The Egyptian Journal of Hospital Medicine (October 2025) Vol. 101, Page 5896-5902

Assessment of Levels of Vitamin B_{12}, Ferritin and Iron in Patients with Viral Warts

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ABSTRACT

Background: Viral warts are benign epidermal proliferations resulting from infection with the human papillomavirus (HPV). **Objective:** This study aimed to determine the potential utility of evaluating serum vitamin B_{12}, ferritin and iron in patients with viral warts. **Methods:** This case–control study was conducted at the Dermatology, Andrology & Sexually transmitted diseases (STDs) Outpatient Clinics on 60 participants divided into two groups: Group I included 30 patients with clinically diagnosed viral warts. Group II composed of 30 age- and sex-matched healthy individuals as controls. Serum vitamin B_{12} and ferritin levels were measured using ELISA kits, and serum iron was assessed by the colorimetric ferrozine method.

Results: Plantar warts showed the highest mean vitamin B_{12} levels and genital warts the lowest, serum vitamin B_{12} was the only significant predictor of viral warts, with each 1 pg/mL increase reducing the odds by about 1%.

Conclusions: Our findings highlighted the potential role of vitamin B_{12} in HPV susceptibility and suggest that assessing B_{12} status could aid in managing recurrent or resistant warts.

Keywords: Viral warts, Human papillomavirus, Vitamin B_{12}, Ferritin, Iron.

INTRODUCTION

Viral warts are benign epidermal proliferations resulting from infection with the human papillomavirus (HPV). These lesions are commonly encountered in clinical dermatology and can affect individuals of all ages. Although generally self-limited, warts may become persistent or recurrent in some patients, indicating a possible underlying impairment in host defence mechanisms [(1)].

HPV infects the basal layer of the epithelium through micro-abrasions, leading to localized cellular proliferation. In most individuals, the infection is transient and cleared by the immune system. However, a subset of patients fails to mount an effective immune response, allowing the virus to persist and manifest clinically as cutaneous or mucosal warts [(2)].

Host immunity plays a central role in determining the outcome of HPV exposure. Both innate and adaptive immune responses are required for viral clearance. Adequate immune function depends, in part, on optimal nutritional status, particularly the availability of key micronutrients involved in hematopoiesis and cellular immunity [(3)]. Vitamin B_{12} (cobalamin) is essential for DNA synthesis and neuronal function. It is also involved in lymphocyte proliferation and cytokine production. Deficiency in vitamin B_{12} may result in impaired immune cell function and reduced resistance to viral infections [(4)]. Iron is a critical trace element required for enzymatic reactions, oxidative metabolism, and immune regulation. Ferritin, the primary intracellular iron-storage protein, serves as an indicator of total body iron reserves. Iron deficiency has been associated with decreased T-cell activity and impaired host defense [(5)].

Alterations in serum levels of vitamin B_{12}, ferritin, or iron can negatively affect the immune system. In the context of viral infections such as HPV, these deficiencies may contribute to lesion development, chronicity, or poor therapeutic response **[(6)].**

Despite the clinical burden of viral warts, limited attention has been given to the potential role of micronutrient status in their pathogenesis. Investigating the relationship between these biochemical markers and HPV-related lesions may offer new insights into host susceptibility and treatment optimization **[(6)].**

This study aimed to determine the potential utility of evaluating serum vitamin B_{12}, ferritin and iron in patients with viral warts.

PATIENTS AND METHODS

This case–control study was conducted through a collaborative effort between the Departments of Dermatology, Andrology & Sexually Transmitted Diseases (STDs) and Medical Biochemistry & Molecular Biology, Faculty of Medicine, Menoufia University.

Purposive sample of sixty participants was recruited and divided into two groups: **Group I (Cases):** Thirty patients presented with clinically diagnosed viral warts including common warts (*verruca vulgaris*), plane warts (*verruca plana*), genital warts (*condyloma acuminata*), and plantar warts (*verruca plantaris*). **Group II (Controls):** Thirty age- and sex-matched apparently healthy individuals with no current or previous history of viral warts.

Exclusion criteria: History of malignant disease, presence of acute systemic infection, metabolic or endocrine disorders (diabetes mellitus, thyroid disease & adrenal disorders), chronic liver disease or hepatic impairment, current or recent (within 6 months) administration of vitamin B_{12}, folic acid, or iron therapy and pregnant or breastfeeding women.

Received: 30/06/2025
Accepted: 02/09/2025

All patients were subjected to history taking, clinical examination and dermatological examination including inspection of the skin, mucous membranes and appendages to determine number of lesions, distribution and anatomical sites affected, morphological classification of warts (common, plane, plantar & genital), complications such as secondary infection or discomfort and laboratory investigations.

Measurement of serum vitamin B_{12} was determined by a quantitative sandwich enzyme-linked immunosorbent assay (ELISA) using a commercially available kit (Human Vitamin B12 ELISA Kit, SunRed Biotechnology Co., Shanghai, China, Cat. No. 201-12-1545). Measurement of serum ferritin concentration was measured by a quantitative sandwich ELISA based on the double-antibody sandwich principle. Measurement of serum iron concentration was determined using the colorimetric ferrozine method, a well-established and specific procedure for spectrophotometric quantification of serum iron.

Sample size calculation: The sample size was calculated to achieve 80% power and a 95% confidence level based on **Tamer *et al.*** [7] who reported lower serum vitamin B_{12} in patients with plantar warts (254.2 ± 104.6 pg/mL) versus controls (341.4 ± 127.8 pg/mL). Using standard formulas for comparing two means ($Z_1-\alpha/2$ = 1.96, β = 0.2, equal group sizes), the minimum sample was 58 (29 per group), rounded to 60 to account for potential dropouts. This ensured adequate sensitivity to detect significant differences.

Ethical approval: The investigation was approved by the Ethical Committee of Menoufia University Hospitals under approval number IRP No. 10/2024DEAM21. Patients were granted informed consents prior to their enrolment in this investigation. The Declaration of Helsinki was adhered to during the research. This investigation comports with the CONSORT recommendations.

Statistical analysis

Data were analyzed using SPSS version 26. Normality was tested with the Kolmogorov–Smirnov test. Continuous data were reported as mean ± SD or median (IQR) and categorical data were expressed as frequency and percentage. Group comparisons used the t-test or Mann–Whitney U test, and categorical variables were assessed with Chi-square or Fisher's exact tests. Correlations between serum vitamin B_{12}, ferritin, and iron levels and clinical variables were evaluated using Pearson's or Spearman's coefficients. Multivariate logistic regression to identify independent predictors of viral wart occurrence. A p-value ≤ 0.05 was considered statistically significant.

RESULTS

The two groups were comparable in age, sex, BMI, residence, occupation, and smoking status. Patients with viral warts had significantly lower vitamin B_{12} levels than controls (mean 122.0 vs 318.7 pg/mL; median 80.1 vs 186.1 pg/mL; p = 0.002). Ferritin and iron levels showed no significant differences, with similar median ferritin values and overlapping serum iron ranges (Table 1).

Table (1): Demographic, baseline characteristics, and serum level of different biomarkers of the studied groups

Characteristic	Group I (Cases, n=30)	Group II (Controls, n=30)	p-value
Age (years)	37.43 ± 14.36	35.40 ± 11.80	0.552
Sex, n (%)			0.592
Male	20 (66.7)	18 (60.0)	
Female	10 (33.3)	12 (40.0)	
BMI (kg/m²)	26.8 ± 3.9	25.3 ± 3.1	0.112
Residence, n (%)			0.796
Urban	13 (43.3)	14 (46.7)	
Rural	17 (56.7)	16 (53.3)	
Occupation, n (%)			0.318
Manual labor	12 (40.0)	8 (26.7)	
Non-manual	11 (36.7)	15 (50.0)	
Unemployed/Student	7 (23.3)	7 (23.3)	
Smoking Status, n (%)			0.455
Non-Smoker	19 (63.3)	21 (70.0)	
Smoker	11 (36.7)	8 (26.7)	
Ex-Smoker	0 (0.0)	1 (3.3)	
Family history of warts, n (%)	5 (16.7 %)	0 (0.0 %)	< 0.001 **
Recurrent warts, n (%)	21 (70.0 %)	0 (0.0 %)	< 0.001 **
Prior treatment history, n (%)	19 (63.3 %)	0 (0.0 %)	< 0.001 **
Serum Vitamin B_{12}	122.00 ± 99.40	318.69 ± 321.02	0.002
Serum Ferritin Levels	150.08 ± 104.84	119.97 ± 39.93	0.153
Serum Iron Levels	101 ± 35	110 ± 28	0.29

Data presented as mean ± SD: Standard Deviation; BMI: Body Mass Index. Equal variances not assumed (Levene's test: F=16.568, p<0.001).

Plantar warts were the most frequent type (43.3%), followed by common warts (26.7%), while genital, plane, and other types were less common. Most cases (80%) had lesions of ≤ 6 months' duration, and two-thirds (66.7%) presented with more than five lesions. Lesions were mainly located on the feet (40%) and hands (23.3%), with the majority showing a progressive disease course (90%) (Table 2).

Table (1): Clinical profile of patients with viral warts (n=30)

Clinical Feature	Category	n	%
Type of wart	**Plantar**	13	43.3
	Common	8	26.7
	Genital	3	10.0
	Plane	2	6.7
	Filiform	1	3.3
	Mixed type	2	6.7
	Other	1	3.3
Duration (months)	**≤ 6 months**	24	80.0
	> 6 months	6	20.0
Number of lesions	**Single**	1	3.3
	2 – 5	9	30.0
	> 5	20	66.7
Size of lesions (mm)	**< 5**	18	60.0
	5 – 10	11	36.7
	> 10	1	3.3
Anatomical site	**Foot**	12	40.0
	Hand	7	23.3
	Genital	3	10.0
	Face	2	6.7
	Multiple sites	2	6.7
	Other	4	13.3
Course of disease	**Progressive**	27	90.0
	Stationary	2	6.7
	Regressing	1	3.3

Data presented as mean ± SD: Standard Deviation; IQR = interquartile range.

Serum vitamin B_{12}, ferritin, and iron levels were compared among the different wart types using the Kruskal–Wallis test. Patients with plantar warts showed the highest mean vitamin B_{12} level, while those with genital warts had the lowest. Ferritin and iron levels showed minor, non-significant variations among wart types. The Kruskal–Wallis H values were 1.42, 2.06 and 1.17 for vitamin B_{12}, ferritin and iron respectively (all $p > 0.05$) indicating no significant differences (Table 3).

Table (3): Comparison of vitamin B_{12}, Ferritin, and Iron levels by wart type in the patient group (n=30)

Wart Type	n	Vitamin B_{12} (pg/mL) Mean ± SD	Ferritin (ng/mL) Mean ± SD	Iron (µg/dL) Mean ± SD
Plantar	13	138.6 ± 118.6	132.9 ± 72.6	1.03 ± 0.38
Common	8	119.4 ± 97.3	198.5 ± 152.7	1.06 ± 0.40
Genital	3	64.9 ± 5.8	101.1 ± 61.2	0.76 ± 0.18
Plane	2	74.4 ± 0.5	111.3 ± 19.1	0.85 ± 0.07
Other types	4	96.5 ± 60.2	125.7 ± 65.2	0.93 ± 0.21
H statistic		1.42	2.06	1.17
p-value		0.84	0.72	0.88

Data presented as SD = standard deviation.

The independent t-test showed no statistically significant difference between short- and long-duration groups in serum Vitamin B_{12}, Ferritin, or Iron (Table 4).

Table (4): Association between wart duration and serum biomarker levels in the patient group (n=30)

Duration Category	n	Vitamin B_{12} (pg/mL) Mean ± SD	Ferritin (ng/mL) Mean ± SD	Iron (µg/dL) Mean ± SD
Short Duration (≤ 6 months)	24	125.71 ± 104.91	144.38 ± 101.16	1.02 ± 0.37
Long Duration (> 6 months)	6	107.56 ± 75.40	173.12 ± 122.98	0.97 ± 0.28
p-value (Independent t-test)		0.782	0.585	0.734

Data presented as mean ± SD, SD = standard deviation.

Pearson correlation analysis demonstrated weak, non-significant correlations between disease duration and serum levels of Vitamin B_{12} ($r = -0.218$, $p = 0.247$), Ferritin ($r = 0.104$, $p = 0.585$), and Iron ($r = -0.065$, $p = 0.734$). Correlation analyses showed inconsistent, weak, and statistically insignificant relationships among vitamin B_{12}, ferritin, and iron in both cases and controls (all $p > 0.05$) (Table 5).

Table (5): Correlation between serum vitamin B_{12}, ferritin, and Iron levels in the patient and control groups

Group	Parameter Pair	Correlation Coefficient (r)	p-value
Case group (n=30)	**Vitamin B12 vs. Ferritin**	0.218	0.247
	Vitamin B12 vs. Iron	0.152	0.421
	Ferritin vs. Iron	0.187	0.322
Control group (n=30)	**Vitamin B12 vs. Ferritin**	-0.104	0.591
	Vitamin B12 vs. Iron	0.217	0.258
	Ferritin vs. Iron	0.086	0.657
Total Sample (n=60)	**Vitamin B12 vs. Ferritin**	0.132	0.318
	Vitamin B12 vs. Iron	0.184	0.163
	Ferritin vs. Iron	0.142	0.285

Presence of viral warts (1 = case, 0 = control), B = regression coefficient; S.E. = standard error; CI = confidence interval, * Significant at $p < 0.05$.

On univariate logistic regression, only serum vitamin B_{12} level showed a statistically significant association with the presence of viral warts ($p = 0.007$). Each 1 pg/mL increase in vitamin B_{12} reduced the odds of having warts by about 0.9% (OR = 0.991, 95% CI: 0.983–0.998). On multivariate logistic regression analysis, low serum vitamin B_{12} remained the only independent predictor of viral warts ($p = 0.005$). Each 1 pg/mL increase in vitamin B_{12} was associated with approximately a 1 % decrease in the adjusted odds of having viral warts (AOR = 0.989, 95 % CI: 0.981–0.997). The overall regression model was statistically significant (Omnibus $\chi^2 = 22.41$, $p = 0.002$) and explained about 40 % of the variance in wart occurrence (Nagelkerke $R^2 = 0.402$) (Table 6).

Table (6): Univariate and multivariate logistic regression analysis for predictors of viral warts

Univariate Logistic Regression						
Predictor	**B**	**S.E.**	**Wald**	**p-value**	**Odds Ratio (OR)**	**95% CI for OR**
Age (years)	−0.020	0.025	0.64	0.424	0.980	0.933 – 1.028
Sex (Male)	−0.281	0.693	0.16	0.686	0.755	0.194 – 2.941
BMI (kg/m²)	0.105	0.078	1.80	0.179	1.111	0.952 – 1.296
Smoking (Yes)	−0.472	0.754	0.39	0.531	0.624	0.141 – 2.768
Vitamin B_{12} (pg/mL)	−0.009	0.004	7.21	**0.007 ***	0.991	0.983 – 0.998
Ferritin (ng/mL)	0.005	0.004	1.22	0.270	1.005	0.996 – 1.014
Iron (µg/dL)	−0.921	1.145	0.65	0.421	0.398	0.041 – 3.846
Multivariate Logistic Regression						
	B	**S.E.**	**Wald**	**p-value**	**Adjusted Odds Ratio (AOR)**	**95 % CI for AOR**
Age (years)	–0.021	0.027	0.60	0.438	0.979	0.929 – 1.032
Sex (Male)	–0.311	0.721	0.19	0.666	0.733	0.178 – 3.012
BMI (kg/m²)	0.121	0.083	2.13	0.145	1.129	0.959 – 1.328
Smoking (Yes)	–0.542	0.784	0.48	0.489	0.582	0.125 – 2.707
Vitamin B_{12} (pg/mL)	–0.011	0.004	7.83	**0.005 ***	0.989	0.981 – 0.997
Ferritin (ng/mL)	0.006	0.005	1.44	0.230	1.006	0.996 – 1.016
Iron (µg/dL)	–1.105	1.241	0.79	0.373	0.331	0.029 – 3.752
Constant	2.198	3.611	0.37	0.543	9.003	—

Presence of viral warts (1 = case, 0 = control). B = regression coefficient; S.E. = standard error; CI = confidence interval. * Significant at $p < 0.05$, Omnibus χ^2 (7) = 22.41, $p = 0.002$ Nagelkerke $R^2 = 0.402$ Classification accuracy = 78.3 %

DISCUSSION

HPV is the most common viral infection of the skin and mucosa, responsible for a wide spectrum of clinical manifestations ranging from benign cutaneous warts to premalignant and malignant lesions. Cutaneous warts, in particular, represent one of the most frequent dermatological problems encountered in clinical practice, affecting both children and adults with variable persistence and recurrence rates. Although most warts are self-limiting, many cases require medical intervention due to pain, cosmetic concerns and treatment resistance. Over the past decade, research has increasingly explored the role of host immunity, genetic predisposition, and nutritional status in determining individual susceptibility to HPV infection and disease severity [8]. Our findings showed that patients with viral warts and controls were comparable in terms of age, sex, BMI, residence, occupation and smoking status, with no statistically significant differences. Comparable results were observed by **Ertuğrul and Aktaş** [9].

They reported no significant age or sex differences between groups, with a similar male predominance in both arms. In contrast, epidemiological surveys often show peak wart incidence in children and adolescents, which differs from our adult-based hospital cohort. For example, a UK population study found the highest prevalence among schoolchildren, with rates up to 33% **[8]**.

Plantar warts were the most common type (43.3%), followed by common warts (26.7%), with other types being less frequent. The majority of patients (80%) had disease duration ≤ 6 months, and 66.7% presented with more than five lesions. Lesions were predominantly on the feet (40%) and hands (23.3%), and most patients showed a progressive course (90%). Our distribution of wart types is partially supported by **Malik *et al.*** [10] who found common warts in 54% and plantar warts in 20% of their Pakistani cohorts. In contrast, our cohort had a higher prevalence of plantar warts compared to common warts, which is opposite to many epidemiological studies. For instance, **Sterling *et al.*** [8] noted common warts as the predominant type (~70%) in community-based surveys. This discrepancy likely reflects differences in population age (our patients were older, where plantar warts are more common) and hospital-based recruitment, where painful plantar lesions prompt medical visits more often than small common warts.

Patients reported significantly higher rates of family history (16.7% vs. 0%), recurrence (70% vs. 0%) and prior treatment (63.3% vs. 0%) compared to controls (p<0.001 for all). Our findings align with those of **Malik *et al.*** [10] who reported positive family history in ~30% of wart cases and emphasized the chronic relapsing tendency of viral warts. By contrast, **Bruggink *et al.*** [11] observed recurrence rates closer to 15% in a randomized trial of wart treatments in Dutch primary care. The higher recurrence in our study (70%) may reflect that our sample included patients with more resistant or long-standing disease, or cultural factors influencing re-presentation to tertiary care.

Patients with viral warts showed significantly lower serum vitamin B_{12} levels than controls (122.0 ± 99.4 vs. 318.7 ± 321.0 pg/mL, p = 0.002). The median B_{12} in cases (80.1 pg/mL) was also far below controls (186.1 pg/mL). Our results are supported by. **Tamer *et al.*** [12] who reported significantly lower mean B_{12} levels in patients (p = 0.010), while ferritin and vitamin D were not different. In contrast, **Demir and Güder** [13] found no significant difference in B12 levels between groups. Their cohort focused on anogenital HPV infection, suggesting that B_{12} deficiency may be more strongly linked to cutaneous rather than mucosal HPV types. Similarly, some epidemiological studies of cervical HPV infection have not consistently shown serum B_{12} deficiency but instead highlighted folate as a more important factor **Sedjo *et al.*** [14] These discrepancies suggest that wart subtype, anatomical site, and host demographics influence the role of B_{12} in HPV infection.

Ferritin levels were slightly higher in wart patients (150.1 ± 104.8 ng/mL) compared to controls (120.0 ± 39.9 ng/mL), but this difference was not statistically significant (p = 0.153). Median ferritin values were nearly identical (111.3 vs. 106.0 ng/mL). Our finding is consistent with **Tamer *et al.*** [12] who found no significant differences in serum ferritin between wart patients and controls. Also, **Ertuğrul and Aktaş** [9] reported comparable ferritin levels (median 17 vs. 20 ng/mL, p = 0.677). In contrast, **Demir and Güder** [13] found that genital wart patients had significantly higher ferritin compared to controls (132.6 vs. 106.8 ng/mL, p<0.05). Their ROC analysis even suggested that ferritin >139 ng/mL increased the risk of persistent genital warts. These results suggest that ferritin may play a different role in mucosal HPV infections compared with cutaneous ones, possibly reflecting site-specific immune–nutrient interactions.

Serum iron levels did not differ significantly between wart patients and controls (1.01 ± 0.35 vs. 1.10 ± 0.28 µg/dL, p = 0.295).

Both groups showed overlapping ranges. This finding aligns indirectly with **Tamer *et al.*** **[12]** and **Ertuğrul and Aktaş** **[9]** who did not find meaningful case–control differences in ferritin or related hematological indices, suggesting that iron deficiency is not a consistent feature of wart patients. On the other hand, some studies of genital HPV infections suggest iron overload, rather than deficiency, may play a role. **Demir and Güder** [13] reported higher ferritin in genital wart patients, suggesting excessive iron stores could promote HPV persistence. Moreover, mechanistic research indicates that both iron deficiency and excess can impair host immunity and viral clearance [15]. Therefore, while cutaneous wart patients in our cohort showed no iron imbalance, the role of iron metabolism in HPV pathogenesis may differ depending on viral type, infection site and host nutritional status.

Our multivariate regression analysis identified low serum vitamin B_{12} as the only independent predictor of viral wart presence (OR = 0.989, 95% CI: 0.981–0.997, p = 0.005), while ferritin, iron, age, sex, BMI, and smoking did not contribute significantly. Furthermore, correlation analyses showed weak and nonsignificant relationships between vitamin B_{12}, ferritin, and iron, reinforcing that B_{12} deficiency acts as an isolated nutritional risk factor rather than reflecting generalized nutritional imbalance. These findings are in strong agreement with several recent studies. **Tamer *et al.*** [12] reported significantly lower vitamin B_{12} levels in wart patients compared to controls (p = 0.010) with no significant difference in ferritin or vitamin D, closely mirroring our results. **Ertuğrul and Aktaş** [9] similarly demonstrated that B_{12} was significantly lower in 70 wart patients versus 70 controls (p = 0.046), while ferritin remained nonsignificant. More recently, interventional studies have supported a causal link [16, 17]. Both **Porcaro *et al.*** [16] and **Tinelli *et al.*** [17] showed that supplementation with vitamin B_{12}, folic acid, EGCG and hyaluronic acid improved HPV clearance and reduced persistence, underscoring the clinical importance of correcting B_{12} insufficiency in HPV-related disease. Mechanistic reviews also confirm this link, **Laganà *et al.*** [18] **and Calcagno *et al.*** [19] highlighted that folate and B_{12} are essential methyl donors for DNA stability and viral genome methylation, reducing HPV integration and progression risk. Thus, convergent evidence supports our conclusion that vitamin B_{12} deficiency is a significant predictor of wart occurrence and persistence. On the other hand, some reports contrast with our results. **Demir and Güder** [13], studying genital warts, found no association with B_{12} but instead reported higher ferritin levels in patients compared to controls ($p < 0.05$). Likewise, **Siegel *et al.*** [20] showed that women with elevated ferritin were less likely to clear oncogenic HPV infections, implicating iron overload as a risk factor for viral persistence. **Chen *et al.*** [21] further observed a nonlinear association between dietary iron intake and HPV infection, suggesting complex iron–immune interactions. These discrepancies may be explained by differences in HPV phenotype (cutaneous vs. genital vs. cervical), host inflammation status and measurement focus (serum vs. dietary vs. storage iron). In our cutaneous wart cohort, ferritin and iron showed no significant predictive value, but this did not exclude their potential role in mucosal HPV persistence where oxidative stress and ferroptosis pathways may be more prominent [22, 23].

LIMITATIONS

The relatively modest sample size from a single tertiary center may reduce generalizability, and the cross-sectional nature of biomarker assessment precludes establishing causality between vitamin B_{12} deficiency and wart occurrence. In addition, the absence of dietary intake data and longitudinal follow-up means the impact of correcting vitamin B_{12} deficiency on wart clearance could not be evaluated.

CONCLUSIONS

There was a significant association between vitamin B_{12} deficiency and viral warts, with low B_{12} emerging as the sole independent predictor after adjusting for demographic and clinical factors. Ferritin and iron levels showed no significant differences. These results highlighted the potential role of vitamin B_{12} in HPV susceptibility and suggest that assessing B_{12} status could aid in managing recurrent or resistant warts, warranting further longitudinal and multicenter studies.

Financial support and sponsorship: Nil.
Conflict of Interest: Nil.

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