

ASSOCIATION OF HELICOBACTER PYLORI INFECTION AND VITAMIN B12 LEVEL AMONG SUDANESE PATIENTS IN KHARTOUM STATE-2018

Ola A. Mohammed¹, Mohammed M. Mohammed¹, Rimaz A. Mohammed¹, Zaienab S. Abdulaziz² and Tarig A. M. Hamid^{1*}

¹Department of Hematology and Immunohematology - Karry University- Khartoum Sudan.

²Department of Hematology and Immunohematology – Sharq Elnile College – Khartoum Sudan.

*Corresponding Author: Tarig A. M. Hamid

Department of Hematology and Immunohematology - Karry University- Khartoum Sudan.

Article Received on 01/06/2018

Article Revised on 22/06/2018

Article Accepted on 12/07/2018

ABSTRACT

Helicobacter pylori is an organism that is reported to cause deficiency of vitamin B₁₂ by producing gastritis and peptic disease which results in vitamin B₁₂ malabsorption, the exact mechanism is unknown but the suggested mechanisms are that *H. pylori* decreases the hydrochloric acid content of the gastric juice which effect on vitamin B₁₂ absorption. A case-control study was conducted in the Military hospital, Khartoum state, Sudan, where *H. pylori* infection was diagnosed. Study done during Feb to April 2018 and included 80 samples, 50 of these samples were collected from patient diagnosed with *H. pylori* as a case group, and 30 samples were collected from apparently healthy individuals as control group. A total of 3 ml venous blood samples were collected to obtain Serum used for estimation of serum B₁₂ level by competitive Immunoluminometric assay among tow studied group. The present study showed that There was a significant decrease of S. B₁₂ level among case compare with control group with a mean (297.7 ± 289.6, 569.8 ± 171.5) respectively (P; 0.007). Also show there is no correlation between S. B₁₂ level in *H. pylori* patient compare with gender, *H. pylori* infection duration, other disease, bloody stool and treatment with P. value (0.949, 0.158, 0.684, 0.339, 0.146) respectively. The study concluded that *Helicobacter pylori* has a significant effect on vitamin B12 level and *H. pylori* infection is a cause of vitamin B12 deficiency. Indicate there is an association between S.B₁₂ deficiency and *H. pylori* infection.

KEYWORDS: *Helicobacter pylori* is malabsorption, Immunoluminometric and *H. pylori* infection.

I. INTRODUCTION

Vitamin B₁₂ deficiency is common seen in clinical practice [Alfawaeir SAA (2013) *et al*]. Vitamin B₁₂ deficiency often goes undetected with manifestations that range from asymptomatic to a wide spectrum of hematologic and/or neuropsychiatric features [Gilsing AM (2010) *et al*], and observed in patients with *Helicobacter pylori* infection [Alfawaeir SAA (2013) *et al*], the reported prevalence for vitamin B₁₂ deficiency in *Helicobacter pylori* infection was 67% of infected cases [Sarari AS (2008) *et al*].

Helicobacter pylori is a bacteria specially adapted to survive in the gastric lumen. *H. pylori* responsible for widespread infection with more than 50% of the world's population infected, even though 80% of those infected have no symptoms. Infection with *H. pylori* has been recognized as a public health problem worldwide and more prevalent in developing than the developed countries [Devrajani, B.R.(2010)*et al*].

Malabsorption plays a major role in the development of different nutritional deficiencies. Stomach plays very

important role especially with respect to vitamin B₁₂. Deficiency of vitamin B₁₂ mostly results from gastrointestinal diseases that impair secretion of intrinsic factor from parietal cells of the gastric glands. Major problem of the stomach include gastritis that leads to the development of peptic ulcers, and gastric cancers. It has been reported that the most common cause of gastric problems is *Helicobacter pylori* [Pietrojusti A (2008), Kuipers EJ (2007) *et al*].

Helicobacter pylori is an organism that is reported to cause deficiency of vitamin B₁₂ by producing gastritis and peptic disease which results in vitamin B₁₂ malabsorption [Lechner K (2005) *et al*]. The exact mechanism is not known but the suggested mechanisms are that *H. pylori* decreases the hydrochloric acid content of the gastric juice [El-Omar EM (1997) *et al*]. Secondly, *H. pylori* neutralizes gastric acidity by producing ammonia, the first required for separating vitamin B₁₂ from the dietary sources [Salgueiro J (2004) *et al*]. Thirdly, *H. pylori* cause decreased secretion of the pepsin (a proteolytic enzyme). The above mentioned three factors are necessary for release of vitamin B12

from the food. [El-Omar EM (1997), Salgueiro J (2004) *et al.*].

Fourthly, *H. pylori* decreases the release of intrinsic factor from the parietal cells that is a necessary factor for the absorption of vitamin B₁₂ from the small intestine [Lechner K (2005), Dierkes J (2003), Carmel R (1997) *et al.*].

A recent review of a number of published studies on the influence of *H. pylori* on nutritional status revealed that the infection appeared to have a definite negative effect on vitamin B₁₂ and vitamin C metabolism [Akcam, M.(2010), Stabler S.(2013), Wu, M.C.(2014) *et al.*].

The present study was planned to observe whether *H. pylori* infection in the gastric mucosa is responsible for vitamin B₁₂ deficiency, so as early detection and eradication of *H. pylori* can prevent the development of complications as gastritis, gastric, duodenal ulcer and consequence vitamin B₁₂ deficiency.

II. MATERIALS AND METHODS

2.1 Study area and population: This case-control study was conducted in the Military hospital, Khartoum state,

Sudan, where *H. pylori* infection was diagnosed. Study done during Feb to April 2018 and included 80 samples, 50 of these samples were collected from patient diagnosed with *H. pylori* as a case group, and 30 samples were collected from apparently healthy individuals as control group.

2.2 Sample collection: A total of 3 ml venous blood samples were collected by using sterile disposable syringes and poured into plain containers, immediately centrifuged to obtain serum which separated. Serum used for estimation of serum B₁₂ level by competitive immunoluminometric assay among two studied group.

2.3 Statistical Analysis All values were expressed as mean \pm SD. Statistical analyses were done using the Student's t-test to assess differences among study groups. The level of significance was set at P < 0.05.

III. RESULTS

The study has been done on 80 participants (31 female and 49 male), 50 patient with *H. pylori* considered as a case group and 30 apparent healthy individual as control group.

Table 1: Demographic data of two studied groups.

	Case Mean \pm SD	Control Mean \pm SD	P. value
Age	32.3 \pm 14.7	29.9 \pm 5.1	0.008

The mean of age among two studied group were found (32.3 \pm 14.7, 29.9 \pm 5.1) (P; 0.008) of case to control group respectively.

Table 2: Comparison of means and SD of serum B₁₂ level among the two studied group.

	Case Mean \pm SD	Control Mean \pm SD	p. value
S. B ₁₂ level	297.7 \pm 289.6	569.8 \pm 171.5	0.007

There was a significant decrease of S. B₁₂ level among case group with a mean of case to control group (297.7 \pm 289.6, 569.8 \pm 171.5) respectively (P; 0.007).

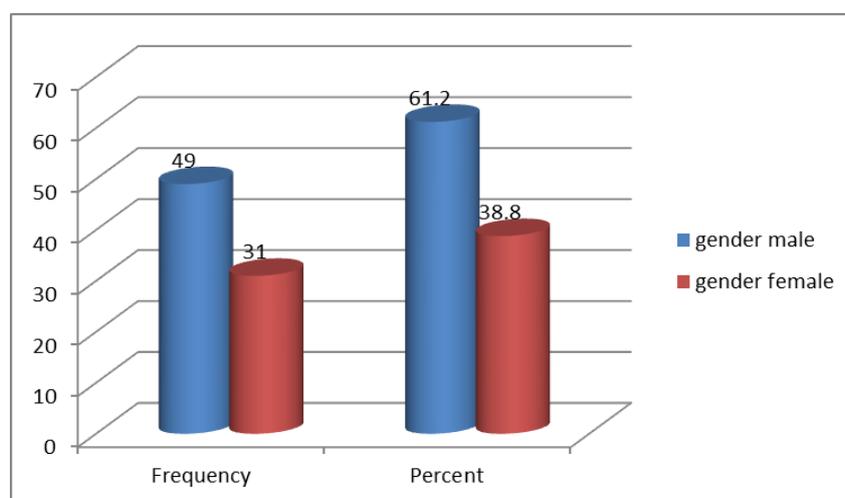


Figure 1: Frequency and percentages of gender among Study population.

The percentage of males was found to be 61.2% while female percentage 38.8% among study population.

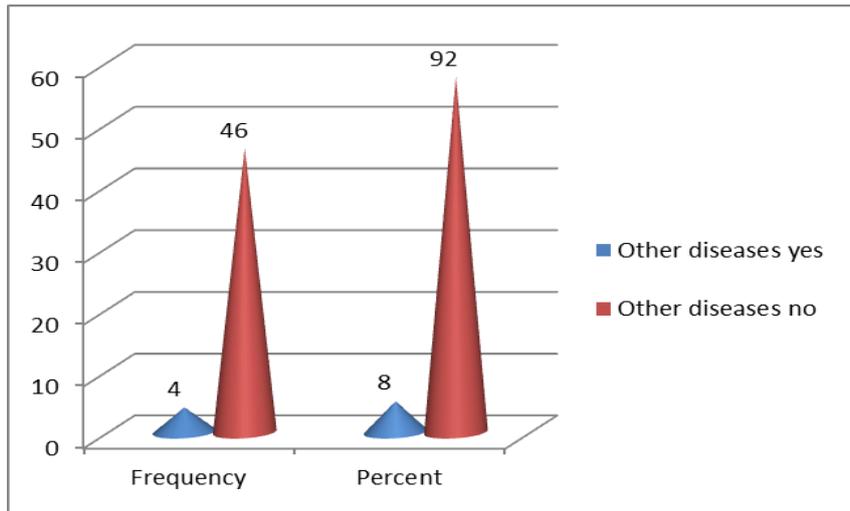


Figure (2): Frequency and percentage of patient with disease other than *H. pylori* infection.

There were 8% of case group has another infection rather than *H. pylori* infection.

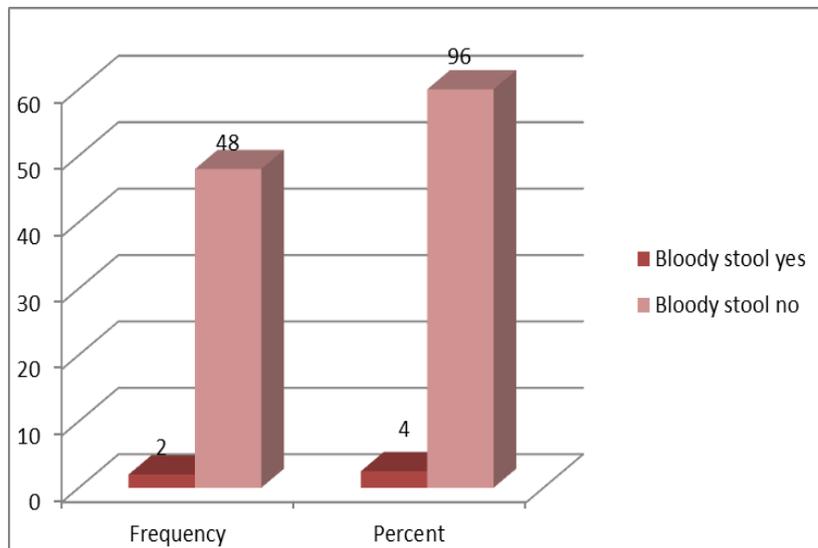


Figure 3: Frequency and percentages of *H. pylori* patient who having a bloody stool.

There were 4% of case group having a bloody stool.

Table 5: Correlation between S. B₁₂ in *H. pylori* patient level with gender, *H. pylori* infection duration, treatment and persistence of other disease among case group.

	gender	Duration	Other disease	Bloody stool	treatment
P. value	0.949	0.158	0.684	0.339	0.146

There is no association between S. B₁₂ level in *H. pylori* patient compare with gender, *H. pylori* infection duration, other disease, bloody stool and treatment with P. value (0.949, 0.158, 0.684, 0.339, 0.146) respectively.

IV. DISCUSSION

Eighty blood sample included in this study, 50 from patient with *H. pylori* infection considered as case group and 30 from Healthy individual included as control group. The mean Result of serum B₁₂ in *H. pylori* patient was significant decrease in case group than control group with (P; 0.007). This agree with previous studies, One

done by [Ahmed.T *et al* 2017] was conducted in Khartoum-Sudan one hundred and twenty blood sample included 60 from patient with *H. pylori* positive as case and 60 from Healthy individual as control group which concluded that serum B₁₂ was found decrease in *H. pylori* positive group (P; 0.00). Our study also agree with study done by [Wesam Mohammed -2016], concluded that the level of Vitamin B12 lower than normal range 200 pg/ml in 21.3% of study population. Also agree with study Done by [kadhim G *et al* -2015 in Malaysia] on one hundred and twenty nine (129) *Helicobacter pylori* infected patients, sixty seven (52%) *H. pylori* infected

patients had normal vitamin B₁₂ level, 31 (47%) were males and 36 (53%) were females. Whereas sixty two (48%) *H. pylori* infected patient had low vitamin B₁₂ level (P; 0.02).

Present study show there is no association between S. B₁₂ level in *H. pylori* patient compare with gender, *H. pylori* infection duration, other disease, bloody stool and treatment with (P; 0.949, 0.158, 0.684, 0.339, 0.146) respectively.

V. CONCLUSION

It is concluded that *Helicobacter pylori* has a significant effect on vitamin B¹² level and *H. pylori* infection is a cause of vitamin B¹² deficiency. Indicate there is an association between S.B₁₂ deficiency and *H. pylori* infection.

ACKNOWLEDGEMENT

Special thanks to Hanan Maki -Bahri teaching Hospital, Khartoum- Sudan.

REFERENCES

1. **Akcam, M.** (2010). *Helicobacter pylori* and micronutrients. Indian Pediatr., 47: 119–126.
2. **Alfawaeir SAA, Abuzaid MB.** Prevalence of vitamin B12 deficiency in *Helicobacter pylori* infected patients in Jordan. J Invest Biochem., 2013; 2(1): 21-5.
3. **Bener A, Micallef R, Afifi M, Derbala M, Al-Mulla HM, Usmani MA.** Association between type 2 diabetes mellitus and *Helicobacter pylori* infection. Turk J Gastroenterol, 2007; 18(4): 225-9.
4. **Devrajani, B.R., Shah, S.A., Soomro, A.A. and Devrajani, T.** (2010). Type 2 diabetes mellitus: A risk factor for *Helicobacter pylori* infection: A hospital based case-controls study. Int. J. Diab. Dev. Ctries., 30: 22-26.
5. **Gilsing AM, Crowe FL, Lloyd-Wright Z, Sanders TA, Appleby PN, Allen NE, et al.** Serum concentrations of vitamin B12 and folate in British male omnivores, vegetarians and vegans: results from a cross-sectional analysis of the EPIC-Oxford cohort study. Eur J Clin Nutr., 2010; 64(9): 933-9.
6. **Goodman KJ, Cockburn M.** The role of epidemiology in understanding the health effects of *Helicobacter pylori*. Epidemiology., 2001; 12: 266.
7. **Langan RC, Zawistoski KJ.** Update on vitamin B12 deficiency. Am Fam Physician., 2011; 83(12): 1425-30.
8. **Sarari AS, Farraj MA, Hamoudi W, Essawi TA.** *Helicobacter pylori*, a causative agent of vitamin B12 deficiency. J Infect Dev Ctries., 2008; 2(5): 346-9.
9. **Stabler, S.** (2013). Vitamin B12 deficiency. N. Engl. J. Med., 368: 149-160.
10. **Troilo A, Mecili M, Ciobanu E, Boddi V, D'Elios MM, Andres E.** Oral vitamin B12: Efficacy and safety data in 31 patients with pernicious anemia and food-cobalamin malabsorption. Presse Med., 2010; 39(12): e273-9.
11. **Rothenbacher, D. and Brenner, H.** (2003). Burden of *Helicobacter pylori* and *H. pylori* related diseases in developed countries: recent developments and future implications. Microbes Infect., 5: 693–703.
12. **Wex T, Venerito M, Kreutzer J, Gotze T, Kandulski A, Malfertheiner P.** Serological prevalence of *Helicobacter pylori* I infection in Saxony-Anhalt, Germany, in 2010. Clin Vaccine Immunol., 2011; 18(12): 2109-12.
13. **Windsor HM, Abioye-Kuteyi EA, Leber JM, Morrow SD, Bulsara MK, Marshall BJ.** Prevalence of *Helicobacter pylori* in Indigenous Western Australians: comparison between urban and remote rural populations. Med J Aust., 2005; 182(5): 210-3.
14. **Wu, M.C., Huang, C.Y., Kuo, F.C., Hsu, W.H., Wang, S.S.W., Shih, H.Y., Liu, C.J., Chen, Y.H., Wu, D.C., Huang, Y.L. and Lu, C.Y.** (2014). The 66 Effect of *Helicobacter pylori* Eradication on the Levels of Essential Trace Elements. Biomed. Research International, 1-5.
15. **Pietrojusti A, Galante A, Magrini A, Bergamaschi A.** *Helicobacter pylori* interference with micronutrients and orally administered drugs: a new mechanism explaining its role in extra gastric disorders. Mini Rev Med Chem., 2008; 8: 135-41.
16. **Kuipers EJ, B MJ.** Acid peptic disease: Epidemiology and Pathobiology. In: Goldman L, Ausiello D, eds. Cecil Medicine. 23rd ed. Philadelphia: Elsevier., 2007: 1009-13.
17. **Lechner K, Fodinger M, Grisold W, Puspok A, Sillaber C.** Vitamin B12 deficiency. New data on an old theme. Wien Klin Wochenschr., 2005; 117: 579-91.
18. **Dierkes J, Ebert M, Malfertheiner P, Luleya C.** *Helicobacter pylori* Infection, vitamin B12 and Homocysteine. Dig Dis., 2003; 21: 237–44.
19. **Carmel R.** Cobalamin, the stomach and aging. Am J Clin Nutr., 1997; 66: 750–59.
20. **El-Omar EM, Oien K, El-Nujumi A, Gillen D, Wirz A, Dahill S, Williams C, Ardill JE, McColl KE.** *Helicobacter pylori* infection and chronic gastric acid hyposecretion. Gastroenterology, 1997; 113: 15-24.
21. **Salgueiro J, Zubillaga M, Goldman C, Barrado A, Martinez Sarrasague M, Leonardi N, Boccio J.** Is there a link between micronutrient malnutrition and *Helicobacter pylori*? Aliment Pharmacol Ther., 2004; 20: 1029-34.