Journal of Pakistan Association of Dermatologists. 2024;**34(1)**:200-207.

Original Article

Vitamin D levels and their relationship with the type of involvement in alopecia areata patients

Sabir Hasanbeyzade, Mustafa Tunca

Department of Dermatology and Venereology, Gulhane Millitary Medical Academy Hospital, Ankara, Turkey.

Abstract

Background Alopecia areata is an autoimmune dermatosis characterized by non-scarring loss of hair, especially of the beard, eyebrows and eyelashes. In this case-control study, we investigated the relationship between vitamin D levels and the disease and the type of involvement (patchy, diffuse, total and universal).

Methods Forty one non-pregnant and over 18 years of age alopecia areata patients, who applied to the dermatology outpatient clinic of our hospital between 08.03.2016 and 31.05.2016 and who gave informed consent after being given verbal and written information about the study, were included in the study. The control group included 41 non-pregnant people over the age of 18 who applied to the outpatient clinic with complaints other than alopecia areata, matched with the patient group in terms of gender and age.

Results There was no statistically significant difference in vitamin D levels between the control and patient groups, as well as between groups separated according to the type of involvement (p=0.06 and 0.72, respectively). There was no significant difference between the 2 groups with normal and low (<30ng/ml) vitamin D in terms of both disease and extent patterns (p=0.11 and 0.37, respectively).When all participants were divided into 3 groups in terms of vitamin D levels: normal, borderline low (20-29.9 ng/ml) and low (<20ng/ml), no statistically significant difference was found in terms of disease and extent patterns (p=0.17 and 0.33, respectively).

Conclusion In our study, no statistically significant connection was found between the disease and the extent of involvement in terms of mean vitamin D levels and low vitamin D levels.

Key words
Alopecia areata; Vitamin D; $25(OH)D_3$.

Introduction

Alopecia is divided into two groups: scarring and scarless. Alopecia areata (AA) is among the non-scarring alopecias. It is a hair loss that suddenly appears in the hair anywhere on the body, especially the scalp, and is seen as sharply defined, round or oval areas without any visible signs of inflammation on the skin. Alopecia areata is observed at a rate of 0.1-0.2% in the general population.[1] It is most commonly observed between the ages of 10 and 40[2] and can be seen in anyone, regardless of race or gender.[3] Although many findings indicate an immunological origin, neither the trigger nor the pathogenic mechanism are clear.[4,5] Etiological factors include the genetic structure of the patient, atopic condition, organ-specific autoimmune reactions, infections, neuropeptides, psychological factors, and environmental factors.[6] Family history is present in 4-28% of patients with AA.[7] The relationship

Address for correspondence
Dr. Sabir Hasanbeyzade, MD,
Dermatovenerology Specialist,
Dermatology and Venerology Department,
Gulhane Millitary Medical Academy Hospital,
Ankara, Turkey.
Email: dr.sabir.hasanov@gmail.com

of AA with HLA class II genes, especially HLA-DR4, DR5 and DQ3, has been demonstrated.[6,7] Hair loss in patients is accompanied by intrafollicular CD8+ and perifollicular CD4+ lymphocytic infiltrate and a decrease in the number of T-suppressor cells.[8]

The incidence of autoimmune diseases such as thyroid diseases and vitiligo, as well as autoimmune glandular syndrome, pernicious anemia, systemic lupus erythematosus, rheumatoid arthritis, ulcerative colitis, lichen planus, myasthenia gravis, celiac disease and polyendocrinopathy syndrome is increased in patients with AA.[9-12] Thomas *et al*.[13] found the frequency of thyroid disease to be 3.2% in the control group and 18.3% in 71 AA patients. The relationship between vitiligo and AA was determined to be 4% in one study.[9]

In the treatment of AA, pharmacological treatments such as topical, systemic or intralesional corticosteroids, minoxidil, immunomodulators, anthralin, cyclosporine A, sulfosalazine, interferon, tacrolimus, pimecrolimus, topical immunotherapy, photochemotherapy, psychotherapy and patient education are recommended, and cosmetic approaches such as local hair accessories can be used.[14-16]

Vitamin D (vit. D) has two sources in the human body. 10-20% of the total vit. D in the human body is taken with food as ergocalciferol (vitamin D_2). The remaining 80-90% of vit. D is cholecalciferol (vitamin D_3), which is synthesized from 7-dehydrocholecalciferol (which is a soluble prohormone with a secosteroid structure) in the epidermis layer of the skin, through ultraviolet-B (UV-B) in the 290-315 nm wavelength of the solar spectrum. Vitamin D_3 coming from the skin and vitamin D_2 entering the body from the gastrointestinal tract through nutrition is converted into 25-hydroxy-vitamin D_3 [$25(OH)D_3$] in the liver parenchyma by the 25-hydroxylase enzyme. $25(OH)D_3$ is the major circulating vit. D metabolite and is the best indicator of serum vit. D.

The role of vit. D in autoimmune diseases has been mentioned in different studies. In this study, we investigated whether there was a difference between AA patients over the age of 18 and the healthy control group in terms of vit. D levels and deficiency, as well as whether there was a difference in the AA group in terms of disease involvement type and vit. D levels and deficiency.

Material and Methods

Ethical approval was received for the study from institutional Clinical Ethics Committee. Participants in the patient and control groups were verbally informed about the study and signed informed consent forms.

Forty one AA patients, over the age of 18, who were not pregnant, who applied to the Dermatovenerology outpatient clinic between 08.03.2016 and 31.05.2016 and agreed to participate in the study by signing the consent form, were included in the study. The control group included 41 people over the age of 18 who applied to the outpatient clinic for reasons other than AA, who were not pregnant and who were matched with the patient group in terms of age and gender.

Those with a history of drug use that may affect bone metabolism, such as calcium and vitamin D, in the last 6 months were not included in the study. We also did not include patients with a BMI (Body Mass Index) >25 as it may affect vitamin D levels.

Informed consent for the study was obtained

from the control group and the patients. Blood samples of the control group and the patients were taken venously between 08:00 and 09:00 in the morning, into a 10 ml red-capped tube and a 4-ml purple-capped EDTA tube. Vitamin D measurement was made by liquid chromatography mass spectrometry (Liquid Chromatography Mass Spectrometry/ Mass Spectrometry: LC-MS/MS) method. Patients were also asked to fill out questionnaires (to collect information about gender, age, disease duration and number of attacks, and family history).

Data collected from the patient and control groups were analyzed on a computer using the SPSS 15.0 (Statistical Package for the Social Science for Windows v. 15.0, Chicago, Illinois, USA) package program. The results were expressed as numbers and percentages for discrete variables. For continuous variables the results were expressed as mean±standard deviation (m±SD) for those with normal distribution, and as median, lowest (min.) and highest (max.) values for those that did not comply with normal distribution. Whether numerical variables followed a normal distribution was examined using Shapiro-Wilk and Kolmogorov-Smirnov tests. Student-t test, one of the parametric tests, was used to compare 2 groups in terms of continuous variables with normal distribution, analysis of variance-ANOVA in independent groups when comparing more than two groups. Mann-Whitney U test, which is a non-parametric test, was used to compare 2 groups for continuous variables that do not show normal distribution, and the Kruskal Wallis for more groups. Chi square test was used to compare data in discrete variable structure. The existence of correlation between variables was examined with Spearman (for discrete variables) and Pearson (for continuous variables) correlation analysis. Results were evaluated at a 95% confidence interval and were considered statistically significant when alpha error was less than five percent ($P < 0.05$).

Results

The study was conducted on 82 volunteers, 41 patients and 41 controls. There were six (14.6%) women and thirty-five (85.4%) men in the patient group, and five (12.2%) women and thirty-six (87.8%) men in the control group. The age of the participants was 26.8±7 years in the patient group, and 26.9±6.9 years in the control group. No statistically significant difference was found between the control and patient groups in terms of gender and age (p=0.75 and 1, respectively **Table 1**). The number of participants who stated that they had any stress factor in their lives was 16 in the control group and 28 in the patient group. Participants in the control group did not describe any other autoimmune diseases. Eight (19.5%) of the participants in the patient group had additional autoimmune diseases.

When the control and patient groups were evaluated in terms of serum vit. D, calcium, phosphorus, albumin and parathormone (PTH), no significant difference was found between the 2 groups in terms of the measured parameters. While the mean value for serum vit. D in the patient group was 32.1±19.5 ng/ml, it was 39.6±15.7 ng/ml in the control group (p = 0.06). The values are summarized in **Table 2**.

Laboratory tests were also evaluated in groups classified according to the type of disease

Table 1 General characteristics.

	Patient (n=41) *n (%)*	*Control (n=41)* *n (%)*	*P value*
Woman/man	6/35 (14.6/85.4)	5/36 (12.2/87.8)	0.75
Age (mean±SD)	26.85±7	26.88±6.95	1
Stress	26 (63.4)	16 (39)	0.03
Autoimmune disease	8 (19.5)	0 (0)	0.003

Table 2 Vitamin D and other measurements in the patient and control groups.

Mean±SD	*Patient (n=41)*	*Control (n=41)*	*P value*
Ca[1] (mg/dl)	9.9±0.3	9.9±0.4	0.28
P[2] (mg/dl)	4.4±2.4	4.7±2.2	0.13
Alb[3] (g/dl)	4.6±0.3	4.7±0.3	0.29
PTH[4] (pg/ml)	32.6±11.3	32.1±11.6	0.65
Vit. D (ng/ml)	32.1±19.5	39.6±15.7	0.058

[1]Calcium, [2]Phosphorus, [3]Albumin, [4]Parathormone.

involvement (patchy AA, diffuse AA, alopecia totalis [AT] and alopecia universalis [AU]). No statistically significant difference was found between the groups separated by involvement in terms of serum calcium, phosphorus, albumin, PTH and vit. D (p=0.35/ 0.27/ 0.11/ 0.76/ 0.72, respectively). Serum vitamin D values were an average of 27.7±12.6 ng/ml in the patchy AA group, 32±15.6 ng/ml in the diffuse AA group, 35.5±30.7 ng/ml in the AT group and 33.7±16.9 ng/ml in the AU group **(Table 3)**.

All study participants were divided into 2 groups in terms of vit. D levels: normal (≥30 ng/ml) and low (<30 ng/ml). It was examined whether there were differences between the separated groups in terms of disease, poor prognostic factors and disease involvement patterns only in the patients, and no difference was found **(Table 4)**.

All study participants were divided into three groups in terms of vit. D levels: normal (≥30 ng/ml), borderline low or deficiency (20-29.9 ng/ml), low or insufficiency (<20 ng/ml). It was examined whether there were any differences

Table 3 Vitamin D and other measurements according to the type of involvement.

Median (min-max)	*Type of involvement*				*P value*
	Patchy AA[1]	*Diffuse AA[1]*	*AT[2]*	*AU[3]*	
Ca (mean±SD)	9.9±0.4	9.9±0.3	9.8±0.2	10±0.3	0.36
P	4.1 (3-16.6)	3.9 (3-5.7)	3.9 (3.1-5.1)	3.5 (3-4.2)	0.27
Alb	4.7 (4.2-5)	4.7 (4.2-5)	4.6 (4.3-4.8)	4.9 (4.3-5)	0.11
PTH	31.5 (12.4-63.8)	32.5 (15.7-57.7)	32 (25.1-66.4)	30.8 (16.2-37.8)	0.76
Vit. D	31.8 (4.3-42.6)	34.8 (7.6-53.2)	26.4 (15.7-120.4)	40.9 (5.6-49.9)	0.72

[1]Alopecia Areata, [2]Alopecia Totalis, [3]Alopecia Universalis.

Table 4 The relationship between low vitamin D and disease, poor prognostic factors and involvement patterns.

		Vitamin D levels n(%)		*P value*
		≥30ng/ml	*<30ng/ml*	
Patient		22 (53.7)	19 (46.3)	0.1
Control		29 (70.7)	12 (29.3)	
Gender	Man	41 (57.7)	30 (42.3)	0.032
	Woman	10 (90.9)	1 (9.1)	
Age (mean±SD)		26.9±7.1	26.8±6.7	0.87
Autoimmune disease	Yes	17 (51.5)	16 (48.5)	0.43
	No	5 (62.5)	3 (37.5)	
Family history	Yes	1 (100)	0 (0)	0.54
	No	21 (52.5)	19 (47.5)	
Nail involvement	Yes	3 (37.5)	5 (62.5)	0.27
	No	19 (57.6)	14 (42.4)	
Atopic dermatitis	Yes	1 (50)	1 (50)	0.72
	No	21 (53.8)	18 (46.2)	
Duration of illness [median (min-max)]		6 m[1] (1 m[1]-15 y[2])	1 y[2] (1 m[1]-19 y[2])	0.64
Number of attacks [median (min-max)]		1(1-4)	1 (1-5)	0.76
Patchy alopecia areata		7 (63.6)	4 (36.4)	0.37
Diffuse alopecia areata		7 (63.6)	4 (36.4)	
Alopecia totalis		3 (30)	7 (70)	
Alopecia universalis		5 (55.6)	4 (44.4)	

[1] month, [2] year

Table 5 The relationship between vitamin D subgroups and disease, poor prognostic factors and involvement patterns.

		Vitamin D levels n(%)			*P value*
		>30ng/ml	*20-29.9 ng/ml*	*<20ng/ml*	
Patient		22 (53.7)	9 (22)	10 (24.4)	0.17
Control		29 (70.7)	8 (19.5)	4 (9.8)	
Gender	Man	41 (57.7)	17 (23.9)	13 (18.3)	0.09
	Woman	10 (90.9)	0 (0)	1 (9.1)	
Age [median (min-max)]		26 (17-51)	26 (17-33)	27 (21-51)	0.60
Stress	Yes	26 (61.9)	8 (19)	8 (19)	0.85
	No	25 (62.5)	9 (22.5)	6 (15)	
Autoimmune disease	Yes	5 (62.5)	2 (25)	1 (12.5)	0.68
	No	17 (51.5)	7 (21.2)	9 (27.3)	
Family history	Yes	1 (100)	0 (0)	0 (0)	0.64
	No	21 (52.5)	9 (22.5)	10 (25)	
Nail involvement	Yes	3 (37.5)	2 (25)	3 (37.5)	0.54
	No	19 (57.6)	7 (21.2)	7 (21.2)	
Atopic dermatitis	Yes	1 (50)	0 (0)	1 (50)	0.60
	No	21 (53.8)	9 (23.1)	9 (23.1)	
Duration of illness [median (min-max)]		6 m[1] (1 m[1]-15 y[2])	1 y[2] (1 m[1]-19 y[2])	10 m[1] (3 m[1]-5 y[2])	0.88
Number of attacks [median (min-max)]		1 (1-4)	1 (1-5)	1.5 (1-3)	0.95
Patchy alopecia areata		7 (63.6)	1 (9.1)	3 (27.3)	0.33
Diffuse alopecia areata		7 (63.6)	1 (9.1)	3 (27.3)	
Alopecia totalis		3 (30)	5 (50)	2 (20)	
Alopecia universalis		5 (55.6)	2 (22.2)	2 (22.2)	

[1] month, [2] year.

between the separated groups in terms of disease, poor prognostic factors and disease involvement patterns only in the patients, and no difference was found **(Table 5)**.

Discussion

There were a total of 41 patients in the study, 6 (14.6%) females and 35 (85.4%) males. In fact, alopecia areata disease affects both genders equally. The reason for the higher number of male patients in this study could be that our hospital was a military hospital and that female patients came to the hospital less often than male patients due to the cosmetic problem of the disease.

Only 3 (8.6%) of the 35 male patients had beard involvement. Beard involvement is seen at a rate of 25-30% (124) in alopecia areata, and the value we found was lower than the general literature information. The reason for this may be that this hospital is a military hospital, soldiers and officers are obliged to shave their beards daily, and therefore they cannot notice the lesions on the beard, especially small lesions.

The patient and control groups, as well as four groups separated according to the type of involvement, were compared in terms of serum calcium, albumin, phosphorus, PTH and vit. D values. There was no significant difference between the groups for any of these values.

When all participants in the control and patient groups were divided into 2 groups, normal and low (<30 ng/ml), according to serum vit. D levels, there was no statistically significant difference between these two groups in terms of demographic characteristics, poor prognostic factors and type of involvement, except for gender. There were fewer women in the low vit. D group. There is no information that low serum vitamin D is less common in women.[17] To

investigate this issue in more detail, larger studies with larger participant groups are needed.

Gade *et al*.[18] found a significant decrease in vitamin D levels in the patients (p=0.001) and a negative correlation between vitamin D and disease severity (p=-0.714, p=0.001) in their case-control study with 45 people in each group. The results of the current study contradict these results.

Marahatta *et al*.[19] found the rate of 25-hydroxyvitamin D deficiency to be higher in the patients in their case-control study (AA group with an average age of 28.37±10.070 and 16 of whom were male, and a control group with an average age of 30.50±9.032 and 15 of whom were male) with 30 people in each group (p=0.01). Vit. D levels were lower in the AA group compared to the control group (12.84 and 29.5, p=0.06). The results of the current study do not coincide with these results.

Rehman *et al*.[20] in their case-control study with 135 people in each group (the mean age was 26±12.89 in the AA group, 26±13.20 in the control group, 91 males and 44 females in each group), they found vitamin D levels in the AA group to be lower (p=0.0004), and there were more participants with vitamin D deficiency in the patient group (98 participants in the patient group and 78 participants in the control group, p=0.01). They found a negative correlation between disease pattern and vit. D levels. The results of the current study do not coincidence with these results.

Siddappa *et al*.[21] in their case-control study with 100 people in each group (age 24.52±10.06 in the AA group, 28.96±11.49 in the control group), they found the mean vit. D levels to be lower in the AA group than in the controls (18.90±8.32 and 28.21±18.32 ng/ml, respectively, p<0.001). Likewise, they observed vitamin D deficiency more frequently in the patients than in the control group (64 patients and 38 controls, p <0.001). The results of the study do not coincide with these results.

Daroach *et al*.[22] in their case-control study with 30 people in each group (age 28.9±9.96 in the AA group, 31.17±9.43 in the controls), found that vit. D levels were 7.65±4.50 ng/ml in the patients and 15.8±11.47 ng/ml in the controls and found difference between the 2 groups in terms of vit. D deficiency (<20 ng/ml, 27 (96.7%) participants in the AA group, 22 (73.3%) participants from controls, p= 0.001). The results of the current study are not similar to these results.

Erpolat *et al*.[23] found no statistically significant difference between the AA and control groups in terms of mean serum vit. D levels and low vitamin D levels in their case-control study with 41 people in each group. The current study showd simila results.

In the study conducted by Yılmaz *et al*.[24] in 2012, 42 participants were included in the patient group (mean age 30.8±8.2, 28 women) and 42 participants were included in the control group (mean age 29.3±7.4, 29 women) and both $1,25(OH)_2D_3$ and $25(OH)D_3$ levels were measured. In the patient group, $1.25(OH)_2D_3$ level was found 40.5±12.8 pg/ml and the $25(OH)D_3$ level was 33.4±17.7 nmol/l, and in the control group, the $25(OH)D_3$ level was found to be 51.2±21.1nmol/l, $1.25(OH)_2D_3$ level was 56±18.4 pg/ml. In the study, a significant difference was found in terms of both values between the 2 groups (p<0.001 for both measurements) and it was shown that the values in AA patients were lower. No difference was found between $1,25(OH)_2D_3$ and $25(OH)D_3$ levels and clinical parameters such as disease duration, nail involvement and severity of

involvement. The current study showed similar results. Autoimmune disease was associated with 8 (19%) AA patients (p<0.001). Similarly, Kılıç *et al*.[25] also found coexistence of autoimmune disease in 17 (13.6%) of AA patients in a study. Ahmed *et al*.[26] also showed the coexistence of AA and autoimmune disease in their own study. In this study, while no accompanying autoimmune disease was described in the control group of 41 people, 8 people (19.5%) in the patient group of 41 described an additional autoimmune disease.

Ogrum *et al*.[27] compared the case group included 40 (21 female, 19 male) alopecia areata patients with a mean age of 31.23±7.34 (21-50) and the control group included 40 (21 female, 19 male) healthy participants in terms of alopecia areata with a mean age of 30.58±7.19 (21-48) in term of 25(OH)D_3 levels, it found no difference between the 2 groups (p>0.05). The significant variable was found to be low at a rate of 97.5% in both groups. No difference was found in terms of gender in 25(OH)D_3 levels (p>0.05). When vit. D levels were grouped as deficient (15-30 ng/ml) and insufficient (<15 ng/ml), no difference was detected between the groups (p >0.05). We did not find any significant difference in vit. D levels between the patient and control groups. However, unlike this study, in our study, we found that there were statistically significantly fewer female patients in the group with low vitamin D.

Conclusion

The current study found, there was no difference in vit. D levels between the groups (patients and controls), as well as between groups separated according to the type of involvement. There was no difference in the groups separated according to vitamin D levels, in terms of disease and involvement type, too. Some of the previous studies on this subject found a relationship between the disease and vit D or between the type of involvement and vit. D, but some did not find a relationship. Therefore, to clarify this issue multicenter studies with larger samples are needed.

Limitations of the study

A deficiency in this study is that the participants' sunscreen use, daily outdoor exposure time, and dressing habits were not questioned. Additionally, the results of our study cannot be generalized to other countries, since the climate and the duration of sunshine will be different in different latitudes.

References

1. Finner AM. Alopecia areata: Clinical presentation, diagnosis and unusual cases. *Dermatol Ther*. 2011;**24(3)**:348-54.
2. De Berker DAR, Messenger AG, Sinclair RD. Disorders of hair. In: Burns T, Breathnach S, Cox N, Griffiths C (Eds). Rook's Textbook of Dermatology. 7th ed. Oxford: Blackwell Science Ltd; 2004. p.3201–321.
3. Rivitti EA. Alopecia areata: a revision and update. *An Bras Dermatol*. 2005;**80(1)**:57-68.
4. Balevi Ş. Alopesi areata. *T Klin Dematol J Int Med Sci*. 2006;**2(31)**:1-18.
5. Wolff H. Diseases of hair. In: Burgdorf WHC, Plewig G, Wolff HH, Landthaler M (Eds). Braun-Falco's Dermatology. 3rd ed. Heidelberg: Springer Medizin Verlag; 2009. p.1029-59.
6. Arca E, Kurumlu Z. Alopesi areatada etyopatogenez, klinik ve tanı. *Dermatose* 2003;**2(3)**:83-9.
7. McDonagh SJ, Tazi-Ahnini R. Epidemiology and genetics of alopecia areata. *Clin Exp Dermatol*. 2002;**27(5)**:405-9.
8. Kalish RS, Gilhar A. The immunology of alopecia areata and potential application to novel therapies. *Dermatol Ther*. 2001;**14(4)**:322-8.
9. Serarslan G, Savaş N, Yenin JZ. Is atopy and autoimmunity more prevalent in patients with alopecia areata? A comparative study. *J*

Eur Acad Dermatol Venereol. 2012;**26(6)**:720-3.
10. Green J, Sinclair RD. Genetics of alopecia areata. *Australas J Dermatol*. 2000;**41(4)**: 213-8.
11. Kılınç I, Alper S, Ceylan C. Alopesi areatalı olgularda hasta profili: Bir retrospektif çalışma. *Ege Tıp Derg*. 2002;**41(1)**:25-7.
12. Wasserman D, Guzman-Sanchez DA, Scott D, McMicheal A. Alopecia areata. *Int J Dermatol*. 2007;**46**:121-31.
13. Thomas EA, Kadyan RS. Alopecia areata and autoimmunıty: a clinical study. *Indian J Dermatol*. 2008;**53(2)**:70-4.
14. Aktaş E. Alopesi areata tedavisindeki yenilikler. *T Klin Dermatol*. 2005;**1(48)**:103-8.
15. Arca E, Kurumlu Z. Alopesi areatada tedavi. *Dermatose*. 2003;**2(3)**:151-8.
16. Gürkan A, Serdaroğlu S, Oğuz O. Ofiyazik modelli alopesi areata hastalarının tedavisinde skuarik asit dibütilester'in (Sadbe) etkinliği. *Dermatose*. 2010;**1(1)**:9-13.
17. Rajiv M, Singh NR. Bexarotene Gel: A New Topical Therapy for Alopecia Areata. *Int J Trichol*. 2010;**2(1)**:66–7.
18. Gade V.K.V., Mony A., Munisamy M., Chandrashekar L., Rajappa M. An investigation of vitamin D status in alopecia areata. *Clin Exp Med*. 2018;**18(4)**:577-584.
19. Marahatta S, Agrawal S, Khan S. Study on serum vitamin D in alopecia areata patients. *J Nepal Health Res Counc*. 2019;**17**:21–5.
20. Rehman F, Dogra N, Wani MA. Serum vitamin D levels and alopecia areata—a hospital based case—control study from North-India. *Int J Trichol*. 2019;**11**:49–57.
21. Siddappa H, Kumar YH, Vivekananda N. Evaluation of association of vitamin D in alopecia areata: A case–control study of 100 patients in a tertiary rural hospital of Southern India. *Indian Dermatol Online J*. 2019;**10**:45-9.
22. Daroach M, Narang T, Saikia UN, Sachdeva N, Sendhil Kumaran M. Correlation of vitamin D and vitamin D receptor expression in patients with alopecia areata: a clinical paradigm. *Int J Dermatol*. 2018;**57**:217–22.
23. Erpolat S, Sarifakioglu E, Ayyildiz A. 25-hydroxyvitamin D status in patients with alopecia areata. *Postepy Dermatol Alergol*. 2017;**34**:248-52.
24. Yilmaz N, Serarslan G, Gokce C. Vitamin D concentrations are decreased in patients with alopecia areata. *Vitamins & Trace Elements*. 2012;**1**:105-9.
25. Kiliç S, Ersoy F, Sanal O, Türkbay D, Tezcan I. Alopecia universalis in a patient with common variable immunodeficiency. *Pediatr Dermatol*. 1999;**16**:305-7.
26. Ahmed I, Masreen S, Bhatti R. Alopecia areata in children. *J Coll Psysicians Surg Pak*. 2007;**170**:587-590.
27. Oğrum A, Nermin B, Arzu KT, Seval K, Hatice ME. Alopesi areatalı hastalarda 25 hidroksi vitamin D_3 düzeyinin değerlendirilmesi. *Türkderm*. 2015;**49**:50-3.