

Serum iron and ferritin levels in alopecia areata

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Background: Alopecia Areata (AA) is a recurrent non-scarring patchy hair loss of autoimmune origin in a genetically susceptible individual although environmental factors may be contributory. The aim of this study is to evaluate serum iron and ferritin levels in patients with AA.

Methods: Fifty patients of AA and 50 age and sex matched control subjects without any hair loss were studied.

Results: No significant difference was found in mean hemoglobin, ferritin, and iron levels in two groups. Serum iron ferritin and hemoglobin levels did not show any significant variations with respect to the extent or duration of the disease, family history, history of atopy, and nail involvement.

Conclusion: In our study, the levels of serum iron and ferritin showed no significant changes in AA patients and seems not to play a role in the etiology of AA.

Keywords: alopecia areata, serum iron, serum ferritin, hemoglobin

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INTRODUCTION

Alopecia Areata (AA) is a recurring, non-scarring inflammatory hair loss that can affect any hair bearing area. Clinically, the prototype lesion is a discoid patch of alopecia without scaling or signs of inflammation. The presence of exclamation mark hair at the periphery of the lesion is pathognomonic. Although immunologic processes and hereditary factors are suggested to play an important role in AA, the specific etiology is unclear^{1,2}. Many theories have been suggested in the etiopathogenesis of AA including infectious, neural, genetic, and organ specific autoimmune hypotheses. There is increasing evidence that AA is a tissue-specific autoimmune disease as it has been associated with various autoimmune disorders³⁻⁵. Another probable mechanism is oxidative stress characterized by increased free radical production exceeding the intracellular antioxidant defense⁶. Environmental factors can trigger the disease^{7,8}. Ferritin has been reported to exhibit different immunological activities such as suppression of antibody production by lymphocytes

and suppression of delayed type hypersensitivity. The ferritin levels are increased in inflammation, infections, malignancies and autoimmune diseases⁹. Since AA is an autoimmune disease, there must be changes in serum ferritin and iron levels. The present study was undertaken to find any association between serum iron, ferritin and other haematological tests with AA.

PATIENTS AND METHODS

This prospective case control study was conducted in the Department of Dermatology, Govt. Medical College Jammu for a period of one year from May 2008 to April 2009. After obtaining informed consents from all the patients included in the study, 50 patients with clinical diagnosis of AA attending the outpatient department (OPD) were recruited. This study was approved by the Institutional Ethical Committee. Detailed history was taken regarding age, sex, activity of the disease, family history, onset, progression, duration of the disease, associated diseases, and any previous treatment. Complete clinical examination was

also performed to detect the pattern of AA, the extent of the involvement (the number and size of the patches) and site (s) involved. Nails were examined for any changes. Exclusion criteria were other autoimmune disorders, pregnancy, known cases of anaemia, any chronic disease, drug use, iron supplementation and other associated diseases that could alter levels of serum ferritin and iron. The control group consisted of 50 age and sex matched individuals without any forms of hair loss attending our OPD for other cutaneous disorders with the same exclusion criteria as for cases. Serum levels of iron, ferritin, total iron binding capacity (TIBC), hemoglobin (Hb) and hematocrit (Hct) were measured in patients and controls. Hemoglobin was measured with hemocue haemoglobin analyser, serum iron/TIBC with the automated AAI-25 colorimetric method and ferritin with the radioimmunoassay method. Any level of hemoglobin lower than normal for sex was regarded as anemia. The levels of ferritin, and iron in AA patients were compared with those of controls. Clinical subtypes of AA were compared with controls and with each other. The statistical analysis was carried out using the arithmetic mean, standard deviation, standard error, and Student's "t" Test. One-sided p-values with the traditional cut-off point of $p < 0.05$ were utilized in order to determine statistical significance. Computer software Stata was used for statistical analysis.

RESULTS

Out of all AA patients, 32 (64%) were male and 18 (36%) were female with a mean age of 25.4 year (range: 8 to 55 years). The duration of hair disease varied from ten days to 100 months, with prolonged duration in the patients having extensive lesions, nail involvement, family history and history of atopy. The family history of AA and atopy was positive in 20% and 16% of the patients,

Table 1. The involvement sites of alopecia areata.

Site of involvement	No of patients
Scalp	35 (70%)
Face	6 (12%)
Scalp+Face	9 (18%)
Total	50 (100%)

respectively. The onset of the disease was sudden in the majority of the patients, followed by slow progression. Sixty percent of the patients had recurrent lesions. The majority of the patients (80%) had used some treatment. The site of involvement has been shown in Table 1.

Nail involvement was seen in 6 patients. Low levels of hemoglobin, ferritin, and iron were detected in 6 (12%), 5 (10%), and 9 (18%) of AA patients, respectively. No significant difference was found between the patients and the controls in mean hemoglobin, ferritin, and iron levels although values were lower in patients (Table 2).

Serum iron ferritin and haemoglobin levels did not show any significant variation in the extent or duration of the disease, family history, history of atopy, and nail involvement.

DISCUSSION

Few studies suggest that iron deficiency can serve as a triggering factor in AA. They suggest that iron deficiency may be a limiting factor if the scalp hairs are in a phase in which re-growth is possible⁵. Rushton et al,¹⁰ indicated that hemoglobin and ferritin levels were decreased in many women with alopecia; these levels generally still fell within the "normal range". Thus, the so-called "normal values" of ferritin and hemoglobin may include women who are physiologically depleted of iron. White et al,⁵ concluded that female patients with AA had an increased incidence of iron deficiency in comparison with the general population. They suggested serum ferritin and iron measurement

Table 2. The comparison of mean value of hematological tests in patients and controls.

Laboratory tests	controls		Patients		Mean difference	Std. error of difference between two means	Ps
	mean	SD ^o	Mean	SD ^o			
Iron	67.47	22.49	60.1	20.1	7	4.26	>.05
TIBC	305	52.8	322	49.19	17	9.92	>.05
Ferritin	43.42	26.58	36.16	23.83	7.26	5.04	>.05
Hb	13.12	1.9	12.50	1.5	0.62	0.34	>.05
Hct	42.82	6.95	40.40	6.62	2.42	1.36	>.05

should be a necessary part of the work-up in patients with AA⁵. None of the men with AA in the study performed by White et al, had iron deficiency anemia. This argues against a real association between iron deficiency and AA. Kantor et al,¹¹ found that mean ferritin levels in patients with AA were significantly lower as compared to individuals without hair loss. The above-mentioned observations are particularly relevant in view of the surprise finding of a significantly lower mean serum ferritin concentration when they would have been expected to have higher levels due to AA which is regarded as an inflammatory condition. These observations are in contrast to our findings. Esfandiarpour et al,⁴ found higher mean levels of serum iron/ferritin, hemoglobin, hematocrit and lower mean levels of TIBC in patients as compared to the controls, but the difference was insignificant ($P>0.05$).

In our study, mean levels of serum ferritin, iron, TIBC, hemoglobin and hematocrit in AA patients were not statistically different from those in the control group, as also reported by Boffa et al³, Esfandiarpour et al⁴, and Mussalo et al,¹² who all suggested that the prevalence of iron deficiency was not significantly increased in patients with AA.

In conclusion, we noted no significant association between AA and iron deficiency and therefore suggest that iron deficiency does not play a role in the etiology of AA. Our conclusion seems logical as AA might be basically an (immunogenetic) autoimmune disease with a genetic base.

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