International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

Original Research Article.

Clinical Correlation: Helicobacter Pylori and Immune Thrombocytopenic Purpura (ITP)

Oras N. Hamad

Department of Anatomy, College of Medicine, University of Misan, Iraq.

ABSTRACT

Background: Thrombocytopenic purpura is a widespread autoimmune blood disorder that can affect people of all ages. It occurs when the immune system produces antibodies that attack platelets, leading to their destruction. In recent years, researchers have proposed that Helicobacter pylori bacteria may play a role in the development of chronic idiopathic thrombocytopenic purpura (ITP). This study aimed to investigate two main aspects: first, to determine how common H. pylori infection is among ITP patients, and second, to assess whether eliminating the bacteria affects the patients' platelet counts.

Materials and Methods: The research involved 34 participants diagnosed with idiopathic thrombocytopenic purpura (ITP), consisting of 5 males and 29 females. The age range of the subjects was 6 to 67 years, with an average age of 38.74 years and a standard deviation of 11.988 years. All patients underwent platelet count testing. Treatment for ITP included either intravenous immunoglobulin (IVIG) or a 60 mg dose of prednisolone. Following the platelet count test, two additional investigations were conducted: an antibody test for Helicobacter pylori and a stool analysis to detect H. pylori antigens. The stool analysis utilized the Helicobacter pylori stool antigen (HpSA) enzyme immunoassay (EIA) method.

Results: The study findings revealed a significant correlation between platelet counts in ITP patients before and after treatment with anti-H. pylori medications. This relationship was observed in patients who tested positive for H. pylori antibodies. The statistical analysis showed a P-value of 0.000, indicating strong statistical significance. The odds ratio (OR) with a 95% confidence interval (CI) ranged from -175,501.6 to -106,439.5. These results suggest that treating H. pylori infection in ITP patients who test positive for the bacteria may have a substantial impact on their platelet counts. The extremely low P-value indicates that the change in platelet counts is very unlikely to be due to chance. The negative values in the confidence interval suggest a decrease in some measures, possibly indicating a reduction in the severity of thrombocytopenia after H. pylori treatment. The research findings demonstrate a significant link between Helicobacter pylori infection and idiopathic thrombocytopenic purpura (ITP). Importantly, the study showed that eliminating H. pylori in infected ITP patients led to meaningful improvements in their condition. Specifically, eradicating the bacteria was associated with remission of ITP symptoms and a notable increase in platelet counts.

Conclusion: These results suggest that H. pylori infection may play a role in the development or persistence of ITP in some patients. Furthermore, the study indicates that testing for and treating H. pylori infection could be a valuable approach in managing ITP, particularly for patients who test positive for the bacteria. This finding could have important implications for ITP treatment strategies, potentially offering a new avenue for improving outcomes in a subset of ITP patients.

Keywords: Thrombocytopenic Purpura (ITP), Autoimmune, H. Pylori, Platelet Count.

***Correspondence to:**

Dr. Oras N. Hamad,
Department of Anatomy,
College of Medicine, University of Misan, Iraq.
Email: orasnlt@gmail.com

Article History:

Received: 06-09-2024, **Revised:** 28-09-2024, **Accepted:** 19-10-2024

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2024.10.6.002	

INTRODUCTION

Immune thrombocytopenic purpura (ITP), formerly known as Werlhof disease, is an autoimmune blood disorder. It's characterized by abnormally low platelet counts due to the immune system's destruction of platelets through autoantibodies. Platelets are crucial for blood clotting, so their depletion can lead to easier bleeding.[1] The British Society of Hematology defines ITP as an autoimmune condition where autoantibodies attach to platelet antigens, causing premature platelet destruction. This process primarily occurs in the spleen and sometimes in the liver, facilitated by the reticuloendothelial system.[2] ITP patients may not show symptoms initially or may experience mild mucocutaneous bleeding. In some cases, life- threatening hemorrhages can occur.[3] While only 5% of patients present with severe bleeding at diagnosis, there's a 15% chance of experiencing severe bleeding

requiring hospitalization within five years of diagnosis. It's important to note that the risk of severe bleeding in ITP isn't uniform across all patients. Factors that can increase this risk include extremely low platelet counts, a history of previous bleeding episodes, and certain coexisting medical condition.[4] Universally known as a causative agent of gastritis, peptic ulcers and a risk factor for the development of gastric adenocarcinoma and mucosa associated lymphoid tissue lymphoma.[5,6] Infection has also been associated with coronary heart disease, acne rosacea, chronic idiopathic urticaria and autoimmune diseases.[7] Recently showed a high prevalence of H pylori infection in patients with ITP and reported a good response to bacterium eradication in most of them.[7] This finding has been confirmed in a further anecdotal ITP case.[8] And successful eradication of H. pylori results in an increase of the platelet count in approximately half of H. pylori positive ITP patients. H. pylori eradication therapy has been recommended as a useful measure in treatment of ITP by both the Maastricht IV Consensus.[9] The possible pathophysiological mechanism of this subset of ITP was mentioned by some authors, they suggested that a cross molecular mimicry between the highly antigenic H. pylori CagA protein and platelet antigens.[10-12]

PATIENTS AND METHOD

This study examined 34 patients diagnosed with Immune Thrombocytopenia (ITP), comprising 5 males and 29 females.

The participants, aged between 6 and 67 years (mean age 38.74 ± 11.988 years), were randomly selected. All patients underwent platelet count testing, as detailed in Table 3. Treatment for ITP involved either intravenous immunoglobulin (IVIG) or prednisolone. The choice between these treatments depended on drug availability at the hospital, as shown in Table 2. Prednisolone was administered at 60 mg daily for three months, followed by a reduced dose of 30 mg daily.

Following the platelet count test, additional investigations included an anti-H. pylori antibody test and detection of H. pylori antigens in stool samples.

The latter used the Helicobacter pylori stool antigen (HpSA) enzyme immunoassay (EIA) method. The study was conducted at Al-zahraa Teaching Hospital in Wasit, Iraq. All participants were included in the study after obtaining approval from the ethics committees of the participating universities and university hospitals.

Informed consent was secured from each participant before their involvement in the study.

Table 1: Frequency and percentage between the male and female

Sex	Frequency	Percentage
Male	5	14.7
Female	29	85.3
Total	34	100

Table 2: Frequency of patients and the type of treatment that received by patients to treat (ITP)

Type of treatment	Frequency	Percentage
Prednisolone	28	82.4
Intravenous immunoglobulin (IVIG)	6	17.6
Total	34	100.0

Table 3: Frequency and count of platelet before and after received the treatment of H. pylori in patients diagnosed with (ITP)

Platelet count before receiving the treatment of anti H. pylori	Frequency	Percentage	Platelet count after receiving the treatment of anti H. pylori	Frequency	Percentage	T-test	OR (95% CI) Lower-Upper
500	1	2.9	10000	1	2.9		
1300	2	5.9	15500	1	2.9		
2000	1	2.9	21000	1	2.9		
2200	2	5.9	24000	1	2.9		
5000	1	2.9	30000	1	2.9		
10500	2	5.9	42000	1	2.9		
11000	6	17.6	45000	1	2.9		
13000	1	2.9	90000	1	2.9		
15000	3	8.8	92000	1	2.9		
17000	4	11.8	98000	2	5.9		
20000	2	5.9	100000	2	5.9		
21000	2	5.9	110000	1	2.9	T= -8.30	
22000	3	8.8	120000	1	2.9	P= 0.000	(-175501.6)- (-106439.5)
24000	1	2.9	145000	3	8.8		
30000	2	5.9	155000	2	5.9		
48000	1	2.9	200000	3	8.8		
		100.0	210000	2	5.9		
			225000	2	5.9		
			230000	1	2.9		
			250000	2	5.9		
			290000	1	2.9		
			310000	2	5.9		
			450000	1	2.9		
Total	34	100	Total	34	100		

***significant at P≤0.05**

Table 4: Frequency of patients diagnosed with (ITP) and H. pylori +ve and the type of treatment that received by patients to treat H. pylori infection

Patients presented with ITP and H. Pylori infections	Percentage	Type of drugs that used to treating the H. pylori infections	Response to treatment (+ve or –ve)	Percentage
34	100	1- Rabiprazole 20 mg /day 2- Clarithromycin 500mg twice daily 3- amoxicillin 500 mg twice daily both for one week	34 +Ve	100

RESULTS

The study revealed a statistically significant relationship between platelet counts in patients diagnosed with Immune Thrombocytopenia (ITP) before and after treatment with anti-Helicobacter pylori medications. This association was observed specifically in patients who tested positive for H. pylori antibodies. The statistical analysis yielded a P value of 0.000, as indicated in Table 3, suggesting a strong correlation between the treatment and changes in platelet levels.

DISCUSSION

For many years, Helicobacter pylori has been recognized as a primary pathogen responsible for numerous health conditions. This bacterium was long considered the sole cause of several gastrointestinal disorders, including gastritis, peptic ulcers, stomach cancer, and mucosa-associated lymphoid tissue (MALT) lymphomas.[15] Otherwise H. pylori has been found to be associated with a number of autoimmune disorders including ITP.[16] A number of research found a high frequency of H. pylori infection in patients with ITP and in most of them an increase in platelet count was observed after H. pylori eradication.[17,18] Many interpretations have been proposed for the pathogenic mechanism, but it's still poorly understood. The research demonstrates that the results of bacterial eradication may depend on genetic factors of the host, environmental factors and on the H. pylori strain. The one of the most accepted hypotheses on the mechanism by which the bacterium could cause ITP is molecular mimicry, its suggest that, the bacterium induces the production of antibodies against antigens of its surface and thus there is a cross-reaction against multiple platelet glycoprotein antigens.[19] In the year 1998 the Italian group reported the relationship between H. pylori infection and ITP when they noticed a significant increase in the platelet numbers in eight of 11 patients in whom the bacterium was eradicated.[20] Emilia, G. et al. in recent report have suggested an association between Helicobacter pylori infection and idiopathic thrombocytopenic purpura (ITP), thy selected 30 ITP patients were investigated. H pylori infection has been documented in 13 patients (43.33%) by 13C urea breath test and confirmed by histologic examination. Bacterium eradication with antibiotics, obtained in 12 of 13 infected patients (92.3%), led to a complete response in 4 (33.33%) and to a partial response (platelets 90 3 109/L- 120 3 109/L) in 2 (16.66%). In a systematic review included determine the effect of H. pylori eradication therapy in patients with immune thrombocytopenic purpura by comparing the platelet response in patients who were, and who were not infected with H. pylori. MEDLINE, EMBASE, Cochrane central registry and abstracts from the American Society of Hematology (from 2003) were searched in duplicate and independently without language or age restrictions. Eleven studies, 8 from Japan, were included enrolling 282 patients with immune thrombocytopenic purpura who received eradication therapy; 205 were H. pylori-positive and 77 were H. pylori-negative. The odds of achieving a platelet count response following eradication therapy were 14.5 higher (95% confidence interval 4.2 to 83.0) in patients with H. pylori infection (51.2% vs. 8.8%). These findings strengthen the causal association between H. pylori infection and immune thrombocytopenia in some patients. Randomized trials are needed to determine the applicability of H. pylori eradication therapy across diverse geographical regions.[21] In case-control study in Latin America involved 32 patients diagnosed with ITP were included in the study. Controls were age and sex matched. Found that the patients with ITP should be initially tested for H. pylori status and if present, infection should be eradicated before initiating a drastic conventional ITP treatment.[17]

CONCLUSION

Our research demonstrates a link between Helicobacter pylori infection and idiopathic thrombocytopenic purpura (ITP). Based on these results, we recommend that patients diagnosed with ITP undergo initial screening for H. pylori. Our findings indicate that eliminating the bacterium can lead to improved platelet counts in affected individuals.

REFERENCES

1. Blanchette, M. and J. Freedman, The history of idiopathic thrombocytopenic purpura (ITP). Transfusion science, 1998. 19(3): p. 231-236.

2. British Committee for Standards in Haematology General Haematology Task Force. Guidelines for the investigation and management of idiopathic thrombocytopenic purpura in adults, children and in pregnancy. Br J Haematol. 2003 Feb;120(4):574-96. doi: 10.1046/j.1365-2141.2003.04131.x.

3. Piel-Julian, M., et al., Risk Factors for Bleeding, Including Platelet Count Threshold, in Newly Diagnosed ITP Patients. Blood, 2017. 130: p. 1041.

4. Adelborg, K., et al., Cardiovascular and bleeding outcomes in a population-based cohort of patients with chronic immune thrombocytopenia. Journal of Thrombosis and Haemostasis, 2019. 17(6): p. 912-24.

5. Forman, D. et al. Association between infection with Helicobacter pylori and risk of gastric cancer: evidence from a prospective investigation. British Medical Journal, 1991. 302(6788): 1302-05.

6. Marshall, B.J., et al., Attempt to fulfil Koch's postulates for pyloric Campylobacter.
Medical Journal of Australia, 1985. 142(8): p. 436-439.

7. Emilia, G., et al., Helicobacter pylori infection and chronic immune thrombocytopenic purpura: long-term results of bacterium eradication and association with bacterium virulence profiles. Blood, The Journal of the American Society of Hematology, 2007. 110(12): 3833-41.

8. Gasbarrini, A., et al. Regression of autoimmune thrombocytopenia after eradication of Helicobacter pylori. The Lancet, 1998. 352(9131): p. 878.
9. Grimaz, S., et al., Resolution of thrombocytopenia after treatment for Helicobacter pylori: a case report. Haematologica, 1999. 84(3): 283-84.
10. Franchini, M. and D. Veneri, Helicobacter pylori infection and immune thrombocytopenic purpura. Haematologica, 2003. 88(10): 1087-91.
11. Huber, M.R., S. Kumar, and A. Tefferi, Treatment advances in adult immune thrombocytopenic purpura. Annals of hematology, 2003. 82: 723-37.
12. Kurtoglu, E., E. Kayacetin, and A. Ugur, Helicobacter pylori infection in patients with autoimmune thrombocytopenic purpura. World Journal of Gastroenterology: WJG, 2004. 10(14): 2113.
13. Greenfield, B., et al., Previously suicidal adolescents: predictors of six-month outcome. Journal of the Canadian Academy of Child and Adolescent Psychiatry, 2008. 17(4): 197.
14. Szumilas, M., Explaining odds ratios. Journal of the Canadian academy of child and adolescent psychiatry, 2010. 19(3): 227.
15. Kandulski, A., M. Selgrad, and P. Malfertheiner, Helicobacter pylori infection: a clinical overview. Digestive and Liver Disease, 2008. 40(8): 619-626.
16. Prelipcean, C.C., et al., Extragastric manifestations of Helicobacter pylori infection. Revista medico-chirurgicala a Societatii de Medici si Naturalisti din Iasi, 2007. 111(3): 575-83.
17. Campuzano-Maya, G., Proof of an association between Helicobacter pylori and idiopathic thrombocytopenic purpura in Latin America. Helicobacter, 2007. 12(3): 265- 73.
18. Rostami, N., et al., Effect of eradication of Helicobacter pylori on platelet recovery in patients with chronic idiopathic thrombocytopenic purpura: a controlled trial. American journal of hematology, 2008. 83(5): 376-81.
19. Stasi, R. and D. Provan, Helicobacter pylori and Chronic ITP. ASH Education Program Book, 2008. 2008(1): 206-11.
20. Stasi, R., et al. Effects of eradication of Helicobacter pylori infection in patients with immune thrombocytopenic purpura: a systematic review. Blood, The Journal of the American Society of Hematology, 2009. 113(6): 1231-40.
21. Arnold, D.M. et al. Platelet counts response to H. pylori treatment in patients with immune thrombocytopenic purpura with and without H. pylori infection: a systematic review. haematologica, 2009. 94(6): 850.

Source of Support: Nil. **Conflict of Interest:** None Declared.

Cite this article as: Oras N. Hamad. Clinical Correlation: Helicobacter Pylori and Immune Thrombocytopenic Purpura (ITP). Int J Med Res Prof. 2024 Nov; 10(6): 7-10.
DOI:10.21276/ijmrp.2024.10.6.002