

## Age-Related Macular Degeneration in Patients with Androgenetic Alopecia: Could the Monocyte/HDL Ratio Be the Link?

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**ABSTRACT** **Introduction:** Both Androgenetic alopecia (AGA) and age-related macular degeneration (AMD) shared the microinflammatory milieu and increased oxidative stress as important criteria in their pathogenesis. The monocyte/high density lipoprotein (HDL) ratio (MHR) seems to be an easy-to-calculate prognostic marker of microinflammation.

**Objectives:** To assess MHR in patients with AGA and its correlation to AMD in these patients, if any.

**Methods:** Forty patients with AGA aged 40 years or more of both sexes and 40 control subjects participated in this case-control study. General, dermatological, and ophthalmologic examination, MHR evaluation and optical coherence tomography (OCT) were performed.

**Results:** The mean MHR was significantly higher in AGA patients ( $6.98 \pm 2.21$ ) than in controls ( $3.82 \pm 0.68$ ) ( $P < 0.001$ ). AMD was significantly higher in patients than controls ( $P < 0.001$ ). Eighty percent of AGA patients were diagnosed with AMD versus 20% of control subjects. The presence of AMD in AGA was significantly related to the degree of severity of AGA in male patients ( $P = 0.02$ ). The MHR was significantly higher in AGA patients found to have AMD ( $9.37 \pm 1.1$  and  $7.01 \pm 1.42$  in the wet and dry type respectively) than those without AMD ( $P < 0.001$ ).

**Conclusions:** AMD may develop more frequently in those with AGA. The MHR seems to be a missing link between both conditions, and could be utilized as a potential biomarker for predicting AMD in AGA patients.

## Introduction

As the name implies, androgenetic alopecia (AGA) is that kind of hair loss characterized by “miniaturization” of hair follicle and caused mainly by an end-organ hyperresponsiveness to circulating androgens, predetermined by hereditary factors [1]. It is the most prevalent cause of hair loss- despite different clinical patterns- in both genders [2].

Also known as pattern hair loss (PHL), AGA strictly follows distinctive patterns in both sexes. Male pattern hair loss (MPHL) is more noticeable in the vertex and frontotemporal regions, whereas female pattern hair loss (FPHL) often preserves the frontal hairline [3].

Apart from the cosmetic implication, there is growing evidence that AGA may be a part of a systemic microinflammation milieu [4]. Research revealed that AGA patients tend to have an atherogenic lipid profile [5].

Known to release the most abundant pro-inflammatory and pro-oxidant cytokines involved in inflammation cascades, monocytes and macrophages have been largely investigated as the main cells in microinflammation [6]. Furthermore, high-density lipoproteins (HDL) were found to protect endothelial cells from the harmful effects of low-density lipoproteins (LDL) and to inhibit the oxidation of LDL particles. As a result, HDL was considered to have antioxidant and anti-inflammatory properties [7]. Based on these facts, the monocyte count to HDL cholesterol level ratio (MHR) has been identified as a simple-to- calculate prognostic indicator of the amount of inflammation and oxidative stress [7].

The MHR has also been investigated in the course of revealing a clear pathogenesis for age- related macular degeneration (AMD), a major factor contributing to visual loss in the geriatric population [8]. AMD development has been linked to several factors, among which are lipid rich deposits (known as drusen) beneath the retinal pigmented epithelium (RPE) and neovascularization [9]. In fact, AMD can be classified into two distinctive types: dry AMD, which is nonexudative or atrophic, and wet AMD, which is exudative or neovascular. The wet type seems to be linked to more severe visual loss [10].

We attempted to find out whether MHR might imply a potential connection between AGA and AMD, and the association between both conditions, if any.

## Methods

This case control study involved 80 individuals of both genders, within two equal groups (40 each). The first group (N = 40) were patients suffering from clinically-diagnosed and trichoscopically-confirmed AGA with a mean age of  $58.75 \pm 9.8$  years. Forty apparently healthy AGA- free individuals were included in the second group as control subjects with a mean age  $55.65 \pm 12.2$  years. Both groups were age ( $P = 0.381$ ) and gender ( $P = 0.752$ ) matched.

Patients were picked out from those attending the outpatient clinic of Dermatology, Venereology, and Andrology in Benha University Hospitals, Egypt, in the period between May 2021 and December 2021, after obtaining an ethical approval from the Local Ethics Committee (MS:4-5-2021) in line with the Helsinki Declaration fundamentals. Signing an informed consent, age  $\geq 40$  years, and abstinence from systemic medications namely steroids or antihyperlipidemic drugs for 6 months prior to the study were prerequisites for participation.

Subjects with any dermatological diseases other than AGA and lactating or pregnant females were rolled out from the study. subjects with any history of blood disease, systemic inflammatory or infectious disease, or cancer were also excluded.

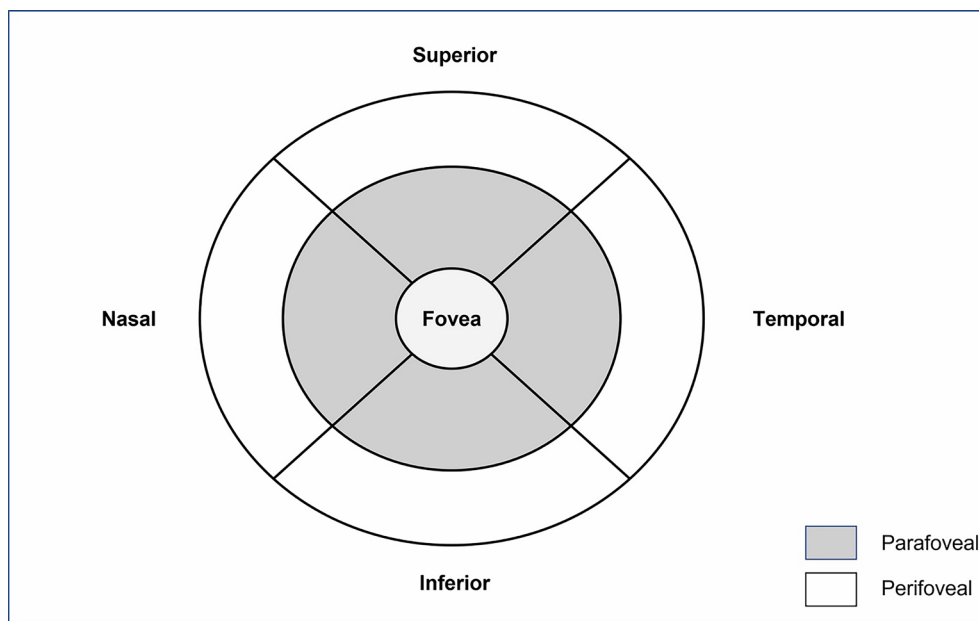
After obtaining a thorough medical history, subjects were generally examined, including for vital signs, and the body mass index (BMI) was measured for each participant using the weight (Kg)/ height<sup>2</sup> (m<sup>2</sup>) formula. The Hamilton-Norwood classification was used to grade MPHL, while the Ludwig scale was utilized in FPHL.

### Ophthalmologic Examination

Slit lamp biomicroscopy using a 90D lens and indirect ophthalmoscopy were performed by an experienced ophthalmologist.

For detection of AMD, optical coherence tomography (OCT) was performed on the macular area to measure foveal thickness, at its central, thickest, and thinnest points both peri- and parafoveal (Figure 1) using the 3D TOPCON® OCT (TOPCON Company).

Before taking the image, mydriatic eye drops were used to dilate the subjects eyes. Throughout the whole OCT



**Figure 1.** Diagrammatic representation of the different regions of the macula assessed by Optical Coherence Tomography.

scanning procedure, patients were told to focus on the intrinsic fixation target. Manual adjusting was done if the patient fixation was not satisfactory, and the image center was not on the fovea. The OCT scans were carried out by an experienced trained ophthalmic technician under the supervision of one of the authors.

The data obtained was evaluated by a qualified retina specialist for the determination of macular thickness and integrity to detect the presence or absence of drusen, foveal, parafoveal, and perifoveal thickness measurements in both eyes (left and right) in order to identify the type of macular degeneration (wet or dry).

### Laboratory Investigations

Estimation of monocyte count and HDL level in venous blood samples was performed for each participant. The monocyte/HDL ratio (MHR) was calculated thereafter.

### Statistical Analysis

The IBM SPSS software programme version 20.0 was used to analyse the data after adding it to the software. The qualitative data were described in terms of numbers and percentages. To confirm that the distribution was normal, the Kolmogorov test was applied. Quantitative data were described using the range (minimum and maximum), mean, standard deviation, median, and interquartile range (IQR) values.  $P < 0.05$  was used as the statistical significance criterion. The tests employed were the chi-square test for categorical variables, the student t-test for quantitative variables that were normally-distributed, the Mann Whitney test for quantitative variables that were

abnormally-distributed, and the comparison between two studied groups. The plotting sensitivity (TP) on the Y axis versus specificity (FP) on the X axis at various cut off levels resulted in the creation of the Receiver Operating Characteristic curve (ROC). The diagnostic effectiveness of the test is indicated by the area under the ROC curve. Areas with performances around 100% are the best for the test, while areas with performances above 50% are considered acceptable. The ROC curve also enables performance comparison between two tests.

## Results

Eighty individuals participated in this case-control study, 40 of them with mean age of  $58.75 \pm 9.8$  years suffering from AGA (the AGA group) and 40 other individuals who were apparently healthy with a mean age group  $55.65 \pm 12.2$  years (serving as a control group). No significant difference was found between the studied groups regarding age ( $P = 0.381$ ), gender ( $P = 0.752$ ), smoking ( $P = 0.288$ ), comorbid hypertension ( $P = 0.311$ ) or occupation ( $P = 0.206$ ). Family history of AGA was positive in 100% of AGA patients and 45% of control subjects. Clinical and anthropometric data of the studied subjects are clarified in Table 1.

Male alopecia was graded by the Hamilton grade (22 patients): grade III and V each was present in 6 patients (27.3%), grade IV and VI each was present in 4 patients (18.2%), while 2 patients (9.1%) had grade VII.

Female alopecia was graded by the Ludwig grade (18 patients): 10 patients (55.6%) had grade III and 8 patients (44.4%) had grade II.

**Table 1. Demographic data of the studied groups.**

Parameter	Groups		Test	
	AGA group N = 40 (%)	Control group N = 40 (%)	t/ $\chi^2$	P
Age (year): Mean $\pm$ SD	58.75 $\pm$ 9.8	55.65 $\pm$ 12.2	0.886	0.381
Gender:				
Female	18 (45%)	20 (50%)	0.1	0.752
Male	22 (55%)	20 (50%)		
Smoking:				
No	26 (65%)	24 (80%)	1.129	0.288
Yes	14 (35%)	16 (20%)		
Occupation:				
Outdoor	24 (60%)	16 (40%)	1.6	0.206
Indoor	16 (40%)	24 (60%)		
Family history:				
Irrelevant	0 (0%)	22 (55%)	15.172	<0.001
Relevant	40 (100%)	18 (45%)		
Hypertension	16 (40%)	10 (25%)	1.026	0.311
Weight (kg)	90.47 $\pm$ 16.73	84.15 $\pm$ 12.39	1.357	0.183
Height (cm)	169.5 $\pm$ 8.26	166.25 $\pm$ 5.68	1.45	0.156
BMI (kg/m <sup>2</sup> )	31.42 $\pm$ 4.84	30.55 $\pm$ 4.97	0.562	0.578

AGA = Aandrogenetic alopecia; BMI = body mass index; SD = standard deviation.

**Table 2. Monocyte counts, HDL levels and their ratio (MHR) among the studied groups.**

Parameter	Groups		Test	
	AGA group Mean $\pm$ SD	Control group Mean $\pm$ SD	t	P
HDL (mg/dl)	42.85 $\pm$ 5.58	53.65 $\pm$ 4.86	4.192	<0.001
Monocytes (per $\mu$ l)	293.0 $\pm$ 87.31	204.0 $\pm$ 37.33	-6.531	<0.001
MHR	6.98 $\pm$ 2.21	3.82 $\pm$ 0.68	6.101	<0.001

AGA = Aandrogenetic alopecia; HDL = high density lipoprotein; MHR = monocyte/ HDL ratio.

All AGA patients (male and female) had a progressive course. The age of disease onset ranged from 25 to 35 years (mean 28.8  $\pm$  2.91 years).

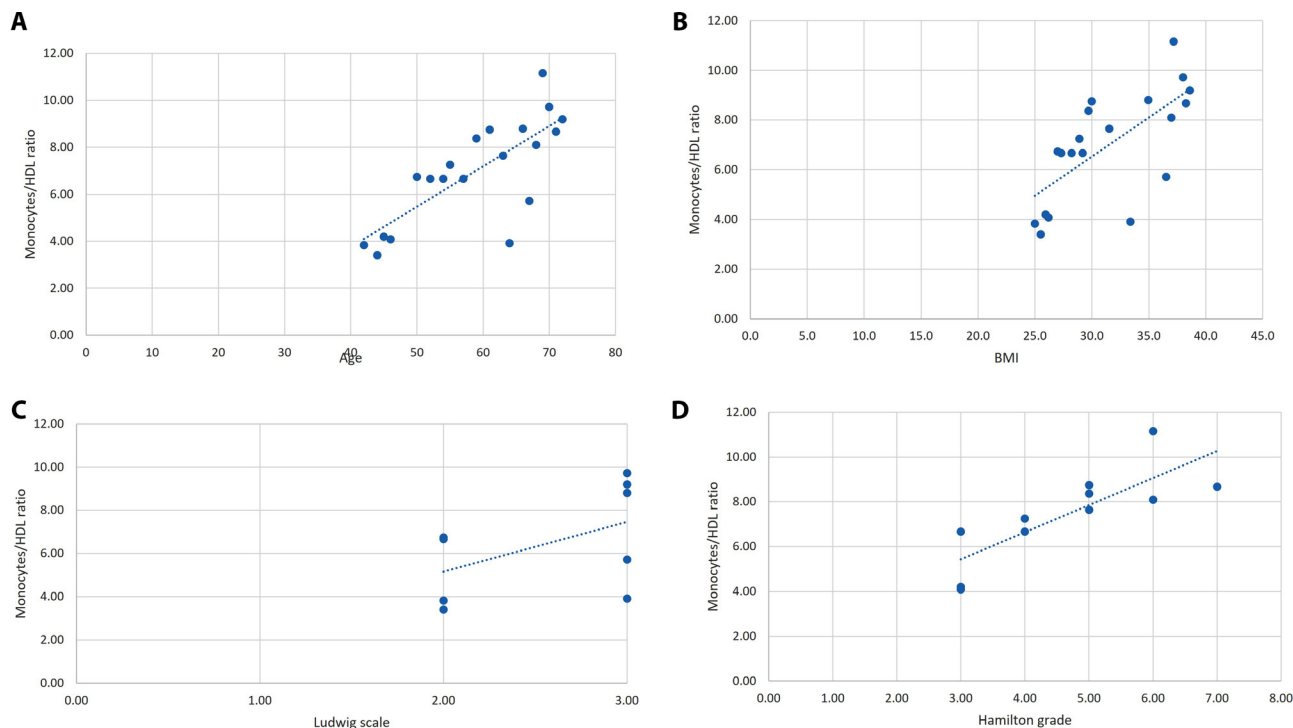
On one hand, monocyte count was significantly higher in AGA patients versus control subjects ( $P < 0.001$ ), and on the other hand, serum HDL was significantly lower ( $P < 0.001$ ). Consequently, the MHR was significantly higher in AGA patients vs. control subjects ( $P < 0.001$ ) (Table 2).

In AGA patients, the MHR correlated significantly and positively with age ( $r = 0.761$ ,  $P < 0.001$ ), BMI ( $r = 0.688$ ,  $P < 0.001$ ), Hamilton grade ( $r = 0.809$ ,  $P < 0.001$ ) and Ludwig grade ( $r = 0.502$ ,  $P < 0.001$ ). No significant difference was found in MHR values between male (7.74  $\pm$  1.76) and female (6.44  $\pm$  2.35) patients ( $P = 0.06$ ) (Figure 2).

Eighty percent of AGA patients met the OCT diagnostic criteria for AMD (22 patients with dry-type, and 10 with

wet-type) versus 20% of the control group (all of them were with wet-type). The presence of AGA increases the risk of AMD by 32-folds when compared with the control group and increases the risk of wet-type AMD by 10-folds (Table 3). Supplementary tables show the significant differences between the OCT findings in both AGA group and control subjects (Tables S1 and S2).

A significant difference was found in the monocyte count, HDL levels and consequently the MHR between patients diagnosed with AMD and those without within the AGA group, with the highest significant difference noted among AGA patients diagnosed with wet-type AMD (Table 4). There was also a significant association between weight – and BMI- on one hand and presence and type of AMD on the other hand, with significantly higher values within the wet-type AMD patients (Table 4).



**Figure 2.** (A-D) Scatter dot graphs showing significant positive correlations between monocyte/HDL ratio (MHR) and Age (A), Body mass index (BMI, B), The Ludwig grade (C), The Hamilton grade in patients with androgenetic alopecia (D).

**Table 3.** Presence of age-related macular degeneration (AMD) among the studied groups.

Parameter	Groups		Test	
	AGA group N = 40 (%)	Control group N = 40 (%)	$\chi^2$	P
Type of AMD:				
Dry	22 (55%)	0 (0%)	18.113	<0.001
Wet	10 (25%)	8 (20%)		
Normal	8 (20%)	32 (80%)		
COR (95% CI)	32 (3.4 – 75.35) (AMD)			<0.001
COR (95% CI)	10 (0.9 – 27.69) (wet AMD)			0.056

AGA = Androgenetic alopecia; AMD = age-related macular degeneration; CI = confidence interval; COR = crude odds ratio.

Furthermore, the presence and type of AMD were significantly related to the grade of AGA in males ( $P = 0.02$ ), with patients diagnosed with wet-type AMD having the highest grades of AGA (66.7% of them were grade VI and 33.3% were grade VII). On the other hand, no significant association was found with the severity of AGA in females as assessed by Ludwig scale ( $P = 0.058$ ). The age of onset of AGA was not related to the presence or type of AMD in affected patients ( $P = 0.556$ ).

significant relation was found between the presence and type of AMD on one hand and age ( $P < 0.001$ ), smoking ( $P = 0.009$ ), or occupation ( $P = 0.024$ ) on the other hand. Wet-type AMD was found to be associated with older age, smoking and outdoor occupation. Whereas no significant

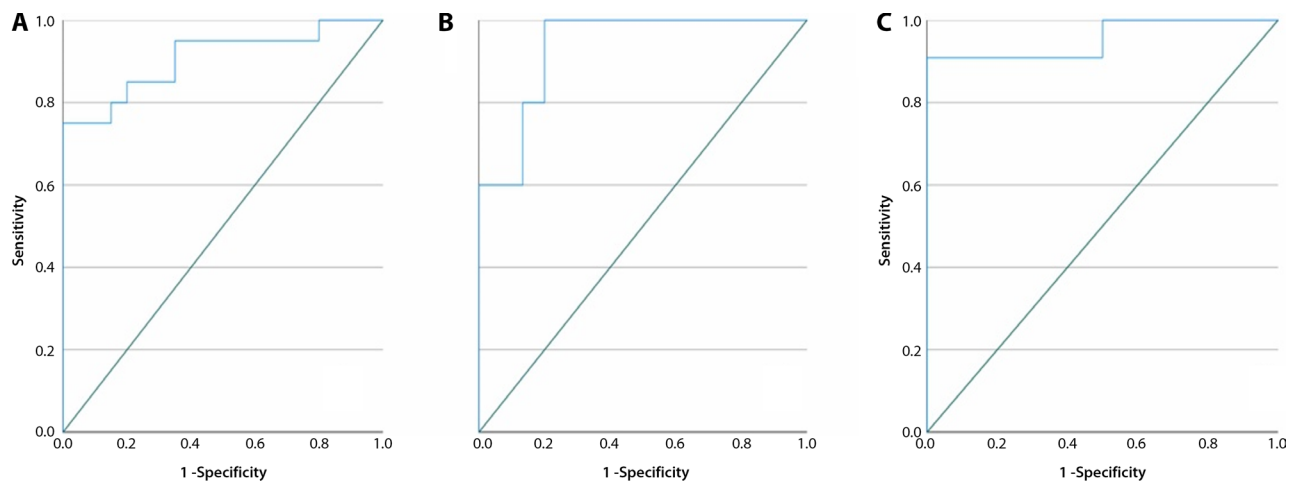
association was detected between presence and type of AMD and gender ( $P = 0.955$ ).

Finally, ROC curve analyses were performed. In the prediction of AGA, the best cutoff value of the MHR was  $\geq 4.041$  with an area under the curve of 0.907, sensitivity 85%, specificity 80%, positive predictive value (PPV) 81%, negative predictive value (NPV) 84.2% and an overall accuracy of 82.5%. The best cutoff value of the MHR in the prediction of wet-type AMD among AGA patients was  $\geq 8.2337$  with an area under the curve of 0.933, sensitivity 80%, specificity 80%, PPV 57.1%, NPV 92.3% and an overall accuracy of 80%. While in prediction of dry-type AMD among AGA patients, the best cutoff of MHR is  $\geq 4.1408$  to  $< 8.2337$  with area under curve 0.955, sensitivity 90.9%,

**Table 4.** Relation between presence and type of age-related macular degeneration (AMD) with monocyte/HDL ratio (MHR), weight, height, and body mass index (BMI) among androgenetic alopecia (AGA) patients.

Parameter	AMD			Test	
	No N = 8	Dry N = 22	Wet N = 10	F	P
	Mean ± SD	Mean ± SD	Mean ± SD		
Monocytes (per µl)	190.0 ± 18.26	290.91 ± 70.21	380.0 ± 26.65	10.587	0.001
Tukey-HSD	P <sub>1</sub> 0.03*	P <sub>2</sub> 0.04	P <sub>3</sub> <0.001		
HDL (mg/dl)	49.0 ± 1.41	41.64 ± 5.75	40.6 ± 3.91	4.12	0.035
Tukey-HSD	P <sub>1</sub> 0.046	P <sub>2</sub> 0.917	P <sub>3</sub> 0.048		
MHR	3.88 ± 1.42	7.01 ± 1.42	9.37 ± 1.1	21.809	<0.001
Tukey- HSD	P <sub>1</sub> <0.001	P <sub>2</sub> 0.007*	P <sub>3</sub> <0.001		
Weight	74.88 ± 9.98	85.64 ± 8.21	113.56 ± 10.59	23.284	<0.001
Tukey HSD	P <sub>1</sub> 0.139	P <sub>2</sub> <0.001	P <sub>3</sub> <0.001		
Height	170.5 ± 9.98	167.45 ± 7.38	173.2 ± 9.12	0.856	0.442
BMI	25.68 ± 0.54	30.61 ± 3.14	37.82 ± 0.69	28.885	<0.001
Tukey HSD	P <sub>1</sub> 0.008	P <sub>2</sub> <0.001	P <sub>3</sub> <0.001		

AMD = age-related macular degeneration; BMI = body mass index; F = One way ANOVA test; HSD = highest significant difference; P<sub>1</sub> = difference between normal and dry AMD; P<sub>2</sub> = difference between dry-type AMD and wet-type AMD; P<sub>3</sub> = difference between wet AMD and normal SD standard deviation.



**Figure 3.** (A-C) Receiver operator characteristics (ROC) curve showing performance of monocytes/HDL ratio (MHR) in the prediction of: Androgenetic alopecia (A), wet-type age-related macular degeneration (B), Dry-type age-related macular degeneration (C).

specificity 7%, PPV 90.9%, NPV 75% and overall accuracy 86.7% (Figure 3).

## Conclusions

The unclear etiopathogenesis is a common feature of both AGA and AMD. Sharing other characteristics, including microinflammatory milieu and increased oxidative stress, makes the potential connection between both diseases likely.

In this case-control study, the MHR was significantly higher in AGA patients versus control subjects ( $P < 0.001$ ).

To date, no data has been published investigating the MHR as an easy-to-calculate prognostic marker in patients with AGA. The MHR as an indicator of inflammation, depends on the pro-inflammatory action of monocytes vs. the anti-inflammatory effect of HDL. Monocyte activation and differentiation into lipid-laden macrophages play a key role in the enhancement of immunological responses in patients with a chronic inflammatory status [11]. On the other hand, HDL was found to counteract the migration of macrophages and deprive them from lipids, thereby reversing the process of lipid deposition in the vessel wall [12]. The MHR had



been investigated as an indicator of systemic inflammation in cardiovascular disease [7]. It is now thought that miniaturization of hair follicles in AGA is a result of an indolent, painless, silent inflammatory process, given the term "micro-inflammation" to be differentiated from the usual inflammatory process [4]. Dyslipidemia in AGA patients has been studied in multiplicity of research, with proved decreased HDL levels in AGA patients in most controlled studies [5]. However, the exact explanation of this finding is yet to be identified. A compromised microvasculature of the dermal papillae of bald scalp was suggested as a possible contributing mechanism to follicle miniaturization, a mechanism similar to atherosclerosis in cardiovascular disease (CVD) linking both conditions [13] and supporting the growing evidence of an atherogenic lipid profile in AGA patients [5].

Numerous studies investigated the MHR as a prognostic marker in various conditions. Bolayir and colleagues found the MHR to be significantly higher in acute stroke patients than healthy individuals and reported the ratio as a significant mortality variable in such patients [14]. According to Karatas et al, the MHR in diabetic nephropathy patients was considerably greater than in normoalbuminuric diabetic patients and healthy controls [15]. Furthermore, a significantly higher MHR was recorded in vitiligo patients in comparison to vitiligo-free controls and was significantly correlated with the severity of the disease as evaluated by the vitiligo extent tensivity index (VETI) score [16]. Moreover, a study by Acikgoz and colleagues detected a significant correlation between the MHR and vascular involvement in patients with Behcet disease, suggesting endothelial dysfunction as a possible mechanism [17]. The MHR was also suggested as an indicator of the systemic inflammatory process in psoriatic patients that correlates with severity of psoriasis as evaluated by The Psoriasis Area and Severity Index (PASI) [18].

In the present work, the MHR correlated positively with severity of AGA as assessed by the Hamilton grade ( $r = 0.809$ ,  $P < 0.001$ ) and the Ludwig grade ( $r = 0.502$ ,  $P < 0.001$ ). Consequently, the ratio may be utilized as a biomarker for AGA severity in both genders.

We also reported a significant positive relation between the MHR and BMI. This is hardly distinguishable from the findings of Usta and colleagues as they reported significantly higher MHR values in obese versus lean subjects when they were studying Polycystic Ovary Syndrome (PCO) patients [11]. This finding supports the hypothesis that obesity is a sub-clinical chronic inflammatory status, where the adipose tissue is invaded by numerous inflammatory cell populations that release cytokines, producing a low-level inflammatory milieu [19].

Among the AGA patients indulged in this study, 32 patients were diagnosed with AMD (both dry and wet types) versus 10 in the controls ( $P < 0.001$ ). Based on the statistical

analysis of the present work data, presence of AGA increases the risk of AMD by 32-folds when compared with control subjects (Table 3). The MHR was found to be significantly higher in AGA patients diagnosed with AMD vs. those who were not ( $P < 0.001$ ). In fact, the MHR has been investigated as a biomarker of systemic inflammation in AMD [20].

In line with the results of the current work, Kucuk and colleagues linked the significantly higher values of MHR to the wet-type AMD [20]. According to reports, monocytes are crucial in the development of the lipid-rich drusen [21] and neovascularization [22] (a key step in development of wet-type AMD), while elevated HDL levels were proven to be protective against development of neovascularization in AMD [23]. Consequently, MHR represents an indicator of the pro/anti-inflammatory balance status in such patients. Given that the composition and pathogenesis of cholesterol-rich subretinal drusen deposits resemble that of atherosclerotic plaques [24], the atherogenic lipid profile increasingly reported in AGA patients [5], and the increased comorbid cardiovascular risk in AGA patients [13], a link among these conditions could be suggested.

The association between sex hormones and development of soft drusen, hence AMD had been investigated earlier in multiple research articles [25,26]. Shin and colleagues suggested increased incidence of macular abnormalities with the use of antiandrogens and stated that the mechanism is not yet well-understood [26]. However, they did reported hair loss in their patients, therefore further investigations are needed to clarify whether the macular abnormalities are associated with the use of anti-androgens, or with hair loss.

Owing to the results of the present research, the AGA male patients diagnosed with wet-type AMD were found to have more severe AGA, making the association between both conditions more plausible. The relatively small size of the included patient samples limits the generalizability of the findings of the present work.

Taken together, the findings of this research suggest that AGA could be associated with increased risk of AMD, the wet-type in particular. We assert that the MHR could represent a potential link between the two diseases and could be a target for future research.

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