

# Serum Ferritin and Vitamin D in Female Hair Loss: Do They Play a Role?

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## Key Words

Female pattern hair loss · Serum iron · Serum vitamin D ·  
Telogen effluvium

## Abstract

**Aim:** Evaluation of serum ferritin and vitamin D levels in females with chronic telogen effluvium (TE) or female pattern hair loss (FPHL), in order to validate their role in these common hair loss diseases. **Methods:** Eighty females (18 to 45 years old) with hair loss, in the form of TE or FPHL, and 40 age-matched females with no hair loss were included in the study. Diagnosis was based upon clinical examination as well as trichogram and dermoscopy. Serum ferritin and vitamin D<sub>2</sub> levels were determined for each participant. **Results:** Serum ferritin levels in the TE ( $14.7 \pm 22.1 \mu\text{g/l}$ ) and FPHL ( $23.9 \pm 38.5 \mu\text{g/l}$ ) candidates were significantly lower than in controls ( $43.5 \pm 20.4 \mu\text{g/l}$ ). Serum vitamin D<sub>2</sub> levels in females with TE ( $28.8 \pm 10.5 \text{ nmol/l}$ ) and FPHL ( $29.1 \pm 8.5 \text{ nmol/l}$ ) were significantly lower than in controls ( $118.2 \pm 68.1 \text{ nmol/l}$ ;  $p < 0.001$ ). These levels decreased with increased disease severity. Serum ferritin cut-off values for TE and FPHL were 27.5 and 29.4  $\mu\text{g/l}$ , respectively, and those for vitamin D were 40.9 and 67.9  $\text{nmol/l}$ . **Conclusion:** Low serum ferritin and vitamin D<sub>2</sub> are associated with hair loss in females with TE and FPHL. Screening to establish these levels in cases of hair loss and supplementing with them when they are deficient may be beneficial in the treatment of disease.

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## Introduction

Women presenting with diffuse hair loss is a very common and challenging problem for dermatologists [1]. Telogen effluvium (TE), whether acute or chronic (i.e. of more than 6 months' duration) is the most common cause, followed by female pattern hair loss (FPHL) [2].

Since nonanemic iron deficiency was first suggested as an etiologic factor for diffuse hair loss in women in 1963, low iron stores have been considered a possible contributing factor in both conditions [3]. Assessment of serum ferritin levels is therefore generally recommended as part of the routine investigation, and dermatologists commonly prescribe iron supplementation in women under the assumption that low iron stores may be causing hair loss. However, contradictory data have so far failed to support this practice [4]. Various observational studies have evaluated the association between decreased ferritin levels and hair loss and have resulted in opposing conclusions [4–12]. As ferritin is also an acute-phase reactant, it is often elevated in the course of various diseases; in this case, a normal C-reactive protein (CRP) can be used to exclude elevated ferritin caused by acute-phase reactions [13].

Interestingly, several authors have pointed to the close relation that exists between the concentration of iron and the vitamin D level [14, 15]. Vitamin D is an inactive precursor, requiring two hydroxylation steps: in the liver, it is converted to 25(OH) vitamin D<sub>2</sub> [25(OH)D<sub>2</sub>, calcidiol],

**Table 1.** Criteria for diagnosis of chronic TE and FPHL

Features	Chronic TE	FPHL
Family history	negative	positive (usually)
Scalp appearance	diffuse hair loss	diffuse thinning over the mid-frontal scalp with widening of the midline and relative sparing of the anterior hair line. The exposed scalp may resemble a Christmas tree pattern.
Bitemporal recession	absent (usually)	present (usually)
Miniaturized hair	absent	present
Hair pull test	positive	negative (except at central scalp)
Trichogram	telogen in excess of 20%	normal or increased telogen less than 20%
Dermoscopy	<20% hair diameter diversity	>20% hair diameter diversity (mainly in androgen-dependent site)

which is the most indicative form of the vitamin D level in the body, and then in the kidney, it is converted to  $1\alpha,25$ -dihydroxyvitamin  $D_3$  (calcitriol), the active hormone [16]. Physiological vitamin  $D_2$  serum level remains controversial and variable. The results of a number of studies suggest that the optimal serum  $25(OH)D_2$  concentration is approximately 75 nmol/l for both skeletal and nonskeletal health outcomes. Vitamin D deficiency is commonly defined as a serum  $25(OH)D_2$  concentration of less than 20–25 nmol/l and insufficiency is defined as a serum  $25(OH)D_2$  concentration of between 25 and 75 nmol/l [17]. Limited studies have been conducted in humans to elucidate the role of vitamin D in the hair cycle [18]. Until now, the connection between vitamin D and TE or FPHL has not been established.

As such studies are still an issue under debate and are very limited in our region, the aim of this work was to shed light on this territory through evaluating the status of both serum ferritin and vitamin D in female patients with chronic TE or FPHL, in the form of a trial, in order to validate the role they play in these common diseases of hair loss and underline the value of supplementing them in such cases.

## Materials and Methods

This prospective, case-controlled study was approved by the Research Ethics Committee, Faculty of Medicine, Cairo University. It was carried out in the Dermatology Outpatients Clinic, Cairo University, from July to October 2011. Written informed consent was obtained from all the patients before their participation in the study.

Eighty adult female patients still in their years of menstruation (18–45 years), with skin phototypes (SPT) 3 and 4, who suffered from hair loss in the form of chronic TE or FPHL were included in this study. The criteria for diagnosis and inclusion are listed in

table 1; only patients with  $\geq 5$  diagnostic criteria of TE or FPHL were included. Forty healthy female volunteers, not suffering from hair loss, and matched to the patient group in terms of age, SPT and socioeconomic status, were also included. The assessment of all included subjects was performed by the same 3 investigators throughout the study.

Exclusion criteria in the study were: patients with less than 5 diagnostic criteria in one diagnosis to avoid overlap cases and the presence of any systemic and/or scalp disease that might be related to hair loss. Subjects on medication that could be related to hair loss (e.g. anticoagulants, retinoids, anticonvulsants and antidepressants) or subjects receiving drugs containing vitamin D, iron or dietary supplements were also excluded. Recent stressful events (i.e. within the last 6 months) like physiological stress, surgical trauma, high fever, chronic systemic illness, hemorrhage, emotional stress, pregnancy or the presence of an elevated level of CRP ( $\geq 4$ ) precluded recruitment. Menopausal subjects were also not included.

All participants were questioned on their medical history including their current clinical status (with regard to the onset and course of their hair loss) and the past (illness, type and dose of drugs and/or changes of these, pregnancy and menstrual cycle). Participants achieved this by answering a preset questionnaire similar to that used by Olsen et al. [5], so as not to miss any detail. General and local examinations were carried out to determine any systemic or local dermatological condition that might be related to hair loss, and thus preclude participation. Recruits with any suspected illness (from their history or clinical examination) further underwent laboratory investigations to verify diagnosis and thereby their participation in the study.

Examination of the scalp was performed for the detection of any scalp abnormalities and to determine the type and pattern of hair loss as well as the severity of the condition. FPHL was graded according to the Ludwig scale findings as mild (Ludwig I), moderate (Ludwig II) or severe (Ludwig III). For TE, we adopted the trichogram findings to grade cases as mild (telogen phase: 20–40% of the hair), moderate (telogen phase: 40–60% of the hair) or severe (telogen phase: >60% of the hair).

One investigator performed the trichograms for all participants by plucking hairs from the vertex area (where the lines from the nose and ear implantation cross) on the fifth day after the last shampoo. The hair was kept in place with clips and 60–80 hairs

were grasped with a rubber-covered hemostat. The hairs were plucked by twisting and lifting of the hair shafts rapidly in the direction of emergence from the scalp. These hair shafts were then cut off 1 cm above the root sheaths and the roots were arranged side by side on a slide and then taped down. The anagen hair bulbs were seen as darkly pigmented, triangular or delta-shaped bulbs at an angle to the hair shaft, and the inner root sheath was present. Hair in the telogen phase was less pigmented with club-shaped hair bulbs and the inner root sheath was absent. These two types of hair were distinguished from one another and the ratio was calculated. Hair in the telogen phase in excess of 20% suggested TE, while <20% increased telogen count suggested FPHL.

Dermoscopic evaluation was performed for each participant by one investigator using a DermLite II pro<sup>®</sup> Dermoscope. To assess the hair diameter diversity, we used the method proposed by de Lacharrière et al. [19] in which the hair diameter clinical scale from center parting within the same area at the vertex was checked with micrometer measurements on corresponding hairs: very thin/thin hair corresponds to 30–40 µm, medium hair to 50–80 µm and thick hair to 90–110 µm. In TE, dermoscopy shows empty follicles, short regrowing hairs and less than 20% hair diameter diversity [19]. The dermoscopic sign of FPHL is diversity in the hair diameter of more than 20% (a clinical sign reflecting the follicle miniaturization) (R), a peripilar brown depressed halo at the follicular opening (in early cases) and yellow dots (in advanced cases) may also be seen. Keeping in mind that the two conditions can be linked, in doubtful cases we compared the dermoscopic patterns of the vertex with the occipital area, as FPHL mainly produces alterations in androgen-dependent areas [20].

A venous blood sample (5 ml) was withdrawn from all participants following the same procedure (i.e. 1 week before menstruation, nonfasting and in the morning). The sample was allowed to clot at room temperature and was centrifuged at 10,000 rpm for 10 min. Serum was stored in two aliquots at –20 °C till the time of the assay. Hs-CRP and ferritin were measured by enzyme solid-phase immunometric assay (ELISA) using a kit supplied by Immunodiagnostic Monobind Inc. (Lake Forest, Calif., USA).

25(OH)D<sub>2</sub> was measured by competitive enzyme immunoassay. Since all circulating 25(OH) D<sub>2</sub> was bound to vitamin D-binding protein *in vivo*, samples were precipitated with precipitation reagent to extract the analyte. The kit was supplied by Immundiagnostik AG, Bensheim, Germany.

#### Statistical Analysis

Data were statistically described in terms of mean ± standard deviation (± SD) or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between the study groups was done using the Student t test for independent samples in comparing 2 groups and the 1-way analysis of variance (ANOVA) test with post hoc multiple 2-group comparisons when comparing more than 2 groups. For comparing categorical data, the  $\chi^2$  test was performed, with the exact test being used instead when the expected frequency was less than 5. The Kruskal-Wallis and Mann-Whitney tests were used when comparing nominal groups (mild, moderate and severe cases). Correlation between different variables was done using the Pearson moment linear correlation equation. Multivariate logistic regression was performed to test for the independent association between age, ferritin and vitamin D in relation to hair loss. The accuracy of ferritin and vitamin D as diagnostic markers was tested using the terms of

sensitivity and specificity. Receiver operator characteristic (ROC) analysis was performed to determine the best cut-off value (considering serum ferritin and vitamin D as screening tests, we searched for a cut-off point based on high sensitivity).  $p < 0.05$  was considered statistically significant. All statistical calculations were done using computer programs SPSS (Statistical Package for Social Science; SPSS Inc., Chicago, Ill., USA) version 15 for Microsoft Windows.

## Results

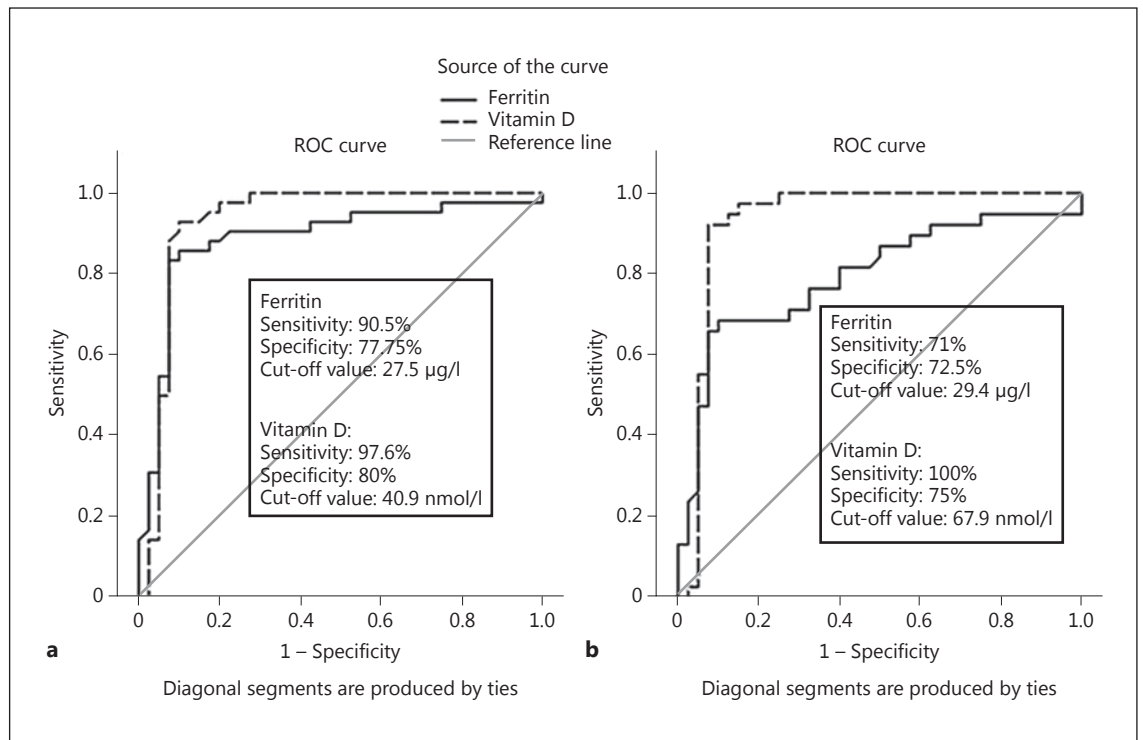
This study included 80 female patients suffering from chronic TE ( $n = 42$ , 52.5%) and FPHL ( $n = 38$ , 47.5%). Their age ranged between 18 and 45 years ( $29.8 \pm 9.3$  years). Forty healthy female controls (age- and SPT-matched) with no complaints about their hair were also included; their age also ranged between 18 and 45 years ( $30.8 \pm 8.56$ ). The participants were included out of a total of 94 cases and 48 controls. The excluded cases either did not meet our inclusion criteria or had nonconfirmed trichogram or dermoscopic findings.

Thirty-seven of the cases (46.3%) were evaluated as mild: 22 were TE (52.4% of the TE cases) and 15 were FPHL (39.5% of the FPHL cases). Nineteen (23.8%) were graded as moderate: 6 were TE (14.3% of the TE cases) and 13 were FPHL (34.2% of the FPHL cases). Twenty-four (30%) were considered severe cases: 14 were TE (33.3% of the TE cases) and 10 were FPHL (26.3% of the FPHL cases).

#### Serum Ferritin in TE and FPHL

The serum ferritin level in TE cases ranged from 2.2 to 131.3 µg/l with a mean of  $14.7 \pm 22.1$  µg/l, while in FPHL cases it ranged from 2.4 to 225.8 µg/l with a mean of  $23.9 \pm 38.5$  µg/l. Both values were significantly lower than that detected for the controls (range 3.2–73.8 µg/l and mean ± SD  $43.5 \pm 20.4$  µg/l,  $p < 0.001$ ). There was no statistical significant difference in the serum ferritin level between TE cases and FPHL cases ( $p = 0.202$ ; table 2).

On comparing the serum ferritin in patients according to the severity of their condition, this study revealed that the serum ferritin level was lowest in the severe forms of hair loss, whether this was TE (range 2.4–23.3 µg/l and mean ± SD  $7.9 \pm 5.4$  µg/l) or FPHL (range 2.4–33.3 µg/l and mean ± SD  $9.7 \pm 9.9$  µg/l). In the moderate cases, TE serum ferritin ranged from 2.2 to 63.6 µg/l with a mean of  $16.7 \pm 27.6$  µg/l, while for FPHL it ranged from 2.4 to 47.4 µg/l with a mean of  $14.9 \pm 14.2$  µg/l. The highest serum ferritin levels were detected in the mild cases of TE (range 2.2–131.3 µg/l and mean ± SD  $22.9 \pm 22.5$  µg/l) and



**Fig. 1. a** ROC analysis to determine the best cut-off values of serum ferritin and vitamin D in diagnosing TE. **b** ROC analysis to determine the best cut-off values of serum ferritin and vitamin D in diagnosing FPHL.

**Table 2.** Serum ferritin and vitamin D<sub>2</sub> levels (mean ± SD) detected in TE and FPHL cases in comparison to controls

	Serum ferritin, µg/l	Vitamin D <sub>2</sub> , nmol/l
<i>TE</i>		
Mean ± SD	14.7±22.1 [2.2–131.3]	23.9±38.5 [2.4–225.8]
Severe	7.9±5.4 (6.85)	9.7±9.9 (5.8)
Moderate	16.7±27.6 (7.75)	14.9±14.2 (7.5)
Mild	22.9±22.5 (20.45)	55.84±41.06 (30.3)
<i>FPHL</i>		
Mean ± SD	28.8±10.5 [11.5–74.4]	29.1±8.5 [12.7–62.0]
Severe	20.5±9.6 (15.6)	24.6±3.9 (22.7)
Moderate	28.4±8.1 (29.3)	29.5±6.5 (27.6)
Mild	31.4±11.2 (30.75)	31.6±11.1 (30.6)
<i>Controls</i>		
Mean ± SD	43.5±20.4 [3.2–73.8]	118.2±68.1 [9.2–250.0]

Data are expressed as mean ± SD, range in brackets and median in parentheses.

FPHL (range 2.6–225.8 µg/l and mean ± SD 55.8 ± 41.1 µg/l). These differences were of no statistical significance in TE cases ( $p = 0.366$ ). In FPHL cases, mean serum ferritin was significantly higher in mild cases than in severe cases ( $p = 0.016$ ) (table 2).

ROC analysis was done to determine the best cut-off values of serum ferritin in diagnosing TE and FPHL. The areas under the curve of serum ferritin were 0.887 in TE and 0.794 in FPHL (95% CI 0.807–0.967 and 0.690–0.898, respectively), with the best cut-off values 27.5 and 29.4 µg/l that achieved sensitivity (90.5 and 71%) and specificity (77.75 and 72.5%) in cases of TE and FPHL, respectively (fig. 1).

#### Serum Vitamin D<sub>2</sub> in TE and FPHL

The serum vitamin D<sub>2</sub> level in TE cases ranged from 11.5 to 74.4 nmol/l with a mean of 28.8 ± 10.5 nmol/l, while in FPHL cases it ranged from 12.7 to 62 nmol/l with a mean of 29.1 ± 8.5 nmol/l. Both values were significantly lower than those detected in the controls (range 9.2–50 nmol/l and mean ± SD 18.2 ± 68.1 nmol/l,  $p < 0.001$ ). There was no statistically significant difference in the se-



rum vitamin D<sub>2</sub> level between TE and FPHL cases ( $p = 0.912$ ) (table 2).

On comparing the serum vitamin D in patients according to the severity of their condition, this study revealed that the serum vitamin D<sub>2</sub> level was lowest in the severe forms of hair loss, whether this was TE (range 11.5–39.1 nmol/l and mean  $\pm$  SD 20.5  $\pm$  9.7 nmol/l) or FPHL (range 20–31.2 nmol/l and mean  $\pm$  SD 24.6  $\pm$  3.9 nmol/l). In the moderate cases, TE serum vitamin D<sub>2</sub> ranged from 12.9 to 32.7 nmol/l with a mean of 28.4  $\pm$  8.1 nmol/l, while in FPHL cases it ranged from 12.7 to 36.8 nmol/l with a mean of 29.5  $\pm$  6.5 nmol/l. The highest serum vitamin D<sub>2</sub> levels among cases were detected in the mild cases of TE (range 12.6–74.4 nmol/l and mean  $\pm$  SD 31.4  $\pm$  11.2 nmol/l) and FPHL (range 14.9–62 nmol/l and mean  $\pm$  SD 31.6  $\pm$  11.1 nmol/l). These differences were of no statistical significance in the TE cases ( $p = 0.203$ ). In the FPHL cases, mean serum vitamin D<sub>2</sub> levels were significantly higher in mild cases in comparison to severe cases ( $p = 0.035$ ) and in moderate cases in comparison to severe cases ( $p = 0.022$ ) (table 2).

The distribution of the TE and FPHL cases is illustrated in table 3 according to the most acceptable cut-off limits for vitamin D<sub>2</sub> [15].

ROC analysis was done to determine the best cut-off values of vitamin D<sub>2</sub> in diagnosing TE and FPHL. The area under the curve of vitamin D<sub>2</sub> was 0.930 in TE and 0.932 in FPHL (95% CI 0.863–0.998 and 0.861–1.002), with the best cut-off values 40.9 and 67.9 nmol/l, that achieved sensitivity (97.6 and 100%) and specificity (80 and 75%) in cases of TE and FPHL, respectively (fig. 1).

There was no correlation between age and serum ferritin ( $r = 0.202$ ,  $p = 0.072$ ), age and vitamin D<sub>2</sub> ( $r = -0.113$ ,  $p = 0.318$ ), serum ferritin and vitamin D<sub>2</sub> ( $r = -0.145$ ,  $p = 0.198$ ) in either TE or FPHL cases or controls.

Multivariate logistic regression analysis was carried out to determine the significant independent risk factors of hair loss (TE and FPHL). We introduced all variables into the analysis, and age, serum ferritin and vitamin D<sub>2</sub> all proved to be significant predictors (OR = 1.175,  $p = 0.000$ ; OR = 0.977,  $p = 0.013$ ; OR = 0.933,  $p = 0.000$ ), respectively.

## Discussion

The results of this study suggest that the reduced hair density seen in chronic TE and FPHL may possibly be associated with low serum levels of ferritin and vitamin D. This was evident from the significantly lower levels in the

**Table 3.** Distribution of the cases according to the most commonly used cut-off limits

	Total number of subjects	Vitamin D <sub>2</sub> deficiency <25 nmol/l	Vitamin D <sub>2</sub> insufficiency 25–75 nmol/l	Vitamin D <sub>2</sub> optimal level 75 nmol/l
TE	42	10 (23.8)	32 (76.2)	–
FPHL	38	13 (34.2)	25 (65.8)	–
Total cases	80	23 (28.8)	57 (71.2)	–
Control	40	2 (5)	9 (22.5)	29 (72.5)

Numbers of patients are represented with percentages in parentheses.

chronic TE and FPHL cases in comparison to the controls, with the lowest levels being reached in the more severe cases according to the trichogram findings (TE) and Ludwig scale parameters (FPHL).

The causes of hair loss are diverse, making it impossible to fix all variables. In this study, we tried to preclude all other factors that might be related to hair loss by following the exclusion criteria so as to be able to focus on the influence of the serum ferritin and vitamin D<sub>2</sub>. Moreover, as depending on clinical examination only is believed to represent a weak point in several hair study designs [5], we utilized several methods including recording a thorough history, answering of a questionnaire, a clinical examination, hair pull test, trichogram and dermoscopy to confirm proper patient selection. We also excluded any participant with CRP >4, so as to confirm the true levels of serum ferritin. With regard to vitamin D research, all included subjects had SPT 3 and 4. The whole study was carried out over 4 months (July–October) with nearly the same climate parameters (all summer months; an Egyptian summer is defined as May–October by many sites). The study was performed on patients from one clinic (Dermatology Outpatients Clinic, Cairo University), serving subjects with the same socioeconomic status, practices and culture.

Our results agree with previous reports [6, 7, 9, 10] demonstrating the association between low iron stores assessed by serum ferritin concentrations and hair loss in women. On the other hand, other studies [5, 11, 12] found no significant link between iron deficiency and hair loss. There is no clear explanation of this discrepancy and additional work is definitely needed to resolve this scientific debate. The existence of several variables, e.g. the study designs adopted in different studies, the variability of the definition of a normal serum ferritin

level in women and the different reference ranges used by different laboratories, are some factors that could contribute in this detected discrepancy [5]. Moreover, the variation in the serum ferritin levels detected in different studies might be attributed to the study population itself. This is because the different patterns of sun exposure of the included subjects [21] as well as the genetic and ethnic variations [22] might all influence the iron status and thereby the serum ferritin levels. Nevertheless, we cannot exclude the concept of serum ferritin being unrelated to hair loss, taking into consideration the reported prevalence of a low serum ferritin level in some populations [23, 24].

All this adds to the complexity of comparing different studies. The lower mean serum ferritin levels of both cases and controls encountered in our study (14.7 µg/l in TE and 23.9 µg/l in FPHL) in contrast to other studies like Kantor et al. [10] (50.1 µg/l in TE and 37.3 µg/l in androgenic alopecia) could further be explained by our specific patient selection, i.e. of female patients in the menstruating phase who are considered to be among the high-risk population for iron deficiency being subjected to menstrual blood loss [8]. Nonetheless, the decreased ferritin levels that we detected in this age group of TE and FPHL patients was still lower than that of their age-matched control subjects.

Although the role of vitamin D in hair disorders and hair follicle cycling has been suggested [25], to the best of our knowledge, no previous studies have examined the relation of vitamin D to TE or FPHL. In this study, its insufficiency/deficiency has been demonstrated in relation to both conditions.

There are several postulated mechanisms by which both iron and vitamin D have possible effects on hair growth. As for the role of iron, from a biological point of view, hair follicle matrix cells are some of the most rapidly proliferating cells in the body. At the cellular level, ferritin levels are increased in nondividing cells, such as stem cells and terminally differentiated cells, whereas rapidly proliferating cells appear to have lower levels of ferritin and higher levels of free iron. This balance of ferritin and iron is at least partially controlled by the transcription factor c-Myc [24]. Overexpression of c-Myc in the cutaneous epithelium results in loss of follicular differentiation and a decrease in stem cells, but whether this phenotype is related to abnormal iron metabolism is still to be determined [25].

Another likely mechanism for the possible effect on of iron on hair growth stems from its requirement as a metabolic cofactor for ribonucleotide reductase, the rate-limiting

enzyme for DNA synthesis. The depletion of iron could prevent proper functioning of this enzyme, resulting in inhibition of proliferation [26]. The inhibition of other iron-dependent enzymes – such as stearyl-CoA desaturase, which, when mutated, causes hair loss in mice and is also present in the human hair follicle [27] – could also contribute to hair loss.

It has been suggested that an optimal concentration of vitamin D is necessary to delay aging phenomena, including hair loss [28]; this might explain the importance of vitamin D in the hair. Moreover, extensive data from animal models clearly show that vitamin D receptor activation plays an important role in the hair follicle cycle, specifically anagen initiation [29]. Interestingly, in vitamin D receptor-ablated mice, it does not seem that normalization of mineral ion homeostasis by means of a diet high in calcium and phosphorous prevents alopecia, suggesting that the mechanism for alopecia is not related to mineral levels but rather to levels of vitamin D [30]. Furthermore, recent data suggested that vitamin D receptor directly or indirectly regulates the expression of genes required for hair follicle cycling, including the hedgehog signaling pathway [31].

Among the ongoing debates, the definition of iron deficiency in hair loss remains an important question. In our work, the cut-off limits were 27.45 and 29.35 µg/l, that achieved a sensitivity of 90.5 and 71% and specificity of 77.75 and 72.5% in cases of TE and FPHL, respectively. Findings by Moeinvaziri et al. [6] led them to suggest that patients with a serum ferritin level of ≤30 µg/l suffering from active hair loss should be given iron therapy. Our study is in agreement with these findings, at least in our part of the world. This is in contrast to other studies which suggest higher cut-off limits such as 40 [7] and 70 µg/l [9] or lower cut-off limits [4] such as 10–15 µg/l.

Interestingly, there was no significant difference in the serum ferritin and vitamin D<sub>2</sub> levels detected in both types of hair loss, despite their variable pathogenesis. This finding supports the previously postulated ‘threshold hypothesis’ [10], which stated that decreased iron stores lower the threshold for developing different types of hair loss and not a certain type. Further investigation is required to determine whether the same concept applies to vitamin D or not.

Obesity, autoimmune, gastrointestinal, hepatic and renal diseases as well as malnutrition are conditions that can be responsible for low serum vitamin D [17] and serum ferritin levels as well as for hair loss. In our study, we attempted to exclude any patients with such abnormalities; however, further large-scale studies with vitamin D

and iron supplementation are needed to properly evaluate their role per se on hair loss. Measuring the serum iron level and the vitamin D binding protein is recommended for more in-depth analysis in future studies. Furthermore, checking this relation in male patients with

androgenetic alopecia would be of interest. Nonetheless, this work shows that serum ferritin, as a representative of the iron status, and vitamin D may both be reduced in female patients with hair loss associated with TE and FPHL.

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