Glutathione S-transferase gene polymorphism, total antioxidant status, and blood pressure changes in androgenic alopecia

Androjenik alopeside glutatyon S-transferaz gen polimorfizmi, total antioksidan kapasite ve kan basıncı değişiklikleri

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Abstract

Aim: To investigate the relationship of glutathione S-transferase gene polymorphism with total antioxidant capacity and blood pressure changes in patients with androgenic alopecia.

Methods: Hamilton-Norwood classification was used for the diagnosis and staging of androgenic alopecia (AGA), and all individuals were evaluated by the same physician. Family history of AGA was questioned; body mass index (BMI), lipid profile, blood pressure (BP) levels, total oxidative stress (TOS), total antioxidant status (TAS), and glutathione S-transferase (GST) gene polymorphisms were evaluated. Polymerase chain reaction (PCR) and Restriction Fragment Length Polymorphism (RFLP) methods were used to detect GST polymorphisms. Blood pressure of the patients was measured by 24-hour ambulatory blood pressure monitoring.

Results: The study included 43 male patients with a mean age of 35.09 ± 10.51 years and 90.6% of the individuals had a family history of AGA. When TOS, TAS, 24-hour BP measurement results (systolic-diastolic BP values) and dipper/nondipper BP status were compared according to GSTT1, GSTM1, GSTP1 I105V, GSTP1 A114V genotypes, the difference was not significant. However, TOS levels were higher in individuals with GSTP1 A114V heterozygous genotype (polymorphism detected) compared to individuals with the normal genotype. In addition, TAS was lower in individuals with GSTT1 null genotype (deletion in both alleles) compared to individuals with GSTT1 gene. Although the difference was not statistically significant, a positive correlation was observed between androgenic alopecia stages and the oxidative stress index (r=0.14).

Conclusion: GST gene polymorphisms may be associated with increased oxidative stress and decreased total antioxidant capacity in patients with androgenic alopecia. Moreover, oxidative stress appears to increase with the progression of AGA stage.

Keywords: Androgenetic alopecia; blood pressure; glutathione S-transferase; oxidative stress; polymorphism

Öz

Amaç: Androjenik alopesi hastalarında glutatyon S-transferaz gen polimorfizminin total antioksidan kapasite ve kan basıncı değişiklikleri ile ilişkisini araştırmak.

Yöntemler: Androjenik alopesi (AGA) tanısı ve evrelemesi için Hamilton-Norwood sınıflaması kullanıldı ve tüm bireyler aynı doktor tarafından değerlendirildi. Ailede AGA öyküsü sorgulandı; vücut kitle indeksi, lipid profili, kan basıncı düzeyleri, total oksidatif stres (TOS), total antioksidan kapasite (TAS) ve glutatyon S-transferaz (GST) gen polimorfizmleri değerlendirildi. GST polimorfizmlerini saptamak için polimeraz zincir reaksiyonu (PCR) ve Restriksiyon Fragman Uzunluk Polimorfizmi (RFLP) yöntemleri kullanıldı. Hastaların kan basıncı 24 saatlik ambulatuvar kan basıncı takibi ile ölçüldü.

Bulgular: Çalışmaya yaş ortalaması 35.09 ± 10.51 yıl olan 43 erkek hasta dahil edilmiş olup, bireylerin %90.6'sında ailede AGA öyküsü bulunmaktadır. TOS, TAS, 24 saatlik kan basıncı ölçüm sonuçları (sistolik-diyastolik KB değerleri) ve dipper/nondipper KB durumu GSTT1, GSTM1, GSTP1 I105V, GSTP1 A114V genotiplerine göre karşılaştırıldığında fark anlamlı değildi. Ancak, GSTP1 A114V heterozigot genotipine (polimorfizm tespit edilen) sahip bireylerde TOS seviyeleri normal genotipe sahip bireylere kıyasla daha yüksekti. Ayrıca, GSTT1 null genotipine (her iki alelde delesyon) sahip bireylerde TAS, GSTT1 genine sahip bireylere kıyasla daha düşüktü. Androjenik alopesi evreleri oksidatif stres indeksi açısından karşılaştırıldığında, fark istatistiksel olarak anlamlı olmasa da pozitif korelasyon bulunmuştur (r=0,14).

Sonuç: Bulgular, GST gen polimorfizmlerinin androjenik alopesi hastalarında artmış oksidatif stres ve azalmış total antioksidan kapasite ile ilişkili olabileceğini; ayrıca, AGA evresinin ilerlemesiyle oksidatif stres düzeylerinin de artış gösterebileceğini düşündürmektedir.

Anahtar Sözcükler: Androjenik alopesi; glutatyon S-transferaz; kan basıncı; oksidatif stres; polimorfizm

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INTRODUCTION

Androgenic alopecia (AGA) is a disease characterized by androgen-induced hair loss in individuals with a genetic predisposition. In AGA, androgens cause the miniaturization of hair follicles that are genetically predisposed to alopecia. This miniaturization is observed in areas that are more sensitive to androgen effects, such as the frontotemporal region and vertex in males and the top of the head in females (1). In addition to genetic and hormonal factors, micronutrient deficiencies, microinflammation, and psychological stress also contribute to the pathogenesis of AGA (2).

The imbalance between free radical or reactive oxygen species production and protective antioxidant systems is referred to as "oxidative stress". While numerous studies in the literature have explored oxidative stress and GST enzyme levels in androgenic alopecia, the association between GST gene polymorphisms and AGA has not yet been investigated. In a study by Prie et al., decreased TAS and increased malondialdehyde levels in plasma samples of AGA patients were considered indicators of oxidative stress in these individuals (3).

The findings of studies examining the relationship between hypertension and AGA are inconsistent; while some studies have identified a strong association between AGA and hypertension, others have not shown such a relationship (4-6). The association of cardiovascular risk factors such as hypertension, smoking, and high body mass index (BMI) with AGA has been investigated; however, no study has simultaneously evaluated hypertension, oxidative stress, and GST gene polymorphism in AGA.

In this study, we aimed to investigate the association of GST gene polymorphism with total oxidative stress, total antioxidant capacity, and blood pressure in androgenic alopecia.

METHODS

Participitants

This cross-sectional study was conducted at Cumhuriyet University Faculty of Medicine Hospital. Male individuals with androgenic alopecia, including patients who applied to the internal medicine outpatient clinic, their relatives, hospital staff, and medical faculty students, were included in the study. The Hamilton-Norwood classification was used for the diagnosis and grading of androgenic alopecia (Figure 1) (7).

Exclusion criteria included being female, the absence of androgenic alopecia findings, and a known history of chronic diseases such as diabetes mellitus and chronic kidney disease. A detailed medical history was obtained for each participant. The study evaluated age, family history, smoking status, BMI, 24-hour ambulatory blood pressure, GST gene polymorphism, serum total antioxidant capacity, total oxidative stress, serum triglyceride, total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), creatinine, and complete blood count.

Informed consent was obtained from all volunteers, and approval was granted by the Cumhuriyet University Clinical Research Ethics Committee (date: 04.06.2013, decision no: 2013-06/04). This research was funded by the Cumhuriyet University Scientific Research Projects Coordination Unit (project no: T-591).

Total genomic DNA isolation from peripheral blood

Genomic DNA was isolated from peripheral blood samples using the GeneMATRIX Quick Blood DNA Purification Kit (EURX). Peripheral complet blood was collected in EDTA tubes, and both patient and control samples were stored at -20°C until analysis. Total genomic DNA was isolated from each blood sample prior to mutation analysis.

Detection of GSTT1 and M1 genotypes by multiplex PCR

The PCR reaction mixture was prepared in a total volume of 25 µl, consisting of 0.100 µg DNA, 0.25 µmol/l dNTP, 0.4 µmol/l GSTM1 primer, 0.8 µmol/l GSTT1 primer, 0.8 µmol/l albumin primer, 5 µl of 10× buffer, 2 mmol/l $MgCl_2$, and 0.5 U DNA Taq polymerase (Fermentas, Lithuania).

PCR amplification was performed under the following conditions: an initial denaturation at 95°C for 15 minutes; followed by 35 cycles of denaturation at 94°C for 1 minute, annealing at 58°C for 1 minute, and extension at 72°C for 1 minute; and a final extension step at 72°C for 10 minutes. The amplified products were subsequently visualized on a 2% agarose gel.

For each sample, the presence of the albumin band was first confirmed. In samples where the albumin band was present, the presence of GSTT1 and GSTM1 bands indicated that the gene was intact, whereas their absence suggested a deletion.

Determination of GSTP1 Exon 5 I1e105Val and Exon 6 Ala114Val Polymorphisms by PCR-RFLP Method

PCR amplification was performed under the following conditions: an initial denaturation at 94 °C for 5 minutes, followed by five cycles in which the annealing temperature was decreased by 1 °C per cycle (94 °C for 30 seconds, 64 °C for 30 seconds, and 72 °C for 30 seconds). This was followed by an additional 25 cycles at 94 °C for 30 seconds, 59 °C for 30 seconds, and 72 °C for 30 seconds.

The polymerase chain reaction yielded PCR products of 433 bp for exon 5 and 420 bp for exon 6. The exon 5 PCR product was digested with 5 units of BsmAI (Fermentas, Lithuania), and the exon 6 PCR product was digested with Aci1 (Fermentas, Lithuania) restriction endonucleases at 37 °C for 16 hours. Following restriction digestion, the resulting fragments were subjected to electrophoresis on a 3% agarose gel.

Blood pressure measurement

Blood pressures of the patients were measured with a 24-hour ambulatory BP monitor.

Total antioxidant status (TAS)

TAS levels were measured using commercial kits provided by Rel Assay Diagnostics. Trolox, a water-soluble analog of vitamin E, served as the calibration standard, and the results were expressed in mmol Trolox equivalents per liter.

Total oxidative stress (TOS)

TOS levels were determined using commercial kits from Rel Assay Diagnostics. Hydrogen peroxide was employed as the calibration standard, and the results were expressed in micromoles of hydrogen peroxide equivalents per liter (μmol H_2O_2 equiv./L).

Oxidative stress index (OSI)

For the calculation of the OSI, defined as the percentage ratio of TOS to TAS levels, TAS values originally expressed in mmol were converted to μmol to match the units used in the TOS assay. The OSI values were expressed in arbitrary units (AU) and computed using the following formula:

OSI = [TOS (μmol H2O2 Eq/L) / TAS (μmol H2O2 Eq/L)] x 100

Statistical analysis

The data obtained from our study were uploaded to the Statistical Package for the Social Sciences package program version 22.0 (IBM Corp., Armonk, NY, USA). When the assumptions for parametric tests were met (Kolmogorov-Smirnov test), the significance test for the difference between two means and analysis of variance (ANOVA) were applied. When parametric test assumptions were not met, the Mann-Whitney U test, Chi-square test, Kruskal-Wallis test, and the correlation analysis were used. The significance level was set at 0.05.

RESULTS

The study included 43 individuals with androgenic alopecia, with a mean age of 35.09 ± 10.51 years (range: 21–62 years). A family history of androgenic alopecia was present in 39 (90.6%) individuals. Only one participant had a history of coronary artery disease. No significant correlation was found between smoking status or BMI and the stages of androgenic alopecia. However, BMI was elevated (>25 kg/m²) in 26 (60.4%) individuals. The parameters assessed in the study are presented in Table 1.

The GSTT1 gene was present in 30 out of 43 participants (69.8%). Likewise, the GSTM1 gene was found in 27 subjects (62.8%), while 16 (37.2%) lacked this genetic variant.

A normal GSTP1 A114V genotype was observed in 37 participants (86%), while 6 (14%) exhibited a heterozygous polymorphism. No homozygous polymorphism was detected. For the GSTP1 I105V gene, a normal genotype was found in 23 participants (53.5%), whereas 17 (39.5%) showed a heterozygous polymorphism and 3 (7%) a homozygous polymorphism.

Comparing TOS, TAS, and blood pressure parameters between those with and without GSTT1 polymor-

Table 1. Demographic data, laboratory findings, and blood pressure measurements of the participants

	n	Minimum	Maximum	Mean	Standart Deviation
Age (years)	43	21	62	35,09	10,51
BMI (kg/m²)	43	20,14	34,93	25,66	3,22
TOS (μmol/L)	43	1,00	41,00	9,30	8,67
TAS (mmol/L)	43	0,23	1,89	1,45	0,35
OSI (AU)	43	0,11	3,02	0,66	0,62
Triglyceride (mg/dL)	43	52,00	463,00	149.02	85,04
Total cholesterol (mg/dL)	42	113,00	266,00	178.74	32,28
HDL (mg/dL)	43	23,00	60,00	41,16	9,21
LDL (mg/dL)	43	61,00	188,00	112,37	26,41
Creatinine (mg/dL)	43	0,60	1,20	0,89	0,14
WBC (10^3/μL)	42	4,90	12,10	6,90	1,70
Hemoglobin (g/dL)	42	13,60	17,70	16,13	0,88
Platelet (10^3/μL)	42	153,00	332,00	247,48	48,18
Systolic BP (mmHg)	40	99,00	138,00	116,92	8,34
Diastolic BP (mmHg)	40	60,00	97,00	73,65	7,26
Pulse (bpm)	40	58,00	111,00	75,55	10,70

n: Number, BMI: Body Mass Index, TOS: Total Oxidative Stress, TAS: Total Antioxidant Status, OSI: Oxidative Stress Index, AU: Arbitrary Units, HDL: High-Density Lipoprotein, LDL: Low-Density Lipoprotein, WBC: White Blood Cell, BP: Blood Pressure, bpm: beats per minute

Table 2. Comparison of TOS, TAS, systolic, and diastolic blood pressure values with GSTT1

GSTT1	TOS x±S	TAS x±S	Systolic BP x±S	Diastolic BP x±S
Present	9.56 ± 8.11	1.49 ± 0.36	117.22 ± 9.36	73.62 ± 7.75
Null	8.69 ± 10.18	1.34 ± 0.32	116.31 ± 5.96	73.69 ± 6.34
p	0.313	0.112	0.750	0.980

TOS: Total Oxidative Stress, TAS: Total Antioxidant Status, GSTT1: Glutathione S-transferase T1, BP: Blood Pressure

Table 3. Comparing of TOS, TAS, systolic, and diastolic blood pressure values with GSTM1

GSTM1	TOS x±S	TAS x±S	Systolic BP x±S	Diastolic BP x±S
Present	9.11 ± 7.69	1.47 ± 0.36	116.58 ± 9.36	74.57 ± 7.60
Null	9.62 ± 10.39	1.41 ± 0.34	117.57 ± 6.25	71.92 ± 6.50
p	0.659	0.479	0.724	0.277

TOS: Total Oxidative Stress, TAS: Total Antioxidant Status, GSTM1: Glutathione S-transferase M1, BP: Blood Pressure

Table 4. Comparison of TOS, TAS, systolic, and diastolic blood pressure values with GSTP1 A114V

GSTP1 A114V	TOS X±S	TAS x±S	Systolic BP x±S	Diastolic BP x±S
Normal (n=37)	8.51 ± 11.04	1.43 ± 0.37	117.06 ± 8.77	74.45 ± 7.38
Heterozygous (n=6)	14.16 ± 12.92	1.52 ± 0.17	116.00 ± 4.74	68.00 ± 2.44
p	p = 0.140	p = 0.888	p = 0.795	p = 0.062

TOS: Total Oxidative Stress, TAS: Total Antioxidant Status, GSTP1: Glutathione S-transferase P1, BP: Blood Pressure

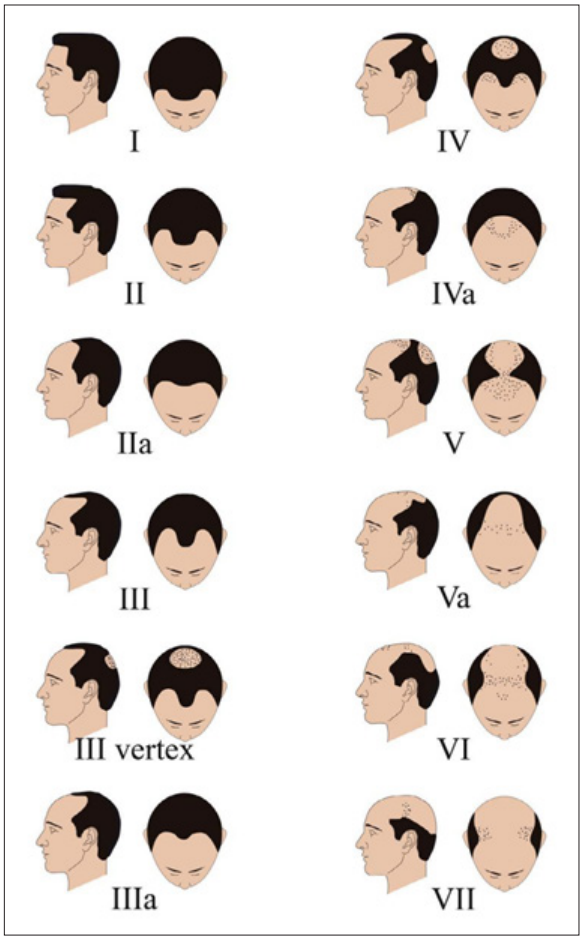

Figure 1. Clinical classification of male-pattern hair loss according to the Hamilton-Norwood scale.

phism showed no statistically significant differences (p>0.05). Total antioxidant status (TAS) was lower in participants with the GSTT1 null genotype than in those with the functional GSTT1 gene. Although not statistically significant (p=0.112), antioxidant capacity tended to decrease when both GSTT1 alleles were deleted (Table 2).

When comparing TOS, TAS, systolic, and diastolic blood pressure values between individuals with and without the GSTM1 polymorphism, no significant differences were found (p>0.05) (Table 3).

Regarding GSTP1 I105V, a significant difference in TOS values was observed (p=0.02). However, the expected outcome is that individuals with the homozygous genotype should have higher TOS levels than those with the normal genotype. Therefore, the results were not considered meaningful. Pairwise comparisons revealed significant differences involving the homozygous group when compared with both the normal and heterozygous groups (p<0.05). In contrast, the difference between the normal and heterozygous groups was not statistically significant (p>0.05).

For GSTP1 I105V, comparisons of TAS, systolic, and diastolic blood pressure values revealed no statistically significant differences (p>0.05). Similarly, for GSTP1 A114V, no significant differences were observed in TOS, TAS, systolic, or diastolic blood pressure values. While these findings did not reach statistical significance, individuals with the heterozygous genotype (those with the polymorphism) exhibited higher TOS levels (14.16 ± 12.92) compared to those with the normal genotype (8.51 ± 11.04) (Table 4).

The oxidative stress index was also compared across androgenetic alopecia stages, and a positive correlation was identified (r=0.14). Despite the lack of statistical significance, the presence of this positive correlation suggests a trend toward increasing oxidative stress levels with advancing AGA stage.

DISCUSSION

In this study, total oxidative stress (TOS), total antioxidant status (TAS), systolic, and diastolic blood pressure values did not differ significantly across the GSTT1, GSTM1, GSTP1 I105V, and GSTP1 A114V genotypes. However, TOS levels were higher in individuals with the GSTP1 A114V heterozygous genotype (indicative of the presence of polymorphism) compared to those with the wild-type genotype. Additionally, TAS levels were lower in individuals with the GSTT1 null genotype (homozygous deletion of the gene) compared to those carrying the GSTT1 gene, although this difference did not reach statistical significance. These findings suggest that GST gene polymorphisms may be associated with increased TOS and reduced TAS in androgenic alopecia.

Similarly, significant associations between oxidative stress and GST gene polymorphisms have been reported in studies on alopecia areata, coronary artery disease, hypertriglyceridemia, and hepatocellular carcinoma (8-11).

When patients were stratified according to the Hamilton-Norwood classification and compared in

terms of oxidative stress index, no statistically significant differences were observed. However, a consistent trend was noted, suggesting that oxidative stress levels tend to increase with advancing stages of AGA. Although this relationship could not be conclusively demonstrated due to the limited number of patients in each stage, it warrants further investigation in studies with larger and more evenly distributed sample sizes across all stages. While certain studies have examined the association between AGA stages and oxidative stress without identifying a significant link, others have proposed that oxidative stress may contribute to the pathogenesis of AGA (12–15). As research in this area advances, it is expected that more consistent and comprehensive findings will emerge.

The majority of patients with androgenic alopecia in our study (90.6%) reported a positive family history. This finding reinforces the hypothesis that genetic factors play a critical role in the pathogenesis of the condition (16,17). Although a family history of AGA increases an individual's risk, the absence of such a history does not exclude the possibility of developing the condition. While an autosomal dominant inheritance pattern with variable penetrance is commonly proposed, not all cases conform to this model, suggesting the involvement of polygenic and environmental factors as well (18,19).

Body mass index (BMI) assessment of individuals with AGA revealed that more than half (53.4%) were classified as overweight. When compared to national data on Turkish men, where the prevalence of overweight is reported to be between 25.9% and 40.4%, the higher proportion of overweight individuals among those with androgenic alopecia is notable (20,21). Given the mean age of the study population (35.09 ± 10.51 years), it can be inferred that they belong to a relatively young age group and may be at increased risk of developing obesity later in life. Therefore, early lifestyle interventions targeting weight management may be advisable for overweight individuals with androgenic alopecia.

A major limitation of this study is the small sample size, compounded by the lack of a control group consisting of healthy individuals without androgenic alopecia.

CONCLUSION

In this study, which investigated the relationship between GST gene polymorphism, total antioxidant capacity, and blood pressure changes in patients with androgenic alopecia, our findings suggest that GST gene polymorphism may be associated with increased oxidative stress and reduced total antioxidant capacity. However, further research is warranted to better understand this relationship.

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Conflict of interest and financial disclosure

The authors declare that they have no conflict of interest to disclose. The authors also declare that this study was financially supported by the Scientific Research Projects Coordination Unit of Cumhuriyet University.

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