Correlation between Clinical Features, Biochemical Parameters, and Histopathological Findings in Women with Patterned Baldness: A Study from North India

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Abstract

Background: Androgenetic alopecia (AGA) is a non-scarring alopecia with a characteristic pattern in genetically predisposed men and women. Hormonal abnormalities namely hyperandrogenism, hair cycle defects, genetic predisposition, and follicular miniaturization have been implicated as the causative factors for AGA. **Aim**: To analyze women with patterned hair loss and correlate their clinical findings with the histopathology and biochemical parameters. **Materials and Methods**: Female patients between 18 and 45 years of age with a history of hair loss on the crown, temporal area, and recession of hairline were clinically examined. These patients were then subjected to histopathological examination, and on confirmation of diagnosis of female pattern hair loss (FPHL), they were included in the study. Their morning blood sample was taken on 3rd–5th day of the menstrual cycle for hormonal analysis. The study was carried out on 30 patients and 30 age- and sex-matched controls. **Results**: A statistically significant difference was observed between the mean values of dehydroepiandrosterone sulfate, prolactin, androstenedione, and free triiodothyronine of cases and controls. The most common histopathological finding in our study was an increase in the percentage of telogen hair. **Conclusion**: The biochemical findings in our study corroborate the role of hyperandrogenism as one of the major etiological factors in FPHL with the role of adrenal androgens being central, and therefore all female patients with FPHL should be evaluated for underlying hormonal imbalances. The role of histopathology in FPHL can be used as a prognostic marker.

Keywords: Androgenetic alopecia, female pattern hair loss, histopathology, hormone profile

INTRODUCTION

Androgenetic alopecia (AGA) is a non-scarring alopecia with a characteristic pattern in genetically predisposed men and women.[1] It presents as progressive thinning and shortening of hair in the affected areas.[2] The pathognomonic feature is hair follicle miniaturization caused by peripheral androgens.[3] In women, it is also known by various synonyms such as female pattern hair loss (FPHL), diffuse hormonal alopecia, and common baldness in women.[4] There is a difference in the pathogenesis of patterned baldness in men and women; therefore, FPHL is not the same as AGA seen in female patients. The role of hyperandrogenemia is not very clear in FPHL, but it has been seen that levels of androstenedione

and dehydroepiandrosterone are raised in some of these cases. Also the classification followed for the patterned baldness in women is different from that in men. AGA in women with male pattern baldness is very rare and its pathogenesis remains an enigma. Histopathological examination of women with patterned hair loss is also required to differentiate between AGA and simple patterned baldness. Hence, we analyzed women with

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patterned hair loss and correlated their clinical findings with the histopathological and biochemical parameters.

MATERIALS AND METHODS

Selection of cases

Female patients between 18 and 45 years of age with a history of hair loss on the crown, temporal area, and recession of hairline were examined clinically for thinning of hair (loss of hair and presence of thin hair) in the department of dermatology. The patients with clinical features of patterned hair loss, who consented for a punch biopsy, were then subjected to histopathological examination for the confirmation of diagnosis. Consequent on histopathological confirmation of patterned hair loss, these patients were included in the study.

Inclusion criteria

- Female patients with patterned hair loss
- Female patients with age between 18 and 45 years
- Female patients with histopathological features suggestive of patterned baldness

Exclusion criteria

- Female patients who are postmenopausal and those in postpartum (up to 1 year), pregnant, and lactation phase
- Female patients who have undergone hysterectomy
- Female patients with alopecia secondary to known endocrinologic disorder (Cushing's disease, Addison's disease, etc.)
- Female patients on hormonal treatment or oral and/or topical hair growth promoters
- Female patients on immunosuppressive drugs, anticancer agents, and steroids

Selection of controls

The control group included an equal number of women, between 18 and 45 years of age, with no clinical signs of hair loss and/or hyperandrogenism. They were either the attendants with the cases or were healthy medical/paramedical health-care professionals.

A detailed medical history along with physical examination was carried out in all patients. History was obtained from each patient using a pro forma, which included age of onset of patterned hair loss, duration, rate of progression, marital status, parity, age at menarche, menstrual irregularities, and presence of symptoms of virilization (i.e., deepening of voice, thinning of scalp hair, seborrhea, and oligomenorrhea). The patients were examined for clinical evidence of acne, hirsutism, acanthosis nigricans, and striae. Body mass index (BMI) was also measured.

A thorough scalp examination was carried out to look for any skin lesions or signs of inflammation and scarring. The extent of patterned hair loss was assessed by noting the severity of hair loss on the crown, any breach of frontal hairline, or the involvement of frontotemporal area. Hair pull test from the involved area was carried out. Approximately 60 hairs were grasped between the thumb and the index and middle fingers. The hairs were then gently but firmly pulled. A negative test (six or less hairs/less than 10% obtained) indicated normal shedding, whereas a positive test (more than six hairs or 10% obtained) indicated definite active shedding of hair. Shampooing should be withheld for 5 days before a pull test. This test helps to differentiate telogen effluvium from patterned hair loss and is negative in patients of FPHL.

Hair was examined with a handheld magnifying glass with 7× magnification to look for any shaft abnormality.

The patients were then classified clinically into Ludwig/Hamilton and Norwood/Olsen pattern of baldness and were subjected to confirmation by histopathological examination.

A punch biopsy from the crown was obtained from all the cases included in the study. The area was cleaned with spirit and draped. Local infiltration anesthesia was given by injecting 2% plain lignocaine solution. A 4-mm cylindrical biopsy punch was used. The biopsy wound was closed with a single suture and a sterile dressing was applied, instructing the patient to keep it dry.

The specimen was placed in 10% formalin solution, which was used as a fixative. The tissue section was processed and hematoxylin and eosin staining was carried out. The histological evaluation in female patients with patterned hair loss included the following:[5]

1. A ratio of terminal to vellus hair of <4:1 along with miniaturization of hair follicles
2. Increase in the percentage of telogen hair (normal of 5%–10%) to 15%–20% in comparison to anagen hair
3. Perifollicular lymphohistiocytic infiltrate around infundibular portion of hair follicle
4. Perifollicular fibrosis

A total of 10 mL of venous blood sample was drawn for hormonal and biochemical assessment after overnight fasting on 3rd–5th day of the menstrual cycle in the mid-follicular phase from the patients with regular menstrual cycles or on any other day from the patients who have not menstruated in the past 1.5 months. Free and total testosterone, sex hormone binding globulin (SHBG), serum albumin, dehydroepiandrosterone sulfate (DHEAS), and 17-hydroxy progesterone (17-OHP) were measured using enzyme-linked immunosorbent assay. Luteinizing and follicle-stimulating hormone (LH/FSH ratio), prolactin, free triiodothyronine (fT3), free tetraiodothyronine (fT4), and thyroid-stimulating hormone (TSH) levels were measured by chemiluminescence.[6]

Pelvic ultrasound for ovaries was carried out between day 3 and 5 of menstrual cycle in all patients. Modified National Institutes of Health (NIH) criteria[7] were used for the diagnosis of polycystic ovarian syndrome (PCOS). The

polycystic ovarian morphology was defined by the presence of 10 or more intermediate follicles each measuring 2–9 mm in diameter and/or increased ovarian volume of more than 10 mL on transabdominal ultrasound.

RESULTS

Demographic features

Of the 30 patients in our study, 16 (53.3%) were in the age group of 28–37 years, followed by 8 (26.6%) in the age group of 18–27 years, and 6 (20%) in the age group of 38–45 years. The mean age at presentation was 31.17 years. The mean age of the control group was 30.6 years. The majority of cases, that is, 21 (70%), noticed the onset of hair fall before the age of 30 years. A total of 13 (43.33%) patients were having hair fall for 1–3 years, whereas 11 (36.66%) patients had been witnessing hair fall for 4–6 years. The remainder of six (20%) patients had hair loss for more than 6 years. The mean age of onset and the mean duration of hair loss were 26.00 and 5.1 years, respectively. The family history of AGA in first-degree relatives was noted in 14 (46%) patients. The mean age at menarche observed in our study was 13.07 years.

Pattern of hair loss

A total of 23 (76.6%) patients had Ludwig pattern [Figure 1A], six (20%) had Olsen pattern [Figure 1B], and a single (3.3%) patient had Hamilton and Norwood pattern [Figure 1C].

Associated clinical features

Acne

A total of 11 (36.67%) patients had acne, and of these 11 patients, six had Ludwig pattern of hair loss, five showed Olsen pattern, whereas none had Hamilton and Norwood pattern. A statistically significant association was found between the different patterns of hair loss and acne ($P < 0.026$).

Acanthosis nigricans

A statistically significant relation was found between different patterns of baldness in women and acanthosis nigricans ($P < 0.034$), with 13 patients having acanthosis nigricans. There were seven patients with Ludwig pattern, five who had Olsen pattern, and one patient who had Hamilton and Norwood pattern.

Hirsutism

In our study, hirsutism was seen in 20 (66.6%) patients of which 13 (43.3%) had Ludwig pattern, five (16.6%) had Olsen pattern of hair loss, and a single patient had Hamilton and Norwood pattern. The association between hirsutism and different patterns of hair loss was not found to be statistically significant ($P > 0.05$).

Polycystic ovarian syndrome

Of the 30 patients, eight were diagnosed as PCOS based on the modified NIH criteria. Of these eight patients, two had unilateral cysts, three had bilateral cysts, and three had bilateral cysts with enlarged ovaries. Five patients of PCOS had Ludwig pattern, two had Olsen pattern, and only one had Hamilton and Norwood pattern. The association among different patterns of hair loss and PCOS was not statistically significant. Of the eight patients who had PCOS, five (62.5%) were overweight with a BMI greater than 24.99 kg/m², and the association between BMI and PCOS was found to be statistically significant ($P < 0.009$).

Laboratory findings

The mean hormone levels in cases and controls is summarized in Table 1. The difference between the mean values of DHEAS, prolactin, androstenedione, and fT3 of cases was found to be statistically significant when compared with that of the control group by *t*-test ($P < 0.05$). No significant association was found between different patterns of hair loss with the levels of prolactin, androstenedione, and fT3.

The chi-square test showed a statistically significant association between raised free testosterone (fT) levels and Hamilton and Norwood pattern of baldness ($P < 0.000$). A single patient with Hamilton and Norwood pattern had significantly raised levels of fT.

Figure 1: Different patterns of hair loss: (A) Ludwig pattern, (B) Olsen pattern, and (C) Hamilton and Norwood pattern

Table 1: Mean hormone levels in cases and controls			
Hormones	Controls (*N* = 30)	Cases (*N* = 30)	PCOS (*N* = 8)
	mean ± SD	mean ± SD	mean ± SD
Free testosterone (range in ng/L)	1.80 ± 0.94 (0.56–4.27	1.54 ± 1.34 (0.29–7.65) (*P* > 0.392)	2.03 ± 2.30 (0.58–7.65) (*P* > 0.66)
Total testosterone (range in nmol/L)	0.54 ± 0.44 (0.06–1.6)	0.729 ± 0.82 (0.2–3.08) (*P* > 0.29)	1.43 ± 1.25 (0.24–3.08) (*P* < 0.002)*
DHEAS (range in µg/mL)	1.21 ± 0.61 (0.48–2.6)	1.7 ± 0.57 (0.84–2.89) (*P* < 0.002*)	1.92 ± .723 (1.04–2.89) (*P* < 0.007)*
17-OHP (range in ng/mL)	0.42 ± 0.30 (0.21–1.2)	0.53 ± 0.46 (0.2–1.8) (*P* > 0.276)	0.73 ± 0.61 (0.22–1.86) (*P* < 0.05)*
LH/FSH	0.92 ± 0.35 (0.37–1.59)	0.97 ± 0.64 (0.26–2.27) (*P* > 0.728)	1.02 ± 0.80 (0.26–2.27) (*P* > 0.59)
Prolactin (range in ng/mL)	10.13 ± 3.92 (4–18.1)	14.09 ± 6.79 (1.4–29.7) (*P* < 0.008*)	15.24 ± 9.69 (1.4–29.7) (*P* < 0.02)*
Free T3 (range in pg/mL)	3.17 ± 0.79 (2–5.3)	3.70 ± 0.66 (2.64–5.37) (*P* < 0.007*)	3.37 ± 0.57 (2.64–4.21) (*P* > 0.51)
Free T4 (range in ng/dL)	1.31 ± 0.40 (0.77–2.2)	1.39 ± 0.55 (0.7–2.99) (*P* > 0.532)	1.38 ± 0.535 (1.07–2.63) (*P* > 0.67)
TSH (range in mIU/mL)	2.59 ± 1.2 (0.6–5.6)	2.58 ± 2.08 (0.015–47.4) (*P* > 0.988)	1.90 ± 1.72 (0.049–4.2) (*P* > 0.21)
Androstenedione (range in pmol/L)	0.96 ± 0.49 (0.12–1.56)	1.7 ± 0.99 (0.18–4.5) (*P* < 0.000*)	2.11 ± 1.07 (1.1–2.9) (*P* > 0.05)
SHBG (range in nmol/L)	72.8 ± 29.5 (19.19–109.4)	63.5 ± 34.8 (8.93–117) (*P* > 0.27)	57.44 ± 38.01 (21.47–114.08) (*P* > 0.22)
Serum albumin (range in g/dL)	4.07 ± 0.469 (3.4–5.5)	4.21 ± 0.389 (3.6–4.8) (*P* > 0.225)	4.33 ± 0.392 (3.8–4.8) (*P* > 0.14)

* denotes a significant result.

Similarly a significant association was found between the total testosterone levels and different patterns of baldness with all the patients with Ludwig pattern of hair loss having normal total testosterone levels and the two patients with raised total testosterone levels had Olsen type and Hamilton and Norwood type of hair loss.

Other clinical features
No significant association was found between different pattern of hair loss and seborrhea, striae, BMI, and menstrual irregularities.

Histopathological findings
The most common histopathological finding in women with pattern baldness, which was reported in our study was an increase in the percentage of telogen hair. This finding was seen in 63% (19) patients. Miniaturization [Figure 2A] and perifollicular fibrosis [Figure 2B] was reported in nine patients each. Perifollicular infiltrate [Figure 2B] was present in 56% (17) of the patients. The chi-square test revealed no statistically significant association of the histopathological findings with different patterns of hair loss (*P* > 0.05). The histopathological findings in different patterns of alopecia are shown in Table 2.

Table 3 shows the relationship between different patterns of hair loss and the histopathological findings. The

histological findings [Figure 2] were variable and a combination of three different findings was seen in five patients, whereas two histological findings were reported in 14 patients. The combination of all the four histological findings was not present in any of the patients in our study.

Discussion
In this study, more than half of the patients (53.3%) were in the age group of 28–37 years. In a study by Lee *et al.*[2] on 445 female subjects with patterned hair loss, it was observed that 252 (56.68%) of the 445 were in the third decade of their life, which is similar to our study.

The majority of the patients, that is, 21 (70%), noticed the onset of hair fall before the age of 30 years. In our study, the mean age of onset was 26.00 years. In a study on Chinese women with patterned baldness by Zhang *et al.*,[8] the mean age of onset calculated in 60 women with patterned hair loss was 29.8 years. Norwood[9] reported that Caucasian women had FPHL, which begins in late 20s. The age of onset in both these studies is similar to the mean age of onset observed in our study. The mean duration of hair loss in our study was 5.1 years. Zhang *et al.*[8] reported the mean duration of FPHL as 4.49 years, which is nearly in agreement with our findings.

In our study, the family history of hair fall in first-degree relatives was noted in 14 (46%) patients. Paik *et al.*[3] in
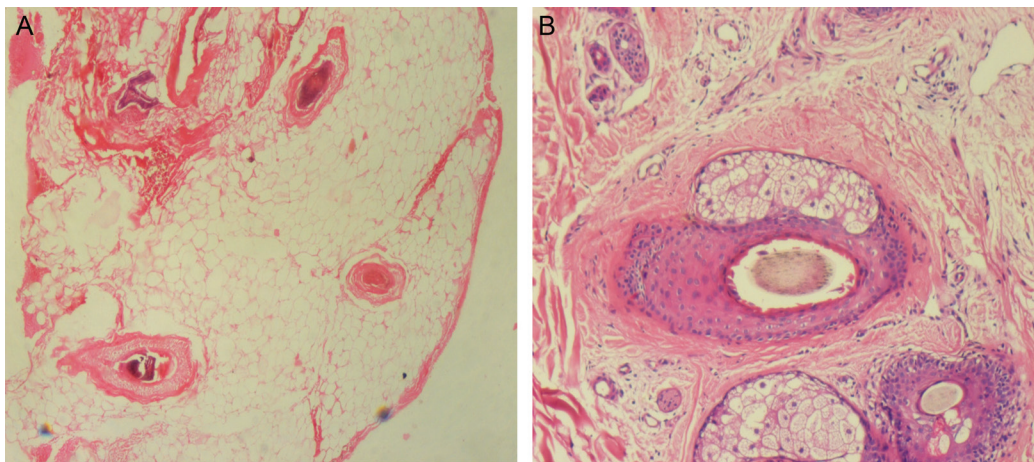
Figure 2: Histopathological findings under 40× magnification with hematoxylin and eosin (H and E) staining. (A) Miniaturized hair follicle. (B) Perifollicular fibrosis and sparse mononuclear inflammatory infiltrate

Table 2: Histopathological findings in different patterns of alopecia

Histopathological findings	Ludwig pattern ($N = 23$)	Olsen pattern ($N = 6$)	Male-type Hamilton and Norwood pattern ($N = 1$)	Total
Miniaturization	5 (55.5%)	3 (33.3%)	1 (11.1%)	9 (30%)
Increase in percentage of telogen hair	13 (68.4%)	5 (26.3%)	1 (5.2%)	19 (63%)
Perifollicular infiltrate	14 (82.3%)	2 (11.7%)	1 (5.8%)	17 (56%)
Perifollicular fibrosis	7 (77.7%)	2 (22.2%)	0	9 (30%)

their study on Korean women with FPHL observed that a positive family history of baldness was present in 45.2% of the patients with patterned baldness and it showed a tendency to increase in third to fifth decade of life.

In our study, hirsutism was seen in 20 (66.6%) patients, whereas previous studies have reported hirsutism in 12%–22% of patients.[10-12] The association between hirsutism and different patterns of hair loss was not found to be statistically significant ($P > 0.05$).

We found acne in 11 (36.6%) of our patients in study. Six patients had Ludwig pattern of hair fall and the remaining five had Olsen pattern. A statistically significant association was found between different patterns of hair loss and acne as of the total six patients with Olsen pattern of hair fall, five (83.33%) had acne ($P < 0.026$). This also implied that women with Olsen pattern of hair loss are more prone to develop acne. Cela *et al.*[10] reported acne in 38 (43%) of the 89 patients with AGA. Moltz[11] in his study on 125 female patients of AGA reported acne in 41.6% of them. The findings of the studies quoted above are similar to those in our study. Literature search did not show any studies linking the different patterns of hair loss with acne.

In this study, 13 (43.3%) patients had acanthosis nigricans. Of these 13 patients, seven (53.8%) had Ludwig pattern of baldness and five (38.5%) had Olsen pattern of hair loss. Only a single patient had the male pattern of baldness. A statistically significant relation was found between

Table 3: Relationship between different patterns of hair loss and the histopathological findings

Histopathological findings	LW	O	H and N	Total
a) Miniaturization	1	0	0	1
b) Increase in percentage of telogen hair	6	1	—	7
c) Perifollicular infiltrate	3	0	0	3
d) Perifollicular fibrosis	0	0	0	0
a + b + c + d	0	0	0	0
b + c + d	1	0	0	1
a +b + c	1	0	1	2
a + b + d	1	1	0	2
c + d	4	0	0	4
b + c	3	2	0	5
a + c	2	0	0	2
a + d	0	1	0	1
a + b	0	1	0	1
b + d	1	0	0	1

LW = Ludwig pattern, O = Olsen pattern, H and N = Hamilton and Norwood pattern

different patterns of baldness in women and acanthosis nigricans ($P < 0.034$) as of the total six patients with Olsen pattern of hair fall, five (83.33%) had acanthosis nigricans. The implicit interpretation of these findings is that women with Olsen pattern of hair fall are more prone to develop acanthosis nigricans. None of the studies, which we have reviewed, have commented on the association between different patterns of hair loss and acanthosis nigricans.

The relationship between PCOS and different patterns of hair loss was not statistically significant ($P > 0.05$).

Futterweit *et al.*[13] studied 109 women with alopecia and they reported a 28% prevalence of PCOS in their study. The difference in the prevalence of PCOS in our study group can be due to the smaller sample size (n = 30).

The difference between the mean values of DHEAS, prolactin, androstenedione, and fT3 of cases was found to be statistically significant when compared with that of the control group by *t*-test ($P < 0.05$). In their study on women with patterned hair loss, Kasick *et al.*[14] reported a significant increase in DHEAS levels in cases compared to that in controls, whereas Montalto *et al.*[15] and Rushton *et al.*[12] did not find a significant increase in the plasma levels of DHEAS in their studies. These findings suggest that the androgens secreted from adrenals may be raised in FPHL with or without other findings of hyperandrogenemia, thus suggesting that lower potency androgens as compared to testosterone are sufficient to cause FPHL. In addition, the biochemical derangement in our study reemphasizes the important role of hyperandrogenism as the main cause of hair loss in FPHL. Futterweit *et al.*[13] published a paper about the prevalence of hyperandrogenism in 109 consecutive female patients with diffuse alopecia. Of them, only two patients had hyperprolactinemia caused by pituitary tumors. Hypothyroidism and hyperprolactinemia as a possible cause of AGA in women was also reported by Schmidt *et al.* in 1989 as he observed that nine patients in their study had increased prolactin levels.[16] The raised levels of prolactin suppress estrogen, and thereby, the potency of androgen increases, which in turn leads to FPHL. In a study carried out by Cela *et al.*[10] on 89 women with patterned baldness, the androstenedione levels were significantly higher in cases than that in controls. Androstenedione is partly secreted from the adrenal cortex and can be peripherally converted to free testosterone and dihydrotestosterone; it is possible that there may be a state of adrenal hypersecretion in patients with FPHL.[16]

All the four histopathological findings were equally distributed among the three different patterns. The histological findings were variable, and a combination of three different findings was seen in five patients, whereas two histological findings were reported in 14 patients. The combination of all the four histological findings was not present in any of the patients in our study. Increase in telogen hair was the most common finding in our study. Lattanand *et al.*[17] found that miniaturization of hair follicles was the most common finding in male pattern hair loss. Olsen also reported miniaturization of hair follicles as the main finding in their study. The different findings in our study can be explained by the smaller sample size. Also, the hair cycles are shorter in patients of AGA, and hence a higher number of telogen hair can be seen, as explained by Whiting.

In a previous study by Whiting[18] on 106 subjects with male pattern AGA (MPAA), it was found that 36% had complicated MPAA (moderate-to-severe perifollicular infiltrate along with fibrosis). On treatment with 2% minoxidil for 1 year, 45% of the patients with complicated MPAA showed no change in hair density, whereas another 45% showed mild regrowth. In comparison, patients with plain MPAA showed mild regrowth in 63% and moderate regrowth in 14%.[17]

In this study, majority of the patients (76%) had Ludwig pattern of baldness. On histopathological examination, it was found that majority of the patients with perifollicular infiltrate (82.3%) and perifollicular fibrosis (77.7%) had Ludwig pattern of hair loss. The implicit interpretations of the aforementioned findings are that the patients presenting with Ludwig pattern of baldness should undergo histopathological examination for the evidence of perifollicular infiltrate and fibrosis, which might serve as a potential prognostic marker for poor response to treatment.

STRENGTH AND LIMITATIONS OF THE STUDY

To the best of our knowledge, this study is the first Indian study to evaluate the clinical patterns of hair loss and the hormonal and histopathological findings in tandem with women with AGA.

The sample size in this study is small for the extrapolation of the results to the general population, but some trends are evident. Histopathological examination of control subjects is also required for better comparison of the results.

A trichogram was not carried out as it is a semi-invasive test and can generate procedural artefacts. In addition, relative values are generated (telogen/anagen ratios), and they are a poor indicator of disease activity or severity.[19] A dermoscopic analysis could not be carried out because of lack of availability of the instrument.

Future studies should aim at including larger number of subjects, and patients should be followed up over a longer duration to validate the prognostic implications of the histopathological findings.

CONCLUSION

The patients who present with FPHL should be advised a hormonal evaluation as adrenal androgens can be raised in these patients despite the absence of clinical signs of hyperandrogenism. Also, patients should be subjected to histopathological evaluation as it can have prognostic implications in deciding the therapeutic approach.

Conflicts of interest

There are no conflicts of interest.

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