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Original Research Article

Study of endocrinal profile and trichoscopic features in female pattern hair loss at tertiary care hospital

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ABSTRACT

Background: Patterned hair loss is one of the commonest conditions for which both men and women seek dermatologist. Though term androgenetic alopecia is used synonymously for female pattern hair loss, role of androgens and other hormones in its causation remains controversial. Also, in literature few studies are present suggesting role of trichoscopy in female pattern hair loss. We sought to investigate role of endocrine disturbances and trichoscopic findings in female pattern hair loss as per Ludwig classification.
Methods: This was a prospective case control study. 100 cases and 100 age matched controls were enrolled in study. Laboratory investigations were done in both groups and trichoscopic examination was done in cases, at frontal and occipital scalp. The data was analyzed using t test, McNemar test and as mean, standard deviation and as percentages.
Results: The difference between the mean value of body mass index, hemoglobin, T4, prolactin, follicle stimulating hormone, testosterone and dehydroepiandrosterone were found to be statistically significant as compared to controls. On trichoscopy, hair diameter diversity was commonest findings, however focal atrichia, 2-3 hairs/hair unit, white dots, honey comb pigmentation (HCP) correlated with grade of hair loss.
Conclusions: Our study supports role of hormonal disturbances in causation of female pattern hair loss and thus we recommend doing tests in early onset and severe degrees of hair loss. Though diagnosis is mainly clinical trichoscopy could be excellent tool which is simple, non-invasive and cost-effective tool and may help to differentiate Female patterned hair loss from other condition like chronic telogen effluvium.

Keywords: Hormones, Patterned hair loss, Ludwig, Trichoscopy

INTRODUCTION

A woman's hair is central to her beauty and sexuality. Long strong and shining hair stands for vitality, youth and health of woman and thus hair loss in woman produces greater psychological distress than in men.

One of the most common hair problems for which both men and women will seek advice from a dermatologist is patterned hair loss (PHL).[1] FPHL is non-scarring alopecia, marked by miniaturization of hair follicles, terminal hair are transformed into vellus hairs, decrease in hair density and thinning of hair over frontal, mid-frontal and vertex region with retention of frontal hair line.[1]

Exact patho-physiology of female pattern hair loss is unknown. Various factors are implicated in causation of disease such as genetic factors, hair cycle defects, hormonal and environmental factors.[2] FPHL may occur alone or may be a part of constellation of androgen related conditions. Few studies have shown association of serum ferritin and thyroid hormones with FPHL but contradictory results are also found in other studies.

Trichoscopy- "dermoscopy of scalp" has gained momentum in field of dermatology in the last one decade. Its use in various scalp and hair disorders is well established; however, few studies are there in literature for FPHL.

Trichoscopic features for FPHL which are described in literature includes variation in hair shaft diameter, increased in number of vellus hairs, yellow dots, white and brown peri-pilar sign, white dots, honey comb pigmentation, areas of focal atrichia and single hair per follicular unit.[3]

As there is paucity of literature of FPHL in India, this study was aimed to investigate the role of endocrinal profile in women with pattern hair loss and to correlate the trichoscopic parameter in female pattern hair loss using Ludwig's scale.

METHODS

Study type

The research was a prospective case control study.

Study place and duration

The study was conducted at the department of dermatology, venereology and leprology, Goa Medical College, Bambolim, Goa. The duration of the study was 1 year (February 2019 to January 2020).

Sample size

The sample size was 100 cases and 100 controls.

Selection criteria

Inclusion criteria

Patients with grade 1-3 FPHL and male type of fronto-temporal recession (FAGA-M) according to Ludwig's classification, age group 18-60 years, patients giving consent.

Exclusion criteria

Any active infection of the scalp, malignancy, on chemotherapy or radiotherapy, suffering from acute illness; and patients not giving consent were excluded from the study.

Procedure of the study

A detailed history was elicited and thorough clinical examination was done of every patient, recorded in a specially prepared proforma. Photographic records were maintained.

Informed consent was taken from patients included in a study. Patients were considered to have female pattern hair loss if they had thinning of hair over the crown, temporal area, recession of hair line, widening of central partition.

Cutaneous examination was done to look for signs of hyperandrogenism like acne, hirsutism, acanthosis nigricans. The pattern of hair loss was noted and was evaluated according to the Ludwig scale into Ludwig I (LW I), Ludwig II (LW II), and Ludwig III (LW III) and in male type fronto-temporal recession (MT).

Hair pull test was done on each patient followed by Trichoscopy with a contact non-polarized dermoscope at 10-fold magnification at hair loss areas in all patients and compared with occipital region (served as control region). Trichoscopic photographs of each patient frontal and occipital (control) region were assessed for typical differentiating features.

As pattern hair loss predominantly affects frontal scalp, thus the relative dermoscopic difference in hair related variables in frontal and occipital areas were only recorded. Patients with similar occipital and frontal dermoscopic findings were considered to be suffering from telogen effluvium instead of FPHL, and thus were excluded from the study.

Age matched controls without history of hair loss was recruited from hospital patients and attendants. The controls were recruited during the same period of time as the cases and using the same exclusion criteria.

In patterned hair loss, trichoscopic examination should be performed in the fronto-parietal area approximately at the cross between the nose line and ear implantation. According to Rakowska et al trichoscopic evaluation of temporal area may be excluded in practice.[4] Furthermore, pattern hair loss–trichoscopic changes seen in the frontal area are more as compared to the occipital region. Thus, the frontal and occipital scalp was examined in the midline (sagittal plane) approximately 3 cm above the hairline at 10X magnification. All the images evaluated for the hair changes are presented below.

Details of criteria used to describe trichoscopic findings are included below.

Variability in hair diameter

Presence of hairs of different width i.e small, medium and terminal hair in one field of vision (as image is 2-dimensional width of hair shaft is used instead of diameter), peri-pilar sign- halo of ~1 mm around the hair follicle, brown peri-pilar sign (BPPS) and white peri-pilar sign (WPPS), white dots- pin point white dots, single hair per follicular unit, 2-3 hairs per follicular unit, yellow dots-empty follicles seen as yellow dots, focal atrichia-~ size of pencil eraser which represents area of total hair loss over scalp, honey comb pigmentation, and scaling.

Morning blood sample was collected for hormonal and biochemical analysis on 3rd to 5th day of the menstrual cycle from the patients with regular cycles or on any other day from the patient who haven't menstruated in the past 2 months or who had menopause.

Diagnosis of PCOS was done based on NIH criteria.

Investigation done included complete blood counts, thyroid function tests and anti-TPO antibodies, serum prolactin, luteinising hormone (LH), follicle stimulating hormone (FSH), oestrogen, progesterone, testosterone, DHEAS, pelvic ultrasonography (USG).

Institutional ethical committee clearance was obtained.

Statistical test

Results were tabulated in excel sheet and statistical analysis was performed using independent t test, McNemar test and analysis of variance (ANOVA) test. Statistical significance was set at p value of <0.05.

RESULTS

Overall demographic and associated features result are explained in the text below.

The baseline parameters of female pattern hair loss and control group is depicted in Table 1.

Table 1: Baseline parameters in FPHL and control group.

Parameters	FPHL group (%)	Control group (%)
Age	33.31±9.42	31.07±9.43
Mean duration of hair loss	14.23±14.62 months	-
Positive family history	53	10
Seborrheic dermatitis	45	30
Acne	12	3
Hirsutism	13	6
Acanthosis nigricans	7	3

Pattern of hair loss

54% (54/100), 29% (29/100), (6/100) and 11% (11/100) of patients had hair loss as per L-I, L-II, L-III and Male type fronto-temporal recession respectively with corresponding age of onset of 32.43±9.553 years, 36.07±8.513 years, 29±8.367 years and 32.73±10.89 years, which were not significant from each other (p<0.236) (Figure 1a-d). However, duration of hair loss among 4 groups were different (L-I→7.94±8.02 months; L-II→20.55±17.079 months; L-III→39±15.987 months; MT→14.91±11.528 months). There was statistically significant difference between stage of hair loss and duration of hair loss (p<0.001).

Also, in our study family history (p=0.078) menstrual history (p=0.812), associated findings like seborrheic dermatitis (p=0.539), acne (p=0.733), acanthosis nigricans (p=0.617) and hirsutism (p=0.916) did not have any statistical significance as per the grading of hair loss.

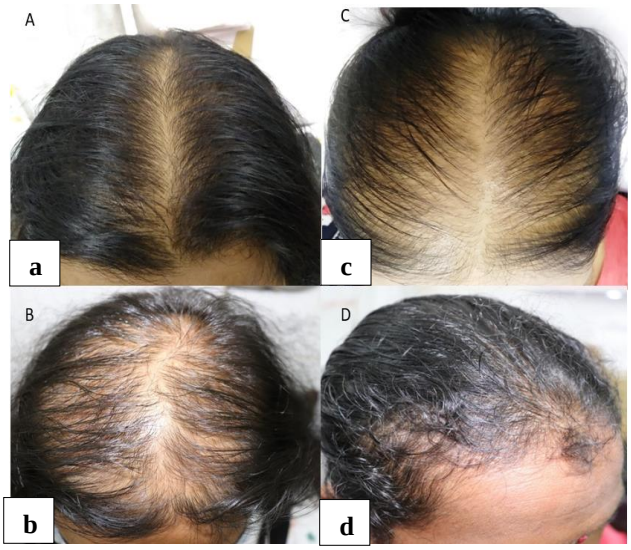

Figure 1: (a)-(d) Female pattern hair loss as per Ludwig classification.
A: L-I, B: L-II, C: L-III, D:MT fronto-temporal recession

Laboratory findings

The results of mean hormone levels in cases and controls are summarized in Table 2.

The difference between the mean value of body mass index, hemoglobin, T4, prolactin, Follicle stimulating hormone (FSH), testosterone and dehydroepiandrosterone (DHEAS) were found to be statistically significant as compared to control group using t test. (p<0.05).

Serum T3, TSH, LH, FSH, prolactin and testosterone had significant association with severity of hair loss (p <0.05).

Trichoscopic findings

Trichoscopic features observed in patients included in our study were hair diameter diversity 97% (97/100), brown peri-pilar sign 25% (25/100), white peri-pilar sign 32% (32/100), focal atrichia 28% (28/100), one hair per follicular unit 88% (88/100), 2-3 hair per follicular unit 22% (22/100), yellow dots in 2% (2/100), scaling in 57% (57/100), white dots in 17% (17/100) and honey comb pigmentation 40% (40/100) (Figure 2a-f).

Table 3 depicts comparison of trichoscopic findings over frontal and occipital scalp.

The incidence of hair diameter diversity, brown peri-pilar sign, White peri-pilar sign, one hair per follicular unit, white dots, scaling and honey-comb pigmentation were found to be statistically significant (p≤0.001). However, only focal atrichia, 2-3 hairs/HU, white dots, honey comb pigmentation (HCP) correlated with grade of hair loss (p<0.05). Tables 4 and 5 depicts trichoscopic findings in each grade of FPHL. No significant association was found between endocrinal profile and trichoscopic findings.

Table 2: Mean hormone levels (cases and controls).

Parameters	Controls (n=100) Mean±SD	Cases (n=100) Mean±SD	t	P value
Age (years)	31.07±9.43	33.31±9.42	-1.681	0.094
BMI	20.86±1.51	21.94±2.63	-3.57	<0.001
Hb	11.11±1	11.64±1.03	-3.686	<0.001
T3	2.49±15.11	2.44±9.72	0.027	0.978
T4	7.55±2.14	8.2±1.9	-2.264	0.025
TSH	2.93±9.9	2.01±2	0.91	0.364
FT3	2.57±0.6	2.4±0.71	1.804	0.073
FT4	1.02±0.22	1.02±0.28	-0.074	0.941
Anti-TPO	7.07±28.03	11.36±36.67	-0.929	0.354
PRL	15.04±7.25	18.82±16.24	-2.128	0.035
Oestrogen	118.68±64.76	102.9±67.42	1.687	0.093
Progesterone	0.88±2.31	1.74±4.22	-1.806	0.073
LH	7.76±5.6	9.46±9.08	-1.592	0.113
FSH	6.02±3.69	12.84±22.35	-3.009	0.003
Testosterone	0.46±0.24	0.83±0.47	-7.067	<0.001
DHEAS	145.29±82.03	186.44±81.86	-3.55	<0.001

Table 3: Comparison of trichoscopic findings over frontal and occipital scalp.

S. no.	Parameter	Frontal	Occipital	P
1	HDD	97	7	<0.001*
2	BPPS	25	7	<0.001*
3	WPPS	32	2	<0.001*
4	FA	28	-	-
5	1 hair / FU	88	11	<0.001*
6	2-3 hairs/FU	22	86	0.52
7	YD	2	-	>1
8	Scaling	57	15	<0.001*
9	HCP	40	2	<0.001*
10	WD	53	13	<0.001*

P value is calculated using McNemar's test, significant results are marked with asterisk

Table 4: Incidence of trichoscopic findings in each stage of FPHL.

Parameters	HDD (%)	BPPS (%)	WPPS (%)	FA (%)	1 hair/FU (%)
L-I	51/54 (94.4)	10/54 (18.5)	19/54 (35.1)	6/54 (11.1)	46/54 (85.1)
L-II	29/29 (100)	7/29 (24.1)	11/29 (37.9)	14/29 (48.2)	28/29 (96.6)
L-III	6/6 (100)	6/6 (100)	2/6 (33.3)	1/6 (16.7)	5/6 (83.3)
MT	11/11 (100)	11/11 (100)	6/11 (54.5)	1/11 (9.09)	3/11 (27.2)

Table 4: Incidence of trichoscopic findings in each stage of FPHL.

Parameters	2-3 hairs/FU (%)	WD (%)	YD (%)	Scaling (%)	HCP (%)
L-I	12/54 (22.2)	22/54 (40.7)	2/54 (0.3)	34/54 (62.9)	15/54 (27.8)
L-II	3/29 (10.3)	21/29 (72.4)	0	16/29 (55.1)	16/29 (55.1)
L-III	1/6 (16.7)	4/6 (66.6)	0	1/6 (16.7)	2/6 (33.3)
MT	9/11 (81.8)	6/11 (54.5)	0	6/11 (54.5)	7/11 (63.6)

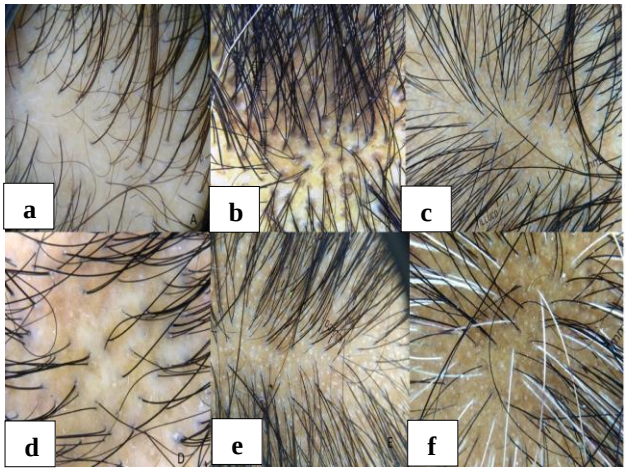

Figure 2: Various trichoscopic features (a) hair diameter diversity, (b) brown peripilar sign, (c) white peripilar sign, (d) focal atrichia and single hair per follicular unit, (e) white dots, and (f) honeycomb pigmentation and brown peripilar sign.

DISCUSSION

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

Video-dermoscopic criteria for diagnosing FPHL are recently given by Rakowska et al which also helps to differentiate FPHL from telogen effluvium.[4] But to diagnose patients as FPHL using these criteria require specialised instrument and dedicated software. Also due to cost factors these sophisticated instruments are not available with most of the dermatologist.

Thus, we planned to evaluate and compare frontal and occipital scalp of patients as occipital part would not be affected in patterned hair loss using hand held dermoscope at 10× magnification. This would help us in diagnosing FPHL with convenience of rapid, low cost using hand held dermoscope.

The results of prevalence studies might be hampered as a result of lack of universally accepted criteria for diagnosing FPHL. Difference incidences of positive family history could be explained by underlying genetic factors which play a role in aetiology of FPHL due to difference in ethnicity. Also, racial differences could be contributory for variation of results.

Results of studies suggesting role of hyperandrogenism in FPHL are discordant and thus dermatologist prefers to use term ‘female pattern hair loss’ instead of androgenetic alopecia. However, many women with features of hyperandrogenism complains of scalp hair loss indicating possible role of androgens in FPHL.

Hyperandrogenism can be explained clinically as well as biochemically. Clinical hyperandrogenism is defined as cutaneous signs and symptoms like acne, and hirsutism whereas biochemically it is defined as elevation in the serum free testosterone, total testosterone or DHEAS above normal laboratory reference range.

Quinn et al showed that there is no difference in hyperandrogenaemia with or without AGA.[5] However, Cela et al reported significant role of testosterone, androstenedione in causation of AGA.[6]

Despite of negative association between cutaneous markers of androgen excess like hirsutism, acne, seborrhoea, acanthosis nigricans, elevated serum testosterone levels was found in 43% of our cases and was significant statistically when compared with control group ($p<0.001$).

In our study DHEAS serum level was found to be normal, but it did achieve statistical significance on comparing with control group. Serum DHEAS itself does not lead to causation of alopecia but it acts as a target for peripheral metabolism of potent androgen.

Also, DHEAS has action on hair follicle directly as G6PD inhibitor, thus inhibiting synthesis of nucleic acid, which suggest that low adrenal is sufficient to cause FPHL with or without increased androgens level.

All patients with signs of androgenism should be evaluated with caution as DHEAS level may not be elevated in all females with adrenal overproduction of DHEAS.

In study done by Ramatulasi et al mean value of serum testosterone, DHEAS and TSH were statistically significant.[7] Similar results were found in a study done by Tandon et al.[8] Studies done by Montalto et al and Rushton et al did not observe any increase in plasma DHEAS levels.[9,10]

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

Interestingly serum androgen levels do not reflect local androgen role in growth of hair follicle thus implicating that serum level of androgens may not be elevated in all subjects.[11,12]

Prolactin and TSH interacts at various levels with the androgen metabolism. In our study 21 patients had high PRL level and 13 patients had hyperthyroidism and 6 patients had hypothyroidism. Futterweit et al in his study observed 2 out 109 patients had hyperprolactinemia.[13] In study done by Ramatulasi et al all patients had normal prolactin level.[7] Prolactin acts as stimulator of adrenal

androgen secretion, also inhibits 5-alpha-reductase thus increasing level of free testosterone and DHEAS and levels of sex hormone binding globulin is decreased.[14]

Hyperprolactinemia can cause oestrogen deficiency by causing ovarian dysfunction. As oestrogens are major anti-androgens it can cause alopecia even in females with normal androgen levels.

Thyroid receptors are present on outer root sheath of hair and it regulates frequency of hair cycle. It has been observed that decrease in thyroid levels reduces length of anagen phase and elevated thyroid levels leads to thin hair.[14]

FPHL can be the manifestation of hypothyroidism or hyperthyroidism thus screening of T3, T4, TSH is recommended. In our study 13 patients had hyperthyroidism and 6 patients had hypothyroidism which was newly diagnosed. 6 patients in case group were previously diagnosed with thyroid disorders and were on treatment for the same.

Schimdt et al in their study reported 23% patients had elevated TSH level.[15] Also similar results (28.5%) were reported by Ramatulasi et al.[7]

The increase prevalence of FPHL after menopause and also increased level of oestrogen in pregnancy suggests the stimulatory role of oestrogen on hair growth. However further studies are needed to prove role of oestrogen in FPHL as data in literature is sparse.

Progesterone tends to influence hair growth by both peripheral as well as central action. At local level it inhibits 5-alpha-reductase thus decreasing conversion of testosterone to more potent DHT. Also it inhibits LH secretion which in turn decreases androgen synthesis.[14]

Increased levels of LH can lead to increase in serum testosterone levels which in turn can cause patterned baldness. Also, LH stimulates adrenal gland to produce androstenedione which is a weak androgen.

Increased levels of LH also suggests that hypophyseal-adrenal axis plays a role in pathogenesis of patterned alopecia.[16]

As per recent S1 guideline for evaluating a case of patterned hair loss if hormonal dysregulation is suspected the free androgen index estimation is more appropriate test.[17]

Investigating role of androgens in FPHL should be undertaken with certain precautions: look for presence of signs and symptoms of hyperandrogenism; diagnosis of underlying endocrinal disorder to be made based on hormonal assay; and suitable control group should be used to compare alopecic women and not just to check for higher levels exceeding reference laboratory range.

Not all patients with FPHL have elevated levels of androgens and increased levels are more common in patients with signs and symptoms of hyperandrogenism. Isolated FPHL should not be considered as sign of hyperandrogenism if serum androgen levels are normal.

FPHL in patients with normal level of androgens could be due to increased androgens receptors or increased androgen sensitivity.[10]

Table 6: Comparison of present study with similar studies done in past.

Parameter	Present study	Ramatulasi et al[7]	Tandon et al[8]	Zhang et al[20]
Mean age of onset	33.31±9.42 years	33±4.5 years	31.17 years	34.4±10.6 years
Mean duration of onset	14.23±14.62 months	2-4 years	5.1 years	4.49±3.76 years
Positive family history (%)	53	56	46	45
Hirsutism (%)	13	40	66.6	-
Acanthosis nigricans (%)	7	37	43.3	-
Acne (%)	12	28.5	36.6	-
PCOS (%)	13	27.14	26.6	1.66

Table 7: Comparison of various trichoscopic findings with other studies done in past.

Parameter	Present study	Zhang et al[20]	Ramatulasi et al[7]	Ummiti et al[19]
Hair diameter diversity (%)	97	-	93	-
BPPS (%)	25	37	47	40
WPPS (%)	32	26.7	44.2	68
Yellow dots (%)	2	1.67	57	88
Focal atrichia (%)	28	56.7	18.6	24
HCP (%)	40	60	61.7	80
White dots (%)	17	21.7	41.4	-

Trichoscopy has gain momentum in the field of dermatology. Rakowska et al have established hair shaft diameter diversity as one of the major criteria to diagnose FPHL on trichoscopy.[4] Also termed as 'anisotrichosis' and it correlates histo-pathologically with miniaturisation of hair.

Bhamla et al in their study suggested that single parameter of hair diameter diversity can diagnose Ludwig's I FPHL in 75% if this is present in >20% hairs.[18] Hair diameter diversity was statistically significant when frontal scalp was compared to that of occipital. Thus, occipital area of patient's scalp can serve as control to compare and use these criteria as screening tool for FPHL.

Peripilar sign is halo of ~1 mm surrounding follicular opening. Colour of the halo varies from white to brown. According to literature peripilar sign is unique to AGA but in our study, we found it in 25% of patients only. It could be due to concealment of finding due to colour of scalp in Indian population.

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

In our study we reported only 2% of patients had yellow dots which was contradictory to study done by Ramatulasi et al (57%) and Ummiti et al (88%).[7,19] Ummiti et al suggested that difference in incidence in this observation could be due to different phenotypes of skin with variation in scalp pigmentation and activity of sebaceous gland.[19] In addition Zhang et al suggested that this could be due to easy recognition of white dots as compared to yellow dots.[20]

Yellow dots represent dilated infundibulum of follicle which contains keratinous material and sebum. Yellow dots are one of major criteria on trichoscopy suggested by Rakowska et al.[4]

Focal atrichia correlated with advanced stage of FPHL in study done by Zhang et al.[20]

White dots were seen in 17% and single hair per follicular unit in 88% of our patients. The normal scalp each follicular unit comprises one to four terminal hairs with one to two vellus hair. However, majority of our patients showed increased in the number of single hairs per follicular units with predominant prevalence in the frontal area suggesting increase thinning of hair and helps in diagnosis of FPHL.

White dots were reported by Zhang et al as white spots of size 0.2-0.3 mm uniformly distributed; histo-pathologically they correspond to eccrine gland opening on scalp.[20] Sweat glands have highest activity of 5α-reductase thus they can undergo hypertrophy under influence of elevated androgen level. Zhang et al also suggested that this feature could be due to hypertrophy of sweat glands under influence of elevated androgen level thus suggesting role of androgen in causation of FPHL.[20]

These white dots can be seen in healthy individuals also with Fitzpatrick skin photo-types IV– VI as they become evident on the contrasting background of the pigmented network.[21] White dots correlated with stage of hair loss in our study.

Limitations

Our study lacks histo-pathological correlation however past studies done have already established correlation of trichoscopic and histo-pathological changes.

CONCLUSION

Thus, based on results of our study we recommend hormonal profile as baseline investigation in patients with FPHL. The probability of underlying altered gonadal profile should be considered as it can present without any manifestations. It is recommended to do hormonal investigations in patients especially with early onset and advanced hair loss and treating clinician should be aware of possibility of underlying endocrinological conditions which may affect the hair re-growth in FPHL.

Various findings on trichoscopy like hair diameter diversity, BPPS, WPPS, focal atrichia, white dots, honey comb pigmentation can be used as non-invasive aid in diagnosis of FPHL and differentiate it from CTE. As pattern hair loss spares occipital region, we can conclude that patients own occipital region can serve as control for diagnosing FPHL.

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Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Singal A, Sonthalia S, Verma P. Female pattern hair loss. Indian J Dermatol Venereol Leprol. 2013;79(5):626.
2. Ramos PM, Miot HA. Female pattern hair loss: a clinical and pathophysiological review. An Bras Dermatol. 2015;90:529-43.
3. Mani S, Manickam N, Gopalan K. Role of dermoscopy in the diagnosis of alopecia. J Pak Assoc Dermatol. 2018;28(3):320-8.
4. Rakowska A, Slowinska M, Kowalska-Oledzka E, Olszewska M, Rudnicka L. Dermoscopy in female androgenic alopecia: method standardization and diagnostic criteria. Int J Trichology. 2009;1(2):123.
5. Quinn M, Shinkai K, Pasch L, Kuzmich L, Cedars M, Huddleston H. Prevalence of androgenic alopecia in patients with polycystic ovary syndrome and

characterization of associated clinical and biochemical features. Fertil Steril. 2014;101(4):1129-34.
6. Cela E, Robertson C, Rush K, Kousta E, White DM, Wilson H, et al. Prevalence of polycystic ovaries in women with androgenic alopecia. Eur J Endocrinol. 2003;149(5):439-42.
7. Ramatulasi S, Malladi MA, Satya RS, Srujan K, Kumar AA. Clinico-laboratory and trichoscopic evaluation of various patterns of female baldness–A prospective case control study. Indian J Clin Exp Dermatol. 2019.
8. Tandon S, Arora P, Gautam RK, Bhardwaj M, Garga U, Sharma N. Correlation between clinical features, biochemical parameters, and histopathological findings in women with patterned baldness: a study from North India. J Cutan Aesthetic Surg. 2019;12(1):42.
9. Montalto J, Whorwood CB, Funder JW, Yong ABW, Callan A, Davies HE, et al. Plasma C19 steroid sulphate levels and indices of androgen bioavailability in female pattern androgenic alopecia. Clin Endocrinol (Oxf). 1990;32(1):1-12.
10. Rushton DH, Ramsay ID, James KC, Norris MJ, Gilkes JJH. Biochemical and trichological characterization of diffuse alopecia in women. Br J Dermatol. 1990;123(2):187-97.
11. Lee AT, Zane LT. Dermatologic manifestations of polycystic ovary syndrome. Am J Clin Dermatol. 2007;8(4):201-19.
12. Rathnayake D, Sinclair R. Innovative use of spironolactone as an antiandrogen in the treatment of female pattern hair loss. Dermatol Clin. 2010;28(3):611-8.
13. Futterweit W, Dunaif A, Yeh HC, Kingsley P. The prevalence of hyperandrogenism in 109 consecutive female patients with diffuse alopecia. J Am Acad Dermatol. 1988;19(5):831-6.
14. Grymowicz M, Rudnicka E, Podfigurna A, Napierala P, Smolarczyk R, Smolarczyk K, et al. Hormonal effects on hair follicles. Int J Mol Sci. 2020;21(15):5342.
15. Schmidt JB, Schurz B, Huber J, Spona J. Hypothyroidism and hyperprolactinemia as a possible cause of androgenetic alopecia in the female. Z Hautkr. 1989;64(1):9-12.
16. Sanke S, Chander R, Jain A, Garg T, Yadav P. A comparison of the hormonal profile of early androgenetic alopecia in men with the phenotypic equivalent of polycystic ovarian syndrome in women. JAMA Dermatol. 2016;152(9):986-91.
17. Blume-Peytavi U, Blumeyer A, Tosti A, Finner A, Marmol V, Trakatelli M, et al. S1 guideline for diagnostic evaluation in androgenetic alopecia in men, women and adolescents. Br J Dermatol. 2011;164(1):5-15.
18. Bhamla SA, Dhurat RS, Saraogi PP. Is Trichoscopy a reliable tool to diagnose early female pattern hair loss? Int J Trichology. 2013;5(3):121.
19. Ummiti A, Priya PS, Chandravathi PL, Kumar CS. Correlation of trichoscopic findings in androgenetic alopecia and the disease severity. Int J Trichology. 2019;11(3):118.
20. Zhang X, Caulloo S, Zhao Y, Zhang B, Cai Z, Yang J. Female pattern hair loss: clinico-laboratory findings and trichoscopy depending on disease severity. Int J Trichology. 2012;4(1):23.
21. Abraham LS, Pineiro-Maceira J, Duque-Estrada B, Barcaui CB, Sodré CT. Pinpoint white dots in the scalp: dermoscopic and histopathologic correlation. J Am Acad Dermatol. 2010;63(4):721-2.

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