BENHA MEDICAL JOURNAL bmj
Print ISSN 1110-208X
Online ISSN 2357-0016

original article

The Effect of vitamin D Therapy on Eradication Rates of Helicobacter Pylori Infection

Amira K. El-Alfy , Basma A. Badr, Mohamed A. Hewedi, A R Mohamed

Department of Internal medicine, Benha faculty of medicine, Benha University, Egypt

Correspondence to:
Mohamed A. Hewedi, Department ofinternal medicine, Benha faculty of medicine, Benha University, Egypt.

Email:

mohamedhewedi36@gmail.com

Recieved: 16 Novembre 2022

Accepted: 6 December 2022

Abstract

Background: Helicobacter pylori (H. pylori) has been shown to be a key contributor to chronic gastritis. Peptic ulcer disease, gastric adenocarcinoma, and gastric lymphoma are all linked to this condition, which affects 50% of the world's population. Aim of the work : To assess the efficacy of adding vitamin D supplement in eliminating helicobacter pylori infection. **Methods:** A total of 50 Egyptian patients with dyspeptic symptoms lasting at least one month were involved in this case study. Patients were categorized into two groups. Group A: 25 patients with helicobacter pylori infection ; they received clarithromycin-based triple treatment and a vitamin D supplement Group B : 25 individuals with Helicobacter pylori infection ; they received triple clarithromycin treatment without vitamin D supplementation. **Results:** There was a statistically significant difference (p= 0.031) in the efficacy of vitamin D between the two groups, with 84% of patients in group A showing favourable outcomes whereas only 56% of the patients in group B exhibited positive results. **Conclusion:** As a result, supplementing clarithromycin-based triple therapy with vitamin D3 may increase eradication rates for H. pylori infections substantially.

Keywords: vitamin D therapy; eradication rates; helicobacter pylori infection

Introduction

Chronic gastritis has been linked to helicobacter pylori (H. pylori) infection. Peptic ulcer disease, gastric adenocarcinoma, and gastric lymphoma are all linked to this condition, which affects 50% of the world's population. H. pylori infection is linked to 76–95 percent of stomach malignancies and 90 percent of duodenal ulcers (1).

If, H. pylori infection is not treated, ulcers and related consequences will continue to occur, according to the National Institutes of Health Consensus Development Conference's conclusion that patients with H. pylori infection should undergo antimicrobial treatment. In 2007, the American College of Gastroenterology estimated that the cure rate for H. pylori infections was 70–85 percent with the use of a proton pump inhibitor (PPI), clarithromycin, and amoxicillin or metronidazole (2).

A recent comprehensive evaluation found that sequential and conventional triple treatment had a cure rate of 84%. As well as 75.1% of the population have an antibiotic resistance to H. pylori strains, which is considered to be the most significant factor impacting cure rates. It is becoming more and more common for H. pylori strains to be resistant to antibiotics. To top it all off, the effectiveness of an H. pylori infection therapy is largely dependent on the host's genetic makeup, namely the presence of cytochrome P450 2C19, interleukin 1B, and multidrug-resistant transporter 1 (3).

In the fight against infectious diseases like H. pylori infection, a critical role is played by a person's own immune system. Secosteroid hormones like vitamin D provide a wide range of beneficial impacts on the body. For many years, it has long been known that vitamin D plays a vital role in the control of calcium and bone homeostasis. But recent studies have shown that vitamin D is also crucial in the regulation of cell proliferation and differentiation. AMPs (antimicrobial peptides) have been shown in recent research to be produced by immune cells when vitamin D is present. This suggests that vitamin D may help modulate the immune response to a variety of viral disorders. The infected macrophage is unable to create enough 1,25-(OH)2D to control the synthesis of AMP cathelicidin when it is vitamin D deficient (4).

Vitamin D has been shown to have an antibacterial effect on mycobacterium tuberculosis-infected macrophages, but cathelicidin, which has broad-spectrum antimicrobial action against gram-negative and gram-positive bacteria, viruses, and fungi, is also widely recognised. There is evidence that vitamin D may have antibacterial properties, and a lack of it may have negative consequences on health and lifespan in general. In addition to boosting innate immunity, vitamin D may also lessen the risk of infection through influencing the production of AMPs and cytokines. As a result, vitamin D has a powerful systemic antibacterial impact by enhancing the activity of monocytes and macrophages. Most illnesses seem to benefit from a vitamin D-rich condition (5).

Vitamin D supplement was used in this research to show that helicobacter pylori infection may be eliminated with treatment.

Patients and methods

Patients:

This case study enrolled 50 sequential Egyptian patients with dyspeptic symptoms for at least 1 month.

Patients will be collected from benha university hospital (inpatient & outpatient),

Benha ,Egypt between april 2021 and july 2021.

Patients will be divided ito 2 groups:

1.Group A: include 25 patients with helicobacter pylori infection with clarithromycin based triple therapy and vitamin D supplement.

2.Group B: include 25 patients with helicobacter pylori infection with clarithromycin based triple therapy and without vit D supplement.

All patients were treated with clarithromycin based triple therapy for 14 days.Helicobacter pylori infection eradication was determined via stool antigen test performed 4 weeks after the end of therapy.

Inclusion criteria:

Patients aged between 18 to 80 years old with helicobacter pylori infection diagnosed by positive stool antigen test for helicobacter pylori infection.

Exlusion criteria:

- Current use of PPIS or vitamin D supplement.
- Known hypersensitivity to PPIS or antibiotics.
- Patients who have previously received helicobacter pylori eradication treatment,corticosteroids/immunosuppressive treatment ,antibiotics,anti-inflammatory and acid suppressive treatment in the prior 2 months.
- History of systemic inflammatory or autoimmune disorders, gastric surgery,renal failure,liver cirrhosis and malignancies.

Methods:

All participants will be subjected to the following:

Full history taking including:

- History of smoking.
- History of vomiting.
- History of alchol intake.
- Dyspeptic symptoms.
- Drug history.
- Diet history.
- Past history of any medical condition.
- Past history of hospital admission.

Full clinical examinations including:

General examination:

- Vital signs.
- Body mass index.
- Dehydration.

Local (abdominal) examination:

- Inspection.
- Palpation:for epigastric tenderness.
- Percussion : of abdomen.
- Auscultation : for intestinal sounds.

Treatment protocol:

All patients were treated with clarithromycin based triple therapy(clarithromycin 500 mg,amoxicillin 1000 mg and esmoperazole 20 mg) twice daily for 14 days.

Recommended Vit D supplement prophylactic dose given to 25 patients was 3000 IU.

Prophylactic dose of Vit D is:

- (19-75) 800-2000 IU/day.
- (>75) 2000-4000 IU/day.

Therapeutic dose of vitamin D is:6000 for eldery IU/day.

(6)

Laboratory investigations will include:

A) Stool antigen test for helicobacter pylori infection before and after vitamin D therapy.

B)Complete blood count(CBC):

- HB(gm/dl).

- WBC(C/mm3).
- Platelets count(C/mm3).

C)LFTS.
D)KFTS.

Ethical consideration:

All patients had informed consent that they were involved in the study. An approval from the research ethics committee in Benha Faculty of Medicine was obtained.

The approval number of the local ethical committee: Ms.4_3.2021.

Statistical Methods

Data management: The clinical data were recorded on a report form. These data were tabulated and analyzed using the computer program SPSS (Statistical package for social science) version 26 to obtain:

Descriptive data: Descriptive statistics were calculated for the data in the form of:

1. Mean and standard deviation $(\pm SD)$. for quantitative data.
2. Frequency and distribution for qualitative data.

Analytical statistics: In the statistical comparison between the different groups, the significance of difference was tested using one of the following test:

1- Student's t-test: - Used to compare mean of two groups of quantitative data.

Inter-group comparison of categorical data was performed by using chi square test (X2-value) and fisher exact test (FET).

A P value <0.05 was considered statistically significant (*) while >0.05 statistically insignificant P value <0.01 was considered highly significant (**) in all analyses.

Results

Table (1) shows that there was statistically significant increase in Hb., TLC, platelets and albumin after intervention compared to before intervention (p<0.001) while, there was statistically significant decrease in urea and serum creatinine after intervention compared to before intervention (p<0.001). There was no statistically significant difference in INR before and after intervention.

Table (2) shows that there was no significant difference between group A who took vitamin D and group B who did not take vitamin D regarding hemoglobin level, TLC and platelets count before and after treatment (p>0.05). In group A, who took vitamin D, there was significant increase in hemoglobin level, TLC and platelets count after treatment compared to before treatment (p<0.001, <0.001 & 0.044 respectively). In group B, who did not take vitamin D, there was significant increase in hemoglobin level , TLC and platelets after treatment compared to before treatment (p<0.001 & 0.026 & 0.073 respectively).

There was no significant difference between group A who took vitamin D and group B who did not take vitamin D regarding urea and serum creatinine levels before and after treatment (p>0.05). In group A, who take vitamin D, there was significant decrease in urea and serum creatinine levels after treatment compared to before treatment (p<0.001 &<0.001 respectively). In group B, who did not take

vitamin D, there was significant decrease in serum creatinine after treatment compared to before treatment (p=0.020) (Table,3).

There was no significant difference between group A who took vitamin D and group B who did not take vitamin D regarding ALT, AST, INR and albumin levels neither before nor after treatment (p>0.05). In group A, who took vitamin D, there was significant decrease in ALT and AST levels after treatment compared to before treatment (p<0.001 &<0.001 respectively). Meanwhile, there was significant increase in albumin level after treatment compared to before treatment (p<0.001). In group B, who did not take vitamin D, there was significant increase in albumin after treatment compared to before treatment (p<0.001) (Table,4).

Table (5) showed a significant improvement at the end of the study in cases of group A compared group B (p= 0.031) as 84% patients in group A showed negative results while in group B 56% patients had negative results.

Table (1): Distribution of the studied cases as per laboratory finding.

		Studied patients (No. = 50)					P-value
		Mean	**± SD**	**Median**	**Min.**	**Max.**	
HB (g/dl)	**Before**	12.78	1.49	12.60	10.40	15.40	**<0.001**
	After	13.27	1.52	13.05	10.50	15.50	
TLC (*10^9/L)	**Before**	7.03	1.35	7.36	4.15	9.30	**<0.001**
	After	7.78	1.35	7.90	5.00	9.90	
PLT (*10^9/L)	**Before**	254.12	79.08	243.50	122.00	431.00	**<0.001**
	After	262.94	76.76	260.00	144.00	418.00	
Urea (mg/dl)	**Before**	29.98	3.77	30.00	22.00	38.00	**<0.001**
	After	28.64	4.10	28.00	20.00	37.00	
Creatinine (mg/dl)	**Before**	.85	.11	.90	.70	1.10	**<0.001**
	After	.77	.13	.80	.60	1.10	
ALT (U/L)	**Before**	22.84	2.90	23.00	18.00	30.00	**<0.001**
	After	21.24	3.35	20.00	15.00	28.00	
AST (U/L)	**Before**	27.60	3.97	28.00	21.00	36.00	**<0.001**
	After	25.84	5.03	25.00	17.00	35.00	
INR	**Before**	1.10	.05	1.10	1.00	1.20	0.715
	After	1.10	.07	1.10	1.00	1.30	
Albumin (g/dl)	**Before**	4.53	.15	4.50	4.20	4.90	**<0.001**
	After	4.62	.14	4.60	4.40	4.90	

p≤0.05 is considered statistically significant, p≤0.01 is considered high statistically significant, SD= standard deviation, comparison between before and after done by Wilcoxon test

Table (2): Comparison between the two studied groups regarding CBC before and after.

	Group (A) (No. = 25)			Group (B) (No. = 25)			Test value**	P-value
	Mean	± SD	Median	Mean	± SD	Median		
				Hb. (g/dl)				
Before	12.84	1.45	12.90	12.72	1.56	12.30	0.418	0.686
After	13.40	1.47	13.90	13.14	1.59	12.90	0.554	0.580
p-value* (before / after)		**<0.001**			**<0.001**			
				TLC (*10^9/L)				
Before	6.78	1.35	6.55	7.28	1.32	7.69	1.379	0.168
After	7.72	1.38	7.90	7.84	1.35	7.90	0.330	0.741
p-value* (before / after)		**<0.001**			**0.026**			
				Platelets (*10^9/L)				
Before	255.20	72.33	246.00	253.04	86.79	241.00	0.262	0.793
After	263.32	71.82	260.00	262.56	82.88	260.00	0.117	0.907
p-value* (before / after)		**0.044**			0.073			

*p≤0.05 is considered statistically significant, p≤0.01 is considered highly statistically significant * Wilcoxon Signed rank Test **. Mann- Whitney U Test. SD: standard deviation,*

Table (3): Comparison between the two studied groups regarding kidney function tests before and after treatment.

	Group (A) (No. = 25)			Group (B) (No. = 25)			Test value**	P-value
	Mean	± SD	Median	Mean	± SD	Median		
				Urea (mg/dl)				
Before	30.60	3.48	30.00	29.36	4.01	30.00	1.242	0.214
After	28.56	3.86	28.00	28.72	4.40	28.00	0.098	0.922
p-value* (before / after)		**<0.001**			0.088			
				S. creatinine (mg/dl)				
Before	.88	.11	.90	.83	.12	.80	1.401	0.161
After	.77	.12	.80	.78	.14	.70	0.193	0.847
p-value* (before / after)		**<0.001**			**0.020**			

*p≤0.05 is considered statistically significant, p≤0.01 is considered highly statistically significant * Wilcoxon Signed rank Test **. Mann- Whitney U Test. SD: standard deviation,*

Table (4): Comparison between the two studied groups regarding liver function tests before and after.

	Group (A) (No. = 25)			Group (B) (No. = 25)			Test value**	P-value
	Mean	± SD	Median	Mean	± SD	Median		
ALT (U/L)								
Before	23.20	3.19	23.00	22.48	2.58	22.00	0.675	0.500
After	21.00	3.23	20.00	21.48	3.53	21.00	0.492	0.623
p-value* (before / after)		**<0.001**			0.067			
AST (U/L)								
Before	27.68	4.39	28.00	27.52	3.58	28.00	0.078	0.938
After	25.20	5.02	24.00	26.48	5.07	26.00	0.896	0.370
p-value* (before / after)		**<0.001**			0.122			
INR								
Before	1.09	.06	1.10	1.10	.05	1.10	0.366	0.715
After	1.09	.07	1.10	1.11	.07	1.10	0.748	0.455
p-value* (before / after)		0.671			0.339			
Albumin (g/dl)								
Before	4.52	.16	4.50	4.54	.14	4.50	0.472	0.637
After	4.63	.13	4.60	4.61	.15	4.60	0.897	0.369
p-value* (before / after)		**<0.001**			**<0.001**			

*p≤0.05 is considered statistically significant, p≤0.01 is considered highly statistically significant * Wilcoxon Signed rank Test **. Mann- Whitney U Test. SD: standard deviation,*

Table (5): Comparison between the two studied groups regarding stool antigen test for helicobacter pylori infection.

		Group (A) (No. = 25)		Group (B) (No. = 25)		Test value	P-value
		N	%	N	%		
H. Pylori stool antigen test	**Negative**	21	84.0%	14	56.0%	X^2= 4.667	**0.031**
	Positive	4	16.0%	11	44.0%		

p≤0.05 is considered statistically significant, p≤0.01 is considered high statistically significant, SD= standard deviation, comparison between groups done by and Chi- Square test

Discussion

The results of this research demonstrate that there was a statistically significant rise in Hb., TLC, platelets, and albumin after intervention, whereas there was a statistically significant drop in urea and serum creatinine, AST& ALT after intervention. Before and after the intervention, there was no statistically significant variation in INR values.

That's what the new research on CBC found, at least Hemoglobin, TLC, and platelet counts did not change significantly between groups A and B who took vitamin D or did not (p>0.05) before and after therapy in group A. Hemoglobin, TLC, and platelet counts all increased significantly following therapy in group A (*p*=0.001, 0.001, and 0.044, respectively) in comparison to

baseline levels. A substantial rise in haemoglobin and TLC was seen in group B after therapy compared to before treatment (p=0.001 and 0.026).

There was no significant difference in urea and creatinine levels before and after treatment between groups A and B (p>0.05) when it came to vitamin D supplementation. Urea and serum creatinine levels dropped significantly in group A following vitamin D administration (p=0.001 and p0.001, respectively). As a result of therapy, serum creatinine levels in group B decreased significantly (p=0.020) compared to before treatment.

In terms of liver function tests, there was no significant difference between groups A and B regarding ALT, AST, INR, and albumin levels before or after therapy (p>0.05). Vitamin D therapy significantly reduced the ALT and AST levels in group A when compared to baseline (p=0.001 & 0.001). After therapy, albumin levels rose significantly (p= 0.001) compared to before treatment. There was a substantial increase in albumin after therapy compared to before treatment in group B, (p=0.001).

According to another study, our findings were confirmed by their investigation, which found that the serum levels of AST and ALT were dramatically reduced following treatment. 46.6 percent and 45.7 percent of the patients with baseline levels beyond the normal range had their AST and ALT levels fall within the normal range (7).

After H. pylori treatment, there were 14 patients with a full response, 20 with a partial response, and 31 with no response (47.7%), 12 of which were eliminated and 19 of which were not eradicated.

All biochemical indicators and reported cirrhotic consequences were significantly reduced following treatment of H. pylori infection (all p=0.05) by group of researchers (8).

Stool antigen tests for H. pylori infection were performed on all of the participants in our study. Compared to group B, group A showed a significant improvement at the conclusion of the trial (p= 0.031) as 84% of patients in group A exhibited negative findings, but only 56% of patients in group B showed negative results. Vitamin D. effectiveness was compared among the participants investigated. In group A, 84 % of patients exhibited good outcomes, compared to 56% of patients in group B, (p= 0.031), indicating a substantial difference in the effectiveness of vitamin D.

Another research, splited their patients into two groups and confirmed our findings: Amoxicillin, clarithromycin, and esomeperazol were administered for two weeks to patients in group A (n=75). Amoxicillin, clarithromycin, and esomeperazol were given to 75 patients in group B (n=75) for two weeks in addition to 1,25-hydroxy vitamin D3 for four weeks. In group A, eradication was accomplished by PP in 46 of 62 patients (74.19%) and by ITT analysis in 46 of 75 (61.33%) whereas in group B, eradication was achieved by PP in 60 of 68 patients (88.13%) and by ITT analysis in 60 of 75 (80%). There was a statistically significant difference in the

eradication rates of group B and group A (p=0.012 in ITT and p=0.029 in PP). The mean blood 25-OH vitamin D levels were 28.910.6 in group A and 28.39.8 in group B, with no statistically significant differences (p=0.268) between the two groups (9).

As another study found, 46 of 62 (74.19%) and 46 of 75 (61.33%) H. pylori eradication was achieved in group A by PP and ITT analyses, respectively. But in group B, eradication was accomplished in 60 of 68 (88.23%) and 60 of 75 (80%) patients using PP and ITT analysis, respectively. There was a significant difference in H. pylori eradication rates (p = 0.012 and p = 0.029 in ITT and PP analyses, respectively) when vitamin D3 was added to the clarithromycin-based triple treatment (10).

H. pylori may be eradicated by supplementing with vitamin D, according to a number of studies. The vitamin D3 breakdown product (VDP1) affects only H. pylori in a selective manner, while having no effect on the survival of Enterobacteriaceae bacteria, Pseudomonas aeruginosa, or Staphylococcus aureus in any way (11).

According to several reports, dimyristoyl-phosphatidylethanolamine (DMPE) is an important component of the cell membrane of H. pylori, and VDP1 dissolves bacteria by coming into contact with DMPE of the H. pylori membrane. Alkyl of indene, a result of vitamin D breakdown, was shown to link up with H. pylori's di-14:0 DMPE, according to a separate research. Alkyl's absence indicated H. pylori was no longer germicidal, and thus causes the breakdown to occur (12). Serum vitamin D levels have been linked to H. pylori eradication in many studies Researchers found a clear link between H. pylori eradication attempts that failed and low levels of vitamin D in the blood (13).

More than 70% of patients were successfully eradicated and less than 30% were unsuccessful, according to another study (10). Eradication failure patients had considerably lower levels of 25[OH]D3 than treatment success patients (14.74.5 vs 27.417.1; p = 0.001). In addition, 30 of the 30 patients in the failed therapy group had 25[OH]D3 levels below normal, compared to only 10 of the 10 patients in the successful treatment group (p= 0.001).

Group of researchers also found a decline in the number of H. pylori (+) patients in the highest 25(OH) vitamin D quartiles (p =0.010) when 25(OH) vitamin D levels rose (14).

It was also reported by another study that the H. pylori eradication rate in individuals with blood vitamin D levels of 10ng/mL and 10ng/mL was substantially different (71.7% and 87.3%), respectively. Vitamin D deficiency was shown to be an independent risk factor for eradicating H. pylori (odds ratio 0.381, 95% confidence range 0.183-0.791; p = 0.010) in a multivariate analysis (15).

Ten papers were eventually included in a meta-analysis. H pylori-positive patients had lower average 25(OH)D levels than H pylori-negative patients (SMD = 0.53 ng/mL, 95 percent CI = (0.91, 0.16 ng/mL))

individuals. There was a statistically significant difference in 25(OH)D3 levels between those who successfully eliminated H pylori and those who did not (SMD = 1.31 ng/mL, 95 percent CI = [0.60, 2.02 ng/mL]). The elimination rate of H. pylori was also reduced in vitamin D-deficient patients (OR = 0.09, 95 percent CI = [0.02, 0.41]). According to the meta-sensitivity analysis, the findings were consistent and credible (16).

Conclusion

H. pylori eradication rates might be greatly improved by complementing clarithromycin-based triple therapy with vitamin D3, as evidenced by our study findings.

References

1. **V Mahachai, RK Vilaichone, R Pittayanon, J Rojborwonwitaya, S Leelakusolvong, M Maneerattanaporn, et al.** Helicobacter pylori management in ASEAN: The Bangkok consensus report. J Gastroenterol Hepatol. 2018; vol.33(1),pp.37-56.
2. **A Sokwala, MV Shah, S,Devani G Yonga.** Helicobacter pylori: a randomized comparative trial of 7-day versus 14-day triple therapy. S Afr Med J. 2012; vol.102,pp. 368–71.
3. **L Gatta, NVakil. D Vaira, Scarpignato C.** Global eradication rates for Helicobacter pylori infection: systematic review and meta-analysis of sequential therapy. Bmj. 2013; vol. 347,pp. f4587.
4. **MT Kitson, SK Roberts.** D-livering the message: the importance of vitamin D status in chronic liver disease. J. Hepatol. 2012; vol. 57(4),pp.897–909.
5. **DA Youssef, CW Miller, AM El-Abbassi.** Antimicrobial implications of vitamin D. Dermatoendocrinol. 2011; vol. 3(4),pp. 220-229.
6. **Holick MF.** The vitamin D deficiency pandemic: Approaches for diagnosis, treatment and prevention. Rev Endocr Metab Disord. 2017 Jun;18(2):153-165.
7. **Salehi H, Minakari M, Yaghoutkar A, Tabesh E, Salehi M, Mirbagher L.** The effect of Helicobacter pylori eradication on liver enzymes in patients referring with unexplained hypertransaminasemia. Adv Biomed Res. 2014 May 28;3:131.
8. **Abdel-Razik A, Mousa N, Elhelaly R, Elzehery R, Hasan AS, Abdelsalam M, et al**. *Helicobacter pylori* as an Initiating Factor of Complications in Patients With Cirrhosis: A Single-Center Observational Study. Front Med (Lausanne). 2020 Mar 24;7:96.
9. **M EL Shahawy.** The Efficacy Of Supplementation Of Vitamin D3 On The Eradication Rates Of Clarithromycin-Based Triple Therapy For Helicobacter Pylori Infection. Joint Event on Global Pharma Summit & 2nd International Conference on Gastroenterology and Hepatology November. 2018; vol. 8,pp. 23-24.
10. **El Shahawy MS, MH Hemida, I El Metwaly, ZM Shady.** The effect of vitamin D deficiency on eradication rates of Helicobacter pylori infection. JGH Open. 2018; vol. 2(6),pp. 270-275.
11. **Hosoda K, H Shimomura, K Wanibuchi.** Identification and characterization of a vitamin D_3 decomposition product bactericidal against Helicobacter pylori. Sci Rep. 2015; vol. 5,pp. 88-60.
12. **Wanibuchi K, K Hosoda, M Ihara.** Indene compounds synthetically derived from vitamin D have selective antibacterial action on Helicobacter pylori. Lipids. 2018; vol 53,pp. 393-401.
13. **Yildirim O, T Yildirim, Y Seckin, P Osanmaz, Y Bilgic, R Mete.** The influence of vitamin D deficiency on eradication rates of Helicobacter pylori. Adv Clin Exp Med. 2017; vol. 26,pp. 1377-1381.
14. **Mut Surmeli D, Surmeli ZG, Bahsi R, Turgut T, Selvi Oztorun H, Atmis V, et al.** Vitamin D deficiency and risk of Helicobacter pylori infection in older adults: a cross-sectional study. Aging Clin Exp Res. 2019 Jul;31(7):985-991.
15. **Han C, Ni Z, Yuan T, Zhang J, Wang C, Wang X, et al**. Influence of serum vitamin

D level on Helicobacter pylori eradication: A multi-center, observational, prospective and cohort study. J Dig Dis. 2019 Aug;20(8):421-426.

16. **Yang L, He X, Li L, Lu C.** Effect of vitamin D on Helicobacter pylori infection and eradication: A meta-analysis. Helicobacter. 2019 Oct;24(5):e12655.

To cite this article: Amira K. El-Alfy , Basma A. Badr, Mohamed A. Hewedi, A R Mohamed. The Effect of vitamin D Therapy on Eradication Rates of Helicobacter Pylori Infection. BMFJ 2023;40(30):640-650.