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Study the Relationship Between Vit.D and Some Biomarkers in Female Hair Loss Patients Compared to Normal Subjects

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Annotation: Background: Female Pattern Hair Loss (FPHL) is a common non-scarring form of alopecia among women, often associated with psychosocial distress. Although its exact pathophysiology remains unclear, recent research highlights potential roles of micronutrients, neurotransmitters, and metabolic factors, such as vitamin D, dopamine, and lipid profiles.

Methodology: A case-control study was conducted from December 2024 to March 2025 at Al-Qurna General Hospital and Al-Faihaa Teaching Hospital in Basra, Iraq, involving 65 female FPHL patients and 65 healthy age-matched controls. Participants underwent clinical evaluation and blood sampling to assess serum levels of dopamine, vitamin D, ferritin, and lipid profile components using ELISA and Cobas e411. Statistical analyses were performed using SPSS v26 with significance set at $p \leq 0.05$.

Results: The study found significantly lower serum levels of vitamin D and ferritin in FPHL patients compared to controls ($p < 0.001$). Dopamine levels

showed no significant difference between groups ($p = 0.34$). Lipid profile analysis revealed significantly lower total cholesterol and triglyceride levels in patients ($p = 0.03$ and $p < 0.001$, respectively), with no significant changes in HDL, LDL, or VLDL levels. Subgroup analysis by age within the patient group indicated age-related variations in lipid parameters but not in vitamin D or dopamine levels.

Conclusion: The findings suggest that vitamin D deficiency and altered lipid metabolism may play significant roles in the pathogenesis of FPHL, while serum dopamine appears not to be directly implicated. These results support the potential benefit of monitoring vitamin D and lipid status in women with FPHL and highlight the need for further investigation into metabolic contributors to this condition.

Keywords: FPHL, Hair loss, Vit. D, Dopamine, Lipid profile.

Introduction

Female pattern hair loss (FPHL) is commonly seen in women. Although in most cases it is a non-scarring, non-inflammatory condition, loss of hair density often leads to disfigurement, afflicting most patients. The disease is clinically characterized by gradual loss of hair in the central and forehead regions of the scalp, but preservation of the forehead hairline (Tsutsui et al., 2022). This pattern differs from that seen in male androgenetic alopecia (M-AGA), which is often characterized by a receding frontal hairline, followed by baldness and possibly central hair loss. Although the appearance may be different, the final follicular changes in both disorders are similar, such as perifollicular microinflammation, increased number of atrophic follicles, reduced sebaceous gland volume, and reduced anagen-telogen ratio (Bertoli et al., 2020; Lolli et al., 2017).

The pathophysiology of FPHL has not been fully elucidated. Evidence suggests it may involve genetics, sex steroid hormones, and environmental factors (Ramos & Miot, 2015). The final pathological manifestations of M-AGA and FPHL are similar, both of which are follicle miniaturization, characterized by an increase in the proportion of vellus hairs and a decrease in the ratio of terminals to vellus. Along this process, a reduction in the size of the sebaceous glands, an increase in the number of microfollicles, and a decrease in the anagen- telogen ratio can easily be observed (Bertoli et al., 2020; Redler et al., 2017).

Vitamin D is a fat-soluble vitamin essential for calcium homeostasis, immune modulation, and cellular differentiation. It has been increasingly implicated in hair follicle biology, particularly in

the anagen phase of the hair growth cycle. The active form of vitamin D, calcitriol, binds to the vitamin D receptor (VDR) in keratinocytes, promoting follicular proliferation and preventing premature follicular regression (Kálmán et al., 2020). Low serum levels of vitamin D have been associated with various forms of hair loss, including androgenetic alopecia and telogen effluvium, in women (Gerkowicz et al., 2017). Furthermore, vitamin D deficiency may exacerbate autoimmune conditions such as alopecia areata, characterized by immune-mediated destruction of hair follicles (Rasheed et al., 2013).

Dopamine, a catecholamine neurotransmitter, is predominantly known for its role in regulating mood, reward, and motor functions. Emerging evidence also suggests that dopamine might influence hair follicle activity through its effects on stress and hormonal pathways (Zhang et al., 2019). Chronic stress elevates dopamine metabolism, leading to dysregulation in hypothalamic-pituitary-adrenal (HPA) axis signaling, which can disrupt hair growth cycles Stress-induced hair loss, or telogen effluvium, is thought to involve heightened dopamine turnover, which exacerbates follicular miniaturization (Muller et al., 2020)

Methodology:

This is a case-control study conducted from December 2024 to April 2025. The study included 65 female patients with hair loss. They were diagnosed by specialist physician in the Al-Qurna General Hospital and Al-Faihaa Teaching Hospital, Basra governorate, southern of Iraq. Also, the study included 65 healthy persons as control. People with thyroid gland abnormalities and cancer were excluded. Patients and controls in this study ranged in age from 16 to 40 years, Participants' Socio-demographic data, including age, BMI, residence and education level, were collected using a standardized questionnaire. After collecting 5 ml of venous blood from each of the 130 participants (65 patients and 65 controls), the blood was centrifuged to obtain serum for the measurement of dopamine, Vit.D and lipid profile were measured by using Enzyme-linked immunosorbent assay (ELISA) and Cobas e411.

Statistical Analysis

The data was analyses using the Statistical Package for the Social Sciences (SPSS) version 26, developed by IBM, SPSS Inc, USA. The study employed an independent sample t-test for parametric variables and a Mann-Whitney U-test for nonparametric variables. The outcome was deemed statistically significant based on a p-value of less than or equal to 0.05.

Results:

The Table (1) display the Socio-demographic characteristics of the studied groups show that a non-significant difference in age distribution (P=0.80) between the group of healthy control and patient's female. Also, for the body mass index between the groups, no statistical significance was shown BMI (P=0.10).

A significant difference in the education level (P<0.05) between groups, the education the entire study samples show the college study groups was the highest in patients (60.0%). For the Residence between the groups no statistical significance was shown (P=0.38).

Table (1): Socio-demographic characteristics among the study groups

Characteristic		Patients group (n=65)	Control group (n=65)	P. value
Age (year)		26.08 ± 5.86	25.83 ± 5.39	0.80 ^{NS}
BMI (kg/m^2)		26.98 ± 4.46	25.36 ± 2.71	0.10 ^{NS}
Education level	**Primary**	7 (10.8%)	19 (29.2%)	0.02
	Secondary	19 (29.2%)	19 (29.2%)	
	College	39 (60.0%)	27 (41.5%)	
Residence	**City**	28 (43.1%)	33 (50.8%)	0.38 ^{NS}
	Countryside	37 (56.9%)	32 (49.2%)	

NS: Non-significant, t-test, Chi-square test

Significant difference at P<0.05

The values represent mean ± SD.

The Table (2) display a non-significant difference in the levels of dopamine among female patients group compared to the healthy control female (P=0.34). While there is a significant increase in the serum levels of Vit.D in patients group compared to healthy control (13.18±4.25 vs. 36.33±9.20, P<0.001)

Table (2): Dopamine and Vit. D levels between patients and control group

Parameters	Patients group (n=65) Mean ± SD	Control group (n=65) Mean ± SD	P. value
Dopamine	441.27 ± 206.68	407.77 ± 183.25	0.34 NS
Vit. D	13.18 ± 4.25	36.33 ± 9.20	<0.001

Significant difference at P<0.05

The values represent mean ± SD.

NS: Non-significant

Regarding lipid profile in control and patients' groups, it shows a significant increase in each of Total Cholesterol (TC) and Triglyceride (TG) between the two groups (153.58 ± 30.48 vs. 164.13 ± 25.95, *P*=0.03, and 100.03 ± 58.15vs. 146.99 ± 26.88, *P*<0.) respectively.

While there are non-significant changes in each of High-density lipoprotein (HDL) (P=0.58), Low density lipoprotein LDL (P=0.29) and Very low-density lipoprotein VLDL (P=0.84) levels between the two groups that seen in table (3).

Table (3): Lipid profile levels between patients and control group

Parameters	Patients group (n=65) Mean ± SD	Control group (n=65) Mean ± SD	P. value
T.C (mg/dL)	164.13 ± 25.95	153.58 ± 30.48	0.03
TG (mg/dL)	146.99 ± 26.88	100.03 ± 58.15	<0.001
HDL (mg/dL)	33.01 ± 10.26	33.84 ± 6.62	0.58 NS
LDL (mg/dL)	90.28 ± 22.66	91.16 ± 27.25	0.29 NS
VLDL (mg/dL)	21.18 ± 12.06	23.59 ± 13.70	0.84 NS

Significant difference at P<0.05

The values represent mean ± SD.

NS: Non-significant

A non-significant difference in the levels of dopamine and Vit.D in age sub groups of patients group as shown in table (4).

Table (4): Dopamine and Vit. D levels in patients group according to age

Parameters	16-23 years Mean ± SD	24-31 years Mean ± SD	>31 years Mean ± SD	P. value
Dopamine	404.64 ± 200.51	465.23 ± 224.32	456.33 ± 184.02	0.56 NS
Vit. D	13.36 ± 4.58	12.65 ± 3.77	13.90 ± 4.70	0.65 NS

Significant difference at P<0.05

The values represent mean ± SD.

NS: Non-significant

The statistical results of the patients group showed a significant increase in (24-31) years age subgroup in the levels of Total cholesterol (T.C), and significant increase in (>31) year age subgroup in the levels of Triglyceride (TG) P= (<0.01), in age sub group and show no Significant difference in level of High-density lipoprotein (HDL).

Also show a significant difference in the levels of Low-density lipoprotein (LDL) and Very low-density lipoprotein (VLDL) P= (<0.01) in age sub groups of patients group as shown in table (5).

Table (5): Lipid profile levels in patients group according to age

Parameters	16-23 years Mean ± SD	24-31 years Mean ± SD	>31 years Mean ± SD	P. value
T.C (mg/dL)	140.58 ± 32.45	173.20 ± 30.12	165.87 ± 41.11	<0.01
TG (mg/dL)	105.49 ± 83.92	133.05 ± 55.49	172.48 ± 73.71	<0.01
HDL (mg/dL)	34.34 ± 6.10	34.57 ± 6.37	31.47 ± 7.96	0.35 ^{NS}
LDL (mg/dL)	75.38 ± 19.29	103.87 ± 29.55	95.61 ± 21.76	<0.01
VLDL (mg/dL)	18.37 ± 9.08	23.92 ± 13.15	31.92 ± 17.53	<0.01

Significant difference at P<0.05

The values represent mean ± SD.

NS: Non-significant

Discussion

Several studies investigating dopamine levels in women with FPHL compared to healthy controls have yielded inconclusive or non-significant results.

A study by Trüeb et al. (2003) suggested that neuroendocrine imbalances, including altered dopaminergic signaling, could contribute to hair loss. However, direct evidence implicating dopamine specifically remains scarce. Another controlled study by Sawaya and Shapiro (2000) found that while androgen receptor activity was altered in FPHL, dopamine concentrations in serum did not differ significantly between women with FPHL and healthy individuals. This aligns with the broader literature indicating that dopamine may exert indirect rather than primary effects on hair follicle cycling, possibly through its influence on prolactin secretion or the hypothalamic-pituitary-adrenal (HPA) axis (Hardy et al., 2006).

In another investigation, Miteva and Tosti (2012) noted that while psychological stress is a known exacerbating factor for FPHL, peripheral dopamine levels do not consistently correlate with stress-induced hair loss patterns. This suggests that while dopamine may participate in the broader neuroendocrine landscape, its serum concentration alone may not serve as a reliable biomarker for FPHL.

Furthermore, a neuroimaging-based study by Jezova et al. (2016) showed that central dopaminergic activity did not significantly differ in female patients with chronic hair loss complaints versus controls, further reinforcing the notion that peripheral dopamine may not play a direct causative role in FPHL pathophysiology.

The results of the current study revealed that serum Vitamin D were significantly lower in FPHL patients than control group.

A comprehensive case-control study conducted in China evaluated serum 25(OH)D levels among 657 women with FPHL and 2,070 healthy controls. The findings revealed that FPHL patients had significantly lower vitamin D levels compared to controls ($P < 0.0001$), indicating a strong correlation between vitamin D deficiency and FPHL (Zhao et al., 2020).

Similarly, a study by El-Shafie et al. (2016) assessed serum vitamin D levels in 31 women with FPHL and 31 age-matched healthy controls. The results showed that FPHL patients had markedly lower mean serum vitamin D levels (18 ± 7.57ng/mL) compared to controls (35.09±15.39 ng/mL;P< 0.001). Furthermore, the study observed a significant decrease in vitamin D levels correlating with the severity of hair loss, as classified by Ludwig's degrees.

In a South Indian, a study by Nayak et al. (2016) focusing on young adults, 81.8% of female participants with diffuse hair fall exhibited vitamin D deficiency, compared to 45.5% in the control group. This significant difference (P = 0.007) underscores the potential role of vitamin D deficiency in hair loss among young women

A meta-analysis by Chen et al. (2024) encompassing 23 studies with 3,374 non-scarring alopecia patients and 7,296 healthy controls further corroborated these findings. The analysis revealed that patients with non-scarring alopecia, including FPHL, had significantly lower serum 25(OH)D levels and a higher incidence of vitamin D deficiency compared to healthy individuals

The current results agree with Abdel Fattah et al. (2016) who demonstrated that women with FPHL had significantly higher total cholesterol and triglyceride levels. It’s also agree with Amer et al. (2021) who found increase in the levels of cholesterol. And in agreement with Farajzadeh et al. (2010) who found non-significant difference in the levels of HDL and LDL.

The current study disagrees with Amer et al. (2021) who found increase in the levels of LDL and decrease in HDL in Egyptian patients with FPHL. It’s also disagree with Farajzadeh et al. (2010) who found non-significant difference in the levels of cholesterol and TG. This inconsistency may be partially attributed to the different target populations studied and to the failure to control for other confounding factors

A study by Azziz et al. (2000) reported that women exhibiting signs of androgen excess, including FPHL, frequently present with abnormal lipid profiles, this metabolic imbalance may contribute to or reflect the hormonal milieu involved in the pathogenesis of FPHL.

The exact mechanism linking dyslipidemia to FPHL remains to be fully elucidated. One plausible explanation involves the role of chronic low-grade inflammation and endothelial dysfunction associated with high cholesterol levels. Elevated TC can lead to oxidative stress and microvascular compromise, potentially reducing blood flow to the scalp and contributing to follicular miniaturization (Barros et al., 2020).

Additionally, cholesterol is a precursor for steroid hormone biosynthesis, including androgens, which are known to play a role in FPHL. Thus, altered cholesterol metabolism may influence local androgen production or receptor sensitivity in scalp follicles (Yip et al., 2009).

Moreover, dyslipidemia is a recognized feature of metabolic syndrome, which has been increasingly associated with various dermatological conditions, including FPHL. Studies like that of Bakry et al. (2021) found that women with FPHL were more likely to meet criteria for metabolic syndrome, including hyperlipidemia, compared to controls. These findings suggest that FPHL might not solely be a cosmetic concern but a marker of broader metabolic dysfunction.

The association is further supported by evidence from cross-sectional analyses, such as the work of Trüeb (2010), who proposed that FPHL may be a dermatologic expression of underlying metabolic alterations, particularly in postmenopausal women. As estrogen levels decline, lipid profiles typically worsen, potentially exacerbating hair loss in predisposed individuals. This observation underscores the importance of evaluating lipid status in women presenting with hair thinning, especially in the absence of overt hyperandrogenism.

In a comparative analysis, Narad et al. (2019) also reported a higher prevalence of elevated triglyceride levels in Indian women with FPHL than in age-matched healthy individuals. Their work aligns with that of Su et al. (2017), who found a positive association between androgenetic alopecia and serum triglycerides in a large cross-sectional Chinese cohort. Together, these studies

point toward a consistent metabolic profile characterized by increased triglycerides, potentially serving as a clinical marker for systemic assessment in FPHL patients.

Conclusion

It could be concluded that vitamin D deficiency and altered lipid metabolism may play significant roles in the pathogenesis of FPHL, while serum dopamine appears not to be directly implicated. These results support the potential benefit of monitoring vitamin D and lipid status in women with FPHL and highlight the need for further investigation into metabolic contributors to this condition

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