# Evaluation of Adipocyte Fatty acid Binding Protein (A-FABP) Serum Level in Male Androgenetic Alopecia Patients

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# ABSTRACT

Male androgenetic alopecia (AGA) is the utmost frequent kind of male baldness.

**Objective:** This study aimed to determine serum levels of A-FABP in individuals with AGA and their putative link to the severity of AGA and other metabolic diseases. **Methods:** This case-control research comprised 60 male patients with AGA and twenty healthy volunteers of the same age and gender. Medical history, clinical examination, and laboratory testing were performed for all individuals. A-FABP serum levels were determined for all subjects. Human A-FABP ELISA kit was used to assess A-FABP in the serum. Participants' A-FABP serum levels were analysed. **Results:** AFABP levels in AGA patients were substantially higher than in controls.

**Conclusion:** The pathogenesis of AGA may be aided by the presence of serum A-FABP. With its high sensitivity, specificity, and accuracy rate, it might be regarded a biomarker for early illness detection.

Keywords: Adipocyte, Fatty acid binding protein, Androgenetic alopecia, AFABP.

### INTRODUCTION

Patients who are genetically susceptible to androgenetic alopecia have a non-scarring, gradual hair follicles shrinkage of. A genetic susceptibility and the androgens presence are both critical etiological variables for AGA, according to Brockschmidt et al. <sup>[1]</sup> research. Testosterone levels may contribute to the development of thrombosis and atherosclerosis and hypercholesterolemia <sup>[2]</sup>.Adipose tissue produces and secretes a range of peptides known as adipokines that affect weight gain, insulin sensitivity, fat metabolism, and vascular functSion. Adipose fatty acid binding protein (A-FABP) has been proposed as another adipokine that is generated and released by adipose tissue <sup>[3]</sup>.Data from both rats and humans reveal that A-FABP is produced into the circulation by adipose tissue, which has been demonstrated to influence the inflammatory cytokines production <sup>[4]</sup>. The obesity development, insulin resistance, diabetes mellitus, atherosclerosis, and hypertension are all associated to elevated levels of A-FABP<sup>[5]</sup>.

Serum level of A-FABP in individuals with AGA and its putative link to the severity of AGA and other metabolic diseases were the focus of this investigation.

# PATIENTS AND METHODS

For this case-control study, twenty age-matched healthy volunteers of the same gender and sixty male patients who presented with AGA participated in this study. They were chosen from patients attending the Outpatient Clinic of Benha University Hospitals, Department of Dermatology, Venereology, and Andrology. The research was conducted between May 2019 and October 2021.

**Inclusion criteria:** The study included male patients with AGA over 18 years old.

**Exclusion criteria:** Patients who had medical history of smoking, who had active malignancy, who

had a systemic disease (such as cardiovascular disease, diabetes mellitus and hypertension) and

chronic liver or renal disease, or who were receiving anti-hyperlipidemic drugs.

# All participants were divided into two groups:

- Group A: Sixty male patients with AGA.
- Group B: Twenty age- and gender-matched volunteers as control group.

All participants were undergoing complete clinical examination, detailed history taking, and laboratory measurement. All cases were tested for adipocyte fatty acid binding serum level determination. Serum A-FABP was measured using "Human adipocyte fatty acid binding protein (A-FABP) ELISA Kit".

Ethical Consideration: The study was done after approval from the Ethical Committee Faculty of Medicine, Benha University Hospitals (approval code: RC 12-5-2019). All cases provided written informed consents prior to blood sample collection (patients and normal volunteers in the control group). This study was conducted in accordance with the Helsinki declaration.

#### Statistical methods

The statistical analysis and management of data were conducted utilising SPSS version 28 (Armonk, New York, United States of America: IBM). The normality of quantitative data was evaluated by employing the Shapiro-Wilk test for controls and the Kolmogorov–Smirnov test for cases in addition to utilising direct data visualisation techniques (for both). In accordance with the results of tests for normality, numerical data were condensed into medians and ranges or means and standard deviations. Numerical and percentage summaries were compiled for the categorical data. Comparing quantitative data between study groups required the use of the Mann-Whitney U test for non-normally distributed numerical variables and the independent t-test for normally distributed numerical variables. Using the Chi-square test, categorical data were compared. A ROC analysis was performed in order to distinguish between controls and cases using A-FABP. The optimal cut-off point, Area under curve (AUC) with 95% confidence interval, and diagnostic indices were computed. Spearman's correlation was employed to establish correlations.

Various parameters of A-FABP were compared through the utilisation of Mann-Whitney U test. An analysis of logistic regression was performed to forecast androgenic alopecia. The odds ratio and confidence interval with a 95% degree of accuracy were computed. Each statistical test had a dual-sided design. P values  $\leq 0.05$  were deemed to be statistically significant.

#### RESULTS

There were non-significant differences between patients and control group regarding age (P = 0.194) and body mass index (P = 0.057) (Table 1)

**Table** (1): General characteristics in patients and control group

			( <b>n</b> = 20)		
Age (vears)	an ± SD	32 ±8	29 ±9	0.194	
Body Mass Me	ean ±	$27.64 \pm$	25.7 ±	0.057	
Index S	SD	3.96	3.68	0.037	

Independent t-test was used.

The average AGA onset age was  $23 \pm 7$  years. The median disease duration was 9 years and ranged from 1 to 27 years. About patients, three-quarters showed progressive course (76.7%), and one-quarter showed stationary course (23.3%). About one-quarter (26.7%) received previous treatment. All patients showed positive family history. Regarding Hamilton-Norwood Grade, the most frequent grade was grade I (40.0%), followed by grade II (33.3%), while the least frequent grade was grade V (5.0%) (Table 2).

Tuble (2). Chillear characteristics in patients						
Clinical characteristics						
Age of AGA onset (years)	Mean $\pm$ SD	$23\pm7$				
AGA duration	Median (range)	9 (1 - 27)				
Course	Stationary n (%)	14 (23.3)				
	Progressive n (%)	46 (76.7)				
Previous Treatment	n (%)	16 (26.7)				
Positive family History	n (%)	60 (100.0)				
Hamilton – Norwood Grade	I n (%)	24 (40.0)				
	II n (%)	20 (33.3)				
	III n (%)	7 (11.7)				
	IV n (%)	6 (10.0)				
	V n (%)	3 (5.0)				

<b>Table (2):</b> Clinical characteristics in patients	Table (2)	Clinical	characteristics	in	patients	
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The A-FABP was measured in serum of all participants. AFABP was higher significantly in AGA patients than controls.

#### DISCUSSION

Serum levels of A-FABP in individuals with AGA and their putative link to the severity of AGA and other metabolic diseases were the focus of this investigation.

In this study, no statistically significant difference was observed in BMI between the patients and the control group. BMI values did not differ significantly between AGA patients and controls, which is in accordance with the findings of **Arias-Santiago** *et al.*<sup>[6]</sup> and **Mumcuoglu** *et al.*<sup>[7]</sup>. There were no significant variations in BMI between the AGA group and the control group in the research by **Ozbas Gok** *et al.*<sup>[8]</sup>.

When compared to **Bakry** *et al.* <sup>[9]</sup>, who evaluated androgenetic alopecia as well as metabolic syndrome and insulin resistance, our study found no correlation. When it came to BMI, there were substantial differences between the patients and the controls. Because majority of the patients evaluated by **Banger** *et al.* <sup>[10]</sup> were diabetic, they discovered that AGA patients had a higher BMI than controls. **Banger** *et al.* <sup>[10]</sup> reported that participants had a considerably higher family history of AGA in comparison with controls in the present investigation, and this was confirmed in the current analysis. Patients with AGA had a considerably greater level of A-FABP than those without the condition.

There was a strong association between A-FABP and Hamilton-Norwood Grade (r = 0.842 & P 0.001) in the present research. While, no statistically significant association was observed between A-FABP and age, BMI and age of disease onset, or disease duration.

Involvement of FABP's in AGA has not been widely researched in published literatures, as far as we know. In contrast, A-FABP was strongly associated to insulin resistance (IR), obesity, type 2 diabetes, atherosclerosis, hypertension, and cardiovascular disease. Research has demonstrated that individuals diagnosed with type 2 diabetes mellitus and obesity exhibit serum adipocyte fatty acid binding protein (FABP) levels that are 2.5 times higher in magnitude compared to individuals without diabetes. Additionally, an association was observed between FABP levels and BMI, blood glucose, and insulin levels. It was observed that FABP and HDL levels exhibited an inverse association [11].

Apolipoprotein B/apolipoprotein A1 ratio (A-FABP/A1 ratio) and serum adipocyte fatty acid binding protein (A-FABP) in dyslipidemic patients were shown to be associated with the common carotid artery intima media thickness (IMT) in cases with metabolic syndrome. Atherosclerosis and maybe metabolic syndrome may be caused by A-FABP's <sup>[12, 13]</sup>.

Because FABPs are crucial for fatty acid metabolism, **Zhang** *et al.* <sup>[14]</sup> hypothesised that FABPs

may have a key role in controlling obesity-induced inflammasome signaling. It has been claimed that lipid homeostasis may be disrupted, which may contribute to organelle stress.

Peroxisomes, endoplasmic reticulum, and mitochondria are all involved in the metabolism of lipids <sup>[15]</sup>. **Todd** *et al.* <sup>[16]</sup>, stated that organelle stress in hair follicle cells may result in the production of reactive oxygen species by the endoplasmic reticulum and mitochondria, thereby inciting inflammatory signaling cascades, oxidative damage, and stress. If the balance between endoplasmic reticulum (ER) and mitochondrial homeostasis in the hair follicle is not restored, apoptosis may ensue.

Perifollicular inflammation in the hair follicles top third has been shown to be a pathogenic factor in AGA, even though the illness is considered noninflammatory clinically. Patients with androgenetic alopecia have dermal papilla cells that have been shown to have high levels of oxidative stress, which is associated to inflammation in biological systems<sup>[17]</sup>.

# CONCLUSION

A-FABP may have a role in the pathophysiology of AGA, according to our findings. A biomarker for early illness detection with a high specificity, accuracy rate, and sensitivity might also be examined.

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