatment is vital.[6]

Female pattern hair loss

This term was first described by Behrman.[5] It is preferred to use FPHL for androgenetic alopecia in females owing to the uncertain relationship between androgens and this entity. It is a slowly progressive, non-scarring alopecia seen most commonly in females aged 20-40 years, characterized by a reduction in hair density over the crown and frontal scalp with retention of the frontal hairline.[2,6]

Epidemiology

In the study by Birch et al conducted in Royal Hallamshire hospital, Sheffield, UK, 337 women aged 18-99 years, who presented to a general dermatology clinic with complaints unrelated to hair growth, 6% (< 50 years) were diagnosed as having female pattern hair loss. This incidence increased to 38% in women above 70 years.[15] Sinclair et al in Australia, have noted prevalence of FPHL is approximately 12% amongst women aged between 20 years to 29 years.[2] Thereafter the incidence was 25% by 49 years, 41% by 69 years, and 50% by 70 years of age and was recorded among 43% of women aged above 80 years.[2] A study conducted by Venning and Dawber found that 87% of 254 premenopausal women and 63% of 310 post-menopausal women without specific complaints of hair shedding had Ludwig grade I-III hair loss. Gan and Sinclair used a photographic scale to grade FPHL pictorially and determined that 53 of 267 (20%) women under 50 years of age had FPHL compared to 90/450 (42%) women over 50 years.[16] A study conducted by Norwood showed increased incidence of FPHL with age among 145 of 568 women under 50 years old, as compared to 26% of 438 women over 50 years old.[16] Birch et al have reported incidence of Ludwig pattern of hair loss as 6% in women under age of 50 years and in 38% of women ≥70 years among total 377 women presenting to a dermatology clinic with complaints unrelated to hair loss.[16]

Etiopathogenesis

Exact cause of FPHL is not known. To date genetic predisposition, iron deficiency and hormonal imbalances have been proposed as probable etiopathologic factors in FPHL.[17] The role of androgens in FPHL not clearly understood.[2] Normal androgen level is seen in most of the women with patterned hair loss and hence other androgen independent mechanisms are likely to be involved in the development of

FPHL.[2,12] Recent studies have reported that some of these women have increased level of serum adrenal androgen dehydroepiandrosterone sulfate.[3] The pattern of hair loss in both men and women may be mediated by locally synthesized estrogen acting in a paracrine fashion to inhibit hair growth.[18] The histopathological hallmark common to both male and female pattern hair loss is miniaturization of hair follicles with progressive transformation of terminal hair follicles into vellus-like follicles.[2] Hair follicle miniaturization is mediated through interaction between androgens, their respective receptors and enzymes like 5α reductase and P450 aromatase.[4] Kasick et al have reported an increased dehydroepiandrosterone sulfate level in women with FPHL. Decreased sex hormone-binding globulin in women with patterned hair loss was noted in a study conducted by Georgala.[19] Carey et al proposed that a single gene abnormality that affects androgen action or production, causing patterned hair loss with different thresholds for phenotypic expression in females and males. It is to note that hyperandrogenemia per se does not necessarily lead to FPHL.[19]

There is limited and inconclusive data on the genetics of FPHL. Autosomal dominant trait with incomplete penetrance and polygenic inheritance are the proposed mechanism.[12] Olsen et al excluded iron deficiency as being a causative factor of FPHL in Caucasians and a low estrogen to androgen ratio in the presence of genetic susceptibility has been found to be of significance in the same population.[17] Zhang et al have concluded that the course of FPHL is not affected by the level of serum ferritin.[17] Kantoor et al in Philadelphia, USA, have found that the mean serum ferritin levels in patients with androgenetic alopecia were significantly lower than levels in women without hair loss.[20]

Clinical features

The essential feature of FPHL is the characteristic pattern of the hair loss.[16] The pattern of hair loss observed in women is different from that of men. Women may develop FPHL any time after the onset of puberty.[2] Women develop diffuse thinning over the mid-frontal scalp with relative sparing of the anterior frontal hair-line.[16] The thinning is most easily appreciated when the hair is parted in the midline, and the exposed scalp may resemble a Christmas tree.[2] Female pattern hair loss may present initially with either episodic or continuous hair shedding, prior to any noticeable reduction in hair volume.[2] Alternatively some women may present with diffuse thinning over the crown, being unaware of any increase in hair shedding.[2] These women usually have normal menstrual cycle.[3] Most women with FPHL, however do not have any signs or symptoms of hyperandrogenemia and typically have normal serum androgen levels.[19] Less than 40% of the cases may have hyperandrogenism and may manifest through hirsutism, severe or recalcitrant acne, oligomenorrhea, infertility, acanthosis nigricans and galactorrhea.[6]

Examination of the scalp shows a widening of the central parting with diffuse reduction of hair density over the frontal scalp.[2,10] Typically the frontal hairline is usually preserved although some women may develop bitemporal recession.[2] In FPHL bitemporal recession is usually less pronounced and vertex bald spots are almost never seen.[2] It begins postpubertally, and may run a course independent of mid-frontal hair loss.[2] Traditionally, severity of FPHL can be graded by Ludwig scale (Figure 1). It divides severity of hair density reduction over the crown into three grades.[2]

Grade I: Perceptible thinning of the hair on crown, limited in front by a line situated 1-3cm behind the frontal hair line (figure 4).

Grade II: Pronounced rarefaction of the hair on the crown within the area seen in grade I (figure 5).

Grade III: Full baldness (total denudation) within the area seen in grade I and II.

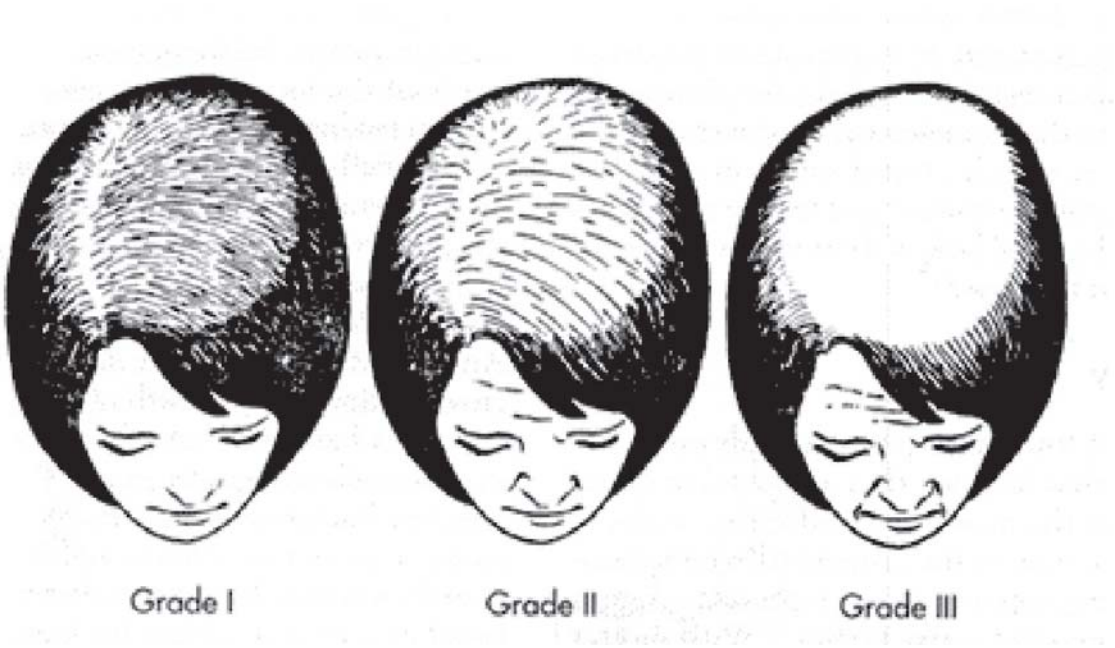


Figure 1: Ludwig scale

Savin density scale classifies FPHL into 8 stages of increasing crown balding, in addition to a special subcategory to detect frontal anterior recession.[2] Sinclair developed 5 point visual analogue scale which assesses the degree of hair loss using the midline parting which is a simplification of the widely accepted Savin density scale.[2] Female pattern hair loss without any treatment invariably leads to progressive reduction in hair volume over the frontal scalp.[2]

Investigations

Hair pull test, hand held epiluminescent microscopy and global photography are important in the diagnosis of FPHL.[21] Hair pull test may be positive in active early hair loss in central scalp but it is generally negative in a patient with long standing hair loss.[22] Female pattern hair loss and CTE can be best distinguished by biopsy.[2]

Epiluminescent microscopy demonstrates miniaturization of hair follicles, which is the hallmark of FPHL and reduction in the average number of terminal hair per follicular unit is seen over the mid-frontal scalp when compared with the occipital scalp.[21]

A horizontally sectioned 4 mm scalp biopsy rather than vertical is the most reliable method for diagnosing FPHL. During biopsy the punch should follow the direction of the hair shafts and is taken deep into the subcutaneous fat where anagen hair bulbs are located.[22] On horizontal scalp biopsy, the ratio of terminal to vellus hair of <4:1 is diagnostic of FPHL whereas a ratio of >8:1 is indicative of chronic telogen effluvium. Ratios of 5:1 to 7:1 are considered indeterminate.[21] Sinclair et al compared reliability of 3 scalp biopsies sectioned horizontally in 207 women with diffuse hair loss, with 2 biopsies, one sectioned horizontally and the other vertically in 305 women. Application of these diagnostic criteria has achieved accurate positive diagnostic results in 98% of women with triple horizontal biopsies compared to 79% with single horizontal biopsy.[23] Multiple scalp biopsies may be needed to reduce the risk of an indeterminate result or of underestimating FPHL.[14] Many of the indeterminate cases may evolve into androgenetic alopecia with time.[14]

Diversity of hair shaft diameter, peripilar signs, and empty follicles are trichoscopic features of FPHL. It has been suggested that diversity of hair shaft diameter >20% is diagnostic of FPHL.[24]

Phototrichograms are performed using a TrichoScan which includes epiluminescence microscopy and automatic digital image analysis. It provides information on hair calibre, density and growth rate. This noninvasive technique in future with improved sensitivity may reduce the need for a diagnostic scalp biopsy in some patients.[21]

It is not routinely necessary to follow extensive metabolic and endocrinological work-up. In presence of menstrual disturbance, impaired fertility and signs of hyperandrogenism, the patient should be investigated for polycystic ovary syndrome.[21]

Treatment

Currently topical minoxidil is the only androgen-independent medication in wide spread use.[2] Topical minoxidil 2% solution is used for mild to moderate FPHL (Ludwig stage I and II).[6] It acts by altering the hair cycle, causing premature termination of telogen and probably prolonging anagen.[2] Antiandrogens may be useful, especially in cases of FPHL with hyperandrogenism. Systemic antiandrogens like spironolactone (100-300mg/day), flutamide (250-500mg twice or thrice a day) and cyproterone acetate (100mg/day) on days 5-15 of menstrual cycle and ethinyl estradiol 50µg/day on 5^{th}-25^{th} days have shown some effectiveness in FPHL.[6] But no thorough large placebo controlled trials have been conducted.[6] In premenopausal women with FPHL associated hyperandrogenism, systemic finasteride has shown some benefit. Topical preparations of finasteride 0.05% have a potential role in the

treatment of both male and female pattern hair loss. Fluridil is a novel topical antiandrogen. This compound is highly hydrophobic, with high local efficacy and tolerance. Fluridil 2% solution not only prevented the progression of FPHL but also increased hair diameter in an open clinical study.[25]

Hair transplantation can be the treatment option for moderate cases of FPHL (Ludwig II). Candidates ideal for hair transplantation are those women with high density donor hairs (>40 follicular unit/cm^2) in some areas and extensive loss or thinning at frontal or mid frontal scalp only.[2,6] In mild FPHL (Ludwig I) pre and post transplantation hair is difficult to appreciate; therefore they are not optimal candidates for hair transplantion.[6] Severe cases of FPHL (Ludwig III) usually do not respond to minoxidil, antiandrogens and surgery. In these cases hair transplantation surgery is not possible as the entire scalp including the donor area is susceptible, and may suffer from hair loss. In advanced cases hair replacement surgery may be required.[2]

Cosmetic aids are an integral part of management options because much of the morbidity of FPHL lies in body image disturbance.[2] Hair prosthesis (wig, hair extention, hair piece), hair cosmetics (tinted powders, lotions, sprays) are used for severe FPHL (Ludwig stage III). It can also be used in mild to moderate cases of FPHL as adjuvant to medical therapy.[6] Different hairstyling options can be followed by a woman's individual hair stylist, and it may take 3 or 4 different haircuts before a woman finds her optimal look. Camouflaging products can be used as it covers exposed areas on the scalp and hide visible hair loss. Hair building fibers, scalp spray thickeners, alopecia masking lotion, and topical shading are most commonly used products.[2] Scalp reduction involves bringing hair-bearing skin of scalp closer together by removal of the central scalp skin affected by alopecia, and it is less popular as

compared to hair transplantation. Sometimes it can be performed along with hair transplantation to optimize cosmetic outcomes.[2]

A variety of laser and light sources have been tried for treatment of hair loss, with varied success rates. The idea of using laser therapy was adopted from experimental observations that low-powered ruby laser can increase hair growth in mice. Paradoxical increase in hair growth has been seen with use of 810-nm pulsed laser and intense pulsed light intended for hair removal. The mechanism of low-level laser on hair growth is yet to be known.[25] Prostaglandin analogues and botulinum toxin are new treatment options for women in FPHL.[25]

Monitoring clinical response

Response to treatment can be monitored practically by encouraging periodical scalp photographs preferably by a trained medical photographer before treatment and every 6 months thereafter, thus allowing subtle improvements to be easily observed. Another way of monitoring is to suspend drug treatment temporarily after 12 months and observe for an increased amount of hair loss 4–6 weeks later, indicating successful treatment.[2]

Chronic telogen effluvium

It was first described by David A Whiting in 1996 in Washington DC.[6] CTE is characterized by an abrupt onset of diffuse hair loss of scalp persisting for more than six months. This condition predominantly affects healthy women in the fourth to fifth decade of life.[4] It may be primary chronic telogen effluvium or may be secondary to various causes.[11]

Epidemiology

The exact incidence of CTE is not known. Garcia-Hernandez MJ reported incidence of CTE as 67% in post-menopausal women out of 109 female study subjects.[26]

Etiopathogenesis

Various causes of CTE are:

- Iron deficiency
- Protein energy malnutrition
- Zinc deficiency
- Hypothyroidism and hyperthyroidism
- Chronic renal failure
- Liver failure
- Systemic lupus erythematosus and other connective tissue disorders
- Drugs (Heparin, colchicine, methotrexate)[5,9]
- Diabetes mellitus[5]

Iron deficiency is the most common cause one can come across as compared to others.[9]

The exact pathogenesis of chronic telogen effluvium is uncertain, but it may be due to shortening of the anagen phase of the hair cycle without miniaturization of hair follicles, synchronization phenomena of the hair cycle or premature teloptosis.[6,9,11] It

has been suggested that shedding is not noticeable until the length of anagen phase is reduced by 50%, with a subsequent doubling of telogen hairs. The patient should be assured that this type of shedding will not lead to baldness.[9,12]

Clinical features

Chronic telogen effluvium may be triggered by an acute TE. In primary CTE no specific triggering agent is evident. It predominantly affects women.[9] The presentation of this type of hair loss tends to be distinctive.[9] The typical patient is an otherwise healthy woman between 30 to 50 years with dense scalp hair (figure 6).[6,9,10] It is characterized by abrupt, excessive, alarming, diffuse shedding of hair that runs a fluctuating course over several years.[6,9,10] Patient often gives history of long hairs during childhood, suggestive of a long anagen phase, and they report a high density of hair prior to the onset of hair loss.[10] Patients insist that previously they had more hair and are distressed by the prospect of going bald. Many patients frequently bring large ball of hair for showing to clinician. However, no clinically obvious finding can be established. This condition tends to run a fluctuating course, at times reflecting seasonal periodicity in the growth and shedding of the hair with a maximal proportion of telogen hairs at the end of summer and beginning of autumn. In the long run the disorder appears to be self-limiting. It is of importance to reassure the patients that this condition represents exaggerated shedding rather than actual hair loss.[9] Chunks of hairs are usually seen in the bathroom, pillow, hair brush and comb. Usually patients will display a hand-full of hair to corroborate the complaint of excessive shedding (figure 7). Thinning of hair is not a feature of their ponytail thickness. Family history of androgenetic alopecia is usually absent.[10]

On examination moderate to severe bitemporal recession may be seen figure 8).[6] There is no widening of the central part, as is common in androgenetic alopecia. Hair pull test is positive commonly over the vertex and occipital scalp.[10] Diagnosis of CTE cannot be excluded by one single negative hair pull test.[10] If the insult is prolonged and/or regularly repeated, it results in diagnostic difficulty.[11]

Investigation

Histopathological picture is normal except for a slight increase in the telogen hair follicles.[6] To exclude androgenetic alopecia scalp biopsy is usually required.[11] The biopsy should be at the level of sebaceous gland.[14] Normal terminal to vellus hair ratio is 7:1. A ratio of more than 8:1 is considered as diagnostic of CTE.[14] In a study conducted by Sinclair R in 2002, out of 305 women with chronic diffuse hair loss 18% were diagnosed as CTE on single biopsy and 23% had intermediate features. Literature concerning iron deficiency as a cause of FPHL remains controversial. Iron deficiency has been reported in majority of women complaining of diffuse hair loss, but this probably has been overestimated. More recent data suggests that in most women with CTE, there is no direct relationship between low serum ferritin (>20µg/l) and hair loss.[9] Light microscopic examination shows club hairs.[9]

Treatment

If a particular cause for chronic diffuse hair loss is identified, such as hypothyroidism or iron deficiency, then suitable treatment should be given. If any drug is under suspicion as the cause of hair loss, then the patient should be advised to stop taking that drug, or that has to be substituted with an alternate drug which has lesser risk of causing hair loss. If the diet is inadequate then it should be rectified.[27] It is said that CTE is a self-limiting process, which may resolve spontaneously in 3-10

years, but there is no evidence to substantiate this assertion.[6] Patient is usually anxious to have some form of treatment.[27] The natural history of CTE is poorly characterized and the prognosis is less certain.[6] No specific drug is available for the treatment of CTE. Topical minoxidil 2% has been suggested in anticipation that it will prolong anagen growth.[6]

Anagen effluvium (AE)

Anagen effluvium is a type of hair loss that follows the administration of cytotoxic drugs, radiation treatment or various chemical agents and is characterized by hair breakage rather than hair loss. Hair shafts are abruptly thinned at the time of maximum drug effect leading to Pohl-Pinkus constriction. Hair shafts break at about the same time when the thin portion reaches the scalp surface. Even though the hair loss can be quite distressing to patients and even family members, dermatologists, have little to do with anagen effluvium.[28] Chemotherapy induced alopecia has an estimated incidence of 65% and is considered to be one of the most traumatic aspects of chemotherapy in female patients.[25]

Etiopathogenesis

Anagen effluvium is a hair loss that occurs during the anagen phase of growth because hair bulb cell divide rapidly and are sensitive to cytotoxic agents. Cytotoxic drugs impair the mitotic and metabolic processes in actively growing hair follicles, leading to thinning of the shaft, which becomes fragile and susceptible to fracture with minimal trauma. The molecular mechanism of anagen effluvium or chemotherapy-induced hair loss has been associated with premature apoptosis-driven hair follicle regression, and p53, Fas and c-kit are the involved factors.[28] These agents can impair or totally disrupt the anagen cycle and cause varying degree of hair follicle

dystrophy. The net result is either anagen hairs that break off within the hair follicle or at the level of scalp (secondary to a weak point in the structurally inferior hair shaft) and are then shed without roots, or dystrophic anagen hairs that are easily dislodged from the usual follicular moorings.[29] Various causes of anagen effluvium has been presented in table 2.

Table 2 : Various causes of anagen effluvium[28, 30]

Chemotherapeutic agents	**Alkylating agents** Melphalan, chlorambucil, busulfan, cyclophosphamide, dacarbazine, cisplatin **Antimetabolites** Methotrexate, 5-flurourcil, hydroxyurea, cytarabine, 6-mercaptopurine **Vinca alkaloids** Vincristine, vinblastine, paclitaxel, docetaxel **Topoisomerase inhibitors** Topotecan, etoposide, doxorubicin, daunorubicin, idarubicin **Antitumor antibiotics** Mitomycin-C, actinomycin D, bleomycin
Plants (cytotoxic derivatives)[30]	Tubers of Gloriosa superba (colchicine), Spines of Lecythis (selen ocystothionine), Leucaena glauca (mimosine), Abrus precatorius (abrin)
Ionizing radiation[3]	Teleradiotherapy
Nutrition	Protein energy malnutrition[4,29]
Other drugs	Warfarin, dextran, thallium, arsenic, gold, bismuth, levodopa, colchicine, mercury intoxication, boric acid intoxication, thallium poisoning, exposure to toxic dosage of colchicine

Clinical features

A careful history is an important key to identify triggers in any patient with diffuse hair loss.[8] Hair loss usually begins 7-10 days after the initiation of chemotherapy and becomes most clinically apparent in 1-2 months. Hair loss can continue over next 3-4 weeks.[28] It mostly affects scalp hairs and to a variable degree terminal hairs at other sites such as eyebrows, eyelashes, axillary and pubic hairs.[25] Anagen effluvium is a reversible condition, and hair regrowth begins several weeks after the cessation of chemotherapy. Hair loss is known to start from the area of mechanical friction such as crown and side of the head above the ears because these areas come in contact with bed linens, pillow and head covering. Nearly 85% of the total number of anagen hairs are shed after chemotherapy and scalp hairs those are in the telogen phase are not affected (figure 9).[28] When hair regrows, approximately 65%of the patients experience a change from their previous hair. Some patients experience alteration in the colour, texture or type of hair.[28]

Regrowth of hairs after radiation therapy depends upon type, depth, and dose-fractionation but it commonly leads to permanent follicular destruction, most likely as a result of irreversible hair follicle stem cell damage leading to scarring alopecia. In fact, this scarring alopecia may progress long after radiation therapy has been discontinued; possibly due to persistent radiation-induced inflammatory changes that progressively damage hair follicle stem cells. Low dose cytotoxic agents more often cause only telogen effluvium, because they induce premature catagen. High dose busulfan which is used in the preparatory treatment for bone marrow transplantation may lead to permanent alopecia due to irreversible damage to hair follicle stem cells.[29] In a study by Korean author Jung Yun S, 20 among the 38 female patients of anagen effluvium had patterned hair loss. They did not notice any significant

difference in the pattern of hair loss depending upon age, associated symptoms and chemotherapeutic agent groups.[28] Hair loss resembling androgenetic alopecia and changes in the structure and colour have been reported with tamoxifen therapy.[30,31]

Investigations

Clinically diffuse hair loss can be diagnosed by hair pull test.[13] The hair pull test is positive in anagen effluvium. Light microscopic examination shows dystrophic anagen hairs with tapered ends and thinning or constriction of the hair shafts called Pohl-Pinkus constriction (figure 10).[8, 31]

Treatment

If the insult ceases, growth of hairs restarts within weeks.[8] Various measures have been tried in order to prevent hair loss. Topical minoxidil has been found to decrease the duration of hair loss caused by chemotherapy. Minoxidil is not effective in preventing initial hair loss due to chemotherapeutic agents. It should not be used in patients undergoing chemotherapy for hematological malignancies with a curative intent.[25] Scalp cooling has been reported as an effective method of preventing chemotherapy-induced alopecia.[30] It involves cooling of the scalp with cold air or liquid. It produces vasoconstriction of the scalp vessels leading to reduced blood flow to the follicles during chemotherapy thus minimizing concentration of the antineoplastic agent in plasma.[25] However it may not be effective when multiple drug regimes or very high doses of individual drugs are used. There are no specific guidelines on optimal method, temperature, and duration of scalp cooling at present.[25] In animal models, topical agents such as imuvert, cyclosporin A and 1,25-dihydroxyvitamin D3 have prevented chemotherapy-induced hair loss.[30]

A battery of investigations may be helpful in diagnosing a case of diffuse hair loss. Hemoglobin, peripheral blood smears and serum ferritin level are estimated to detect iron deficiency anemia. Thyroid stimulating hormone, T3 and T4 levels are done to rule out associated thyroid function anomalies. Stool is tested for parasites and occult blood if there is history suggestive of chronic blood loss.

From review of literature it is evident that studies on etiological factors of diffuse hair loss in women are inadequate. Diffuse alopecia in women may bring such psychological stress that it may cause emotional upset and leads to personal, social and work related problems. As many as 40% of women may suffer from marital disharmony as a consequence of diffuse hair loss.[4] Thus it is evident that it is a disorder of utmost importance and knowledge about various factors precipitating diffuse hair loss will be helpful both for clinician as well as for the patients.

METHODOLOGY

SOURCE OF DATA:

A hospital-based, cross-sectional study to determine various causes of diffuse hair loss in women was conducted in the department of Dermatology Venereology and Leprosy of B.L.D.E.U's Shri. B.M. Patil Medical College Hospital and Research Centre, Bijapur, Karnataka. One hundred and eighty female patients complaining of diffuse hair loss were recruited from the out patient section of the department. The study was conducted between the period of October 2011 to September 2013.

METHOD OF COLLECTION OF DATA:

Inclusion criteria:

All adolescent girls and adult women who presented with the complaints of diffuse hair loss, attending the out patient section of the department of Dermatology, Venereology and Leprosy were included in this study.

Exclusion criteria:

On thorough clinical and histopathological examination, patients found to have specific pathological disorders of hair, like alopecia areata/alopecia universalis and cicatricial alopecia were excluded.

METHOD:

All the patients were explained about the non-invasive methods which would be followed during the study. Informed consent was taken from all patients or parents, in case of teen-aged patients. Detailed history was taken from all women presenting with diffuse hair loss, with special emphasis upon the following;

1. Major febrile illness or psychological stress in the recent past (3 months)
2. History of chronic blood loss (bleeding piles, menorrhagia, peptic ulcer disease)
3. Any history of drug intake 3-4 months prior to the onset of hair loss
4. Passage of worms in stool
5. History suggestive of hyper/ hypothyroidism (intolerance to heat or cold, rough skin, palpitation, change in voice, insomnia, fatigue, tremor etc.)
6. History of crash diet in the recent past

On clinical examination specific signs for anemia, jaundice, and thyroid swelling were recorded. Diagnosis of diffuse hair loss was done by scalp examination and hair pull test. It was done by grasping 40-60 closely grouped scalp hairs with thumb and index finger. Gentle traction was applied as the hairs were pulled firmly and slowly from the scalp.

Wherever feasible, patients were advised to collect the shed hairs from combing and head bath for the last 24 hours in a plastic bag and count those meticulously to report to the investigator.

LABORATORY INVESTIGATIONS:

Following laboratory investigations were carried out for all patients.

- Hemoglobin (gm%)
- Peripheral blood smear

- Thyroid stimulating hormone levels - It was estimated by ultra sensitive sandwich chemi luminescent immune assay. T3 and T4 level were estimated if indicated by altered TSH level. It was assayed by competitive chemi luminescent immuno assay.

- Serum ferritin – It was estimated by fully automated bidirectionally interfaced chemi luminescent immuno assay.

Following tests were done wherever indicated;

- Erythrocyte sedimentation rate.

- Occult blood in stool.

- Stool microscopy for ova, parasite, cyst.

- Scalp biopsy and microscopic examination of hair.

All the clinical and laboratory data were recorded in patient proforma (enclosed). All patients were provided topical and/or systemic treatment as indicated by clinical and investigational results.

STATISTICAL ANALYSIS:

The observations pertaining to the parameters under study group was expressed in percentage.

Collected data was presented with mean ± 2SD

To find the correlation between diffuse hair loss and various laboratory parameters, Chi-square test and Fischer exact test were applied.

ETHICAL CLEARANCE:

Institutional ethical committee clearance was undertaken for the study.

Figure 2: Telogen effluvium

Figure 3: Photomicrograph showing shed telogen hair on light (10X)

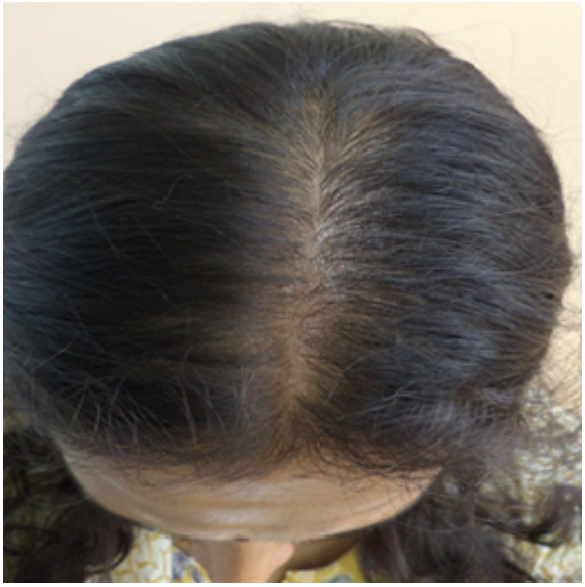


Figure 4: Female pattern hair loss grade I

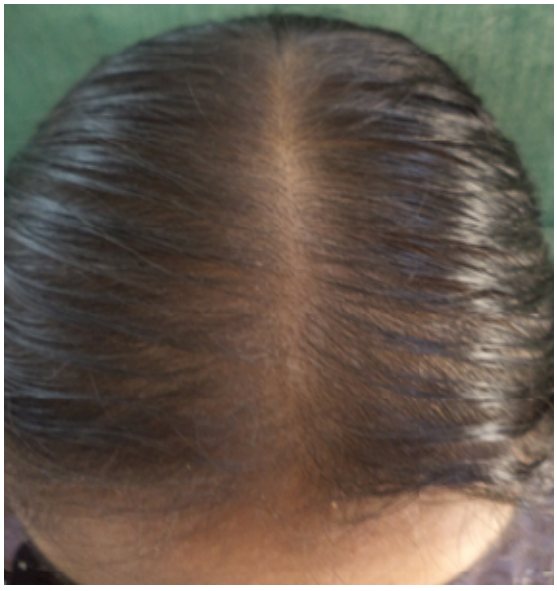


Figure 5: Female pattern hair loss grade II

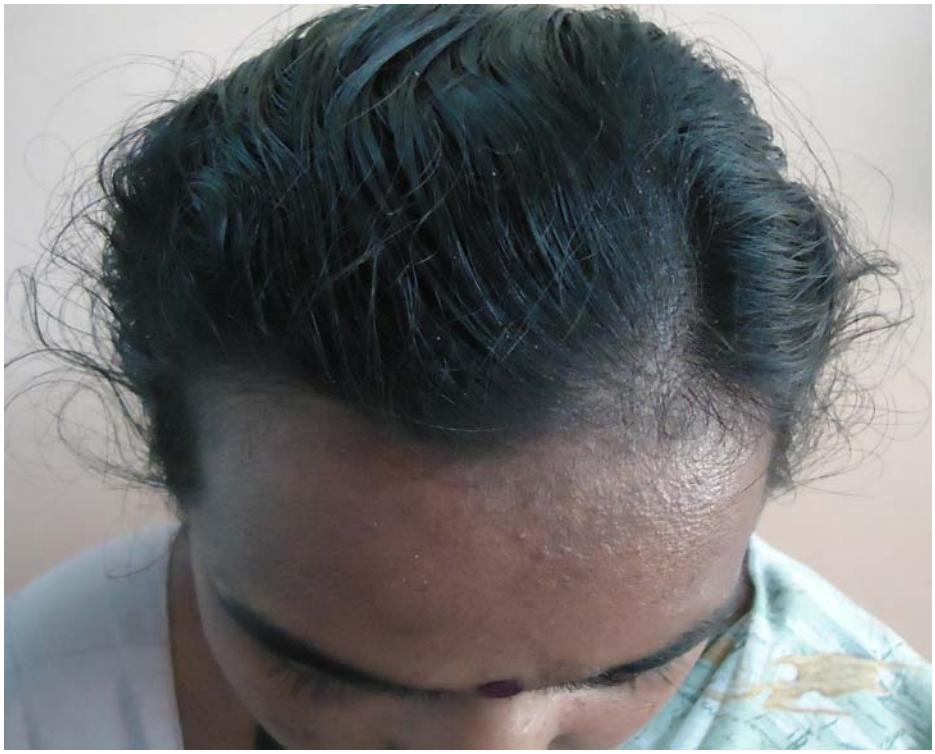


Figure 6: Chronic telogen effluvium: bitemporal recession

Figure 7: Chronic telogen effluvium: a bunch of shed hairs brought for inspection

Figure 8: Chronic telogen effluvium: hair recession and hair thinning in right temple

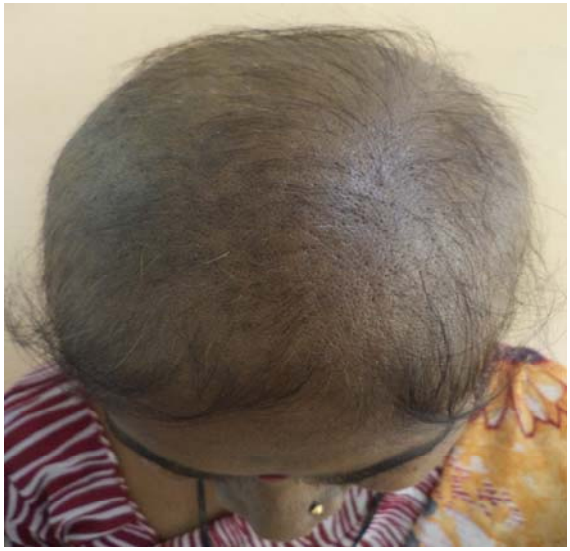


Figure 9: Anagen effluvium

Figure 10: Photomicrograph showing shed anagen hair (10X)

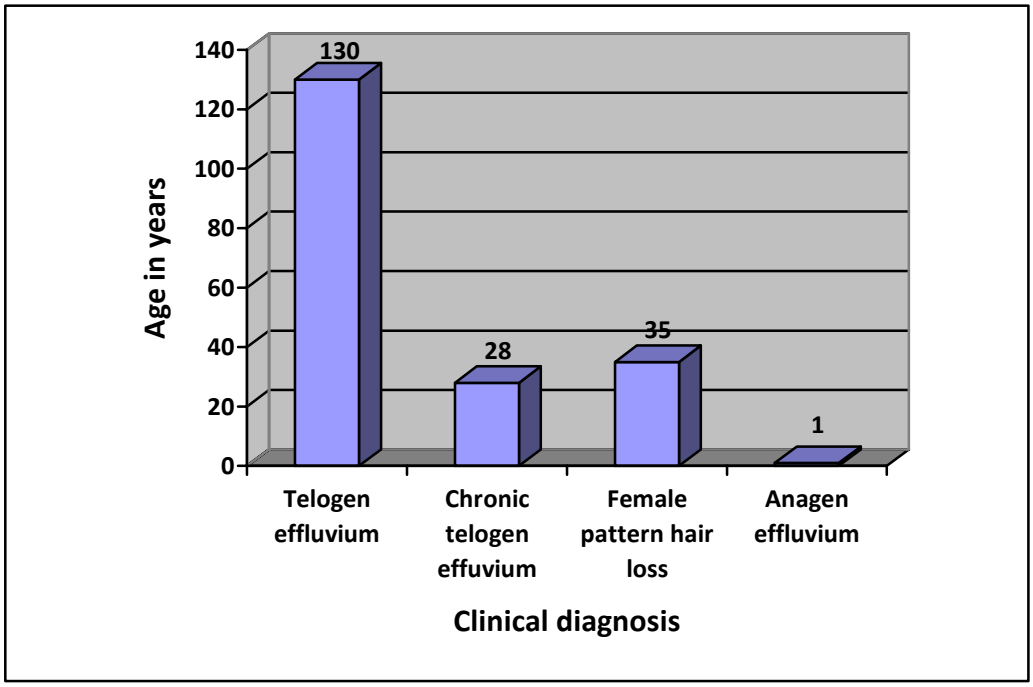
RESULTS

A total of 180 patients with diffuse hair loss were examined during the study period. Out of 180 patients, 130 (66.6%) had telogen effluvium, 28 (14.3%) had chronic telogen effluvium, 35 (19.4%) had female pattern hair loss and 1 (0.5%) had anagen effluvium (Table 3 and figure 11).

Table 3: Clinical types of diffuse hair loss

Clinical types	No.	%
Telogen effluvium	130	66.6
Chronic telogen effluvium	28	14.3
Female pattern hair loss	35	19.4
Anagen effluvium	01	0.5

Figure 11: Clinical types of diffuse hair loss



Age-wise distribution of patients with diffuse hair loss:

The age of the women with diffuse hair loss ranged from 12 to 55 years (mean age 30.21 years). Incidence of diffuse hair loss was highest among 20-30 years. Age-wise distribution of diffuse hair loss among the study subjects has been depicted in table 4 and figure 12. Various clinical types of hair loss in different age groups has been presented in table 5 and figure 13.

Table 4: Age-wise distribution of diffuse hair loss

Age (in years)	No of patients
10-20	29
21-30	92
31-40	39
41-50	14
51-60	6
Total	**180**

Figure 12: Age-wise distribution of diffuse hair loss

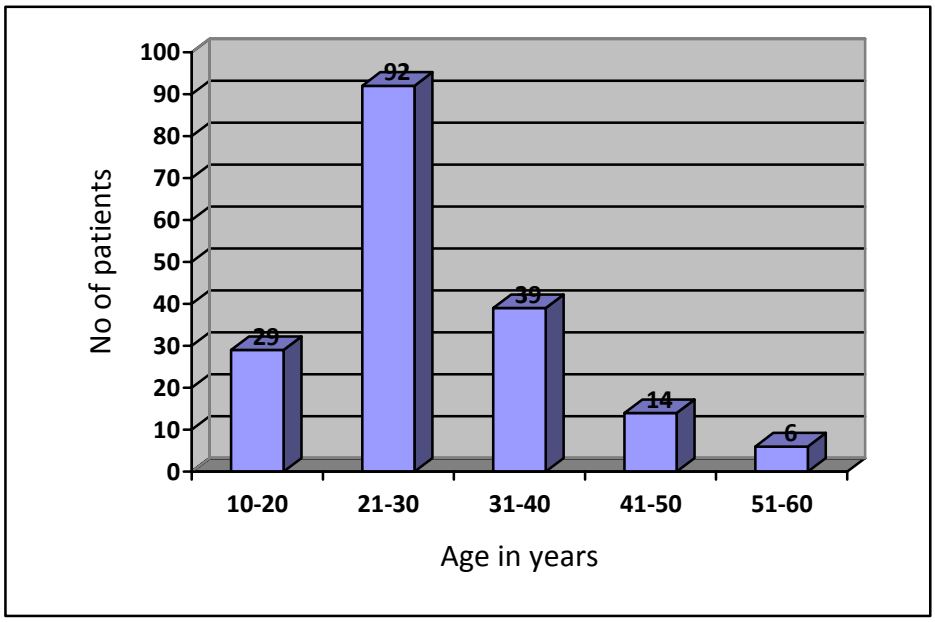
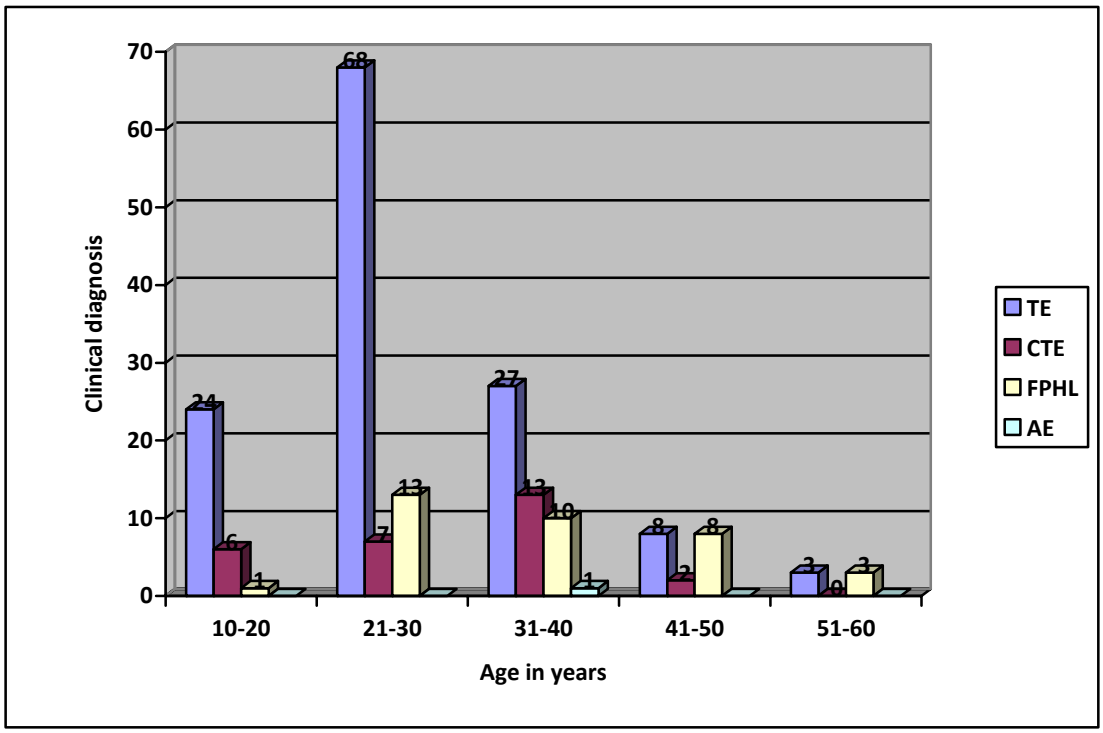


Table 5: Clinical types of hair loss in various age groups of the study subjects

Clinical types	**Age-groups in years**					
	10-20	**21-30**	**31-40**	**41-50**	**51-60**	**Total**
TE	24	68	27	08	03	130
CTE	06	07	13	02	00	28
FPHL	1	13	10	08	3	35
AE	0	0	01	0	0	01
Total	**30**	**94**	**45**	**18**	**8**	**194**

Figure 13: Clinical types of hair loss in various age groups



Telogen effluvium:

Out of 180 patients with diffuse hair loss 130 (66.6%) had telogen effluvium. The age of the patients ranged from 12 years to 54 years (mean age 25.9 ± 7.99 years). Incidence of telogen effluvium was highest in the age group of 21 to 30 years. Out of 130 patients, 24 were in the age group of 10-20 years, 68 in 21-30 years, 27 in 31-40 years, 8 in 41-50 years and 3 in 51-60 years.

Clinical history:

Duration of hair loss varied from 7 days to 6 months. Psychological stress was the most common probable etiological factor (n=48, p = <0.05) followed by fever in 16 cases, drug intake in 12 cases, topical application in 8 cases, crash diet in 8 cases, child birth in 7 cases, chronic blood loss in 6 cases, preceding surgery in 3 cases,

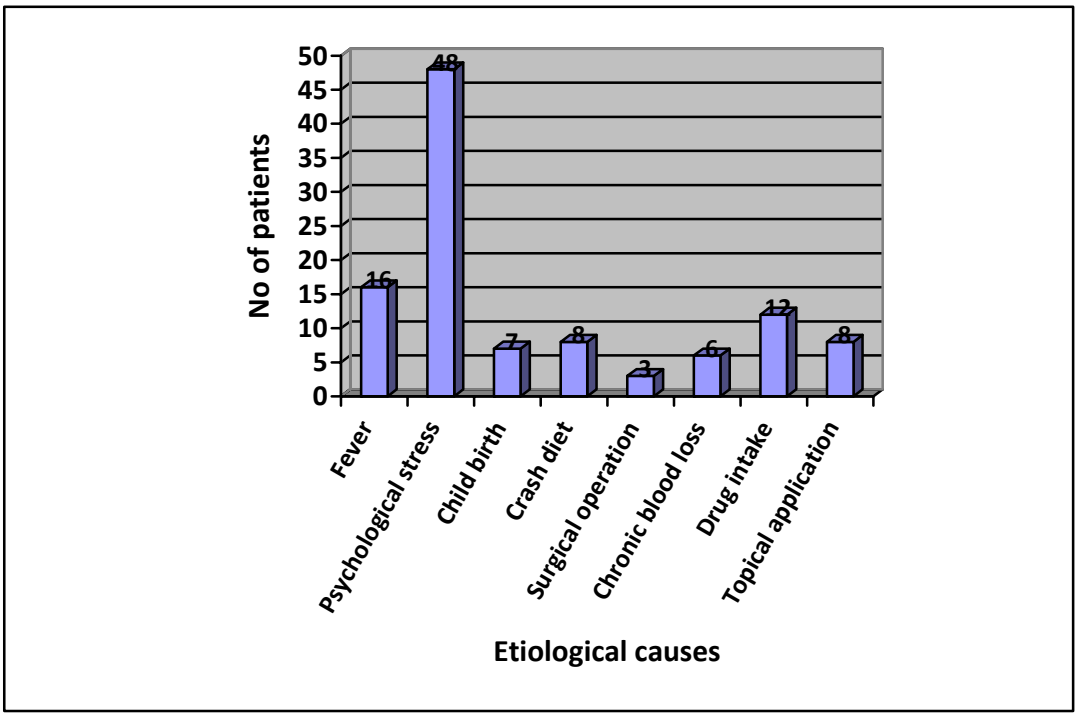
worm infestation in 1 case (Table 6 and figure 14). More than one factor was recorded in 10 (7.69%) cases. No probable etiological factor could be elicited in 32 (24.61%) patients.

Table 6: Probable etiological causes of telogen effluvium in the study subjects

Sl. NO	Probable clinical causes	No of patients	%
1	Fever	16	12.30
2	Psychological stress	48	36.92
3	Child birth	7	5.38
4	Crash diet	8	6.15
5	Surgical operation	3	2.30
6	Chronic blood loss	6	4.61
7	Drug intake	12	9.23
8	Topical application	8	6.15

Out of 130 patients 8 patients gives history of application of topical medications which included hair dye, mehendi and various native medications following which they probably developed allergic contact dermatitis and subsequently hair loss.

Figure 14: Probable etiological causes of telogen effluvium in the study subjects



Clinical examination and investigations:

On scalp examination hair thinning was seen in 46 (35.38%) patients. Hair pull test was positive in 54 (41.53%) patients (Table 7). Positive tug test in 13 (10%) patients and bitemporal recession was observed in 16 (12.30%) patients. On microscopic examination telogen hair was seen in all 130 (100%) cases.

Table 7: Hair pull test in telogen effluvium patients

Hair pull test	**No of TE patients (n=130)**	**Percentage (%)**
Positive	54	41.53
Negative	76	58.46

Complete hemogram was done in 83 out of 130 patients. Low hemoglobin (<12gm/dl) was observed in 50 (60.24%) patients (p = <0.05) (Table 8). Peripheral smear examination revealed microcytic hypochromic picture in 21 (25.30%) patients, normocytic hypochromic picture in 4 (4.87%) patients, dimorphic and macrocytic anemia in 1 (1.21%) patient each. Normocytic normochromic blood picture was noted in 56 (68.29%) patients (Table 9 and figure 15).

Table 8: Hemoglobin level in patients with telogen effluvium

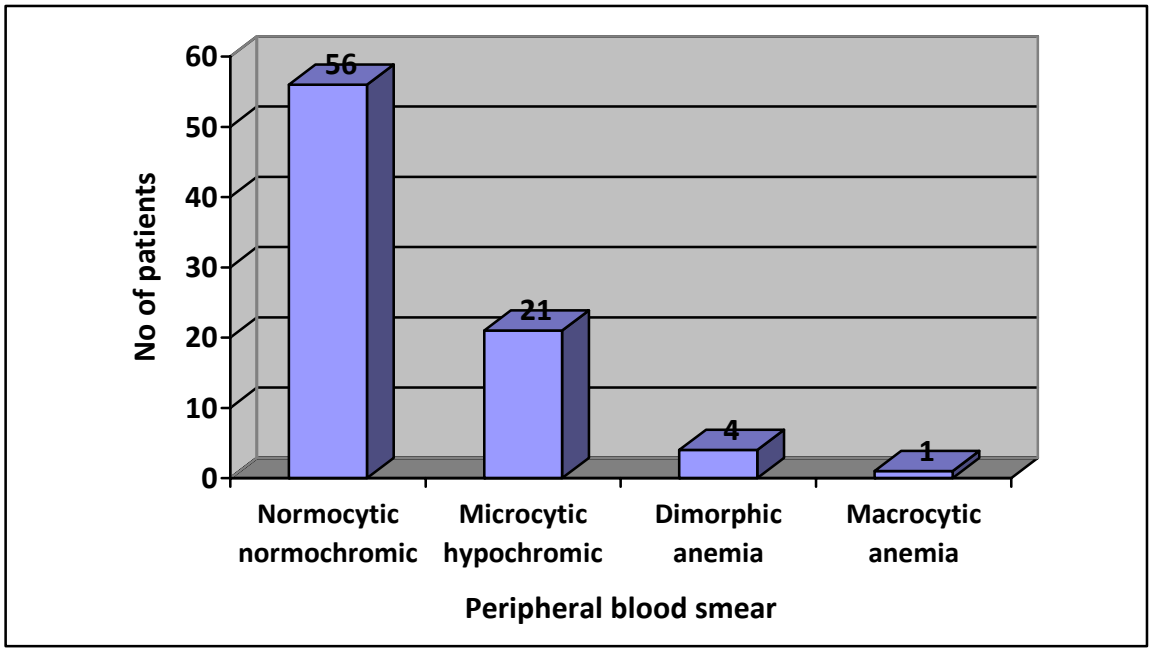
Hemoglobin	No of cases (n=83)	%	p value
Normal (>12gm/dl)	33	39.75	0.035
Low (<12gm/dl)	50	60.24%	

(p = <0.05 is significant, p = >0.05 is not significant)

Table 9: Peripheral blood smear in patients with telogen effluvium

Peripheral blood smear	No of Patient with TE (n=83)	Percentage (%)
Microcytic hypochromic	21	25.30
Normocytic hypochromic	4	4.87
Dimorphic anemia	1	1.21
Macrocytic anemia	1	1.21
Normocytic normochromic	56	68.29
Total	**83**	

Figure15: Peripheral blood smear in patient with telogen effluvium

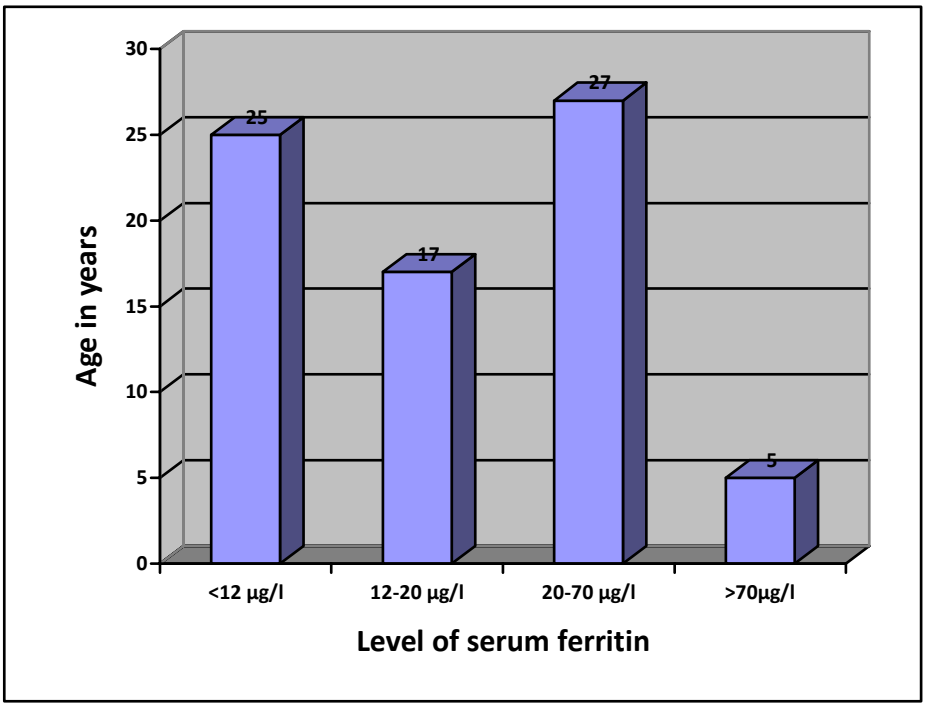


Serum ferritin estimation was done in 74 out of 130 patients with TE. Serum ferritin level of patients with TE has been presented in table 10 and figure 16. Serum ferritin level <70µg/l was seen in 69 (93.24%) patients (p = >0.05).

Table 10: Serum ferritin level in patients with telogen effluvium

Serum ferritin	**No of patients (n=74)**	**Percentage (%)**
<12 µg/l	25	33.78
12-20 µg/l	17	22.97
20-70 µg/l	27	34.48
>70 µg/l	5	6.75
Total	**74**	

Figure 16: Serum ferritin level in patients with telogen effluvium



Thyroid function test was done in 74 out of 130 patients with TE; Hyperthyroidism was recorded in 3/74 (4.05%) patients and hypothyroidism in 2/74 (2.70%) patients.

Stool examination was done in 33 patients; evidence of parasitic infestation was seen in 2(6.06%) patients.

Female pattern hair loss:

Age incidence and distribution:

Total 35 patients were diagnosed with female pattern hair loss. The age of onset of female pattern hair loss varied from 20-55 years. Grade of FPHL in study subjects has been presented in table 11. Incidence of FPHL was highest in the age group of 21 to 30 years. Age-wise distribution of FPHL has been presented in table 12 and figure 17. Age of the patient with FPHL type I ranged from 20 to 55 years (mean

age 28.87±8.81 years) and FPHL type II was 22 to 55 years (mean age 36.75±8.60 years). Family history of patterned baldness was noted in 14 (38.8%) patients.

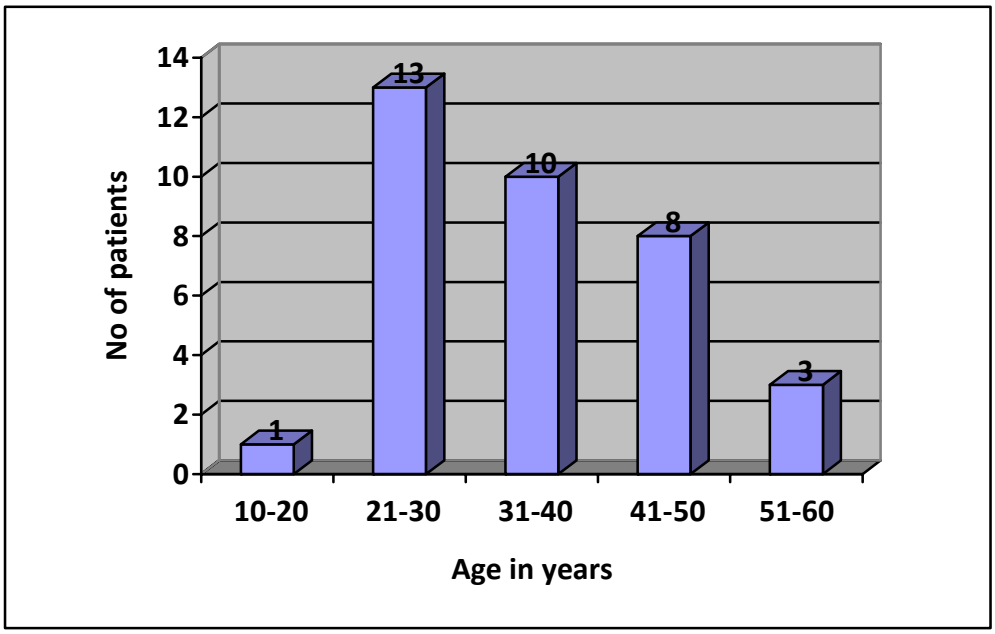
Table 11: Type of FPHL in study subjects

Type of FPHL	No. of patients with FPHL (n=35)	Percentage (%)
Type I	15	42.85
Type II	20	57.14
Type III	0	0
Total	**35**	

Table 12: Age-wise distribution of the patients with FPHL

Age	No of patients with FPHL (n=35)	Percentage (%)
10-20	1	2.85
21-30	13	37.14
31-40	10	28.57
41-50	08	22.85
51-60	03	8.57
Total	**35**	

Figure 17: Age-wise distribution of the patients with FPHL



Clinical examination and investigations:

On examination, bitemporal recession was noted in 3 (8.33%) patients. Positive hair pull test was seen in 13 (36.11%) patients (Table 13). On microscopic examination telogen hair was seen in all 35 (100%) patients.

Table 13: Hair pull test in patients with FPHL

Hair pull test	No. of patients with FPHL (n=35)	Percentage (%)
Positive	13	37.14
Negative	22	62.85

Complete hemogram was done in 20 out of 35 patients. Low hemoglobin was observed in 6 (30%) patients (P = <0.05). Peripheral blood smear examination showed normocytic normochromic picture in 16 (80%) patients, microcytic hypochromic and normocytic hypochromic picture, each in 2 (10%) patients (Table 14).

Table 14: Hemoglobin level and peripheral blood smear findings in patients with FPHL

No of patients with FPHL (n=20)				
Hemoglobin		**P value**	**Peripheral blood smear**	
Normal (>12gm/dl)	14(70%)	0.02	Normocytic normochromic	16(80%)
			Microcytic hypochromic	2(10%)
Low (<12gm/dl)	6(30%)		Normocytic hypochromic	2(10%)

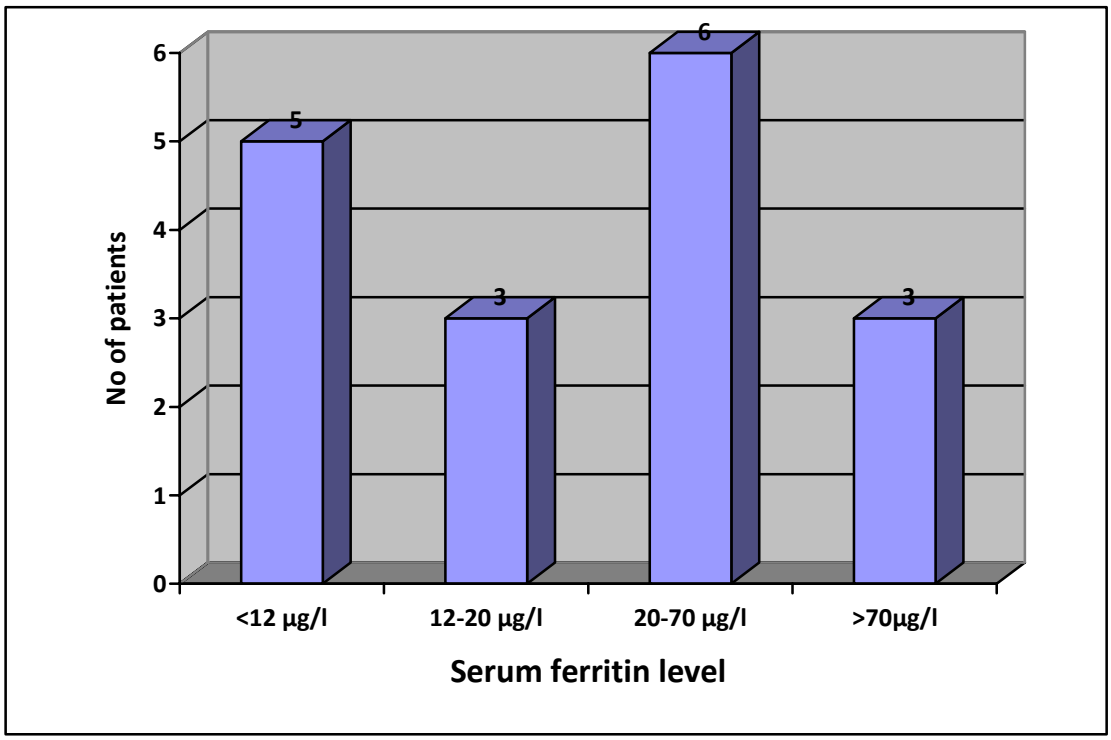
(p = <0.05 is significant, p = >0.05 is not significant)

Out of 35, 17 patients underwent serum ferritin estimation. Value of serum ferritin level in patients with FPHL has been presented in table 15 and figure 18. Serum ferritin level <70µg/l was seen in 14 cases (p = >0.05).

Table 15: Serum ferritin level in patients with FPHL

Serum ferritin	No of patients with FPHL (n=17)	Percentage (%)
<12 µg/l	5	29.41
12-20 µg/l	3	17.64
20-70 µg/l	6	35.29
>70 µg/l	3	17.64
TOTAL	**17**	

Figure 18: Level of serum ferritin in patients with FPHL



Thyroid function test was undertaken in 17 out of 35 patients. One each of the 17 patients had biochemical evidence of subclinical hypothyroidism and hyperthyroidism.

Chronic telogen effluvium:

Age incidence and distribution:

Among 180 patients with diffuse hair loss, 28 (15.55%) patients were diagnosed as CTE with age of the patients ranging from 15 - 45 years (mean age 26.74 years). Incidence of CTE was highest in the 30-40 years of age group. Out of 28 patients with CTE, 6 were in 10-20 years, 7 in 21-30 years, 13 in 31-40 years and 2 in 41-50 years of age group (Table 16).

Table 16: Age-wise distribution of patients with CTE

Age group in years	No of patients with CTE (n=28)	Percentage (%)
10-20	06	21.42
21-30	07	25
31-40	13	46.42
41-50	02	7.14
Total	**28**	

Clinical history:

Out of 28 patients, 5 patients had positive history in favor of probable etiological causes of CTE. The probable etiological causes of chronic telogen effluvium elicited from history were, drug intake (n=1, 3.57%), diabetes (n=2, 7.14%), protein energy malnutrition (n=1, 3.57%) and zinc deficiency (n=1, 3.57%) (Table 17). Family history of patterned baldness was recorded in 18 (64.28%) patients.

Table 17: Probable etiological causes of chronic telogen effluvium (Based on history)

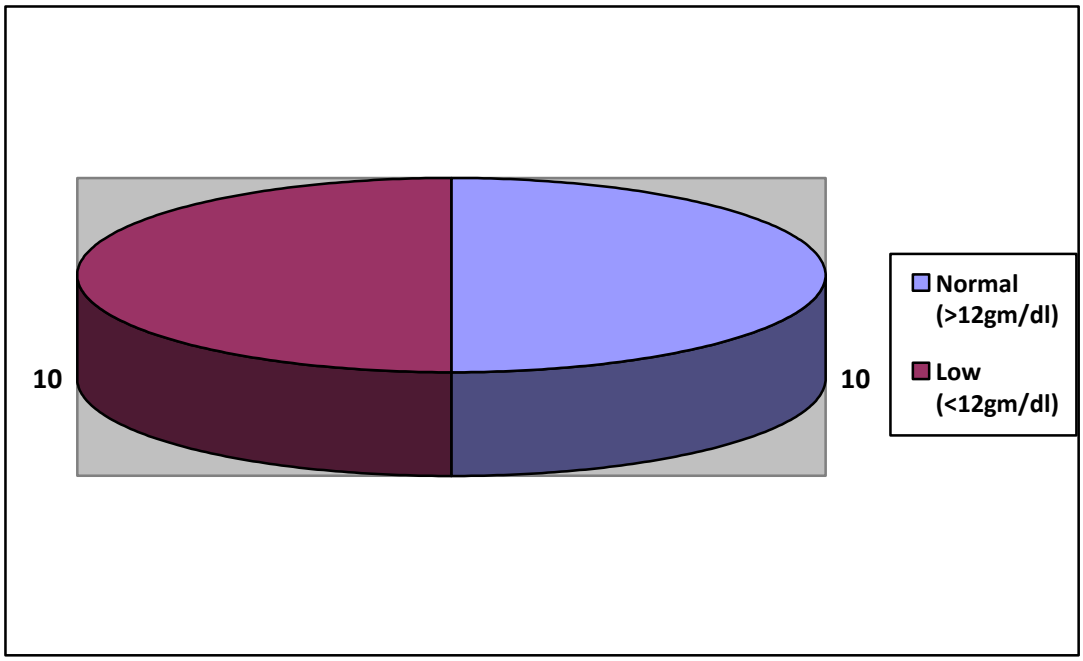
Etiological causes	No of patients (n=28)	Percentage (%)
Idiopathic	23	82.14
Drug intake	1	3.57
Diabetes	2	7.14
Protein energy malnutrition	1	3.57
Zinc deficiency	1	3.57

Clinical examination and investigation:

On scalp examination, thinning of hair was noted in 5 (1.78%) patients and bitemporal recession in 5 (17.85%) patients. Hair pull test was positive in 10 (35.71%) patients.

Complete hemogram was done in 20 out of 28 patients; low hemoglobin (<12gm/dl) level was seen in 10 patients (p = >0.05) (Figure 19). On peripheral smear examination, microcytic hypochromic anemia was seen in 5 patients, normocytic hypochromic smear in 2 patients and normocytic normochromic blood picture in 13 patients.

Figure 19: Hemoglobin level in patients with CTE

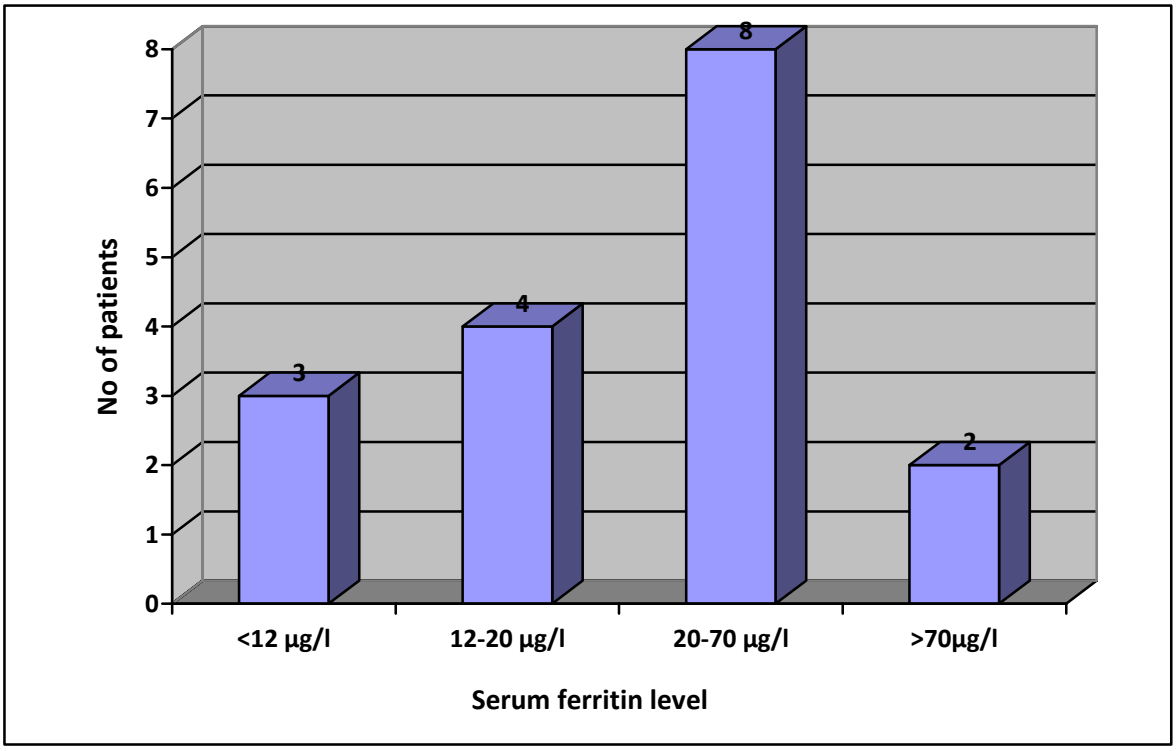


Serum ferritin was estimated in 17 out of 28 patients. Serum ferritin level of patients with CTE has been presented in table 18 and figure 20. Serum ferritin level <70µg/l was seen in 12 cases (p = >0.05). Thyroid function test was undertaken in 17 out of 28 patients but all of them were found to be euthyroid.

Table 18: Serum ferritin level in patients with CTE

Serum ferritin	No of patients with CTE (n=17)	Percentage (%)
<12 µg/l	3	17.64
12-20 µg/l	4	23.52
20-70 µg/l	8	47.05
>70 µg/l	2	11.76
TOTAL	**17**	

Figure 20: Serum ferritin level in patients with CTE



Anagen effluvium: One of the 180 patients presented with anagen effluvium. She was on chemotherapy (cyclophosphamide, adriamycin, 5-flurouracil) for carcinoma of breast. On examination, hair pull test and tug test were positive. On microscopic examination, dystrophic anagen hairs were observed.

DISCUSSION

Dermatologists are frequently confronted with hair related problems in their day-to-day practice. Hair loss has little physically harmful effects, but may lead to psychological consequences, including high levels of anxiety and depression. Hair loss is a common disorder, with an estimated life time prevalence of 1.7%; however, this figure is not a reliable estimate, as very few epidemiological studies have been published in this regard, owing partly to under-reporting.[1]

There are various causes of diffuse hair loss of which most common causes are telogen effluvium, female pattern hair loss and chronic telogen effluvium; the rest of the causes are not common and can be relatively easily diagnosed through history and clinical examination. Women presenting with diffuse hair loss is a very challenging problem for dermatologists. Diffuse hair loss may be multifactorial. Only few studies have been carried out to enlist the causes of diffuse hair loss in Indian females.[7] Till date the exact incidence or prevalence of telogen effluvium and chronic telogen effluvium remains unknown.

Telogen effluvium

In the normal scalp, 90–95% of the hair follicles are in the anagen phase and the remainder (5–10%) in the telogen phase with about 100-150 hairs being shed daily. Only a few follicles will be in the transitional or catagen phase. The biological clock that determines the end of the anagen phase and the beginning of the catagen/telogen phase is a complex phenomenon whose molecular basis is being unveiled. Various metabolic alterations such as pregnancy, malnutrition and other stressful conditions are capable of influencing the biological clock within hair

follicles, and it is possible for abnormally large number of hair follicles to enter the telogen phase simultaneously.[32]

Jain et al studied 100 cases of diffuse hair loss and observed telogen effluvium in 92% cases. Probable etiological causes of hair loss in their study were fever in 33%, psychological stress in 30%, systemic illnesses in 23%, child birth in 22%, pain abdomen and passage of worms in stool in 14%, crash diet in 14%, surgical operation and increased blood loss in 12% and drug intake in 12% cases.[7] Most common cause of telogen effluvium was fever (33%) in the above study. In present study, psychological stress was the commonest cause of hair loss (36.92%) and there was a statistically significant correlation ($p < 0.05$) between these two variables. Psychological stress was the precipitating factor for telogen effluvium in 30 cases in the above-quoted study. The causes of stress were varied; marital disharmony in 2 cases, husband's illness in 3 cases, lack of children in 4 cases, death of family member in 7 cases, parental illness in 3 cases, education related stress in 3 cases, financial constraint in 4 cases, and, stress of hair loss itself, in 4 cases.[7]

Rustom et al studied 50 cases of telogen effluvium, the probable etiological causes found in their study being psychological stress in 21 (42%) cases, fever in 11 (22%) cases, child birth in 5 (10%) cases and surgical operation in 3 (6%) cases.[13] On investigation authors noted low hemoglobin level (< 12 gm/dl) in 25 (50%) cases[13] where as in the present study it was found in 50/83 (60.24%) patients, and there was a statistically significant correlation ($p = <0.05$) between these two variables. On routine stool examination gastrointestinal parasitic infestation was found in 12 cases in the above study, which included *Entamoeba coli* in 6 cases, *Giardia lamblia* in 4 cases, and *Entamoeba histolytica* and *Ascaris lumricoides* in 1 case each.[13] In the present study parasitic infestations were seen in 2 cases (6.06%). Authors did not

notice concomitant drug intake or endocrinopathy as an etiological factor of diffuse hair loss, whereas in the present study, drug intake, hyperthyroidism and hypothyroidism were encountered as probable causes of telogen effluvium in 12/130 (9.23%), 3/74 (4.05%) and 2/74 (2.70%) cases respectively. Rustom et al could not find any probable etiological causes of diffuse hair loss in 15/50 (30%) patients based on history[13] as compared to the present study, where in 32 (24.61%) patients did not give any history pertaining to probable causes of diffuse hair loss. Rustom et al noted family history of patterned baldness in 26/50(52%) of their cases,[13] whereas in the present study it was found in 61/130 (46.92%) cases. Probable etiological causes of telogen effluvium based on the history in present study, study by Jain et al[7] and Rustom et al[13] has been depicted in table 19.

Table 19: Probable etiological causes of telogen effluvium

Sl. NO	Probable etiological causes	Present study	Study by Jain et al[7]	Study by Rustom et al[13]
1	Fever	12.30 %	33%	22%
2	Psychological stress	36.92 %	30%	42%
3	Child birth	5.38 %	22%	10%
4	Crash diet	6.15 %	14%	-
5	Surgical operation	2.30 %	13%	6%
6	Chronic blood loss	4.61 %	-	-
7	Drug intake	9.23 %	12%	-
8	Topical application	6.15 %	-	-

The association of low serum ferritin level and hair loss has been debated over the years. There has been controversy over the serum ferritin cut-off level below which it can be defined as nutritional deficiency, triggering hair loss.[33] Ferritin is a highly conserved protein complex that plays an important role in iron storage and is recognized as the main iron-binding protein in non erythroid cells. Intracellular ferritin is synthesized by the smooth endoplasmic reticulum. Serum ferritin is synthesized by the rough endoplasmic reticulum and glycosylated at Golgi apparatus before being secreted. Generally, serum ferritin is directly related to intracellular ferritin and thus to total body iron stores. Only iron deficiency causes very low serum

ferritin concentrations; therefore a low ferritin concentration is very specific for iron deficiency.[34]

Using serum ferritin level as a marker for iron storage deficiency, the definition of iron deficiency (but not specifically iron deficiency anemia) in various studies has ranged from a serum ferritin level of ≤15µg/l to <70µg/l. According to World Health Organization (WHO), anemia is defined as serum hemoglobin level <12gm/dl.[35]

In a prospective cohort study by Sinclair R (2002), in 194 female subjects aged 11 to 72 years with telogen effluvium, 12 (6%) were found to have serum ferritin level ≤20µg/l. All of the patients had normal hemoglobin concentration.[33]

In the present study, the observed values of serum ferritin level were as follows; <12µg/l in 25/74 (33.78%) patients, 12-20 µg/l in 17/74 (22.97%) patients, 20-70 µg/l in 27/74 (36.48%) patients and >70µg/l in 5/74 (6.75%) patients. Serum ferritin level was <70µg/l in 69/74 patients, which was statistically non-significant (p = >0.05). Kantor et al compared the serum ferritin levels of female patients with different etiological causes of hair loss, e.g., telogen effluvium, androgenetic alopecia, alopecia areata and alopecia totalis/ universalis with healthy control group.[36] The mean serum ferritin levels were lower in subjects with androgenetic alopecia and alopecia areata compared with healthy control subjects, but not lower when compared with those with TE or alopecia totalis/ universalis.[36]

Female pattern hair loss

Fewer than 45% of women have a head full of hair throughout the life.[2] Female pattern hair loss is characterized by a reduction in hair density over crown and

frontal scalp with retention of frontal hair line. Prevalence of female pattern hair loss increases with advancing age. Affected women may experience psychological distress and impaired social interaction. In most of the cases the diagnosis can be made clinically and medical management is possible.

Gan et al noted increased prevalence of FPHL with increasing age, from approximately 12% amongst women aged between 20 to 29 years to over 50% among women over the age of 80.[2]

In the present study total 35 patients were diagnosed as FPHL. Among them 15 (42.85%) patients had type I and 20 (57.14%) patients had type II, but none had type III hair loss. In a study by Birch et al conducted at Royal Hallamshire hospital, Sheffield, UK, 337 women aged 18-99 years, who presented to a general dermatology clinic with complaints unrelated to hair growth, 6% of women less than 50 years were diagnosed as having female pattern hair loss.[15] This incidence increased to 38% in women above 70 years.[15] In our study no patient above 70 years was diagnosed as FPHL. Gan et al[2] noted increased incidence of FPHL upto 70 years of age and decreased incidence thereafter, whereas in the present study, a lower incidence of FPHL was recorded in the older age group. This may be due to reduced concern regarding hair loss in the older age group in this population, who are mostly rural. Out of 35 patients, the mean age of onset of hair loss in our study was 31.60 years as compared to 34.4 years in the study by Zhang et al.[17] Female pattern hair loss type III or Sinclair grade 5 is a severe form of FPHL which is uncommon and affects less than 1% of women.[2] In our study we did not notice any case of FPHL type III. Severe bitemporal recession as observed in male androgenetic alopecia is also uncommon in women in whom the frontal hairline is usually preserved.[2] In this study, we noted mild bitemporal recession in 3(8.57%) patients. In a study of 60 female patients of

patterned baldness, by Zhang et al, family history of androgenetic alopecia was present in 27/60 (45%) patients;[17] in the present study such history was noted in 14/35 (38.8%) patients. Mean duration of hair loss of in our patients was 1.5 years, as compared to 4.49 years in the study by Zhang et al.[17]

In this study low hemoglobin level (<12gm/dl) was noted in 6/20 (30%) patients compared to the study by Zhang et al, where 5/60 (8.3%) patients had low hemoglobin.[17] Only 2/20 patients showed microcytic hypochromic peripheral blood smear in the present study.

Out of 17 patients, 2 had thyroid dysfunction of which one had subclinical hypothyroidism and the other had hyperthyroidism. However, in the study by Zhang et al, of the 17 patients with FPHL, slight deviation of TSH level was noted in 2(11.76%) patients, but T3, T4 levels were within normal limits.[17]

In our study we divided the patients based on their serum ferritin levels into 4 categories:

- <12µg/l (iron deficiency)
- 12-20 µg/l (iron depletion)
- 20-70 µg/l (serum ferritin level lower than required for normal hair cycle)
- >70 µg/l (normal ferritin level)

Serum ferritin levels were estimated in 17 out of 28 patients with FPHL. Fourteen (82.35%) out of these 17 patients had serum ferritin level < 70 µg/l. This is higher compared to the findings of the study by Zhang et al[17], where only 35% of the FPHL patients had serum ferritin level <70 µg/l. In our study we did not find significant correlation between FPHL and serum ferritin

levels. This is similar to the findings of the study by Zhang et al.[17] Serum ferritin level of the patients with FPHL in our study and that of Zhang et al[17] has been compared in table 20.

Olsen EA et al studied 285 women with FPHL, out of which 215(75.4%) showed serum ferritin level < 70 µg/l[34] as compared to 14/17 (82.35%) in our study. The mean hemoglobin and serum ferritin level was 11.2gm/dl and 36.11 µg/l respectively, as compared to the values 13.64gm/dl and 61.01µg/l in study by Olsen EA et al.[34]

Table 20: Comparison of serum ferritin level in present study and study by Zhang et al[17]

Serum ferritin	**Study by Zhang et al[17] (n=60)**	**Present study (n=17)**
<12 µg/l	4	5
12-20 µg/l	2	3
20-70 µg/l	15	6
>70 µg/l	39	3
Total	**60**	**17**

In another case-control study by Aydingoz et al, the authors compared 10 female subjects with FPHL and 46 healthy controls.[37] The study found no difference in the prevalence of depleted iron stores or in the prevalence of iron deficiency anemia in both the groups.

Chronic telogen effluvium:

Chronic telogen effluvium is a diffuse, generalized form of hair loss of unknown cause that is common in middle aged women. It affects the entire scalp. It often starts abruptly and is alarming to the patient as large number of hairs are shed.[27] In the present study among 180 patients with diffuse hair loss 28 were diagnosed as chronic telogen effluvium. Duration of hair loss varied from 6 months to 8 years. Incidence of CTE was highest in 31-40 (46.42%) years of age group. Out of 28 patients with CTE, 6 (21.42%) patients were in 10-20 years, 7(25%) patients were in 21-30 years, 13(46.42%) patients were in 31-40 years and 2 (7.14%) patients were in 41-50 years of age group. Premenopausal women (96.4%) were most commonly affected, in contrast to the study by Garcia-Hernandez, in which it was predominantly seen among post-menopausal women (67%).[26] Olsen et al studied 96 women of CTE, of whom 58 (60%) were premenopausal and 38(40%) were postmenopausal.[34]

In our study probable etiological causes of CTE based on history were drug intake in 1(3.57%) patient, diabetes in 2(7.14%) patients, protein energy malnutrition in 1(3.57%) patient, and zinc deficiency in 1 (3.57%) patient. On scalp examination hair thinning was noted in 5 (1.78%) patients, bitemporal recession in 5 (17.85%) patients and hair pull test was positive in 10 (35.71%) patients.

Of the twenty eight patients with CTE in our study, 20 underwent hemoglobin estimation, and 10 (50%) of them were found to have a low level (<12gm/dl), which was not statistically significant (p = >0.05). Serum ferritin level was done in 17 patients, and the levels were as follows;

- <12µg/l in 3 (17.64%) patients
- 12-20µg/l in 4 (23.52%) patients

- 20-70µg/l in 8 (47.05%) patients
- >70µg/l in 2 (11.76%) patients.

Fifteen out of these 17 patients had serum ferritin level <70µg/l, which was statistically non-significant (p = >0.05). In the study by Olsen et al[34], 72 (75%) out of 96 patients had serum ferritin level <70µg/l, as compared to 15/17 (88.23%) patients in our study. Mean serum ferritin level of the patients with CTE in our study was 39.39µg/l, as compared to 51.81µg/l in the study by Olsen et al.[34] Rushton et al studied 200 women with CTE out of which 95% had serum ferritin level less than 70µg/l.[38] Thyroid profile was also done on 17 of the 28 patients in our study and showed no abnormal values.

Anagen effluvium

Anagen effluvium is typically reversible. Severity of hair loss depends on the route of administration as well as the dose and frequency of administration. Hair shedding usually begins at 1-3 weeks after initiation of chemotherapy. Due to long anagen phase, the scalp is the most common location for hair loss, while other terminal hairs are variably affected depending on the percentage of hairs in anagen phase.

Out of 180 patients only one was diagnosed as anagen effluvium. The patient was started on cyclophosphamide, adriamycin and 5-flurouracil regimen for carcinoma of breast. After 3 weeks of initiation of chemotherapy she noticed hair loss. On scalp examination, diffuse hair loss was seen. The World Health Organization criteria for anagen effluvium is grade 0 = no loss, grade 1 = mild hair loss, and grade 2 = pronounced or complete hair loss. According to WHO criteria, the present case belonged to grade 2.[39]

CONCLUSION

A hospital-based cross-sectional study was conducted to determine various causes of diffuse hair loss and to elicit their correlation with various laboratory parameters. Diagnosis of patients was mainly based on clinical findings. Various laboratory investigations for determining iron deficiency anemia, thyroid dysfunction and parasitic infestation were done.

The following conclusions were drawn from the study:

1. Telogen effluvium were the most common type of diffuse hair loss in women followed by female pattern hair loss and chronic telogen effluvium.
2. Telogen effluvium most commonly affected individuals in the age group of 20-30 years. Psychological stress and hemoglobin level was found to be significantly associated with telogen effluvium whereas serum ferritin level had no statistically significant correlation. This may be because of the fact that, estimation of serum ferritin level was possible to do only in few patients.
3. Female pattern hair loss most commonly affected individuals in the age group of 20-30 years. Female pattern hair loss type III was rarely encountered. Relationship between hemoglobin levels and FPHL was statistically significant whereas serum ferritin level had no statistical significance.
4. Chronic telogen effluvium most commonly affected individuals in the age group of 30-40 years. In most of the patients it was idiopathic. No significant relationship was observed between CTE, hemoglobin level and serum ferritin.
5. Anagen effluvium was a rare cause for diffuse hair loss.

Diffuse hair loss is a common clinical presentation. The diagnosis can usually be established with a history, particularly focusing on the chronology of events, examination of the bulbs of the shed hairs, and a few simple screening blood tests.

Once the diagnosis has been established, treatment appropriate that diagnosis is likely to arrest the hair loss in all cases except chronic telogen effluvium. Patients with chronic telogen effluvium can, however, be consoled by the fact that their condition is no progressive and self-limiting.

SUMMARY

A hospital-based, cross-sectional study for determining causes of diffuse hair loss in women was conducted during the period of October 2011 to September 2013. All adolescent girls and women who presented with diffuse hair loss were included in the study. Detailed history was taken from all study subjects with special emphasis upon the major febrile illness, psychological stress or any surgery or child birth 3 months prior to the onset of hair loss. History of chronic blood loss, recent past history of crash diet, detailed history of drug intake was also recorded.

Specific signs of anemia, jaundice, and thyroid swelling were recorded in general clinical examination. Type of hair loss, hair thinning and temporal recession was noted on scalp examination. Hair pull test was performed. Specific laboratory investigations for determining iron deficiency anemia, thyroid dysfunction and parasitic infestation were done.

Following are the salient findings of this study:

- Telogen effluvium was the most common type of diffuse hair loss in women followed by female pattern hair loss and chronic telogen effluvium. Psychological stress and iron deficiency anemia were most common causes of telogen effluvium. Psychological stress and telogen effluvium were found to be significantly associated.

- Telogen effluvium and female pattern hair loss were most commonly observed in individuals of 20-30 years of age group whereas chronic telogen effluvium in 30-40 years.

- Low hemoglobin level was significantly associated with telogen effluvium and female pattern hair loss but not with chronic telogen effluvium.

- Serum ferritin level had no significant correlation with telogen effluvium, female pattern hair loss and chronic telogen effluvium.

- Anagen effluvium was a rare cause of diffuse hair loss.

A careful clinical history is essential in case of diffuse hair loss. In patients complaining of increased hair shedding, clinicians should investigate for potential triggers from 3 months before the development of hair loss, including intake of medication, systemic illness, crash diet or weight loss. As diffuse hair loss may be multifactorial, it is important to take a detailed history, need for thorough clinical examination and to do appropriate investigations to find out causative factors and treat them appropriately.

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ANNEXURES

PROFORMA:

SCHEME OF CASE TAKING

Name: SL NO:

Age Date:

Guardian name: IP NO/ OP NO:

Occupation: Family income:

Address:

1. Complaints with duration

2. Male pattern baldness in the family

Father	: YES/NO	Mother	: YES/NO
Grandfather:	YES/NO	Grandmother:	YES/NO
Brother	: YES/NO	Sister	: YES/NO

3. Menstrual history : Menopause : Yes / No

Menorrhagia : Yes / No

4. If patient is pregnant : YES / NO

5. If patient is lactating : YES / NO

6. Last child birth :

7. No of children :

8. Passing worms in stool: YES / NO

9. H/o crash diet: YES/NO If YES, duration:

Cause:

10. H/o of chronic blood loss: YES/NO If YES, duration:

Cause:

11. Any history of

Fatigue : YES/NO

Lethargy : YES/NO

Anxiety : YES/NO

Hoarse voice : YES/NO

Intolerance to heat : YES/NO

Intolerance to cold : YES/NO

Palpitation : YES/NO

Constipation : YES/NO

Disturbed sleep : YES/NO

Loss of appetite : YES/NO

Tremor: YES/NO

- Feeling tired: YES/NO
- Cheilosis: YES/NO
- Impaired concentration: YES/NO

12. Past history (3 month):

Fever:

Psychological stress:

Systemic illnesses

Child birth: YES/NO

Surgery: YES/NO

Abortion: YES/NO

Drugs: YES/NO

If YES, Name of the drug -

Diabetes : YES/NO

Hyper/hypothyroidism: YES/NO

Hypertension: YES/NO

Weight loss: YES/NO

Asthma: YES/NO

Others:

13. General Physical Examination :

Weight:	BP:	Hair pull test:
Pallor:	Koilonychia:	Cyanosis:
Icterus:	Clubbing:	Lymphadenopathy:
Pedal edema:	Tremor:	Pulse rate:

14. Scalp examination :

Hair thinning: Present / Absent

Hair loss type: diffuse/ FPHL – Ludwig's scale - Grade 1
Grade 2
Grade 3

Bitemporal recession: YES / NO

15. Systemic Examination :

Cardiovascular system :

Respiratory system :

Central nervous system :

Per abdomen :

16. Diagnosis :

17. Investigations: Date

TSH -

T_3 -

T_4 -

Serum ferritin -

Hemoglobin -

Peripheral smear-

Stool test:

Occult blood-

Parasite, ova, cyst-

Scalp biopsy-

Hair pull test-

Microscopic examination of hair-

Hair collection-

1 day

2 day -

3 day -

4 day -

5 day -

6 day -

7 day -

SAMPLE INFORMED CONSENT FORM

BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR-586 103

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT: - A CROSS SECTIONAL STUDY TO DETERMINE VARIOUS CAUSES OF DIFFUSE HAIR LOSS IN WOMEN.

PG GUIDE : - DR. APARNA PALIT.

PG STUDENT : - DR. SHASHIKANT.

PURPOSE OF RESEARCH:-

I have been informed that this project will study the causes of diffuse hair loss in women.

BENEFITS:-

I understand that my participation in this study will help the investigator to study the various causes of diffuse hair loss which will help in the management of the disease.

PROCEDURE:-

I understand that relevant history will be taken and I will undergo detailed clinical examination after which necessary investigations will be done whenever required.

RISK AND DISCOMFORTS:-

I understand there is no risk involved and I will experience minimal pain during the procedures performed.

CONFIDENTIALITY:-

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs, videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:-

I understand that I may ask more questions about the study at any time concerned. Dr. Shashikant is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:-

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that Dr. Shashikant may terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician, if this is appropriate.

INJURY STATEMENT:-

I understand that in the unlikely event of injury to me resulting directly from my participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I understand that by my agreement for my participation in this study, I am not waiving any of my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that I undergo and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent for my participation as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

ETHICAL CLEARANCE CERTIFICATE

Ethical Committee BIJAPUR-586103 OUT WARD No 87/2011 Date 21-10-11 B.L.D.E.'s Shri B. M. PATIL Med.cal College

B.L.D.E. UNIVERSITY'S

SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 20-10-2011 *at* 10-30 am *to scrutinize the Synopsis/Research projects of postgraduate/undergraduate student/Faculty members of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis/Research project has been accorded Ethical Clearance.*

Title "A cross sectional study to determine various causes of diffuse hair loss in women"

Name of P.G./U.G. student/Faculty member Dr. Shashikant Dept of Dermatology

Name of Guide/Co-investigator Dr. Aparna Palit, Prof Dermatology

DR.M.S.BIRADAR,
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Chairman
Ethical Committee
BLDEA'S Shri. B.M. Patil
Medical College
Bijapur-586103

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

KEY TO MASTER CHART

A – Anagen hair

Ab – Absent

AE – Anagen effluvium

BT – Bitemporal

CTE – Chronic telogen effluvium

D – Iron deficiency (Serum ferritin <12µg/l)

DL – Iron depletion (Serum ferritin 12µg/l - 20µg/l)

DMA – Dimorphic anemia

FPHL – female pattern hair loss

H - High

Hb – Hemoglobin

H/o – History of

HL – Hair loss

L - Low

LF – Low serum ferritin (Serum ferritin 20µg/l - 70µg/l)

MA – Megaloblastic anemia

MCHC –Microcytic hypochromic

MS – Microscopy

N – Normal

NCHC – Normocytic hypochromic

NCNC – Normocytic normochromic

ND – Not done

Neg – Negative

P – Present

PHL – Pattern hair loss

Pos – Positive

PS – Peripheral smear

Sl. No – Serial number

T – Telogen hair

TE – Telogen effluvium

TR – Trophozoites

TSH – Thyroid stimulating hormone

Yrs – Years

I – grade I

II – Grade II

III – Grade III

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
1	24	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
2	48	0.5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
3	30	2	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
4	23	6	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
5	18	2	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
6	22	6	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
7	25	2	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
8	36	3	Ab	Ab	P	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
9	23	6	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
10	19	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
11	22	0.5	P	Ab	P	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	P	Ab	Ab	Neg
12	36	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
13	28	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Neg
14	21	24	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	AB	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Neg
15	21	0.5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
16	22	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
17	20	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
18	22	4	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
19	22	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
20	34	6	P	Ab	P	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	P	P	Ab	Ab	Neg
21	28	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Neg
22	42	3	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	P	P	Ab	Neg
23	21	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
24	30	2	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
25	18	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
26	30	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
27	28	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	P	Ab	Neg
28	45	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Neg
29	17	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
30	31	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Neg
31	22	6	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
32	30	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Neg
33	35	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	Neg
34	24	0.5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
35	17	3	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Neg
36	40	0.5	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
37	21	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
38	38	5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
39	23	1	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
40	21	1	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
41	14	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
42	22	2	P	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
43	30	2	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Pos
44	26	0.5	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
45	17	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
46	40	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Pos
47	22	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
48	18	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
49	25	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
50	34	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Pos
51	34	0.5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
52	21	12	P	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
53	22	0.25	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
54	20	4	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
55	33	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
56	42	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
57	32	4	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
58	20	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Pos
59	17	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
60	12	6	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	P	Ab	Ab	Pos

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
61	22	48	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Neg
62	30	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
63	30	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	P	P	Ab	Neg
64	38	24	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Pos
65	28	48	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
66	20	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Pos
67	23	36	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Ab	Neg
68	21	2	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
69	38	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
70	35	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
71	34	24	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
72	15	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
73	31	3	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
74	25	5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Ab	Pos
75	25	0.25	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
76	27	0.5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Pos
77	35	72	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Neg
78	22	24	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
79	22	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
80	28	108	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Neg
81	40	0.5	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	P	P	Ab	Ab	P	Ab	Ab	Pos
82	24	24	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	Pos
83	29	0.5	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	P	P	Ab	P	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
84	32	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
85	20	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
86	45	24	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	Ab	P	P	P	Ab	Pos
87	54	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
88	27	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
89	22	36	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Pos
90	19	20	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
91	15	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
92	23	84	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Neg
93	22	6	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Neg
94	33	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Neg
95	42	12	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Neg
96	22	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	P	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
97	20	6	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Neg
98	30	2	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Pos
99	32	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
100	22	3	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Pos
101	35	96	Ab	Ab	Ab	Ab	Ab	AbAb	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
102	32	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	P	P	Ab	Pos
103	38	36	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
104	26	2	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Pos
105	24	4	P	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	P	Ab	Ab	Ab	P	Ab	Ab	Neg
106	29	7	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
107	22	3	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
108	29	5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	Ab	P	P	Ab	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
109	21	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
110	27	3	P	Ab	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	P	Ab	Ab	Pos
111	23	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Neg
112	30	6	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Neg
113	25	6	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Pos
114	18	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Pos
115	23	2	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
116	26	0.5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
117	28	24	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
118	16	4	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Pos
119	26	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
120	32	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
121	16	3	Ab	Ab	Ab	Ab	AbAb	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Neg
122	35	0.25	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Pos
123	25	3	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Neg
124	25	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	P	Pos
125	16	3	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	P	P	Ab	Ab	Pos
126	30	0.5	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	P	P	P	P	Pos
127	22	6	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Pos
128	19	3	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	Ab	Ab	Neg
129	18	0.25	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Neg
130	44	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	Ab	Ab	P	Ab	Ab	Neg
131	21	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
132	20	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
133	40	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
134	32	0.25	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
135	16	0.5	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	P	Neg
136	50	2	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	Pos
137	38	6	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	Pos
138	28	0.25	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
139	21	5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
140	55	12	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
141	20	2	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
142	15	0.5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
143	40	6	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	P	Ab	Pos
144	18	2	P	Ab	AbAb	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
145	28	2	Ab	Ab	P	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Pos
146	23	6	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
147	33	60	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
148	30	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	P	Neg
149	21	4	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
150	42	4	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	P	Ab	Ab	Pos
151	26	0.5	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
152	35	3	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	P	Ab	Ab	Pos
153	20	5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	P	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Neg
154	15	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
155	18	3	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Neg
156	21	12	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Pos

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
157	40	36	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
158	25	5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
159	54	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	Ab	P	P	Pos
160	27	2	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	AbAb	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Pos
161	27	1	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Neg
162	30	0.5	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	P	P	Ab	
163	19	2	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Pos
164	21	3	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
165	32	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
166	26	8	P	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos
167	20	24	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
168	30	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Pos

Sl. No	Age in yrs	Duration of HL (in months)	Family h/o PHL	Menstrual H/O		Pregnancy	Lactation	Worms in stool	H/o crash diet	H/o chronic blood loss	H/o Fever 3 months before onset of hair loss	Psychological stress	H/o child birth 3 months before onset of hair loss	H/o surgery 3 months before onset of hair loss	H/o Diabetes	H/o thyroid dysfunction	H/o drug intake	Pallor	Koilonychia	Scalp examination				Hair pull test
				Menopause	Menorrhagia															Hair thinning	Type of hair loss		BT recession	
																					Diffuse	FPHL		
169	21	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Pos
170	20	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
171	22	1	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Pos
172	20	5	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	P	P	Ab	Ab	Pos
173	20	4	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
174	21	12	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Neg
175	30	24	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
176	23	0.5	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Pos
177	20	2	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	P	Ab	Ab	Neg
178	31	72	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	P	Neg
179	32	2	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	P	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Neg
180	21	1	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Ab	Pos

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	N	NCNC	N	-	-	D	ND	TE
Neg	T	ND	ND	ND	ND	ND	ND	ND	TE
Neg	T	N	NCNC	N	-	-	DL	ND	TE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	ND	ND	ND	ND	ND	ND	ND	TE
Pos	T	L	MCHC	N	-	-	D	ND	TE
Neg	T	L	NCNC	N	-	-	LF	ND	TE
Neg	T	L	MCHC	N	-	-	D	ND	TE
Neg	T	L	NCNC	N	-	-	ND	ND	TE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	N	NCNC	H	-	-	LF	ND	TE
Neg	T	L	NCNC	N	-	-	LF	ND	TE+FPHL II

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	N	NCNC	N	-	-	LF	ND	TE+FPHL II
Neg	T	N	NCNC	N	-	-	LF	ND	CTE
Pos	T	L	MCHC	N	-	-	D	N	TE
Neg	T	N	NCNC	N	-	-	DL	N	TE
Neg	T	L	NCNC	N	-	-	LF	ND	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	D	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Pos	T	L	MCHC	N	-	-	D	ND	TE+FPHL I
Neg	T	L	MCHC	N	-	-	D	N	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL I

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Pos	T	L	NCHC	N	-	-	DL	N	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	LF	ND	TE
Neg	T	L	MCHC	ND	-	-	ND	ND	CTE
Neg	T	N	NCNC	N	-	-	ND	ND	TE
Neg	T	N	NCHC	N	-	-	N	N	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	N	NCHC	N	-	-	DL	N	TE
Neg	T	N	NCNC	N	-	-	ND	ND	FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	MCHC	N	-	-	D	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	L	NCHC	N	-	-	DL	N	TE
Pos	T	L	MCHC	N	-	-	D	ND	TE
Pos	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	N	NCNC	N	-	-	ND	ND	TE
Neg	T	N	NCNC	N	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	LF	ND	FPHL I
Neg	T	N	NCNC	L	-	-	N	N	TE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II
Neg	T	N	NCNC	N	N	N	LF	ND	TE
Neg	T	ND	ND	ND	ND	ND	ND	ND	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	N	NCNC	N	-	-	LF	ND	CTE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	N	NCNC	N	-	-	DL	ND	CTE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	N	NCNC	N	-	-	DL	ND	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II
Neg	T	N	NCNC	N	-	-	DL	ND	TE
Neg	T	L	MCHC	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Pos	T	L	MCHC	N	-	-	D	ND	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	N	NCNC	N	-	-	D	ND	FPHL I
Neg	T	ND	ND	ND	-	-	LF	ND	CTE
Neg	T	L	MCHC	N	-	-	DL	ND	TE+FPHL I
Neg	T	N	NCNC	N	-	-	LF	ND	FPHL II
Pos	T	ND	ND	ND	-	-	ND	ND	CTE
Pos	T	L	MCHC	N	-	-	D	N	CTE
Pos	T	ND	ND	ND	-	-	ND	ND	CTE
Neg	T	N	NCNC	N	-	-	D	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	MCHC	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	CTE
Neg	T	N	NCNC	N	-	-	LF	TR	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	L	NCNC	N	-	-	D	TR	TE
Neg	T	ND	ND	ND	-	-	LF	ND	TE
Neg	T	L	ND	ND	-	-	LF	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	DL	N	FPHL II
Neg	T	N	NCNC	N	-	-	LF	N	FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL I
Neg	T	N	NCNC	N	-	-	LF	N	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	N	NCNC	H	N	N	N	N	TE+FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL I
Neg	T	ND	ND	ND	-	-	ND	ND	CTE
Neg	T	N	NCNC	N	-	-	DL	ND	TE
Neg	T	L	MCHC	ND	-	-	ND	N	CTE
Neg	T	N	NCNC	L	N	N	D	ND	TE+FPHL I
Neg	T	N	NCNC	ND	-	-	ND	ND	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	TE+FPHL II
Neg	T	N	MCHC	N	-	-	DL	ND	CTE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	L	NCNC	N	-	-	LF	N	TE+FPHL I
Neg	T	L	NCNC	N	-	-	LF	ND	TE
Neg	T	N	NCNC	H	-	-	LF	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	TE+FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	N	NCNC	N	-	-	LF	N	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL I
Neg	T	ND	ND	ND	-	-	ND	ND	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	N	NCNC	N	-	-	LF	N	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	N	NCNC	N	-	-	ND	ND	TE
Neg	T	L	MCHC	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Pos	T	L	MCHC	N	-	-	D	ND	TE
Neg	T	L	MCHC	N	-	-	D	ND	TE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II
Neg	T	N	NCNC	N	-	-	N	N	TE
Neg	T	L	NCNC	L	-	-	N	N	CTE
Neg	T	L	NCNC	N	-	-	DL	N	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	DL	N	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	D	N	TE
Pos	T	L	MA	N	-	-	ND	ND	TE+FPHL I
Neg	T	N	NCNC	N	-	-	N	N	TE
Neg	T	L	NCHC	N	-	-	DL	ND	TE
Neg	T	L	NCNC	N	-	-	LF	N	TE
Neg	T	L	NCNC	L	N	H	DL	N	TE
Neg	T	L	MCHC	N	-	-	D	N	TE
Pos	T	ND	ND	ND	-	-	ND	ND	CTE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	L	MCHC	N	-	-	D	N	TE
Neg	T	L	NCNC	N	N	N	LF	N	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	N	N	TE+FPHL I
Neg	T	L	NCNC	N	N	N	DL	N	TE+FPHL II
Neg	T	N	NCNC	ND	-	-	ND	N	TE
Neg	T	L	NCNC	N	N	N	LF	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II
Neg	T	N	NCNC	N	-	-	LF	N	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	MCHC	N	N	N	D	N	TE+FPHL I
Neg	T	ND	ND	ND	-	-	ND	ND	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Pos	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCNC	N	-	-	DL	ND	TE
Neg	T	L	NCNC	N	N	N	LF	N	CTE
Neg	T	L	NCNC	H	N	N	D	ND	CTE
Neg	T	L	NCNC	N	N	N	LF	N	TE
Neg	A	N	NCNC	N	-	-	N	N	AE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Pos	T	L	NCNC	N	-	-	D	N	TE
Neg	T	L	NCNC	N	N	N	DL	N	TE
Neg	T	ND	ND	ND	-	-	ND	ND	CTE
Neg	T	N	NCNC	N	-	-	LF	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL I

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL II
Neg	T	L	MCHC	N	-	-	D	N	TE
Neg	T	L	MCHC	N	N	N	N	N	FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE+FPHL II
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Pos	T	L	DMA	ND	-	-	ND	ND	TE
Pos	T	L	MCHC	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	CTE
Pos	T	L	NCNC	ND	-	-	ND	ND	CTE
Neg	T	L	NCNC	N	N	N	D	N	TE

Tug test	Hair root	Investigations						Stool MS	Diagnosis
		Hb	PS	Thyroid profile			Serum ferritin		
				TSH	T3	T4			
Neg	T	L	MCHC	N	-	-	D	N	TE
Neg	T	L	MCHC	N	-	-	D	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	L	NCHC	N	N	N	D	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	ND	ND	ND	-	-	ND	ND	FPHL I
Neg	T	ND	ND	ND	-	-	ND	ND	CTE
Neg	T	ND	ND	ND	-	-	ND	ND	TE
Neg	T	N	NCNC	N	-	-	DL	ND	TE
Neg	T	N	NCNC	N	N	N	D	ND	FPHL I
Neg	T	ND	ND	N	N	N	ND	ND	TE
Neg	T	L	NCNC	N	N	N	DL	ND	TE