

Evaluation of Galectin-3 level in Male Androgenetic Alopecia Patients

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Abstract

Background: Androgenetic Alopecia areata (AGA) is one of the most prevalent forms of hair loss, especially among men. Terminal hair transforms into intermediate hair and then vellus hair over time in people with AGA, a hereditary condition. Study participants and healthy volunteers will be compared for their blood levels of galectin-3. **Methods:** This Sixty males with androgenetic alopecia (age 18) and twenty and six age-matched healthy controls were studied in a case-control study. Serum galectin-3 levels were measured with other laboratory parameters in a thorough assessment of all patients. **Results:** The Patients and controls did not vary significantly in terms of age ($P = 0.082$), body mass index (BMI) ($P = 0.478$), or obesity ($P = 0.517$). Sixty percent of patients compared to twenty-five percent of controls smoked ($P = 0.007$). The median serum galectin-3 was substantially higher in patients than controls (20.38 ng/ml, 8.63 ng/ml respectively; $P < 0.001$). A favourable association between serum galectin-3 and the course of the illness was found ($r = 0.438$, $P 0.001$) **Conclusion:** The The results of this investigation revealed that AGA patients, regardless of age or body mass index, had elevated blood galectin-3 levels.

Keywords: Revealed, AGA patients, body mass index (BMI)

Introduction

Androgenetic Because of androgen and genetic factors, alopecia causes follicular shrinkage and patterned hair loss (Yang et al., 2014; Choi et al., 2017). Hair loss often begins in the third or fourth decade of life, although it may begin at any time after puberty and worsens with time (Di Loreto et al., 2014). Hereditary androgen deficiency (AGA) causes terminal hair to gradually transform into finer, shorter hair (Vora et al., 2019). Concomitancy between AGA and several disorders, such as cardiovascular disease and hypertension, has been found (Herrera et al., 1995). Numerous epidemiological studies have revealed a link between AGA and increased cardiovascular risk in those with an early start (Lotufo et al., 2000). The -galactoside-binding lectin known as galectin-3 is expressed on the cell surface and plays an essential role in cell proliferation, apoptosis control, inflammation, fibrosis, and host defence throughout a wide range of tissues and organs. Serum and urine have detectable levels of galectin-3, making it a useful biomarker (Akira et al., 2020).

The purpose of this investigation was to examine galectin-3 levels between male androgenetic patients and healthy controls.

Subject and Methods

This case control research was undertaken on sixty AGA patients and twenty age and sex matched healthy volunteers as controls. They came from the Dermatology, Venereology, and Andrology (DVA) Outpatient Clinic at Benha University.

This research followed the criteria outlined in the Helsinki declaration and was approved by the Research Ethical Committee at Benha Faculty of Medicine.

Criteria for inclusion

Male patients with varying degrees of AGA. All of the participants in this research were above the age of 18 and gave their informed consent.

Criteria for exclusion

Patients with a known septic focus, diabetes mellitus, cardiovascular disease, hypertension, a history of active malignancy, a history of taking immunosuppressive treatment, a history of other dermatological diseases, and patients on systemic therapy for AGA for less than one month prior to the study were not included in this study.

Methods

All participants were put through

Full documentation of the past:

Personal history, current condition history, medication history, systemic illness and endocrine issue history, prior AGA treatment history, and AGA family history were all included.

Check for any systemic disorders; this is a broad assessment. The BMI was determined by using the following formula to analyse the measured body weight and height.

$BMI = \text{Mass kg} \div \text{height m}^2$

The following estimated body mass index (BMI) categories were derived: underweight 18, normal weight = 18.5-24.9, overweight 25-29.9, and obese 30+. (WHO, 2008).

Regional Diagnosis

Classification by Hamilton and Norwood was used to determine the degree of AGA (Hamilton, 1951; Norwood, 1975).

Studies Conducted in a Laboratory

Sampling consisted of drawing 5 ml of peripheral blood from each participant using a conventional venipuncture method under strict sterile circumstances. Serum was allowed to

coagulate at room temperature for 10-20 minutes, centrifuged for 20 minutes at 2000-3000 r.p.m., and then refrigerated at - 20o C until use.

Concentrations of Galectin-3 in Serum

Galectin-3 concentrations were determined using the ELISA technique, using a commercially available, human-specific ELISA kit, as per the manufacturer's instructions.

Statistical Methods

Data The most recent version of SPSS was used for all administrative and statistical processing (IBM, Armonk, New York, United States). The Kolmogorov-Smirnov test was used to check for normality in the quantitative data (for patients), while the Shapiro-Wilk test was used (for controls) and direct data visualisation techniques were also used (for both). Summaries of numerical information included medians, ranges, and standard deviations. Numbers and percentages were used to summarise the categorical information. Independent t-tests or Mann-Whitney U tests were used to compare normally distributed and

non-normally distributed numeric variables across research groups. Chi-square analysis was used to compare categorical variables. ROC analysis was done for utilising serum galectin-3 in distinguishing AGA patients. The diagnostic indices, optimal cutoff point, and area under the curve were determined along with a 95% confidence interval. Spearman's correlation was used to find associations between levels of serum galectin-3 and a variety of other variables. We used the Mann Whitney U test and the Kruskal-Wallis test to compare serum galectin-3 levels across several criteria. Predictions of AGA were made using a logistic regression model. Both the odds ratio and the 95% CI were determined. All of the statistical analysis was unreliable.

Results

Smoking was statistically significant in patients (60 percent vs. 25%; $P = 0.007$) but not in controls. Patients and controls did not vary significantly from one another in terms of age ($P = 0.082$), body mass index ($P = 0.478$), or obesity ($P = 0.517$). Table 1: Patient and Control Group Demographics

		Patients (n = 60)	Control (n = 20)	Test	P
Age (Years)	Mean \pm SD	41 \pm 9	38 \pm 5	t = -1.772	0.082
Smoking	n (%)	36 (60 %)	5 (25%)	$X^2 = 7.335$	0.007
BMI (Kg/m ²)	Mean \pm SD	29.85 \pm 4.26	28.88 \pm 5.52	t = - 0.720	0.478
Obesity	n (%)	29 (48.3 %)	8 (40%)	$X^2 = 0.419$	0.517

Age and body mass index were tested using a t = independent t-test.

Analysis of the correlation between smoking and weight gain using $X^2 =$ Chi-square * $P < 0.05$

The average onset age for AGA was 26. All the patients had AGA that started before the age of 35. Disease durations varied widely, from 4 years at the low end to 26 years in the high. Grade III was the most common Hamilton-

Norwood categorization, accounting for 31.7% of all cases, while grade I was the least common (3.3 percent). With regard to the degree of AGA, almost two-thirds of patients had either mild or moderate AGA (43.4% and 45.2%, respectively), while 11.7% had severe AGA. Almost three-quarters of patients reported a similar case in their family. All patients reported no previous history of systemic illnesses.

Clinical features of AGA patients are listed in Table 2.

Clinical characteristics

Age of AGA onset (years)	Mean \pm SD	26 \pm 4
Onset of AGA	Early onset n (%)	60 (100%)
Disease duration (years)	Median (range)	14 (4 - 36%)
	I	n (%) 2 (3.3%)
Hamilton-Norwood classification	II	n (%) 5 (8.3%)
	III	n (%) 19 (31.7%)
	IV	n (%) 15 (25%)
	V	n (%) 12 (20%)
	VI	n (%) 7 (11.7%)

Severity of AGA	Mild	n (%)	26 (43.3%)
	Moderate	n (%)	27 (45%)
	Severe	n (%)	7 (11.7%)
Family history of AGA		n (%)	45 (75%)
History of systemic diseases		n (%)	0 (0%)

Grades I–III for mild, grades IV–V for moderate, and grades VI–VII for severe AGA (grade VI, VII)

Discussion

Androgenetic The most common kind of hair loss is alopecia (Blume-Peytavi, et al., 2011). Early catagen hair follicles showed signs of significant endogenous autophagy impairment and enhanced apoptosis (Liu et al., 2021). Some research suggests that AGA is linked to cardiovascular risk factors such as hypertension (Bakry et al., 2015), metabolic syndrome (Swaroop et al., 2019), and central obesity (Vora et al., 2019). Mast cells, histiocytes, and macrophages are just few of the immune cells that produce galectin-3, and it's crucial in a wide variety of ways. Heart failure is only one of several cardiac illnesses for which galectin-3 may be useful as a diagnostic and prognostic biomarker (Hara et al., 2020). Consistent with Neveen et al., we found that the serum galectin-3 level in AGA patients was considerably greater than control (2020). The present research demonstrated a positive association between galectin-3 levels and illness duration, while Neveen et al (2020). The disparity in findings across research may be due to the studies' respective sample sizes (20 male patients). Galectin-3 was shown to have no correlation with body mass index in this investigation. These findings corroborated those of Dag et al. (2019), who examined the relationship between galectin-3 and body mass index (BMI) in a sample of 53 adolescents (52 of whom were obese) and 33 (who were of normal weight) for comparison. Wiegert et al. (2010) observed that serum galectin-3 levels were greater in obese individuals with type 2 diabetes compared to overweight and normal weight groups, hence this finding contradicted their findings. Ohkura et al. (2014) revealed that galectin-3 level was linked with IR in type 2 diabetes but not with BMI.

Conclusion

The Patients with AGA, independent of age or body mass index, were shown to have significantly higher levels of blood galectin-3. There may be a connection between galectin-3's higher levels in AGA patients and its possible function in fibrogenesis or apoptosis. The presence of elevated galectin-3 in AGA

patients may be indicative of an increased cardiovascular risk.

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