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FREQUENCY OF DYSLIPIDEMIA IN MALE PATIENTS PRESENTING WITH ANDROGENIC ALOPECIA AT TERTIARY CARE HOSPITAL, KARACHI

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ABSTRACT

Because androgenetic alopecia is the most common cause of hair loss in men, it is sometimes referred to as "Common baldness." The disease has a polygenic inheritance pattern, and the main pathogenic event is the miniaturization of hair follicles. Androgen levels are often normal, indicating a role for end organ hyperreactivity to androgen, but androgenetic alopecia requires at least physiological amounts of circulating androgens to occur.

Objective: To determine the frequency of dyslipidemia in male patients presenting with androgenic alopecia at Dr. Ruth K.M Pfau Civil Hospital, Karachi.

Place and Duration: This Cross-Sectional Study was held in the Department of Dermatology, Dr. Ruth K.M Pfau Civil Hospital, Karachi, Pakistan for Six months after the approval of synopsis from May 31, 2021 to November 30, 2021.

METHODS: All patients who met the criteria of inclusion and visited Civil Hospital, Karachi were studied. After discussing the study's process, hazards, and benefits, informed consent was obtained. The blood sample was taken sterilely after a 12-hour overnight fast and labeled for dyslipidemia testing at a hospital laboratory. All obtained data were entered into the proforma at the end and used electronically for study.

Results: Mean ± SD of age was 46.9±7.9 years. Diabetes mellitus was documented in 45 (25.6%) patients. Dyslipidemia was found to be in 114 (64.8%) patients.

CONCLUSION: It is to be concluded that dyslipidemia is commonly prevalent in patients presenting with androgenic alopecia. More prospective and well-controlled clinical trials are needed to validate these findings.

Key Words: Androgenic Alopecia, Dyslipidemia, Metabolic Syndrome, Triglyceride

INTRODUCTION

The most prevalent kind of alopecia is AGA, which causes progressive, symmetric, pattern hair loss. A single dominant autosomal gene causes androgenic alopecia, or male pattern baldness, in most males. Genes cause susceptibility to dihydrotestosterone (DHT) in some scalp regions[1-2]. DHT may shorten the hair cycle's growth, or anagen, phase from 3-6 years to weeks or months. This shrinks follicles and produces fewer, finer anagen hairs. DHT synthesis is controlled by 5-alpha reductase. Age, genetics, and androgen are other contributors. Hair on the scalp grows around half an inch per month. About 15% of hair grows, while 85% rests. Men with AGA lose hair in two stages: bitemporal retraction of the frontal hair line and diffuse vertex thinning. Central vertex hair loss causes baldness over time. The risk of CAD from dyslipidemia is widely documented. Many studies have linked AGA to cardiovascular disease risk factors, despite its aesthetic nature. These patients may have hypertension, overweight, aberrant lipids, insulin resistance, carotid atheromatosis, diabetes, or elevated heart disease mortality. Other emerging risk variables include serum lipoprotein-a, homocysteine, and adiponectin[3-4]. Liver low-density lipoprotein (LDL) Lp (a) has a lipid core connected to glycoprotein and apo-lipoprotein-A. Its adhesiveness to atheromatous plaque on the arterial wall and inflammatory action via monocyte and macrophage activation suggest it is linked to endothelial dysfunction. Recent investigations have linked AGA to CAD risk, but pathogenesis is unclear[5-6]. Arias-Santiago et al. identified dyslipidemia in 66.2% of androgenic alopecia patients. Human hair is considered the most significant element of the body since it shows personality and confidence[7-8]. The study examines dyslipidemia in androgenic alopecia-afflicted men. Since Pakistani genetics, lifestyle, and diet are distinct from the rest of the globe, there is little local data on this topic. This data will help create dyslipidemia screening strategies for androgenic alopecia patients. Moreover, early dietary modifications, lifestyle changes and development of effective management plan would help in prevention of cardiovascular complications in patients at risk.

METHODS

This Cross-Sectional Study was held in the Department of Dermatology, Dr. Ruth K.M Pfau Civil Hospital, Karachi, Pakistan for Six months after the approval of synopsis from May 31, 2021 to November 30, 2021. The required sample size came out to be 176 patients. By taking prevalence of dyslipidemia in male patients with androgenic alopecia to be 66.2, margin of error=7% and confidence level 'C.I'=95%. This sample size was calculated using the WHO software and sampling technique used was Non-Probability, Consecutive Sampling.

Inclusion Criteria

- Patients who were suffering from androgenic alopecia for more than one year but not treated for hair growth within 6 months were included as per operational definition.
- Male gender.
- Age 20-60 years.

Exclusion Criteria

- Patients with history of alopecia areata, scarring alopecia and psoriasis.
- Patients with history of hormone replacement therapywith testosterone.
- Patients with history of corticosteroids for > 1 month (chronic corticoid therapy).
- Patients underwent treatment with antilipidemic drugs.

▪ Patients with history of stroke, asthma, renal impairment and chronic obstructive pulmonary disease and CCF were excluded.

The College of Physicians and Surgeons Pakistan approved this study. The study included patients from Dr. Ruth KM Pfua's Outpatient Dermatology Department at Civil Hospital, Karachi, who had operationally defined androgenic alopecia. Study was conducted with institutional ethical review committee approval. All patients gave informed consent for sample assignment and research use. The duration of androgenic alopecia and demographic data (age and residency status) were recorded at study entry. Participants' height in meters was measured on a wall-mounted scale and weight to the closest kilogram was measured on a weighing machine At study entry, patients' weight (kg) and height (m) body mass indexes were calculated. After a 12-hour overnight fast, a sterile blood sample was transferred to a hospital-standardized laboratory with correct labeling and the desired examination for free. Operationally, the patients had dyslipidemia. The proforma attached as an annexure contained quantitative variables (age, height, weight, BMI, triglycerides, cholesterol, LDL, HDL, and duration of androgenic alopecia) and qualitative variables (residence status, diabetes mellitus type II, hypertension, smoking status, obesity status, family monthly income status, educational status, occupational status, family history of androgenic alopecia, and dyslipidemia

SPSS Version 20 was used to evaluate demographic data, which included mean and standard deviation for age, height, weight, BMI, triglycerides, cholesterol, LDL, HDL, and androgenic alopecia duration. Data was provided as mean ± SD for normally distributed variables and median (IQR) for non-normal variables. Qualitative variables like residence, diabetes mellitus type II, hypertension, smoking, obesity, family monthly income, educational status, occupational status, family history of androgenic alopecia, and dyslipidemia were calculated as frequencies and percentages. To determine the effect of effect modifiers on the outcome variable (dyslipidemia), age, residence status, diabetes mellitus type II, hypertension, smoking status, obesity status, family monthly income status, educational status, occupational status, family history of androgenic alopecia, and duration were stratified. Following stratification, Chi-square/fisher test was used. $P < 0.05$ was considered significant.

RESULTS

In this study, 176 patients were included to assess the dyslipidemia in male patients presenting with androgenic alopecia at Dr Ruth KM Pfau, Civil Hospital, Karachi and the results were analyzed as:

The distribution of continuous variables was tested by applying Shapiro-Wilk test, age (P=0.061), weight (P=0.128), height (P=0.091), body mass index (P=0.235), triglyceride (P=0.173), cholesterol (P=0.422), LDL (P=0.452), HDL (P=0.117) and duration of androgenic alopecia (P=0.085) as shown in TABLE 1.

TABLE # 1: DESCRIPTIVE STATISTICS FOR DISTRIBUTION OF CONTINUOUS VARIABLE n=176

VARIABLES	MEAN±SD	P-VALUE
Age Group	46.9±7.9	0.061
Weight	67.5±9.7	0.128
Height	1.62±0.7	0.091
Body mass index	26.8±5.9	0.235
Triglyceride	157.8±24.5	0.173
Cholesterol	201.2±30.1	0.422
LDL	125.4±16.9	0.452
HDL	39.7±6.8	0.117
Duration of Androgenic Alopecia	2.3±1.1	0.085
Mean ± SD of age	46.9±7.9	
Mean ± SD of weight	67.5±9.7	
Mean ± SD of height	1.62±0.7	

Mean ± SD of body mass index	26.8±5.9
Mean ± SD of triglycerides	157.8±24.5
Mean ± SD of cholesterol	201.2±30.1
Mean ± SD of LDL	125.4±16.
Mean ± SD of HDL	125.4±16.9
Mean ± SD of HDL	39.7±6.8
Mean ± SD for duration of androgenic alopecia	2.3±1.1

Educational status showed that 9 (5.1%) patients were illiterate, 26 (14.8%) had primary education, 85 (48.3%) had secondary level education while 56 (31.8%) had higher level education as shown in TABLE 2.

TABLE # 2: FREQUENCY OF EDUCATIONAL STATUS n=176

EDUCATIONAL STATUS	FREQUENCY	PERCENTAGE
Illiterate	9	5.1%
Primary	26	14.8%
Secondary	85	48.3%
Higher	56	31.8%

Stratification of age group, diabetes mellitus type II, hypertension, smoking status, obesity status, family monthly income status, educational status, occupational status, family history of androgenic alopecia and duration of androgenic alopecia was done with respect to dyslipidemia in order to assess statistical difference from TABLE [12-22].

TABLE # 3: STRATIFICATION OF AGE GROUP WITH DYSLIPIDEMIA n=176

AGE GROUP [In Years]	DYSLIPIDEMIA		P-VALUE
	Yes	No	
20 – 40	19 (10.8%)	40 (22.7%)	0.0001
>40	95 (54.0%)	22 (12.5%)	

TABLE # 3: STRATIFICATION OF DIABETES MELLITUS TYPE II WITH DYSLIPIDEMIA n=176

DIABETES MELLITUS	DYSLIPIDEMIA		P-VALUE
	Yes	No	
Diabetic	35 (19.9%)	10 (5.7%)	0.034
Non-Diabetic	79 (44.9%)	52 (29.5%)	

TABLE # 4: STRATIFICATION OF HYPERTENSION WITH DYSLIPIDEMIA n=176

HYPERTENSION	DYSLIPIDEMIA		P-VALUE
	Yes	No	
Hypertensive	87 (49.4%)	16 (9.1%)	0.0001
Non-Hypertensive	27 (15.3%)	46 (26.1%)	

TABLE # 5: STRATIFICATION OF SMOKING STATUS WITH DYSLIPIDEMIA n=176

SMOKING STATUS	DYSLIPIDEMIA		P-VALUE
	Yes	No	
Smoker	59 (33.5%)	16 (9.1%)	0.001
Non-Smoker	55 (31.3%)	46 (26.1%)	

TABLE # 6: STRATIFICATION OF OBESITY STATUS WITH DYSLIPIDEMIA n=176

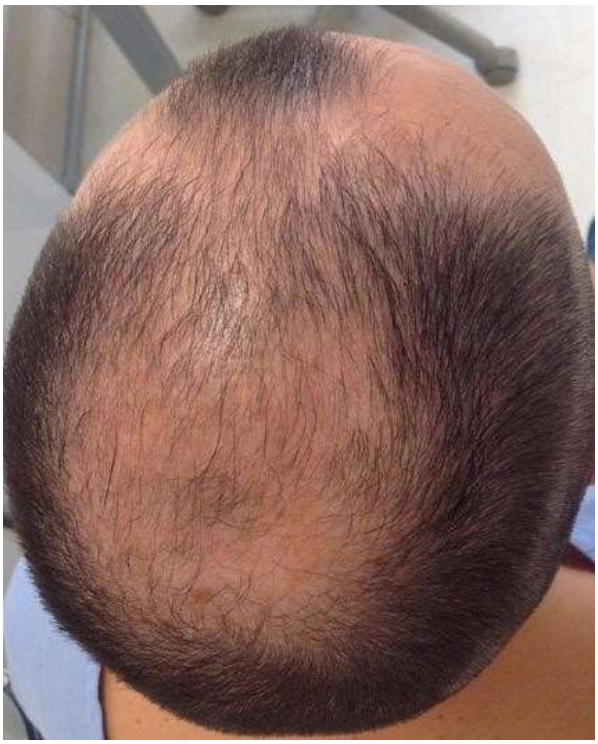
OBESITY STATUS	DYSLIPIDEMIA		P-VALUE
	Yes	No	
Obese	41 (23.3%)	19 (10.8%)	0.477
Non-Obese	73 (41.5%)	43 (24.4%)	

TABLE # 7: STRATIFICATION FOR FAMILY HISTORY OF ANDROGENIC ALOPECIA WITH DYSLIPIDEMIA, n=176

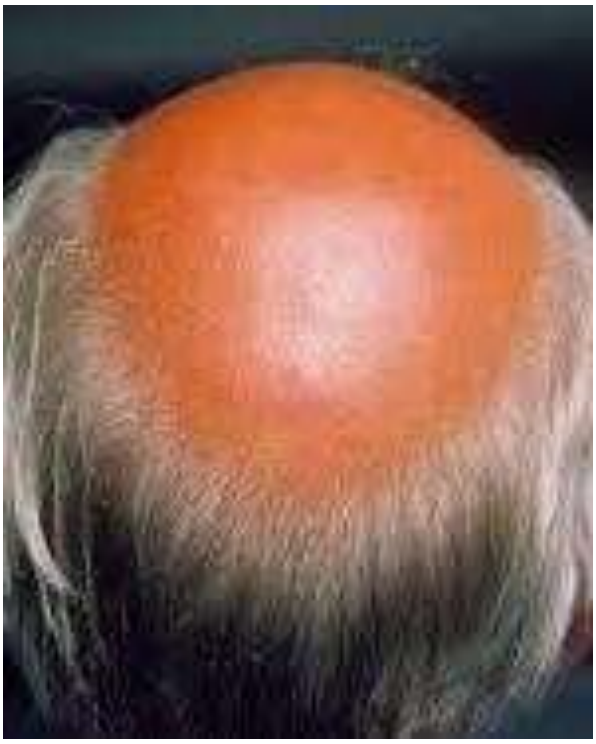
FAMILY HISTORY	DYSLIPIDEMIA		P-VALUE
	Yes	No	
Positive	29 (16.5%)	13 (7.4%)	0.506
Negative	85 (48.3%)	49 (27.8%)	

TABLE # 8: STRATIFICATION FOR DURATION OF ANDROGENIC ALOPECIA WITH DYSLIPIDEMIA, n=176

DURATION [In Years]	DYSLIPIDEMIA		P-VALUE
	Yes	No	
1 – 3	104 (59.1%)	36 (20.5%)	0.0001
>3	10 (5.7%)	26 (14.8%)	



Male-pattern Androgenetic Alopecia with Loss of Hair from Frontal, Temporal and Central Scalp Areas



Male-pattern Androgenetic Alopecia

DISCUSSION

Androgenetic alopecia is diffuse, symmetric, and progressive hair loss in both sexes. It is a polygenic, androgen-dependent autosomal dominant condition[9-10]. The prevalence is 50% in men

and 30% in women. Hair loss begins between 12 and 40, although frequency rises with age. DHT shrinks hair follicles in genetically sensitive people, causing patterned hair loss. In addition to psychological effects, it lets UV radiation to reach the scalp, increasing actinic damage and cardiovascular and prostate hyperplasia risk[11].

The clinical pattern of androgenetic alopecia (AGA) is defined by follicular shrinkage and is caused by systemic androgens and hereditary factors. AGA causes most hair loss in both men and women. Ethnicity affects prevalence[12-13]. White males have it more often and severely than Asian and black guys. The rate rises with age. Hamilton found 30% frequency in men at 30 and 50% at 50. Hair loss usually starts in the 3rd and 4th decades, however it can start after puberty and progress. Men and women have diverse phenotypes[14]. Women have diffuse dilution while preserving the frontal hairline. Hairline draws back from bitemporal areas and vertex baldness occurs in men. Clinical symptoms differ between sexes, although etiology is the same. Environmental variables may worsen AGA, although studies show that overweight and smokers are more likely to have it. Family history was associated with more severe hair loss in both genders and earlier illness onset in males[15-16].

It is unclear how AGA and dyslipidemia are linked. This association may be explained by altered peripheral androgen sensitivity. Five peripheral tissue alpha reductase enzymes convert testosterone to dihydrotestosterone, which has five times the affinity of androgen receptors[17]. The artery wall, blood arteries, and adipose tissue contain five alpha reductase enzymes and androgen receptors. Increased androgen sensitivity may cause AGA and metabolic syndrome symptoms. More research is needed on this topic. Chronic microinflammation is the second explanation for AGA and dyslipidemia. Perifollicular inflammatory infiltration in AGA may be a local sign of metabolic syndrome-causing systemic inflammation. Proinflammatory cytokines alter cholesterol transport and apolipoproteins, causing serum lipid abnormalities[18-19].

Our study found a mean age of 46.9±7.9 years. NA Biquees et al. reported a 46.87±10.58-year age. Another study found an age of 47.1±8.4 years. Rizvi et al. discovered a mean age of 51.8±16.51 years.

Androgenic alopecia (AGA) is the most frequent kind of alopecia in men and women, but its frequency and pathogenic processes have mostly been studied in men. Cotton et al. proposed that male AGA may increase CHD risk[20-21]. Some later research, mostly in male patients, support this link. Only a few studies have examined AGA and CHD in women.

The pathogenetic pathways of atherosclerosis are well understood, however the relationship between alopecia and atherosclerosis is unclear[22-23]. Higher cholesterol and triglycerides contribute to atheromatous plaque along with other processes. HDL-C shields the arterial wall from endothelial adhesion, monocyte migration, etc. and helps reverse cholesterol transport. The poor lipid profile of men and women with AGA may explain its link to CHD[24]. In men with AGA who received coronary artery bypass graft or percutaneous transluminal coronary angioplasty, Matilainen et al. discovered increased LDL-C and triglycerides[25].

A major study demonstrated that total cholesterol/HDL-C ratio is an even better metabolic marker for CHD risk than increased LDL-cholesterol/HDL-cholesterol ratio. Sharrett et al. found that high triglycerides and low HDL-C cause atheroma to become atherothrombosis. These two cardiovascular risk factors must be controlled in subclinical disease patients[26].

CONCLUSION

It is to be concluded that dyslipidemia is commonly prevalent in patients presenting with androgenic alopecia. More prospective and well-controlled clinical trials are needed to validate these findings.

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