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THE ASSOCIATION BETWEEN VITAMIN D LEVELS AND INTESTINAL PARASITES

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Abstract

Immune system is known to be affected by vitamin D deficiency. In this study, we investigated whether there was a significant association between vitamin D levels and intestinal parasites.

A total of 239 patients treated at the internal medicine outpatient clinics with gastrointestinal complaints were included in this study. Demographic characteristics of the patients were recorded. Examination of stool samples were repeated three times within 10 days. Cellophane band method, native-lugol, sedimentation and modified Kinyoun's acid fast stain methods were used for the detection of parasites in the stool samples. The samples were prepared, stained and examined under a microscope. Vitamin D levels were examined in the blood samples collected from the patients. Patients having vitamin D level under 20 ng/ml were diagnosed with vitamin D deficiency.

Binary logistic regression analysis showed that serum vitamin D level was significant informative variable for only Iodamoeba and Cyclospora. No association between vitamin D level and the presence of other intestinal parasites was found. The risk of Iodamoeba positivity was 2.54 times higher in the patients with a serum vitamin D level under 20 ng/ml ($p < 0.01$). Similarly, the risk of Cyclospora positivity was 2.44 times higher in the patients with a serum vitamin D level under 20 ng/ml compared to those with a serum vitamin D level over 20 ng/ml ($p < 0.01$).

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The risk for positivity of Iodamoeba and Cyclospora that are among the intestinal protozoa is high for people with vitamin D deficiency.

Key words: vitamin D, Iodamoeba, Cyclospora, intestinal parasites, vitamin D deficiency

Introduction. Vitamin D is a group of fat-soluble sterols, which can be synthesized endogenously. It plays a role in bone mineralization, calcium and phosphorus metabolism [1]. Besides calcium and bone metabolism, vitamin D has been shown to have important biological functions, like cell differentiation, inhibition of proliferation and immunomodulation. 1.25 (OH)2 D3, which is the active form of vitamin D, needs the presence of high-affinity vitamin D receptor (VDR) in order to exert its effect. After binding to this receptor, vitamin D regulates gene transcriptions that mediate its biological effects. While VDR is structurally present in antigen presenting cells (macrophages, dendritic cells), it emerges after the activation in lymphocytes [2]. Moreover, the presence of 1α hydroxylase activity in these cells in addition to VDR supports the opinion that vitamin D plays an immune modulator role [2–4]. Studies have found that vitamin D insufficiency is common in many northern regions [3]. Furthermore, it is accepted as a global epidemic because the incidence of vitamin D deficiency increases and it is associated with many chronic diseases [1]. Studies have presented vitamin D insufficiency in more than 50% and severe vitamin D deficiency in 16% of the adult population in England [5], vitamin D deficiency and insufficiency by 28.16% in Brazil [6], and vitamin D deficiency by 51.8% and vitamin D insufficiency by 20.7% in Ankara province of Turkey [7]. According to the literature, vitamin D insufficiency is seen by 20–30% in South America and Africa, and by 92% in North America [3].

Recent studies have reported that vitamin D deficiency and insufficiency are associated with metabolic syndrome, cancer, infectious diseases, cardiovascular diseases and autoimmune diseases [3,8–13]. It has been reported that vitamin D insufficiency negatively affects immune system, contributing to the pathogenesis of some infectious diseases. In addition, vitamin D has been found to have an immune modulator role in the control of monocytes, macrophages and dendritic cells as well as various immune cells, including T-lymphocytes and B-lymphocytes [2–4,14].

Intestinal parasitic infections are an important public health problem in developing countries and in countries with a tropical climate. Gastrointestinal system findings such as abdominal pain, diarrhea, nausea/vomiting, constipation, loss of appetite and skin rash are often observed in parasitosis. It has been shown that parasites can produce free oxygen radicals such as superoxide and hydrogen peroxide, and contain enzymes producing these radicals. Accordingly, we aimed to investigate the association between vitamin D levels and the incidence of intestinal parasites in this study.

Material and methods. The necessary approval was received from the local ethics committee before the beginning of the study. Patients treated at the internal medicine outpatient clinic with gastrointestinal complaints were informed

about the study. The patients were informed about that the examination should be repeated three times at different time intervals within 10 days, when no parasite was detected at the first examination. Those accepting to participate were included in the study.

Material collection. Patients treated at the internal medicine outpatient clinic of Ordu University Medical Faculty Training and Research Hospital with gastrointestinal complaints and accepting to bring samples to Department of Parasitology were included in the study. Stool collection containers were given to the patients and they were informed that 3–4 soup spoons of stool samples in patients with diarrhea and wallnut size of stool samples in patients without diarrhea should be put into the container. The container should be closed tightly and it should be directed to the parasitology laboratory within one hour. In addition, the cellophane band method used in the diagnosis of *Enterobius vermicularis*, of which eggs are not seen in the stool, was performed before toilet or bath. The cellophane band was cut in a length of 10–15 cm, placed on a glass baguette or pen with the adhesive surface being outward, touched around anus of the patient, and the eggs were provided to adhere to it. The band was smoothly adhered on a clean slide and examined under a microscope. For confidentiality of the patients were informed in detail how to perform the administration. Examinations were repeated three times with 3–4 days intervals in order to infer that the patient was not infected by parasites.

Examination of the collected materials. Cellophane band, native-lugol, sedimentation and modified Kinyoun's acid fast stain methods were used for the diagnosis of parasites in the stool samples [15]. The samples were prepared, stained and examined under a microscope. Patients with parasite positivity were treated and called for follow-up after the treatment.

Examination of blood samples. Blood samples were collected in anticoagulant free gel tubes to measure vitamin D level. Blood samples were centrifuged at 1800 g for 15 min and plasma and serum samples were obtained. Vitamin D levels were measured via a colourimetric method using an Abbott original reagent with Abbott Architect I 2000-SR autoanalyzer.

Definition of vitamin D deficiency: We categorized 25 (OH) D levels as follows: deficiency and insufficiency were determined as serum levels < 20 ng/ml and normality as serum levels ≥ 20 ng/ml [1,18].

Statistical analysis. For vitamin D levels, normality of data and homogeneity of variance were controlled by Kolmogorov–Smirnov test and Levene's test, respectively. Pearson's (χ^2) or likelihood-ratio chi-squared (LRχ^2) test was used to compare categorical variables and independent samples t-test was used to compare the means of the groups. Binary logistic regression analyses were used to estimate the odds ratio (OR) at 95% confidence interval (CI). A p value < 0.05 was considered statistically significant. All statistical analyses were performed using SPSS v25 statistical software (IBM Inc., Chicago, IL, USA).

Results. In this study, a total of 239 patients, 178 (74.5%) being female and 61 (25.5%) male were examined for intestinal parasites and serum vitamin D levels. The mean vitamin D concentration was 17.80 ± 14.55 ng/ml. The overall prevalence was 71.5% for ones having vitamin D < 20 ng/ml and 28.5% for ones having vitamin D ≥ 20 ng/ml. Table 1 shows the baseline characteristics of the participants included in the study. Vitamin D levels of the patients showed difference in terms of only working status ($p < 0.01$). The frequency distribution of vitamin D groups was almost the same in patients who were working. However, among the patients who were not working, the frequency of those with a vitamin D < 20 ng/ml was nearly three times higher than the patients with a vitamin D level ≥ 20 ng/ml. No significant difference was found between the groups in terms of

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Baseline characteristics according to vitamin D status

Variables		Vitamin D levels				p
		< 20 ng/ml		≥ 20 ng/ml		
Gender	Female	132	(77.2%)	46	(67.6%)	0.127
	Male	39	(22.8%)	22	(32.4%)	($\chi^2 = 2.332$)
Age	15–34 years	22	(12.9%)	5	(7.4%)	0.449
	35–50 years	51	(29.8%)	20	(29.4%)	($\chi^2 = 1.601$)
	> 50 years	98	(57.3%)	43	(63.2%)	
Marital status	Single	9	(5.3%)	1	(1.5%)	0.335
	Married	133	(77.8%)	56	(82.4%)	($\text{LR}\chi^2 = 2.184$)
	Widow	29	(17.0%)	11	(16.2%)	
Economic status	High	11	(6.4%)	5	(7.4%)	0.632
	Middle	126	(73.7%)	53	(77.9%)	($\text{LR}\chi^2 = 0.888$)
	Low	34	(19.9%)	10	(14.7%)	
Educational status	Illiterate	55	(32.2%)	19	(27.9%)	0.670
	Primary school	89	(52.0%)	34	(50.0%)	($\text{LR}\chi^2 = 1.552$)
	High school	18	(10.5%)	9	(13.2%)	
	University	9	(5.3%)	6	(8.8%)	
Settlement	Village	125	(73.1%)	47	(69.1%)	0.873
	Town	3	(1.8%)	1	(1.5%)	($\text{LR}\chi^2 = 0.700$)
	District	11	(6.4%)	4	(5.9%)	
	City	32	(18.7%)	16	(23.5%)	
Working status	Unemployment	149	(87.1%)	47	(69.1%)	0.001^{**}
	Working	22	(12.9%)	21	(30.9%)	($\text{LR}\chi^2 = 10.704$)
Chronic Diseases	No	79	(46.2%)	31	(45.6%)	0.932
	Yes	92	(53.8%)	37	(54.4%)	($\chi^2 = 0.007$)

χ^2 – Pearson Chi-Square; $\text{LR}\chi^2$ – Likelihood Ratio Chi-Square; $^{**}p < 0.01$

the other baseline characteristics ($p > 0.05$). There was no significant difference between vitamin D groups with respect to having chronic disease ($p > 0.05$).

In this study, *Blastocystis* spp., *Iodamoeba buetschlii*, *Entamoeba coli*, *E. histolytica*, *Dientamoeba fragilis*, *Giardia intestinalis*, *Chilomastix mesnili*, *Enterobius vermicularis*, *Cryptosporidium* spp., *Hymenolepis nana*, *Cyclospora cayetanensis*, *C. cayetanensis*, *Endolimax nana*, *Entamoeba hartmanni*, *Ascaris lumbricoides* and *Taenia saginata* parasites were investigated. *E. histolytica*, *C. mesnili*, *A. lumbricoides* and *T. saginata* were detected in only one patient.

Descriptive statistics regarding blood serum vitamin D levels (ng/ml) of the patients divided into two groups as negative and positive in terms of both the presence and the types of parasites are given in Table 2.

Of the patients, 67.36% were positive for any parasite and 32.64% were negative. According to the independent t-test, the mean serum vitamin D level (ng/ml) was statistically significantly lower in the patients who were positive for any parasite (16.04 ± 0.77) compared to the patients who were negative (21.42 ± 2.37) ($p < 0.05$). However, this difference was not significant in terms of the type of parasites ($p > 0.05$).

Binary logistic regression analysis was used to estimate the odds ratios (ORs) at 95% confidence interval (CI) for the association between vitamin D deficiency and the positivity of gastrointestinal parasites. Table 3 shows ORs (95% CI) of vitamin D deficiency by the presence of intestinal parasites. The calculated ORs show the possibility (risk) of positivity for *Blastocystis* spp., *I. buetschlii*, *E. coli*, *E. histolytica*, *D. fragilis*, *G. intestinalis*, *C. mesnili*, *E. vermicularis*, *Cryptosporidium* spp., *C. cayetanensis* parasites in the group with vitamin D < 20 ng/ml. Logistic regression analysis could not be performed and ORs could not be calculated for *E. histolytica*, *C. mesnili* and *E. vermicularis* parasites due to the lack of sufficient data. Logistic regression analysis showed that serum vitamin D level was a significant informative variable only for *I. buetschlii* and *C. cayetanensis*. No association between vitamin D deficiency and the presence of other gastrointestinal parasites was found. The risk of *Iodamoeba buetschlii* positivity was 2.54 times higher in patients with a vitamin D level < 20 ng/ml ($p < 0.01$). Similarly, the risk of *Cyclospora cayetanensis* positivity was 2.44 times higher in the group with a vitamin D level < 20 ng/ml group compared to the group with a vitamin D level ≥ 20 ng/ml ($p < 0.01$).

Discussion. In this study in which intestinal parasites were compared between the individuals with a vitamin D level < 20 ng/ml and the individuals with a vitamin D level ≥ 20 ng/ml, the risk of *Iodamoeba* positivity, which is among the intestinal protozoa increased by 2.54 times ($p < 0.01$) and the risk of *Cyclospora* positivity increased by 2.44 times ($p < 0.01$) in the individuals with a vitamin D level ≥ 20 ng/ml.

It has been reported that vitamin D has an immune system modulating effect, and immune system may be affected in the case of its deficiency [2–4, 18]. It has been

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Comparison of vitamin D levels in groups with and without gastro-intestinal parasites

		n	(%)	Mean ± SEM	p
Parasite	negative	78	(32.64)	21.42 ± 2.37	0.033*
	positive	161	(67.36)	16.04 ± 0.77	($t = 2.159$)
Blastocystis spp.	negative	161	(67.4)	18.31 ± 1.27	0.435
	positive	78	(32.6)	16.74 ± 1.19	($t = 0.782$)
L. butschlii	negative	232	(97.1)	17.81 ± 0.96	0.938
	positive	7	(2.9)	17.38 ± 5.65	$t = 0.077$
E. coli	negative	201	(84.1)	17.97 ± 1.07	0.666
	positive	38	(15.9)	16.86 ± 1.70	($t = 0.432$)
E. histolytica	negative	238	(99.6)	17.76 ± 0.95	—
	positive	1	(0.4)	26.00 ± —	
D. fragilis	negative	230	(96.2)	17.98 ± 0.97	0.325
	positive	9	(3.8)	13.10 ± 3.28	($t = 0.986$)
G. intestinalis	negative	225	(94.1)	17.94 ± 0.99	0.558
	positive	14	(5.9)	15.58 ± 2.56	($t = 0.587$)
C. mesnili	negative	238	(99.6)	17.81 ± 0.95	—
	positive	1	(0.4)	15.90 ± —	
E. vermicularis	negative	235	(98.3)	17.91 ± 0.95	0.378
	positive	4	(1.7)	11.43 ± 2.58	($t = 0.882$)
Cryptosporidium spp.	negative	163	(68.2)	17.99 ± 1.28	0.771
	positive	76	(31.8)	17.40 ± 1.16	($t = 0.291$)
H. nana	negative	239	(100.0)	17.80 ± 0.94	—
	positive	0	(0.0)	—	
C. cayetanensis	negative	237	(99.2)	17.79 ± 0.95	0.918
	positive	2	(0.8)	18.85 ± 7.95	($t = -0.102$)
E. nana	negative	234	(97.91)	17.84 ± 0.96	0.766
	positive	5	(2.09)	15.88 ± 2.73	($t = -0.298$)
E. hartmanni	negative	236	(98.74)	17.88 ± 0.95	—
	positive	3	(01.26)	11.87 ± 2.263	
A. lumbricoides	negative	238	(99.58)	17.79 ± 0.95	—
	positive	1	(0.42)	19.60 ± —	
T. saginata	negative	238	(99.58)	17.84 ± 0.95	—
	positive	1	(0.42)	9.10 ± —	

— Not calculated; SEM – Standard error of mean; t – Independent samples t-test; *$p < 0.05$

Table 3

Odds ratios (95% confidence intervals) of vitamin D deficiency or insufficiency by the presence of intestinal parasites

	Odds Ratio	[95% CI]	p
Blastocystis spp.	1.014	[0.560–1.836]	0.96
I. buetschlii	2.540	[0.300–21.49]	**0.00****
E. coli	1.020	[0.475–2.89]	0.96
D. fragilis	1.216	[0.296–5.006]	0.78
G. intestinalis	1.368	[0.441–4.236]	0.58
Cryptosporidium spp.	1.285	[0.713–2.435]	0.40
C. cayetanensis	2.435	[0.150–39.480]	**0.00****

CI – Confidence interval; $^{**}p < 0.01$

stated that the risk of developing bacterial, viral and fungal infections as well as parasite related infections may increase in the immunosuppressive conditions [20]. In their review, Gois et al. [3] showed that vitamin D deficiency increases the incidence of tuberculosis, influenza, HIV and fungal infections, and vitamin D replacement contributes to the treatment.

In the literature, vitamin D levels and different infections have been compared and different results have been found. Li et al. [16] found that the expressions of 1α-hydroxylase, an enzyme catalyzing the synthesis of active vitamin D in 16HBE cells of *A. fumigatus*, and vitamin D receptor (VDR) are induced, causing an increase in the production of vitamin D. Also, the studies have reported that vitamin D plays a role as a large signal molecule in the complex mechanisms connecting metabolic functions of the body, cellular survival during stress, and cellular cycle. In general, vitamin D levels increase in infection conditions and it has been stated to help the organism in killing viruses as an anti-inflammatory cytokine [19].

Mostafa Bel-D et al. [17] reported that vitamin D levels were lower in patients with allergic fungal rhinosinusitis compared to the control group and those with nasal polyposis. In addition, Yildirim et al. [14] demonstrated in their study about the eradication of *Helicobacter pylori* (*HP*) that vitamin D deficiency is one of the factors causing failure of HP eradication. In the present study, we investigated whether there was an association between vitamin D levels and *Blastocystis* spp., *Iodamoeba buetschlii*, *Entamoeba coli*, *E. histolytica*, *Dientamoeba fragilis*, *Giardia intestinalis*, *Chilomastix mesnili*, *Enterobius vermicularis*, *Cryptosporidium* spp. and *Cyclospora cayetanensis* positivity. In this study, it was found that the patients with a serum vitamin D level < 20 ng/ml may have a risk for *I. buetschlii* and *C. cayetanensis* positivity ($p < 0.01$). This may be explained by the fact that *I. buetschlii* and *C. cayetanensis* may be more commonly seen in patients with a low level of vitamin D.

In the studies about parasites and vitamin D, it was reported that a diet deficient in vitamins A, D3 and E may cause an increase in *Hymenolepis diminuta* [19]. In the present study, no significant difference was found between vitamin D levels of patients with and without helminth positivity, too ($p > 0.05$). This may be a result from the patient population, the method used, the strains and number of parasites detected.

In an experimental study, vitamin D was found to provide protection against trypomastigotes of *Trypanosoma cruzi*, and reduce histopathological tissue inflammation and the rate of parasites. Also, it was reported that vitamin D may be efficient in prevention of *Plasmodium falciparum* infection [20]. In our study, the positivity of *C. cayetanensis*, which is among the obligate parasites, was significant in the group with a vitamin D level < 20 ng/ml ($p < 0.01$). This may be attributed to the decrease in vitamin D levels, facilitating habitation of the parasite.

In this study, vitamin D level was significantly lower in overall intestinal parasites ($p < 0.05$). This may be explained by the fact that low levels of vitamin D may facilitate habitation of the parasite. Also, the increased risk of *I. buetschlii* and *C. cayetanensis* positivity may be a result from vitamin D deficiency according to the logistic regression analysis (Table 3). In addition, the risk of parasite transmission may be increased because vitamin D levels were lower due to lack of sufficient sunlight exposure depending on climate conditions of the study area.

Conclusion. According to the data obtained in this study, there was a correlation between general presence of parasites and vitamin D levels, while it was found that decreased vitamin D levels may increase the risk of *I. buetschlii* and *C. cayetanensis* positivity according to the logistic regression analysis. No significant difference was found between the other parasites and vitamin D levels. This may be a result from the study setting, method, strains and number of parasites. Accordingly, it was concluded that parasite types and vitamin D levels could be compared with controlled in vivo experiments. In addition, it was thought that vitamin D levels may provide effective data for the treatment of intestinal parasites and protection according to the results.

REFERENCES

[1] Fida F., B. M. Alkan, A. Tosun (2014) Pandemic era: Vitamin D deficiency and insufficiency, Turk. J. Osteoporosis, **72**(20), 71–74.

[2] Ardeniz Ö. (2008) Vitamin D and immune system: Medical education, Turkiye Klinikleri J. Med. Sci., **28**(2), 198–205.

[3] Gois P. H. F., D. Ferreira, S. Olenski, A. C. Seguro (2017) Vitamin D and infectious diseases: Simple bystander or contributing factor?, Nutrients, **9**(7), 651, https://doi.org/10.3390/nu9070651.

[4] Tufan F., B. Kiran, Z. Şahin, M. Çiplak, V. Kiran et al. (2012) Association of 25-OH vitamin D levels with lymphocyte subgroups in the elderly, Turk. Intern. Med. Specialists Assoc. J. Intern. Diseases, **19**, 47–58.

[5] Pereira-Santos M., J. Y. G. Dos Santos, G. Q. Carvalho, D. B. Dos Santos, A. M. Oliveira (2019) Epidemiology of vitamin D insufficiency and deficiency in a population in a sunny country: Geospatial meta-analysis in Brazil, Crit. Rev. Food Sci. Nutr., **59**(13), 2102–2109.

[6] Pearce S. H. S., T. D. Cheetham (2010) Diagnosis and management of vitamin D deficiency, BMJ 2010, 340, https://doi.org/10.1136/bmj.b5664.

[7] Uçar F., M. Y. Taşlipinar, A. Ö. Soydaş, N. Özcan (2012) 25-OH vitamin D levels in patients admitted to Ankara Etlik İhtisas Training and Research Hospital, Eur. J. Basic Med. Sci., **2**(1), 12–15.

[8] Atoum M., F. Alzoughool (2017) Vitamin D and breast cancer: latest evidence and future steps, Breast Cancer (Auckl), **11**, https://doi.org/10.1177/1178223417749816.

[9] Zhu Y., P. P. Wang, G. Zhai, B. Bapat, S. Savas et al. (2018) Association of rs2282679 A > C polymorphism in vitamin D binding protein gene with colorectal cancer risk and survival: effect modification by dietary vitamin D intake, BMC Cancer, **18**(1), 155.

[10] Mahendra A., Karishma, B. K. Choudhury, T. Sharma, N. Bansal et al. (2018) Vitamin D and gastrointestinal cancer, J. Lab. Physicians, **10**(1), 1–5.

[11] Alam U., A. Fawwad, F. Shaheen, B. Tahir, A. Basit et al. (2017) Improvement in neuropathy specific quality of life in patients with diabetes after vitamin D supplementation, J. Diabetes Res., 7928083, https://doi.org/10.1155/2017/7928083.

[12] Umar M., K. S. Sastry, F. Al Ali, M. Al-Khulaifi, E. Wang et al. (2018) Vitamin D and the pathophysiology of inflammatory skin diseases, Skin Pharmacol. Physiol., **31**(2), 74–86.

[13] Hoan N. X., H. V. Tong, L.Ḣ. Song, C. G. Meyer, T. P. Velavan (2018) Vitamin D deficiency and hepatitis viruses-associated liver diseases: A literature review, World J. Gastroenterol., **24**(4), 445–460.

[14] Yildirim O., T. Yildirim, Y. Seckin, P. Osanmaz, Y. Bilgic et al. (2017) The influence of vitamin D deficiency on eradication rates of *Helicobacter pylori*, Adv. Clin. Exp. Med., **26**(9), 1377–1381.

[15] Laboratory in Parasitology (eds M. Korkmaz, Ü. Zeki Ok) (2011) https://www.tmc-online.org/userfiles/file/pdf/Parazitolojide.Lab.Giris.pdf (in Turkish).

[16] Li P., T. Wu, X. Su, Y. Shi (2015) Activation of vitamin D regulates response of human bronchial epithelial cells to Aspergillus fumigatus in an autocrine fashion, Mediators Inflamm., **2015**, 208491, https://doi.org/10.1155/2015/208491.

[17] Mostafa Bel-D., M. S. Taha, T. Abdel Hamid, A. Omran, N. Lotfi (2016) Evaluation of vitamin D levels in allergic fungal sinusitis, chronic rhinosinusitis, and chronic rhinosinusitis with polyposis, Int. Forum Allergy Rhinol., **6**(2), 185–190, https://doi.org/10.1002/alr.21585.

[18] Ardehali S. H., S. Dehghan, A. R. Baghestani, A. Velayati, Z. V. Shariatpanahi (2018) Association of admission serum levels of vitamin D, calcium, phosphate, magnesium and parathormone with clinical outcomes in neurosurgical ICU patients, Sci. Rep., **8**(1), 2965.

[19] Chirumbolo S., G. Bjørklund, A. Sboarina, A. Vella (2017) The role of vitamin D in the immune system as a pro-survival molecule, Clin. Ther., **39**(5), 894–916.
[20] Kaplan M., H. Özavci (2014) Parasitic infections in transplant patients, F.U. Health Sci. Medicine J., **28**(2), 81–92.

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