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Insulin Resistance in Patients with Androgenic Alpoecia

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Abstract
Background: Androgenic Up to half of men and females are susceptible to androgenetic alopecia (AGA), a genetically determined condition caused by an overreaction to androgens. The loss of scalp hair in male-pattern baldness is related to androgen metabolism and the hair development cycle, both of which insulin is thought to regulate (Nabaie et al., 2009). We found significant differences in insulin, glucose, and HOMA-IR between the patients and controls when we defined hyperinsulinaemia as a fasting serum insulin level of > 10 U/mL. Androgenic alopecia patients had elevated insulin, glucose, and HOMA-IR compared to healthy individuals. This shows that individuals with androgenic alopecia may have unique metabolic profiles, which points to possible underlying mechanisms in the aetiology and evolution of AGA. Based on our findings, we know that HOMA-IR is positively correlated with insulin levels, and that a higher HOMA-IR is strongly linked to being female.

Keywords: Androgenic alopecia , Insulin resistance

1.Introduction

Androgenic Alopecia areata (AGA) is the gradual thinning out of scalp terminal hair that may occur at any moment after puberty and follows a distinct pattern in men and women. In men, thinning hair is more noticeable at the vertex and fronto-temporal areas, whereas in women, diffuse apical hair loss manifests as a larger anterior section of the hair and seldom affects the frontal hairline. (1)

Clinically, insulin resistance is defined as the inability of a fixed dose of exogenous or endogenous insulin to stimulate glucose uptake and utilisation to the same extent as it does in a normal population. It reduces glucose synthesis in the liver and impairs insulin's ability to promote glucose transport in skeletal muscle and adipose tissue. The pancreas also generates much more insulin than usual in response to the IR. Hyperinsulinemia causes an increase in sex hormone binding globulin (SHBG) levels, which in turn causes an increase in luteinizing hormone (LH) and follicle stimulating hormone (FSH), which in turn causes an increase in ovarian androgen production and also in their biologically active portion, which may lead to hyperandrogenism (2).

AGA may be caused by hyperinsulinemia brought on by insulin resistance. Subclinical chronic inflammation best describes IR. Hair follicles vulnerable to androgenetic alopecia (AGA) might experience progressive follicular shrinkage and loss as a result of IR, which can reduce Sex Hormone Binding Globulin (SHBG) concentrations by increasing free androgen levels. A study by Wu et al.

A spectrum of disorders sharing a similar phenotypic make up hyperandrogenism, a prevalent endocrine ailment affecting women of reproductive age (5-10% prevalence). Polycystic ovarian syndrome is the most common ailment associated with hyperandrogenism (PCOS). Linked closely to IR, its incidence is 80-85% in women with androgen excess. The wide clinical scenario associated with IR and hyperinsulinemia is highlighted by the signs of hyperandrogenism, which include hirsutism, alopecia, seborrhea, acne, and, in extreme cases, signs of virilization (deepening of the voice, increased muscle mass, clitoromegaly, decreased breast size, and amenorrhea)

. Although the glucose-insulin connection is significant for clinical purposes when assessing IR and hyperinsulinemia, it is also vital to remember that in theory, IR reacts to stimuli other from glucose metabolism (4).

Increasing free androgen levels that operate on AGA-susceptible hair follicles causes gradual follicular shrinkage and hair loss, and IR may reduce Sex Hormone Binding Globulin (SHBG) concentrations, leading to these effects. When administered to those with systemic disorders like hypertension, diabetes, or multiple sclerosis, or those who suffer from polycystic ovarian syndrome, adiponectin has been shown to improve IR and decrease blood glucose levels.

2.Subjects and method

Sixty individuals with moderate to severe androgenetic alopecia were participated in this trial (AGA). Also included was a control group of 40 people who were similar to the study participants in terms of age, gender, and BMI. The patients were recruited at random from the Benha University Hospitals' Dermatology, Venereology, and Andrology outpatient clinic. All participants provided written informed

permission, and the study was approved by the Benha Faculty of Medicine's local ethics committee for research involving human beings (Ms.6.2016).

Each patient had a full medical history taken as part of a thorough clinical examination. This data included demographics, medical history, and an exhaustive account of the patient's experience with androgenetic alopecia and any other skin disorders or medications that may have been significant.

In addition, a complete clinical evaluation was carried out. The body mass index (BMI) was calculated using the following formula: weight (kg) / [height (m) × height (m)], in accordance with World Health Organization (WHO) standards from 2004. Obesity was defined as a body mass index (BMI) of 30 or above, while underweight was defined as a BMI of 18 or below.

In order to determine the clinical type and severity of androgenetic alopecia in each patient, a thorough cutaneous assessment was performed in addition to the general examination.

Patients of both sexes with AGA of varied degrees of severity met the inclusion criteria for the study.

On the other side, exclusion criteria were used to keep the research population consistent. Those with a body mass index (BMI) of 30 or more, those with other autoimmune or inflammatory skin problems, those with other causes of hair loss, pregnant or breastfeeding women, and those with chronic diseases were not included.

Serum insulin levels and fasting blood glucose were measured as part of the laboratory tests given to all participants. Each participant had a clean venipuncture performed using a disposable plastic syringe to draw three millilitres of blood. The blood samples were centrifuged at 1500 rpm for 15 minutes after being put in plain tubes without anticoagulants and allowed to clot at room temperature for 30 minutes. The resultant serum has been frozen and will be tested at a later date.

To determine insulin concentrations, we used a commercially available insulin quantitative ELISA Kit intended for clinical diagnosis (Cat #: E1-072, Immunospec, USA).

Blood glucose levels were tested using the spectrophotometric technique of measuring a red hue at a wavelength of 546 nm, which is the product of an oxidation reaction involving glucose oxidase.

3. Results and discussion

Table (1) Demographic and Anthropometric Data

Variable		**Cases (no.=60)**		**Controls (no.=40)**		**Test**	**P**
		No.	**%**	**No.**	**%**		
Sex	**Female**	30	50.0	24	60.0	$X^2 = 0.97$	0.33
	Male	30	50.0	16	40.0		
		Mean ±SD	**Range**	**Mean ±SD**	**Range**		
Age (years)		25.83±3.06	20-30	24.95±2.13	22-31	t= 1.70	0.09
BMI (kg/m^2)		22.55±1.37	19.5-24.6	22.14±1.14	18.9-23.5	t= 1.56	0.121

Body mass index = BMI; count = number; Definition of "SD" Case-specific clinical data : The average beginning age of sickness among the 26 individuals analysed was (23.383.94) years. Average illness lasted 2.47 years on average. Only 43.3% showed an improving trend, while 56.7% declined. Eighty-three point three percent (50) of individuals with this illness had a favourable family history (Table 1).

Table (2) Present history among all studied cases.

Variable		**Cases (no.=60)**	
		Mean±SD	**Rane**
		No.	**%**
	Age of onset (years)	23.38±3.94	11-29
	Duration (years)	2.47±2.5	0.5-14
Course	Progressive	26	43.33
	Regressive	34	56.67
Family history	Yes	50	83.33

Table (3) laboratory findings of the studied groups.

Variable	Cases (no.=60) Mean ±SD	Range	Controls (no.=40) Mean ±SD	Range	Test	*P*
Insulin (µu/ml)	18.5±3.4	15-35	12.21±6.58	4.18-13	Z= 3.24	0.001 (HS)
Fasting blood glucose(mg/dl)	120.23±6.48	110-130	86.175±8.89	70-102	t=27.91	<0.001* (HS)
HOMA-IR	3.55±2.21	2.5-12	1.01±0.31	0.51-1	t=3.07	0.001 (HS)

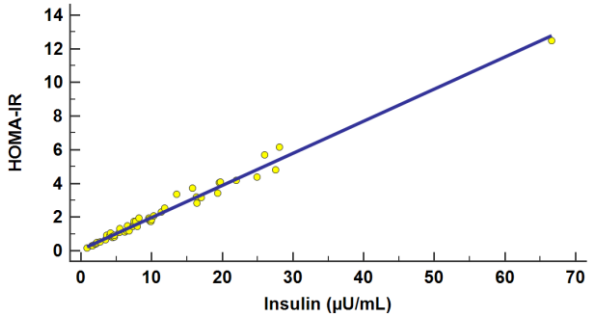

Fig. (1) Insulin levels correlate with HOMA-IR

There were substantial positive connections between HOMA-IR and insulin levels (P 0.001) across the variables tested (Table3 and figure1).
Women's HOMA-IR levels were noticeably greater than men's (Table 1)
On the other hand, we discovered no statistically significant associations between HOMA-IR and calculated parameters across instances.

Case-specific HOMA-IR correlations with calculated parameters are shown in Table 4.

Variable (no.=60)	HOMA-IR *ρ*	*p*
Age	0.224	0.086
BMI	-0.055	0.675
Age of onset	0.178	0.174
Duration	-0.015	0.908
SBP	-0.122	0.353
DBP	-0.172	0.188
Insulin	0.989	<0.001 (VHS)
Glucose	0.230	0.078

Table (5) Association of HOMA-IR levels with other parameters among studied cases.

Variable		HOMA-IR No.	Mean	±SD	Range	Test	*P*
Sex	**Male**	30	1.9	3.0	0.15-12.5	Z=2.1	0.036 (S)
	Female	30	2.2	1.7	0.48-6.18		
Course	**Progressive**	26	2.1	1.8	0.48-6.18	Z=1.3	0.199
	Regressive	34	2.0	2.9	0.15-12.5		
Family history	**No**	10	4.0	4.7	0.52-12.5	Z=1.3	0.190
	Yes	50	1.7	1.5	0.15-6.18		

We found significant differences in insulin, glucose, and HOMA-IR between the patients and controls when we defined hyperinsulinaemia as a fasting serum insulin level of > 10 U/mL. The insulin, glucose, and HOMA-IR values of patients with androgenic alopecia were greater than those of the controls. This shows that individuals with androgenic alopecia may have unique metabolic profiles, which points to possible underlying mechanisms in the aetiology and evolution of AGA.

Consistent with the present investigation, Bakry et al. found that the mean values of FBS, fasting insulin, and HOMA-IR were significantly different between cases and controls. Specifically, those with AGA had higher levels of fasting blood sugar, fasting insulin, and HOMA-IR than the control group (4). Contrary to the findings of Nabaie et al. (2009), who compared AGA patients and controls for serum fasting insulin level, blood glucose, and insulin resistance and found no significant difference between the groups, their data showed a significant difference (5). Possible explanations for the disparities include different total numbers of AGA patients in each study.

Our findings show a favourable association between HOMA- IR and insulin levels, with a higher HOMA- IR being strongly linked to being female.

Cannarella et al. (17) found that males with early-onset androgenic alopecia had substantially higher insulin levels and HOMA index(indicative of insulin resistance) than controls.

They also found that males with androgenic alopecia at a young age had a little lower glycemic and lipid profile and slightly higher body mass index.

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