

ORIGINAL ARTICLE

Medicine Science 2023;12(3):859-64

Does vitamin D deficiency exacerbate hidradenitis suppurativa?

ID Erdal Pala¹, ID Omer Karasahin²¹Atatürk University, Faculty of Medicine, Department of Dermatology and Venerology, Erzurum, Türkiye²Erzurum Regional Training and Research Hospital, Department of Infectious Diseases, Erzurum, Türkiye

Received 24 June 2023; Accepted 15 August 2023

Available online 25.08.2023 with doi: 10.5455/medscience.2023.06.098

Copyright@Author(s) - Available online at www.medicinescience.org

Content of this journal is licensed under a Creative Commons Attribution-NonCommercial-NonDerivatives 4.0 International License.



Abstract

In this study, we aimed to compare vitamin D (25 Hydroxy Vitamin D (25 OH D)) levels in Hidradenitis Suppurativa (HS) patients with healthy control groups, and to investigate its possible relationship with disease severity. Our study is a retrospective cross-sectional study. A total of 36 patients diagnosed with HS who presented to our Dermatology clinic between January 2019 and December 2022 were included. Vitamin D levels were recorded by reviewing the patients' files and compared with healthy control groups. The relationship between the patients' 25 OH D levels and disease severity score was determined. The median age of the patient group was 33.5 (IQR; 25.2-42) years, and 30 (83.3%) were male. The median age of the healthy control group was 34.5 (IQR; 24-42), and 21 (58.3%) were male. In patients with severe 25 OH D deficiency, statistically significant findings were observed with a higher frequency of Hurley-3 disease severity score, whereas patients with mild to moderate deficiency or normal 25 OH D levels showed a higher frequency of disease severity classified as Hurley-1. ($p=0.022$). Additionally, patients with severe vitamin D deficiency had significantly higher C-reactive protein levels and body mass index ($p=0.036$ and $p=0.044$, respectively). Among patients with active lesions such as abscesses, nodules, and fistula, significantly lower vitamin D levels were observed compared to those without such lesions ($p=0.020$, $p=0.032$, and $p=0.017$, respectively). Vitamin D level in patients with HS (13.42 ng/mL (IQR: 8.00-18.65)) compared to the healthy control group (18.00 ng/mL (IQR: 14.12-22.87)) was statistically significant. was significantly lower ($p=0.001$). Vitamin D levels of HS patients are lower than healthy control groups. Inflammatory lesions are more common in patients with severe vitamin D deficiency and the disease tends to be more severe.

Key words: Hidradenitis suppurativa, vitamin D deficiency, severity of illness

Introduction

Hidradenitis Suppurativa (HS) is a chronic, inflammatory disease of varying severity that usually appears after puberty and affects areas of the body where apocrine sweat glands are more abundant. The exact prevalence of the disease is not fully known but is reported to range between 1% and 4% [1]. The lesions of the disease initially start as painful subcutaneous nodules. These nodules can develop into abscesses, and the opening of these abscesses results in the formation of sinuses that produce foul-smelling, purulent, or seropurulent discharge. Ulceration can also occur in abscesses. Double-ended blackheads (pseudocomedones)

are considered pathognomonic for diagnosing the disease. The lesions can heal with fibrosis and band-like scar tissue [2]. While HS primarily affects the axillary, inguinal, and anogenital regions, lesions can also occur in the neck, back, retroauricular area, scrotum, labia, and beneath the breasts. The factors that cause HS have not been determined yet, but it is reported that it may be due to genetic and some environmental factors [3]. The most well-known environmental factors in the etiology of HS are obesity and smoking [4]. In HS, in addition to skin findings, some diseases such as dyslipidemia, polycystic ovary syndrome, psychiatric disorders, cardiovascular diseases and thyroid

CITATION

Pala E, Karasahin O. Does vitamin D deficiency exacerbate hidradenitis suppurativa?. Med Science. 2023;12(3):859-64.



Corresponding Author: Erdal Pala, Atatürk University, Faculty of Medicine, Department of Dermatology and Venerology, Erzurum, Türkiye
Email: erdalpala2525@gmail.com

disorders can be seen [5]. The pathophysiology of the disease has not been fully clarified yet. The basis of the pathogenesis of HS is inflammation resulting from the obstruction of the hair follicle. After occlusion, follicular dilatation and bacterial colonization occur. HS is not an infectious disease, but bacteria play an important role in the pathogenesis of the disease. After dilation, the follicle ruptures, leading to the release of bacteria and keratin into the dermis, which causes chemotaxis of inflammatory cells consisting of lymphocytes and neutrophils. Damage occurs in the pilosebaceous unit and surrounding adnexal structures in conjunction with abscess formation [6]. In HS, there are also disruptions in both innate and adaptive immune responses that contribute to the pathogenesis. Some studies have shown that there are irregularities in the natural immunity of the skin, which is the first line of defense against microorganisms. These studies have shown decreased expression of antimicrobial peptides such as Toll-Like Receptors (TLR) 2, 3, 4, 7, 9, β -defensin 2, 3, 4, ribonuclease 7, in keratinocytes [7,8].

A significant portion of vitamin D (25 Hydroxy Vitamin D (25 OH D)) is synthesized in keratinocytes in the skin. Vitamin D is an important immune system modulator that plays a role in both innate and acquired immunity. It inhibits cell proliferation and has the ability to support antimicrobial activity in the innate immune system while reducing inflammatory activity and the onset of acquired immune system response. It particularly regulates the immune system by suppressing T-cell proliferation. Vitamin D plays a role in the regulation of the innate immune system by increasing the synthesis of TLRs and antimicrobial peptides [9].

Dysregulation in the innate immune system is important in the pathogenesis of HS. Considering the immunomodulatory properties of vitamin D, in this study, we aimed to compare the effect of vitamin D levels of patients with HS on the disease severity score and to compare the vitamin D levels of the patient group and healthy control groups.

Material and Methods

Study design

This study was conducted in a single center and was designed as a retrospective, cross-sectional.

Ethical approval

Our study was carried out in accordance with the Declaration of Helsinki Rules after the approval of the local ethics committee (Dated 29.12.2022 and numbered 6).

Setting

Our study was carried out in the dermatology outpatient clinic of a tertiary university hospital serving approximately 4.5 million

people in the eastern part of Turkey. The study included patients diagnosed with HS who presented to the clinic between January 2019 and December 2022.

Participants and study protocol

A total of 36 patients diagnosed with HS who presented to our hospital's outpatient clinic between January 2019 and December 2022 were included in the study, along with a control group of 36 healthy with similar demographic characteristics. The age range of the patient group was between 18-70 years. The patients' medical records were retrospectively reviewed, and information such as age, gender, socio-demographic characteristics, body mass index (BMI), smoking status, existing lesions at the time of presentation, and disease severity scores were recorded. Patients under the age of 18, pregnant and lactating women, patients with a history of another inflammatory disease, those who had received vitamin D supplementation within the last 6 months, and patients with chronic kidney and liver failure were excluded from the study. Disease severity scoring was performed based on the presence of lesions, categorizing the patients as Hurley 1, 2, or 3. Hurley 1 was assigned to patients with solitary/multiple abscesses without fistula tracts or scarring, Hurley 2 was assigned to patients with solitary/multiple recurrent separate abscesses with fistula tracts and scarring, and Hurley 3 was assigned to patients with multiple interconnected fistula tracts and abscesses in the affected area. The reference values for Vitamin D (25-hydroxycholecalciferol) for the patient and control groups were determined based on our hospital's laboratory values. Evaluation of vitamin D levels was performed with the Cobas 702 biochemistry analyzer (Roche® Diagnostics, Mannheim, Germany) using the electrochemiluminescence immunoassay method. Accordingly, if the vitamin D level was below 10 ng/mL, severe deficiency, 10–24 ng/mL, mild moderate deficiency, and 25–80 ng/mL were considered normal. Since the study was conducted retrospectively and it was designed as an archive scan of all patients' files, it was not necessary and not possible to obtain informed consent from the patients.

Statistical analysis

All data were entered into statistical package program SPSS for Windows v2 (SPSS Inc., Chicago, IL, USA) and analyzes were made using the same package program. The Kolmogorov-Smirnov test was used to assess whether continuous variables followed a normal distribution. Accordingly, continuous variables were presented as median (interquartile range, IQR). Categorical variables from the descriptive data were presented as frequency distribution and percentage. The chi-square test was used to compare categorical variables between groups, and Fisher's exact test was used when the expected count in any cell was less than 5. For the comparison of continuous variables,

non-parametric tests, specifically the Kruskal-Wallis and Mann-Whitney U tests, were employed due to the failure to meet the assumptions of parametric hypothesis testing. A p-value of less than 0.05 was considered statistically significant.

Results

The median age of our patients was 33.5 (IQR: 25.2-42) and 30 (83.3%) were male. The main characteristics of those in the patient group are presented in Table 1. Distribution of regions where active lesions are located is presented in Figure 1. The distribution of regions where active lesions are located is presented in Figure 1. Active lesions were detected in 94.4% (n=34) patients, with a total of 87 lesions identified. The most common locations for active lesions were axilla 33.3% (n=29), inguinal area 23.0% (n=20), and gluteal region 17.2% (n=15). Among these active lesions, 112 different manifestations were observed, with abscess 29.5% (n=33), scar 24.1% (n=27), and nodules 22.3% (n=25) being the most common findings. The distribution of the basic characteristics of the patient group according to the vitamin D level groups is shown in Table 2. Patients with severe vitamin D insufficiency had higher Hurley-3 disease severity scores, while patients with mild-moderate deficiency or normal levels had significantly higher Hurley-1 disease severity scores (p=0.022). Additionally, patients with serious vitamin D insufficiency had significantly higher BMI and C-reactive protein (CRP) levels (p=0.044 and p=0.036, respectively). The relationship between disease severity and vitamin D level, smoking and BMI was assessed in Figure 2. There was a statistically significant relationship between disease severity scores and vitamin D levels (p=0.008). Specifically, patients with Hurley-1 had significantly higher vitamin D levels compared to Hurley-2 and Hurley-3 (p=0.021 and p=0.003, respectively). While disease severity scores increased with increasing BMI, the difference was not statistically significant (p=0.476). No statistically significant difference was found between the disease severity score and smoking status (p=0.755). The distribution of the relationship between vitamin D levels and the anatomical localization and type of active lesions is presented in Table 3. There was no significant difference in vitamin D levels based on the presence or absence of active lesions in any localization (p>0.05). However, in cases with active lesions of abscess, nodules, and fistulas, markedly lower vitamin D levels were observed compared to those without active lesions (p=0.020, p=0.032, and p=0.017, respectively). The median age of the healthy control group was 34.5 years (IQR: 24-42), and 58.3% (n=21) were male. The vitamin D levels of the healthy control group were compared to the patient group and are presented in Figure 3. The patient group (13.42 ng/mL (IQR: 8.00-18.65)) had a lower vitamin D level compared to the healthy control group (18.00 ng/mL (IQR: 14.12-22.87)) (p=0.001).

Table 1. Basic characteristics of patients

Variables	
Age, years, median (IQR)	33.5 (25.2–42)
Gender, male, n (%)	30 (83.3)
Duration of symptoms, months, median (IQR)	60 (24–120)
Age of disease onset, years, median (IQR)	24 (20–33)
Smoking status, present, n (%)	27 (75.0)
Duration of smoking, years, median (IQR)	12 (6–18)
Family history, present, n (%)	4 (11.1)
Severe acne history, present, n (%)	8 (22.2)
Height (cm), median (IQR)	174 (169–176)
Weight (kg), median (IQR)	79.5 (75.2–85.7)
BMI (kg/m²), median (IQR)	27 (24.8–27.9)
Presence of active lesions, present, n (%)	34 (94.4)
Disease activity, active, n (%)	34 (94.4)
Presence of comorbidities, n (%)	
Diabetes mellitus	4 (11.1)
Hypertension	3 (8.3)
Pilonidal sinus	2 (5.6)
Psychiatric illness	2 (5.6)
Joint disease	1 (2.8)
Disease severity score, n (%)	
Hurley–1	10 (29.4)
Hurley–2	12 (35.3)
Hurley–3	12 (35.3)
Laboratory values, median (IQR)	
ESR	10 (5–24.5)
CRP	11.4 (3.2–31.8)
WBC count	10.48 (8.52–11.98)
Vitamin D	13.4 (8.0–18.6)

BMI: body mass index, CRP: c-reactive protein, ESR: erythrocyte sedimentation rate, WBC: white Blood Cell

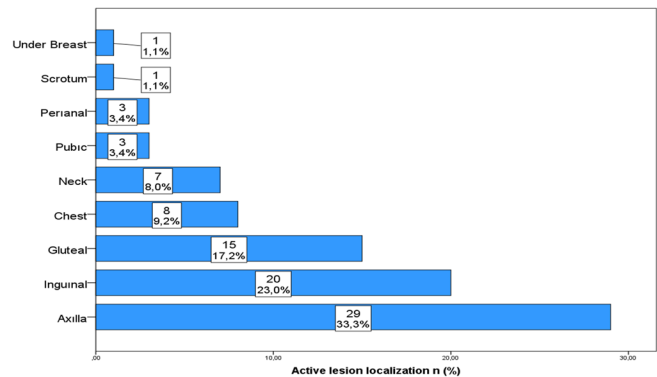


Figure 1. Distribution of active lesion locations

Table 2. Distribution of basic characteristics of patients according to vitamin D level groups

Variable	Severe Deficiency (<10ng/mL)	Mild-Moderate Deficiency or Normal (≥10ng/mL)	P-value
Age (years)	33 (22.5-39)	36 (25-42)	0.478
Gender (male, n (%))	10 (76.9)	20 (87.0)	0.369
Duration of symptoms (months)	72 (36-156)	48 (24-120)	0.337
Age of onset of disease (years)	23 (20-31)	29 (19-35)	0.489
Smoking, present, n (%)	9 (69.2)	18 (78.3)	0.414
Duration of smoking (years)	12 (7.5-16)	11 (5.7-20)	0.860
Family history, present, n (%)	1 (7.7)	3 (13.0)	0.541
BMI	27.4 (26.8-28.5)	26.1 (24.6-27.8)	0.044
Severe acne history, present, n (%)	5 (38.5)	3 (13.0)	0.091
Presence of comorbidities, n (%)	6 (46.2)	6 (26.1)	0.195
Presence of active lesions, n (%)	13 (100)	21 (91.3)	0.402
Disease activity, active, n (%)	13 (100)	21 (91.3)	0.402
Disease severity score, n (%)			
Hurley-1	1 (7.7)	9 (42.9)	0.022
Hurley-2	4 (30.8)	8 (38.1)	
Hurley-3	8 (61.5)	4 (19.0)	
Laboratory, median (IQR)			
ESR	13 (6.5-63)	7 (5-22)	0.072
CRP	22.3 (8.3-56.8)	8.8 (2.6-22.5)	0.036
WBC Count	11.21(9.00-11.81)	10.47 (7.83-12.09)	0.284

BMI: body mass index, CRP: c-reactive protein, ESR: erythrocyte sedimentation rate, WBC: white blood cell

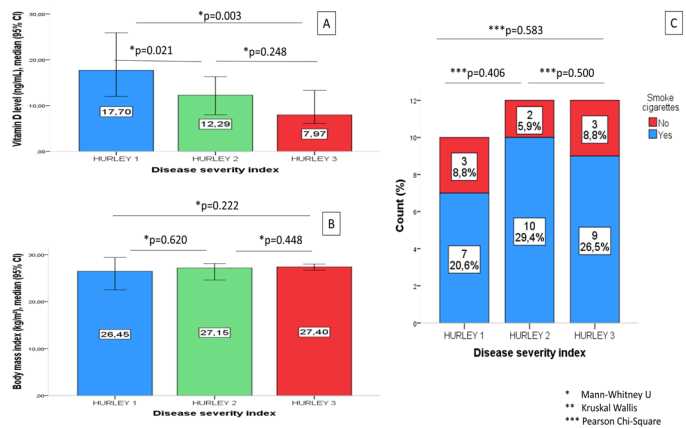


Figure 2. Relationship between disease severity and vitamin D levels (A), body mass index (B), and smoking status (C)

Table 3. Distribution of lesion type and anatomical localization according to vitamin D level

Localization	D vitamin level (ng/mL)		P-value
	Active lesion present	No active lesion	
Gluteal	11.0 (7.7–19.6)	14.6 (8.0–18.5)	0.634
Axilla	13.5 (8.0–18.5)	9.3 (5.3–19.6)	0.675
Inguinal	15.1 (8.5–19.0)	9.8 (6.3–17.4)	0.152
Chest	8.0 (5.9–16.7)	14.8 (9.1–18.8)	0.119
Scrotum	9.1	13.5 (8.0–18.8)	0.531
Under breast	1.5	13.5 (8.0–18.8)	0.092
Pubic	16.9 (15.0–22.0)	12.0 (7.9–18.5)	0.161
Perianal	7.9 (5.8–11.0)	14.6 (8.0–18.9)	0.115
Neck	14.6 (8.0–20.3)	13.3 (7.9–18.5)	0.795
Active lesion type			
Abscess	12.0 (7.9 – 16.7)	25.1 (18.2 – 25.9)	0.020
Nodule	10.4 (7.9 – 15.7)	18.2 (12.0 -25.1)	0.032
Pseudocomedone	13.5 (8.0 – 14.0)	13.3 (7.9 – 18.5)	0.710
Papule	10.0 (5.9 – 18.6)	14.0 (8 – 18.6)	0.546
Scar	13.3 (7.9 – 18.5)	15.0 (8.3 – 20.7)	0.559
Fistula	10.3 (6.6 – 15.0)	16.8 (9.8 – 21.5)	0.017

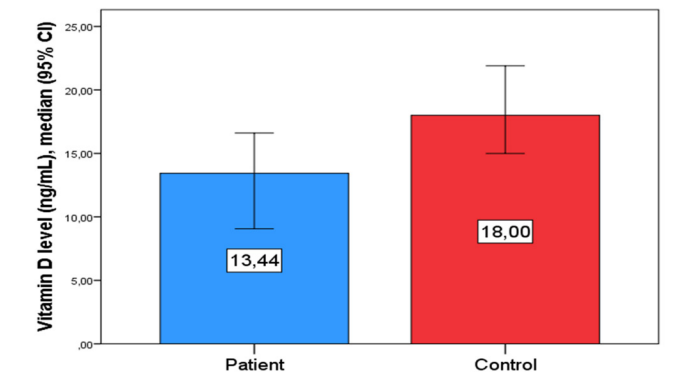


Figure 3. Comparison of vitamin D levels between the healthy control group and the patient group

Discussion

HS is a chronic inflammatory disease with a tendency to recur. While the occlusion of hair follicles is considered the primary reason in its pathogenesis, the involvement of both innate and acquired system dysregulation has also been demonstrated [7]. Vitamin D has an important function in the regulation of proliferation, differentiation, apoptosis, barrier function and innate immunity in the skin, especially in keratinocytes and adnexal structures [9]. Based on this, in our study, we aimed to evaluate the relationship between vitamin D deficiency and disease severity score in patients with HS by comparing their vitamin D levels with those of healthy control groups. There are very few studies evaluating vitamin D levels in patients with HS. According to our knowledge there is no study on this

subject in our country.. Similarly, in the literature, it has been shown that supplementation of vitamin D can lead to a decrease in disease severity in some inflammatory skin diseases such as psoriasis vulgaris and atopic dermatitis [10,11]. This shows the importance of vitamin D function in inflammatory skin diseases. The study evaluating the relationship between HS and vitamin D levels reported that patients with HS had lower vitamin D levels compared to the control group, and the disease severity score (eg Sartorius score), was higher in these patients. This study demonstrated the positive effects of vitamin D supplementation on the clinical condition of the disease [12]. Similarly, in our study, we also demonstrated statistically significant lower vitamin D levels (13.42 ng/mL (IQR: 8.00-18.65)) in patients compared to the control group (18.00 ng/mL (IQR: 14.12-22.87)) ($p=0.001$). However, we did not evaluate the effect of vitamin D administration on the clinical situation in our study. Unlike this study, we used the Hurley classification system for disease severity scoring. In our study, severe vitamin D deficiency was found in 36.1% ($n=13$) of the patients, mild to moderate vitamin D deficiency in 55.6% ($n=20$), and normal vitamin D levels in 8.3% ($n=3$). Vitamin D deficiency is quite common in our country, as it is in many regions around the world [13]. This could be attributed to the geographical conditions, low exposure to sunlight, and high skin photo type of the people in our region. In our study, we hypothesized that the lower vitamin D levels in the patient group compared to the healthy control group may be due to the avoidance of sun exposure by HS patients in public due to their behavior of avoiding undressing in public. In a study examining the relationship between vitamin D levels and disease severity in patients with HS, it was reported that patients with low vitamin D levels had higher disease severity scores [14]. Similarly, in our study, we found that patients with severe vitamin D deficiency had higher frequency of Hurley-3 disease severity score, while patients with mild-moderate vitamin D deficiency or normal levels had higher frequency of Hurley-1 disease severity score. This shows that the more severe the vitamin D deficiency is, the more severe the disease can be in proportion to it. When evaluating the lesion type and anatomical location of the patients, consistent with the literature, we found that lesions were most commonly located in the axilla 33.3% ($n=29$), inguinal region 23.0% ($n=20$), and gluteal area 17.2% ($n=15$). Regarding lesion type, abscesses were the most frequent 29.5% ($n=33$), followed by scars 24.1% ($n=27$) and nodules 22.3% ($n=25$). Unlike the literature, the relationship between vitamin D levels, lesion type and anatomical location was also evaluated in our study. While no statistically significant association was found between the anatomical location of the lesions and vitamin D levels, significant lower vitamin D levels were observed in patients with abscesses, nodules, and fistulas compared to those without these lesions ($p=0.020$, $p=0.032$, and $p=0.017$, respectively). This supports the notion that the number of inflammatory lesions may increase as vitamin D levels decrease. For patients with HS, obesity is a serious risk factor. It is suggested that obesity, due to its increased friction in areas with abundant peripheral fat distribution, can lead to follicular occlusion and initiate

HS [15]. Some studies have reported decreased bioavailability of vitamin D and severe deficiencies in obese patients [16]. In our study, consistent with the literature, statistically significant lower vitamin D levels were found in patients with higher BMI ($p=0.044$). Disease severity increased with increasing BMI, but no statistically significant difference was observed ($p=0.476$). CRP is a highly sensitive positive acute-phase reactant that indicates inflammation and tissue damage [17].

Since HS is a chronic inflammatory disease, elevated CRP levels are expected in these patients. In a study, it was shown that CRP levels were higher in patients with Hurley -3 HS compared to those with Hurley-2 HS [18]. In our study, HS patients with severe vitamin D deficiency had higher CRP levels. This supports the anti-inflammatory activity of vitamin D. In one study, it was mentioned that the administration of vitamin D to HS patients would be beneficial for clinical findings [19]. Another recent study showed that HS patients had lower vitamin D values than a healthy control group [20].

Limitations

First of all, the retrospective nature of the study caused a limitation. Secondly, the small number of patients is another limitation of our study. Thirdly, due to the study being conducted in a single center, the obtained results cannot be generalized, and we believe that prospective studies conducted in multiple centers are needed.

Conclusion

The most important finding in our study is that HS patients have lower vitamin D levels than healthy control groups and that severe vitamin D deficiency is associated with more severe disease. Therefore, in this chronic disease that significantly affects quality of life, we believe that the evaluation of vitamin D levels during initial patient visits should not be overlooked, and vitamin D supplementation should be considered if deficiency is present.

Conflict of Interests

The authors declare that there is no conflict of interest in the study.

Financial Disclosure

The authors declare that they have received no financial support for the study.

Ethical Approval

The study was carried out in accordance with the Helsinki Declaration Rules after approval from the local ethics committee (dated 29.12.2022 and numbered 6).

References

1. Revuz JE, Canoui-Poitrine F, Wolkenstein P, et al. Prevalence and factors associated with hidradenitis suppurativa: results from two case-control studies. *J Am Acad Dermatol.* 2008;59:596-601.
2. Patil S, Apurwa A, Nadkarni N, et al. Hidradenitis suppurativa: inside and out. *Indian J Dermatol.* 2018;63:91-8.
3. Napolitano M, Megna M, Timoshchuk EA, et al. Hidradenitis suppurativa: from pathogenesis to diagnosis and treatment. *Clin Cosmet Investig Dermatol.* 2017;10:105-15.

4. Bukvić Mokos Z, Miše J, Balić A, Marinović B. Understanding the relationship between smoking and hidradenitis suppurativa. *Acta Dermatovenerol Croat.* 2020;28:9-13.
5. Cartron A, Driscoll MS. Comorbidities of hidradenitis suppurativa: a review of the literature. *Int J Womens Dermatol.* 2019;5:330-4.
6. Prens E, Deckers I. Pathophysiology of hidradenitis suppurativa: an update. *J Am Acad Dermatol.* 2015;73:8-11.
7. Hofmann SC, Saborowski V, Lange S, et al. Expression of innate defense antimicrobial peptides in hidradenitis suppurativa. *J Am Acad Dermatol* 2012;66:966-74.
8. Yao Y, Frew JW, Thomsen SF, Ring HC. Antimicrobial peptides in hidradenitis suppurativa: a systematic review. *Br J Dermatol.* 2022;186:236-44.
9. Ismailova A, White JH. Vitamin D, infections and immunity. *Rev Endocr Metab Disord.* 2022;23:265-77.
10. Ingram MA, Jones MB, Stonehouse W, et al. Oral vitamin D3 supplementation for chronic plaque psoriasis: a randomized, double-blind, placebo-controlled trial. *J Dermatolog Treat.* 2018;29:648-57.
11. Ng JC, Yew YW. Effect of vitamin D serum levels and supplementation on atopic dermatitis: a systematic review and meta-analysis. *Am J Clin Dermatol.* 2022;23:267-75.
12. Fabbrocini G, Marasca C, Luciano MA, et al. Vitamin D deficiency and hidradenitis suppurativa: the impact on clinical severity and therapeutic responsiveness. *J Dermatolog Treat.* 2021;32:843-4.
13. Yeşiltepe-Mutlu G, Aksu ED, Bereket A, Hatun Ş. Vitamin D status across age groups in Turkey: results of 108,742 samples from a single laboratory. *J Clin Res Pediatr Endocrinol.* 2020;12:248-55.
14. Guillet A, Brocard A, Bach Ngohou K, et al. Verneuil's disease, innate immunity and vitamin D: a pilot study. *J Eur Acad Dermatol Venereol.* 2015;29:1347-53.
15. van Straalen KR, Vanlaerhoven AMJD, Ardon CB, van der Zee HH. Body mass index at the onset of hidradenitis suppurativa. *J Dtsch Dermatol Ges.* 2021;19:437-9.
16. Wortsman J, Matsuoka LY, Chen TC, et al. Decreased bioavailability of vitamin D in obesity. *Am J Clin Nutr.* 2000;72:690-3. Erratum in: *Am J Clin Nutr.* 2003;77:1342.
17. Wu Y, Potempa LA, El Kebir D, Filep JG. C-reactive protein and inflammation: conformational changes affect function. *Biol Chem.* 2015;396:1181-97.
18. Hoffman LK, Ghias MH, Cohen SR, Lowes MA. Polyclonal hyperglobulinaemia and elevated acute-phase reactants in hidradenitis suppurativa. *Br J Dermatol.* 2018;178:e134-e135.
19. Kelly G, Sweeney CM, Fitzgerald R, et al. Vitamin D status in hidradenitis suppurativa. *Br J Dermatol.* 2014;170:1379-80.
20. Seetan K, Eldos B, Sarairoh M, et al. Prevalence of low vitamin D levels in patients with Hidradenitis suppurativa in Jordan: a comparative cross-sectional study. *PLoSOne.* 2022;17:e0265672.