

# The frequency of alopecia and quality of life in high-school students in rural areas (Sivrihisar, Mahmudiye, Alpu, and Beylikova) of Eskisehir

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## ABSTRACT

**OBJECTIVE:** The aim of the present study was to determine the incidence of alopecia and related factors and the health-related quality of life (HRQoL) in high-school students in rural areas of Eskisehir. This was a cross-sectional study.

**METHODS:** The study was performed between March 2, 2015 and April 30, 2015. A total of 1662 (74.9%) students were included in the study. The questionnaire performed for the purpose and consisted of four sections was filled out by the students themselves under supervision. The HRQoL was evaluated by Short Form—36 (SF-36). Students' hair and scalps were examined by a dermatologist. The acquired data were analyzed by SPSS 20 statistical package program. Chi-square test, Mann–Whitney U test, and logistic regression analyses were used for statistical analyses. A p value  $\leq 0.05$  was accepted as statistically significant.

**RESULTS:** In the present study, the incidence of alopecia was found to be 37.4% (n=622). Alopecia was more frequently seen in male students who have complaints about their scalps and those with a fatty scalp. In the study group, students with alopecia had poor HRQoL in general health perception, vitality, and mental health of SF-36.

**CONCLUSION:** There is a need to provide early diagnosis and treatment to decrease the incidence of alopecia and to improve the quality of life. Health education studies must be performed to increase the awareness of students about alopecia. Integrating hair and scalp examination into school health screening studies, steering the students who have alopecia to the dermatologists, and suggesting students who have fatty scalp regular hair washing will be appropriate.

*Keywords:* Adolescent; alopecia; Eskisehir; quality of life.

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Adolescent (adolescence) period is a process in which rapid physical, biological, psychological, and emotional changes are experienced during transition from childhood to adulthood. The World Health Organization (WHO) defines the 10–19 age group as the adolescent period, and this period may vary from person to person; it is difficult to confine the adolescent period

strictly within a certain age group because of the socio-cultural and socioeconomic characteristics of the societies, geographic, and seasonal features of the living place and dietary habits of the communities [1–4].

According to the WHO's definition of adolescent, it is reported that 1 out of 5 people worldwide is in the adolescent age group, meaning approximately 1.2 billion



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people. Globally, 85% of adolescents live in developing countries. In most countries, adolescents account for 20%–25% of the population, whereas the adolescent age group accounts for 16.8% of Turkey's population [4–7].

While hormonal changes in the adolescent period cause physical growth and pubertal changes, significant psychosocial changes are also seen. This change, which begins in the second decade of life, varies according to gender, person, and society [4].

Adolescent period is a special period where both hair problems and other dermatological disorders are seen frequently because of the hormonal changes in human life and the intense life conditions of collective life as is seen in schools or dormitories. Hair problems lead to depression and anxiety in adolescents and young adults and may affect their health-related quality of life (HRQoL) adversely. Changes in the appearance and quality of hair as wearing, splitting, thinning of hair strands or vice versa, and hypertrichosis (excessive growth of hair) may be defined as hair problems [1, 4, 8].

As generally known, congenital, infectious, autoimmune, genetic, and environmental factors are responsible for hair loss (alopecia). The known risk factors for alopecia are chronic endocrine or allergic diseases, surgery, regular drug use, nutrition, gynecological diseases, and stressful lifestyle [8, 9].

Physicians should identify the problem or any other underlying disease that causes hair loss accurately because of the lack of self-confidence and the quality of life caused by hair loss. Although it is important to evaluate alopecia in primary health care services, it is known that many physicians fail to evaluate and diagnose alopecia [10, 11].

Alopecia is categorized into two groups as those with and without scar, and most of them are cases with scarless alopecia. Androgenetic alopecia (AGA), telogen effluvium (TE), alopecia areata (AA), traction alopecia, and tinea capitis are grouped under the category of scarless alopecia, whereas discoid lupus erythematosus, lichen planus, infection (e.g., bacterial, viral, and fungal)-induced alopecia, and alopecia secondary to trauma or burns are also grouped under the category of scarred alopecia. According to the amount of hair loss, alopecia is also classified as diffuse alopecia and focal alopecia. The most common subtypes of alopecia in adolescents are AGA and TE [9, 10, 12, 13].

The prevalence of alopecia in adolescents worldwide varies widely between 15.5% and 38.5% because of the diversity of societies with respect to their genetic, eth-

nic, and lifestyle characteristics [14–17]. A field research related to the prevalence of alopecia has not been performed in Turkey.

The present study was conducted to determine the prevalence of alopecia among high-school students studying in the rural settlement of Eskisehir, to investigate the presumably related factors, and to evaluate the HRQoL of the study participants.

## MATERIALS AND METHODS

This was a cross-sectional study on high-school students who were receiving education and training in high schools in four districts (Sivrihisar, Mahmudiye, Alpu, and Beylikova) situated at the center of Eskisehir province between March 2, 2015 and April 30, 2015. The total population of the research region is 46,869 people. The region is at a medium socioeconomic level, and generally, the livelihood of the people is agriculture and animal husbandry [18, 19].

The study constituted of high-school students grades 9–12 living and receiving education and training in the schools situated in the center of the districts of Sivrihisar, Mahmudiye, Alpu, and Beylikova. A total of 14 high schools were situated in the district centers, and a total of 2220 students were receiving education and training in these high schools. In our study, any sampling was not performed, and it was aimed to reach all students.

The study was approved by the ethics committee, and necessary permissions and appointments were received from the Provincial Directorate of National Education and High Schools. The students were allowed to attend and gather in the classes during the day and hours of the appointment. Students were informed about the subject and purpose of the study. Oral informed consent was obtained from the students who agreed to participate in the study.

The previously prepared questionnaire forms were filled up by the students themselves under surveillance in accordance with the purpose of the study. The questionnaire form consisted of four sections. Questions about some of the sociodemographic characteristics of the students were included in the first section. Some factors that are thought to be related to the presence of alopecia comprised the second section. Results of hair and scalp examination were recorded in the third section. The fourth section consisted of questions about Short Form—36 (SF-36) scale items. After completing the questionnaire survey, the height and body weights of the students were

measured by the researchers. Then, the students' hair and scalp were examined by a dermatologist. The presence and severity of alopecia were assessed by examination. The severity of hair loss among those with alopecia was grouped as mild, moderate, and severe.

To make the diagnosis of TE, which is one of the subtypes of alopecia, skin pull test was performed [20–22]. According to the dermoscopic examination of hair strands, students having hair strands with a diameter of  $\leq 39 \mu\text{m}$ ,  $40\text{--}79 \mu\text{m}$ , and  $\geq 80 \mu\text{m}$  were evaluated as those with thin, normal, and thick hair strands, respectively [23]. In the present study, the criteria in the Declaration of Helsinki have been complied with.

In our study, SF-36 was used in the evaluation of HRQoL. This scale was developed by Ware et al. in 1992, and its validity and reliability study in Turkey was performed by Kocyigit et al. in 1999 [24, 25]. The SF-36 is a self-evaluation scale, and the status of individuals within the last 4 weeks is taken as a basis. There are eight subdomains of the scale, including physical functioning, physical role, pain, general health, vitality, social functioning, mental role, and mental health. The higher the score from each subdomain, the higher the HRQoL of that subdomain [26, 27].

Data obtained were evaluated using the IBM SPSS statistical package program (International Business Machines for Social Sciences version 20.0; SPSS Inc., Chicago, IL, USA). Chi-square test and Mann–Whitney U test were used for data analysis. Backward Stepwise (Wald) Logistic Regression Analysis was performed with some variables found to be related to the presence of alopecia. A  $p$  value  $\leq 0.05$  was accepted as statistically significant.

## RESULTS

A total of 2220 students were attending the high schools in the research area. Of the 2220 students, 463 could not be found in schools during the study period, and 95 were excluded from the study because they did not accept hair and scalp examination. The study group was composed of 1662 (74.9%) students. The ages of the students ranged from 14 to 19 years, and the average age was  $16.2 \pm 1.1$  years. In our study, the prevalence of alopecia was 37.4% ( $n=622$ ).

The study group consisted of 906 (54.5%) male and 756 (45.5%) female students, and 493 (29.6%) students were aged  $\leq 15$  years. Of the 1662 students, 349 (21.0%)

were smokers, and 179 (10.8%) were drinking alcohol. A total of 262 (15.8%) students had physician-diagnosed chronic diseases. Table 1 shows the distribution of alopecia in the study group according to some sociodemographic characteristics.

Of the 1662 students, 1422 (85.6%) had been living in a core family, and 240 (14.4%) had been living in a large family. More than half of the students reported that their family was at a moderate income level. Mothers of 250 (15.0%) and fathers of 1256 (75.6%) students were employed. Mothers of 1147 (69.0%) students had received primary education (or lower level). However, fathers of 856 (51.2%) students were secondary and high-school graduates, and 927 (55.8%) students were living with their families. Table 2 shows the distribution of alopecia among the study groups according to some parental characteristics.

In the study group, 419 (25.2%) students were complaining of burning, stinging, and itching sensations on the scalp, whereas 835 (50.2%) students were using hair dryer after shower/bath, and 409 (24.6%) students were using chemical hair styling products. Table 3 shows the distribution of alopecia in the study group according to some factors related to hair health.

According to the results of scalp examination, 938 (56.4%) students had thin, 633 (38.1%) had thick, and 91 (5.5%) had normal hair strands. According to the results of scalp examination, 780 (46.9%) students had natural, 563 (33.9%) had greasy, 253 (15.2%) had dry, and 66 (4.0%) had moist hair.

Table 4 presents the distribution of hair strand thickness and scalp type in patients with and without alopecia.

Among the students with alopecia, 385 (23.2%) had AGA, 171 (10.3%) had TE, 40 (2.4%) had triangular (TAA), 37 (2.2%) had traction, 17 (1.0%) had scleral, and 4 (0.2%) had androgenic alopecia. In the study group, the distribution of students with alopecia according to alopecia subtypes is given in Figure 1.

In the study group, the results of logistic regression analysis that is performed with the variables (sex, age group, smoking status, presence of complaint in the scalp, hair thickness, and scalp type) found to be related to the presence of alopecia are given in Table 5.

In our study, according to the results of logistic regression analysis, gender, presence of scalp, and type of scalp were found to be risk factors for the incidence of alopecia.

**TABLE 1.** Distribution of study participants with and without alopecia according to their sociodemographic characteristics

Some sociodemographic characteristics	Alopecia						Test value X <sup>2</sup> ; p
	Yes		No		Total		
	n	%*	n	%*	n	%**	
Gender							<b>15.704; 0.001</b>
Male	378	41.7	528	58.3	906	54.5	
Female	244	32.3	512	67.7	756	45.5	
Age group							<b>8.462; 0.037</b>
≤15 years	172	34.9	321	65.1	493	29.6	
16 years	165	34.4	314	65.6	479	28.8	
17 years	192	40.2	286	59.8	478	28.8	
≥18 years	93	43.9	119	56.1	212	12.8	
Smoking status							<b>9.211; 0.002</b>
Smoker	155	44.4	194	55.6	349	21.0	
Nonsmoker	467	35.6	846	64.4	1313	79.0	
Alcohol consumption							3.241; 0.072
Yes	78	43.6	101	56.4	179	10.8	
No	544	36.7	939	63.3	1483	89.2	
Presence of physician-diagnosed chronic disease							0.168; 0.682
Yes	101	38.5	161	61.5	262	15.8	
No	521	37.2	879	62.8	1400	84.2	
Total	622	37.4	1040	62.6	1662	100.0	

\*Percentage of the row; \*\*Percentage of the column.

In the study group, there were no differences in the scores obtained in the subdomains other than the general health, vitality, and mental health subdomains. Table 6 presents the distribution of the median scores obtained from the subdomains of the SF-36 scale in the study group.

## DISCUSSION

Alopecia, which can be seen in men and women of all ages, is a dermatological problem observed in approximately half of the people throughout their lives. Although alopecia is not a life-threatening condition, hair loss can often lead to adverse social and psychological consequences due to the fact that hair is an important social communication tool, and hair loss adversely affects self-confidence and the quality of life of individuals [10, 11].

Owing to the increase in androgenic hormonal activity and psychosocial changes, the incidence of alopecia in adolescence has increased significantly [4, 28, 29]. In

our study, the incidence of alopecia was 37.4%. In studies conducted in various countries, the incidence of alopecia in adolescents has been reported to range from 15.5% to 38.5% [14–17]. The reasons of the different results reported in various studies may be considered as the differences in sociodemographic, ethnic, genetic, and lifestyle characteristics of the societies in which the studies are conducted and the differences in the diagnostic methods used. AGA is the most common alopecia subtype in adolescence and afterwards. Androgenic hormones trigger alopecia by miniaturizing strong terminal hairs, and as a result, alopecia is expected to be more common in men [29–31]. In our study, the incidence of alopecia was higher in male than in female students ( $p \leq 0.05$ , odds ratio (OR)=1.367). Paik et al. and Xu et al. reported similar results [32, 33]. Agirgol et al. reported that there is no relationship between the frequency of alopecia and gender [34].

There was no correlation between the frequency of alopecia and age group in the study group ( $p > 0.05$ ).

**TABLE 2.** Distribution of study participants with and without alopecia according to some parental characteristics

Some parental characteristics	Alopecia						Test value X <sup>2</sup> ; p
	Yes		No		Total		
	n	%*	n	%*	n	%**	
Family type							
Core	533	37.5	889	62.5	1422	85.6	0.014; 0.906
Large	89	37.1	151	62.9	240	14.4	
Income level of the family							
Poor	36	42.9	48	57.1	84	5.1	2.646; 0.266
Moderate	438	38.1	713	61.9	1151	69.3	
Good	148	34.7	279	65.3	427	25.7	
Employment status of the mother							
Employed	95	38.0	155	62.0	250	15.0	0.042; 0.838
Unemployed	527	37.3	885	62.7	1412	85.0	
Employment status of the father							
Employed	471	37.5	785	62.5	1256	75.6	0.012; 0.911
Unemployed	151	37.2	255	62.8	405	24.4	
Educational level of the mothers							
Primary school and lower	430	37.5	717	62.5	1147	69.0	0.007; 0.936
Secondary school and higher	192	37.3	323	62.79	515	31.0	
Educational level of the fathers							
Primary school and lower	319	39.3	492	60.7	811	48.8	2.466; 0.116
Secondary school and higher	303	35.6	548	64.4	851	51.2	
Place of residence							
With his family	361	38.9	566	61.1	927	55.8	2.063; 0.151
Dormitory	261	35.5	474	64.5	735	44.2	
Total	622	37.4	1040	62.6	1662	100.0	

\*Percentage of the row; \*\*Percentage of the column.

Kyriakis et al. reported results similar to our study [35]. Price et al. reported an increase in the incidence of alopecia with aging [36]. Kim et al. reported that the incidence of alopecia in the 14–18 age group is higher than that in the 10–14 age group [37]. In our study, the relationship between the age group and the incidence of alopecia could not be determined due to the fact that the age groups were very close to each other.

The poor socioeconomic status of the family and the poor environmental characteristics of the living place are risk factors that increase the incidence of alopecia [38]. In our study, there was no relationship between the incidence of alopecia and family type, family income level, parental working status, parental education level, and place of residence ( $p > 0.05$ , for each). Similar studies

have reported the lack of any correlation between the socioeconomic characteristics, such as family type, family income, and parental education, and the incidence of alopecia [39, 40]. Since the schools included in the study were in rural areas and the individuals living there had similar socioeconomic and environmental characteristics, any correlation between the parental characteristics and the incidence of alopecia could not be found.

The chemical substances contained in the cigarette can cause free radicals to break down the capillary structure, reduce the blood supply, and impair nutrition of the hair follicles. Alcohol consumption can also damage the hair follicles by increasing oxidative stress and reducing protein synthesis. Smoking and alcohol consumption may cause alopecia due to these reasons [41–44]. Simi-

**TABLE 3.** Distribution of study participants with and without alopecia according to some factors related to hair health

Some factors related to hair health	Alopecia						Test value X <sup>2</sup> ; p
	Yes		No		Total		
	n	%*	n	%*	n	%**	
Complaints related to the scalp							<b>4.027; 0.045</b>
Yes	174	41.5	245	58.5	419	25.2	
No	448	36.0	795	64.0	1243	74.8	
Use of hair dryer							0.023; 0.879
Users	311	37.2	524	62.8	835	50.2	
Non-users	311	37.6	516	62.8	827	49.8	
Use of hair styling products							1.139; 0.286
Users	144	35.2	265	64.8	409	24.6	
Non-users	478	38.1	775	61.9	1253	75.4	
Total	622	37.4	1040	62.6	1662	100.0	

\*Percentage of the row; \*\*Percentage of the column.

**TABLE 4.** Distribution of students with and without alopecia according to hair strand thickness and scalp type

Hair strand thickness and scalp type	Alopecia						Test value X <sup>2</sup> ; p
	Yes		No		Total		
	n	%*	n	%*	n	%**	
Thickness of the hair strand							<b>7.273; 0.026</b>
Thin	356	38.0	582	62.0	938	56.4	
Normal	22	24.2	69	75.8	91	5.5	
Thick	244	38.5	389	61.5	633	38.1	
Scalp type							<b>20.999; &lt;0.001</b>
Natural	251	32.2	529	67.8	780	46.9	
Dry	97	38.3	156	61.7	253	15.2	
Moist	24	36.4	42	63.6	66	4.0	
Fatty	250	44.4	313	55.6	563	33.9	
Total	622	37.4	1040	62.6	1662	100.0	

\*Percentage of the row; \*\*Percentage of the column.

lar studies have also reported the lack of any relationship between smoking and alcohol consumption and the incidence of alopecia [43–46]. Mosley et al. reported that the incidence of alopecia is higher in smokers [47].

Severi et al. reported that the incidence of alopecia is higher in alcohol consuming individuals, and that alcohol consumption is associated with AGA, especially involv-

ing the forehead and vertex, and not related to the other types of alopecia [43]. Owing to the smaller age of the students in the study group and the time required for the observation of the cumulative effects of cigarette and alcohol on alopecia did not pass, the relationship between smoking and alcohol consumption and the frequency of alopecia might not be found in our study.

**TABLE 5.** Results of logistic regression analysis performed using variables detected to be related to alopecia

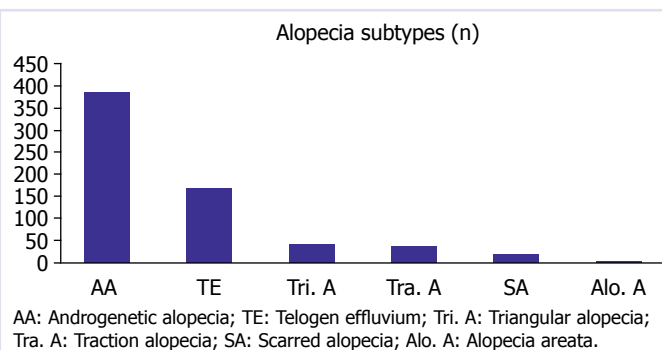
Variables	$\beta$	SE	p	OR	95% CI
Gender (reference: female students)					
Male students	0.313	0.113	<b>0.005</b>	1.367	1.097–1.705
Complaints related to the scalp (reference: none)					
Yes	0.265	0.121	<b>0.028</b>	1.303	1.029–1.650
Scalp types (reference: natural)					
Dry			0.486	1.210	0.708–2.066
Moist			0.100	1.294	0.952–1.759
Fatty			0.001	1.586	1.257–2.001
Constant	–		0.381	0.000	– –
	1.441				

SE: Standard error; OR: Odds ratio; CI: Confidence interval.

**TABLE 6.** Distribution of median scores obtained by study participants with and without alopecia from SF-36 scale subdomains

SF-36 subdomains	Alopecia		Test value z; p
	Yes Median (Min.–Max.)	No Median (Min.–Max.)	
Physical functioning	90.0 (0.0–100.0)	95.0 (0.0–100.0)	1.370; 0.171
Physical role	75.0 (0.0–100.0)	100.0 (0.0–100.0)	1.755; 0.079
Pain	80.0 (0.0–100.0)	87.5 (0.0–100.0)	1.785; 0.074
General health	60.0 (0.0–100.0)	65.0 (0.0–100.0)	<b>1.968; 0.049</b>
Vitality	60.0 (0.0–100.0)	65.0 (0.0–100.0)	<b>2.326; 0.020</b>
Social functioning	75.0 (0.0–100.0)	75.0 (0.0–100.0)	1.397; 0.162
Mental role	66.7 (0.0–100.0)	66.7 (0.0–100.0)	1.396; 0.163
Mental health	56.0 (0.0–100.0)	60.0 (0.0–100.0)	<b>3.007; 0.003</b>

Min.: Minimum; Max.: Maximum.

**FIGURE 1.** In the study group, the distribution of students with alopecia according to alopecia subtypes.

Chronic diseases, such as hypothyroidism, hyperthyroidism, thyroiditis, hypertension, and diabetes, cardiovascular diseases, anemia, vascular structure, and autoimmune diseases cause T lymphocyte-mediated hair follicle damage and increase the risk of the development of alopecia [48–51]. There was no significant difference in the prevalence of alopecia among patients with physician-diagnosed chronic disease ( $p > 0.05$ ). Similar results have been reported in various studies [28, 52, 53]. Akbas et al. emphasized that the incidence of AA is found to be higher in patients with autoimmune thyroid diseases, atopic dermatitis, vitiligo, pernicious anemia, and chronic AA [19]. In the present study, the reasons of not

finding a relationship between the presence of chronic disease and the incidence of alopecia can be related to the small age of the students, the onset of chronic illnesses in general in advanced ages, and the cumulative emergence of the alopecic effects of chronic diseases.

Burning, stinging, and itching sensations felt on the scalp can be caused by scalp lesions, and these lesions may lead to impaired nutrition oxygenation, and integrity of the scalp. In addition, the presence of a complaint or lesion involving the scalp can cause mechanical trauma because of the continuous playing with the hair and scratching of the scalp. It is expected that the presence of complaints related to the scalp will increase the incidence of alopecia due to these effects [19]. In the study group, the frequency of alopecia was higher in those with complaints related to the scalp than in those without ( $p \leq 0.05$ ,  $OR = 1.303$ ). It has been reported that the incidence of alopecia is higher in patients with symptoms, such as pruritus, burning, and stinging sensations, felt on the scalp and those with sensitive scalp skin [19, 54, 55].

The use of a hair dryer, especially at high temperatures, is known to induce alopecia by causing deformities and fractures in the hair shaft. The use of a hair dryer at high temperatures to shape hair causes the hair follicles to burn, creating a special type of alopecia called "hot comb alopecia" [56, 57]. In the study group, there was no difference in the frequency of alopecia between the hair dryer users and non-users ( $p > 0.05$ ). Whiting et al. and Jan et al. reported that the use of a hair dryer at high temperatures causes alopecia due to its traumatic effect [58, 59]. Since the students participating in the study group used a hair dryer at low temperatures to dry their hair after a bath/shower, not for the purpose of hot combing, we did not find any difference in the incidence of alopecia between users and non-users of hair dryers.

Chemical hair styling products, such as jelly, grease, and hairspray, can lead to alopecia by causing irritation and allergic reaction on the scalp and destroying keratin protein, an important component of the hair strand [60, 61]. In our study, no difference was found in the incidence of alopecia among those who used and did not use chemical hair styling products ( $p > 0.05$ ). Similar results were reported in the study by Gummer et al. [62]. Draelos et al. and Zviak et al. reported higher incidence of alopecia among users of chemical hair styling products [63, 64]. In the present study, the reasons of not finding a difference in the frequency of alopecia between users and non-users of chemical hair styling products may be re-

lated to our study group being composed of adolescents. In addition, owing to the shorter follow-up period, we could not observe the side effects of these products, and our study population living in rural areas could not afford for these products due to economic concerns.

Thin hair strand is a sign of miniaturization in the hair follicle, causing the hair to become more fragile, and may lead to alopecia. The development of alopecia is facilitated with greater volume of hair loss with aging in people with thick hair [65, 66]. In the present study, there was no relationship between the thickness of hair follicle and the frequency of alopecia ( $p > 0.05$ ). Several studies have reported that the incidence of alopecia is higher in those with thin hair strands [67, 68]. Courtois et al. and Barman et al. reported that alopecia is more frequently detected in patients with thicker hairs [65, 69]. Different results obtained in various studies may be due to the difference in examination methods.

The sebum, which is produced by the sebaceous glands in the scalp, has a rich fat content and protects the scalp from mechanical trauma as it acts as a physical barrier by providing the lubrication of the scalp. In case of excessive secretion of the sebum, the hair follicles may become clogged, and the oxygenation and blood supply of the hair may be reduced with the potential increase in the risk of alopecia [70, 71]. In the study group, the incidence of alopecia was higher in patients with oily/greasy hair ( $p \leq 0.05$ ,  $OR = 1.586$ ). Ronny et al. and Pireard et al. reported that alopecia is more common in those with oily-haired skin [72, 73]. Banka et al. reported that any correlation is not found between the incidence of scalp type and alopecia [74].

Owing to the increased activation of androgenic hormones during puberty, terminal hairs in hair follicles can turn into miniature hairs and induce the development of AGA. It is known that changes in thyroid and androgenic hormone metabolism play an important role in TE etiology. The most common alopecia subtypes due to hormonal changes occurring during and after adolescence are AGA and TE [28, 30, 75, 76]. In the present study, the incidence rates of AGA and AA were 23.2% and 10.3%, respectively. In some studies, the incidence rates of AGA and TE have been reported to range between 13.1 and 24.5% [32, 36, 77] and 1.6 and 59.7%, respectively [19, 78]. The reasons of the different results reported in various studies may be due to the sociodemographic and ethnic characteristics, lifestyles of the study populations investigated, and different diagnostic methods used in these studies.



Since the body's perception of people is negatively affected by alopecia, their self-esteem decreases, which is known to affect the quality of life adversely leading to psychological and social problems [11, 79]. In the study group, the SF-36 scale scores indicated the presence of a worse quality of life in the general health, vitality, and mental health subdomains of patients with alopecia ( $p \leq 0.05$ , for each). In the other subdomains of the scale, no difference was found with respect to the quality of life among those who were not identified with alopecia ( $p > 0.05$ , for each).

Gulec et al. reported poorer quality of life in the subdomains of vitality and mental health in those with alopecia [80]. Hollanda et al. reported that patients with alopecia are found to have poorer quality of life in the mental health, mental role, social function, and pain subdomains [81].

## Conclusion

In the present study, owing to the high prevalence of alopecia, we deemed it appropriate to conduct health education studies, to integrate hair and scalp examinations into school screening programs, to direct students with alopecia to the dermatologists, to make students with fatty scalp to wash their hair regularly to improve the quality of life of the students in the research region, and to increase the awareness of students so as to increase the rates of early diagnosis and treatment.

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## REFERENCES

1. Stevens-Simon C. Clinical applications of adolescent female sexual development. *Nurse Pract* 1993;18:18, 21, 25–9. [CrossRef]
2. Tecce N, Pane A. [Notes on morbidity of Heine-Medin disease in the province of Naples in the ten-year period 1947-1956]. [Indexed for MEDLINE]. *Acta Med Ital Mal Infett Parasit* 1957;12:324–32.
3. Cotrufo P, Pezzullo C, Tecce N, Villari A. [Preliminary note on the influenzal epidemic in the province of Naples in August & September, 1957]. [Article in Italian]. *Acta Med Ital Mal Infett Parasit* 1957;12:300.
4. Özcebe H. Birinci basamakta adölesan sorunlarına yaklaşım. *Sted* 2002;11:374–7.
5. Başer M. Adölesan cinselliği ve gebelik. *C.Ü. Hemşirelik Yüksekokulu Dergisi* 2000;4:50–4.
6. Colucci Cf, Jacono G, Tecce N. [Influenza epidemic of 1952-53 in the island of Procida; epidemiologic, virologic and serologic research]. [Article in Italian]. *Riv Ist Sieroter Ital* 1954;29:348–60.
7. Tecce N, Villari A. [Statistical contribution to the spread of intestinal parasitosis]. [Article in Italian]. *Acta Med Ital Mal Infett Parasit* 1957;12:150–6.
8. Hawryluk EB, English JC 3rd. Female adolescent hair disorders. *J Pediatr Adolesc Gynecol* 2009;22:271–81. [CrossRef]
9. Grimes DA, Blankenship O, Kremer C, Reese S, Sonstein F. Initial Office Evaluation of Hair Loss in Adult Women. *The Journal for Nurse Practitioners* 2011;7:456–62. [CrossRef]
10. Mounsey AL, Reed SW. Diagnosing and treating hair loss. *Am Fam Physician* 2009;80:356–62.
11. Springer K, Brown M, Stulberg DL. Common hair loss disorders. *Am Fam Physician* 2003;68:93–102.
12. Shapiro J, Wiseman M, Lui H. Practical management of hair loss. *Can Fam Physician* 2000;46:1469–77.
13. Bedocs LA, Bruckner AL. Adolescent hair loss. *Curr Opin Pediatr* 2008;20:431–5. [CrossRef]
14. Khumalo NP, Jessop S, Gumede F, Ehrlich R. Hairdressing and the prevalence of scalp disease in African adults. *Br J Dermatol* 2007;157:981–8. [CrossRef]
15. Pathomvanich D, Pongratananukul S, Thienthaworn P, Manoshai S. A random study of Asian male androgenetic alopecia in Bangkok, Thailand. *Dermatol Surg* 2002;28:804–7. [CrossRef]
16. Rhodes T, Girman CJ, Savin RC, Kaufman KD, Guo S, Lilly FR, et al. Prevalence of male pattern hair loss in 18-49 year old men. *Dermatol Surg* 1998;24:1330–2. [CrossRef]
17. Setty LR. Hair patterns of scalp of white and Negro males. *Am J Phys Anthropol* 1970;33:49–55. [CrossRef]
18. <http://www.alpu.bel.tr/>. Accessed at 19.04.2015.
19. Akbaş A, Kılınc F, Yakut İ, Metin A. Türk Çocuklarında Görülen Saç Hastalıkları: Retrospektif Bir Analiz. *Dermatoz* 2014;4:1–8.
20. Fiedler VC, Gray AC. Diffuse alopecia: telogen hair loss. In: Olsen EA, editor. *Disorders of Hair Growth: Diagnosis and Treatment*. 2<sup>nd</sup> ed. New York: McGraw-Hill; 2003. p.303–20.
21. Olsen EA. *Disorders of hair growth: diagnosis and treatment*. New York: McGraw-Hill, 2003.
22. Sinclair R, Banfield C, Dawber RPR. *Handbook of Diseases of the Hair and Scalp*. Oxford: Blackwell Science; 1999.
23. Oura H, Iino M, Nakazawa Y, Tajima M, Ideta R, Nakaya Y, et al. Adenosine Increases Anagen Hair Growth and Thick Hairs in Japanese women With Female Pattern Hair Loss: A Pilot, Double-Blind, Randomized, Placebo-Controlled Trial. *J Dermatol* 2008;35:763–7. [CrossRef]
24. Koçyiğit H, Aydemir Ö, Ölmez N, Memiş A. Kısa Form-36 (KF-36)'nın Türkçe Versiyonunun Güvenilirliği ve Geçerliliği. *İlaç ve Tedavi Dergisi* 1999;12:102–6.
25. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992;30:473–83. [CrossRef]
26. Carr AJ, Thompson PW, Kirwan JR. Quality of life measures. *Br J Rheumatol* 1996;35:275–81. [CrossRef]
27. Keller SD, Bayliss MS, Ware JE Jr, Hsu MA, Damiano AM, Goss TF. Comparison of responses to SF-36 Health Survey questions with one-week and four-week recall periods. *Health Serv Res* 1997;32:367–84.
28. Bologna JL, Jorizzo JL, Rapini RP. *Dermatologia*. 1<sup>st</sup> ed. Madrid: Elsevier. 2004.
29. Randall V. Physiology and pathophysiology of androgenetic alopecia. *Endocrinology*; 2005. p. 2257–68.
30. Kaufman KD. Androgens and alopecia. *Mol Cell Endocrinol* 2002;198:89–95. [CrossRef]
31. Trüeb RM. Molecular mechanisms of androgenetic alopecia. *Exp Gerontol* 2002;37:981–90. [CrossRef]

32. Paik JH, Yoon JB, Sim WY, Kim BS, Kim NI. The prevalence and types of androgenetic alopecia in Korean men and women. *Br J Dermatol* 2001;145:95–9. [CrossRef]
33. Xu F, Sheng YY, Mu ZL, Lou W, Zhou J, Ren YT, et al. Prevalence and types of androgenetic alopecia in Shanghai, China: a community-based study. *Br J Dermatol* 2009;160:629–32. [CrossRef]
34. Ağırşöl Ş, Baş Y, Azaklı HN, Geyik N, Karaoğlu A, Derviş E, et al. Alopesi Areatada Klinik Özellikler ve Tiroid Otoimmünitesi Sıklığı. *Türkiye Klinikleri J Dermatol* 2013;23:1–6.
35. Kyriakis KP, Paltatzidou K, Kosma E, Sofouri E, Tadros A, Rachioti E. Alopecia Areata Prevalence by Gender and Age. *J Eur Acad Dermatol Venereol* 2009;23:572–3. [CrossRef]
36. Price VH. Androgenetic alopecia in adolescents. *Cutis* 2003;71:115–21.
37. Kim BJ, Kim JY, Eun HC, Kwon OS, Kim MN, Ro BI. Androgenetic alopecia in adolescents: a report of 43 cases. *The Journal of dermatology* 2006;33:696–9. [CrossRef]
38. Sehgal VN, Srivastava G, Aggarwal A, Sethi G, Adhikari T. Alopecia areata in the Indian subcontinent. *Skinmed* 2007;6:63–9. [CrossRef]
39. Bilgiç Ö, Kaya K, Akça ÖF, Polat R. Alopesi Areatalı Hastalarda Psikiyatrik Belirtiler. *Dermatoz* 2011;2:227–9.
40. Güz H, Ay M, Dilbaz N. Bir Grup Dermatolojik Hastalarda Aleksitimi, Depresyon ve Anksiyete. *Düşünen Adam* 2001;14:99–103.
41. Cylwik B, Chrostek L, Szmitkowski M. [The effect of alcohol on iron metabolism]. [Article in Polish]. *Pol Merkur Lekarski* 2008;24:561–4.
42. Delibaş N, Özçankaya R. Serbest radikaller. *SDÜ Tıp Fakültesi Dergisi* 1995;2:11–7.
43. Severi G, Sinclair R, Hopper JL, English DR, McCredie MR, Boyle P, et al. Androgenetic alopecia in men aged 40–69 years: prevalence and risk factors. *Br J Dermatol* 2003;149:1207–13. [CrossRef]
44. Su LH, Chen TH. Association of androgenetic alopecia with smoking and its prevalence among Asianmen: a community-based survey. *Arch Dermatol* 2007;143:1401–6. [CrossRef]
45. Arias-Santiago S, Gutiérrez-Salmerón MT, Castellote-Caballero L, Buendía-Eisman A, Naranjo-Sintes R. Androgenetic alopecia and cardiovascular risk factors in men and women: a comparative study. *J Am Acad Dermatol* 2010;63:420–9. [CrossRef]
46. Su LH, Chen HH. Androgenetic alopecia in policemen: higher prevalence and different risk factors relative to the general population (KCIS no. 23). *Arch Dermatol Res* 2011;303:753–61. [CrossRef]
47. Mosley JG, Gibbs AC. Premature grey hair and hair loss among smokers: a new opportunity for health education? *BMJ* 1996;313:1616.
48. Gathers RC, McMichael AJ. Hair disorders in systemic disease. *Dermatologic Signs of Internal Disease*. Ed. Callen J, Jorizzo J, Bolgnia JL, et al. 4th ed. Elsevier: Saunders. 2003.
49. Özdemir M. Telojen Saç Dökülmesi. *Türkiye Klinikleri Journal of Internal Medical Sciences* 2006;2:6–9.
50. Sperling LC. Hair and systemic disease. *Dermatol Clin* 2001;19:711–26.
51. Goh C, Finkel M, Christos PJ, Sinha AA. Profile of 513 patients with alopecia areata: associations of disease subtypes with atopy, autoimmune disease and positive family history. *J Eur Acad Dermatol Venereol* 2006;20:1055–60. [CrossRef]
52. Olsen EA, Reed KB, Cacchio PB, Caudill L. Iron deficiency in female pattern hair loss, chronic telogen effluvium, and control groups. *J Am Acad Dermatol* 2010;63:991–9. [CrossRef]
53. Paus R, Olsen EA, Messenger AG. Hair growth disorders. *Fitzpatrick's Dermatology in General Medicine*. Wolff K, Goldsmith LA, Katz SI, Gilchrist BA, Paller AS, Leffell DJ, editors. 7. Ed. New York: McGraw-Hill Companies; 2008.p.753.
54. Karakuzu A. Primer sikatriyel alopesiler/Primary cicatricial alopecia. *Turkderm* 2014; 48:56–59. [CrossRef]
55. Sharma VK, Dawn G, Kumar B. Bubble hair: case caused by an overheating hair dryer and reproducibility in normal hair with heat. *Journal of the American Academy of Dermatology* 1994;30:54–60. [CrossRef]
56. Derwiler SP, Carson JL, Woosley JT, Gambling TM, Briggaman RA. Bubble hair. Case caused by an overheating hair dryer and reproducibility in normal hair with heat. *J Am Acad Dermatol* 1994;30:54–60.
57. Wilborn WS. Disorders of hair growth in African Americans. In: Olsen EA, editor. *Disorders of hair growth: Diagnosis and treatment*. New York: Mcgraw-Hill Inc. 1994.p.389–407.
58. Whiting DA. Chronic telogen effluvium: increased scalp hair shedding in middle-aged women. *J Am Acad Dermatol* 1996;35:899–906.
59. Jan V, Roudier-Pujol C. Prise en charge d'une alopecie chez la femme. In: *Annales de dermatologie et de v.n.r.ologie*. Masson; 2000.
60. Niang SO, Kane A, Dieng MT, Sy TN, Diallo M, Ndiaye B. Alopecia in Senegalese women. *Int J Dermatol* 2005;44 Suppl 1:22–3. [CrossRef]
61. Zviak C. Ondulation permanente et d.frisage. *Science des traitements capillaires*. Paris: Masson; 1987. p. 183209.
62. Gummer CL. Cosmetics and hair loss. *Clin Exp Dermatol* 2002;27:418–21. [CrossRef]
63. Draeos ZK. Hair cosmetics. *Dermatol Clin* 1991;9:19–27. [CrossRef]
64. Zviak C. Permanent waving and hair straightening. In: Zviak C, editor. *The Science of hair care*. New York: Marcel Dekker 1986. [CrossRef]
65. Courtois M, Loussouarn G, Hourseau C, Grollier JF. Ageing and hair cycles. *Br J Dermatol* 1995;132:86–93. [CrossRef]
66. Kutlubay Z, Bağlam S, Ergin B, Serdaroğlu S. Male androgenetic alopecia. *Turkderm* 2014;48:36–39. [CrossRef]
67. Birch MP, Messenger JF, Messenger AG. Hair density, hair diameter and the prevalence of female pattern hair loss. *Br J Dermatol* 2001;144:297–304. [CrossRef]
68. Mostafa WZ, Assaf MI, Ameen IA, El Safoury OS, Al Sulh SA. Hair loss in pityriasis versicolor lesions: a descriptive clinicopathological study. *J Am Acad Dermatol* 2013;69:19–23. [CrossRef]
69. Barman JM, Pecoraro V, Astore I. Method, Technic and Computations in the Study of the Trophic State of the Human Scalp Har. *J Invest Dermatol* 1964;42:421–5. [CrossRef]
70. Bologna J, Schaffer J, Cerroni L. *Dermatology*. Vol 1. Madrid: Elsevier; 2004.
71. Yücel A, Aksungur VL, Denli YG, Özpoyraz M. Yağlı Saç ve Bakımı. *Türkiye Klinikleri J Cosmetol* 2004;5:68–70.
72. Pierard-Franchimont C, Pierard GE. Approche physiopathologique de la s.borrh.e du cuir chevelu. In: *Annales de dermatologie et de v.n.r.ologie*. Masson; 1988.
73. Rony HR, Zakon SJ. Effect of endocrine substances on the adult human scalp. *Arch Derm Syphilol* 1945;52:323–7. [CrossRef]
74. Banka N, Bunagan MJ, Dubrule Y, Shapiro J. Wigs and hairpieces: evaluating dermatologic issues. *Dermatol Ther* 2012;25:260–6. [CrossRef]
75. Malkud S. Telogen Effluvium: A Review. *J Clin Diagn Res* 2015;9:WE01–3. [CrossRef]
76. Braun-Falco O, Plewig G, Wolff HH. *Dermatologie und venerologie*. Verlag Berlin Heidelberg: Springer; 2013.
77. Gan DC, Sinclair RD. Prevalence of male and female pattern hair loss in Maryborough. *J Invest Dermatol Symp Proc* 2005;10:184–9.
78. Nnoruka N, Obiagboso I, Maduechesi C. Hair loss in children in South-East Nigeria: common and uncommon cases. *International journal of dermatology* 2007;46:18–22. [CrossRef]
79. Cash TF, Price VH, Savin RC. Psychological effects of androgenetic alopecia on women: comparisons with balding men and with female control subjects. *J Am Acad Dermatol* 1993;29:568–75. [CrossRef]
80. Güleç AT, Tanriverdi N, Dürü C, Saray Y, Akçali C. The role of psychological factors in alopecia areata and the impact of the disease on the quality of life. *Int J Dermatol* 2004;43:352–6. [CrossRef]
81. de Hollanda TR, Sodré CT, Brasil MA, Ramos-E-Silva M. Quality of life in alopecia areata: a case-control study. *Int J Trichology* 2014;6:8–12. [CrossRef]