

Platelet Indices: Impact of Helicobacter Pylori Infection

Esra Polat¹ , Elif Erolu² 

¹Department of Pediatrics, Division of Pediatric Gastroenterology, University of Health Sciences, Ümraniye Training and Research Hospital, İstanbul, Turkey

²Department of Pediatrics, Division of Pediatric Cardiology, University of Health Sciences, Ümraniye Training and Research Hospital, İstanbul, Turkey

ORCID iDs of the authors: E.P. 0000-0002-0185-0344; E. E. 0000-0002-2927-5732.

Cite this article as: Polat E, Erolu E. Platelet Indices: Impact of Helicobacter Pylori Infection. *Cyprus J Med Sci* 2020; 5(2): 136-8.

BACKGROUND/AIMS

Platelets act as reactive immune cells in inflammatory conditions. Mean platelet volume (MPV) and platelet distribution width (PDW) is an indicator of platelet activity and function. We aimed to investigate the effect of helicobacter pylori eradication on platelet markers in patients who were infected by H. Pylori and eradicated.

MATERIAL and METHODS

Fifty-two patients with H. Pylori positivity confirmed by esophagogastroduodenoscopy and biopsy were included in the study. Platelet parameters (platelet count, MPV-mean platelet volume, PDW-platelet distribution range, MPV-mean platelet volume) of the patients were evaluated before and after treatment.

RESULTS

The mean age of the patients was 11.8±4.4. The mean height was 138.6±25.7 cm and the mean weight was 36.4±17.3 kg. The number of platelets was 297673±60409 before treatment and 288514±74108 after treatment (p=0.48). PDW was 15.9±0.66 before treatment and 15±0.32 after treatment (p=0.050). MPV was 9.39±1.41 before treatment and 9.67±1.21 after treatment (p=0.12). The severity of Helicobacter pylori infection was not correlated with platelet count, MPV, PDW.

CONCLUSION

The results from this study demonstrate that eradication of H. Pylori infection has an impact on the platelet indices.

Keywords: Children, Helicobacter pylori infection, platelet functions 19

INTRODUCTION

Helicobacter Pylori (H. Pylori), which is primarily colonized in the human gastric mucosa, is a gram-negative, spiral-shaped, bacterium with flagella (1, 2). H.Pylori is reported to be the most common infectious agent in the world (3). According to meta-analyses, the global prevalence of H. pylori was reported to be 33% and 44.3% (4, 5). The rate is reported to be 80% in developing countries and 10% in developed countries (4,5). As a primary colonization site, H. pylori increases toxic mucosal pH by displaying toxic effects on gastric epithelium and disrupting gastric epithelial functions and mucus secretion that generally protects gastric epithelium.

It is possible that H. pylori infection may be completely asymptomatic. Chronic gastritis, duodenal ulcers, gastric ulcers, gastric cancers, and MALT (mucosa-associated lymphoid tissue) lymphomas have been previously associated with H.Pylori infection (6). Without peptic ulcer disease, H. pylori-associated gastritis rarely presents as a symptom during childhood but may lead to more serious complications in the future. Detection of H.Pylori in biopsy specimens taken from the antrum and corpus (with Sydney score) is currently the gold standard for diagnosis in patients undergoing upper gastrointestinal endoscopic evaluation for organic causes of abdominal pain. In the case of a positive finding, eradication success should be evaluated by urea breath test (CI3) or stool test following standard eradication therapy (at least 15 days after discontinuation of proton pump inhibitor-PPI and at least 1 month after discontinuation of anti-biotic therapy) (6).

H.pylori has been shown to trigger a local and systemic immune response. It is also known to play a role in the etiopathogenesis of some diseases with extra-gastric location. These include iron deficiency anemia, coronary heart disease, immune thrombocytopenic purpura (ITP), dermatological diseases (Acne rosacea, prurigo pigmentosa, prurigo chronica multiformis, chronic idiopathic urticaria), neurodegenerative diseases and autoimmune diseases (7).

Platelets play a primary role in hemostasis and coagulation. Inflammation is known to be an important stimulus for platelets. The presence of inflammation causes the release of cytokines and chemokines from the platelet membrane. MPV and PDW are both indicators of platelet function and activation. In the presence of thrombopoietic stress, an increase in MPV occurs through stimulation of the megakaryocytic series. It has been stated before that MPV is a useful indicator in evaluation of platelet function and activation and may help in early diagnosis of some diseases through changes in platelet structure and number. (8). It is a well known fact that platelet count increases with H. pylori treatment in chronic ITP patients (9). Based on this information, we planned to investigate the effect of treatment on platelet markers in patients with H.Pylori detected in esophagogastroduodenoscopy due to treatment-resistant dyspeptic complaints.

MATERIAL and METHODS

Ethics committee approval was received from Umraniye Research and Training Hospital. Fifty-two patients who tested positive for H.Pylori and had an esophagogastroduodenoscopy performed for treatment-resistant dyspepsia in a pediatric gastroenterology clinic in two different centers were included in the study. Platelet parameters included platelet count, MPV-mean platelet volume, PDW-platelet distribution range, and MPV-mean platelet volume of the patients and were evaluated before treatment. First-line eradication therapy (Amoxicillin + Clarithromycin + Lansoprazole) was given to patients whose biopsies were positive for H. pylori. H. pylori eradication status was evaluated by stool H. pylori antigen test one month after the end of the treatment. The blood counts of the patients who were successful in eradication treatment were repeated three months after the end of the treatment and platelet parameters were re-evaluated subsequently. Patients who were not successful during the first line eradication treatment and who subsequently needed a secondary eradication treatment were excluded from the study.

RESULTS

The mean age of the patients was 11.8 ± 4.4 (30 girls, 22 boys). The mean height was 138.6 ± 25.7 cm and the mean weight was

36.4 ± 17.3 kg. The number of platelets was 297673 ± 60409 before treatment and 288514 ± 74108 after treatment ($p=0.48$). PDW was 15.9 ± 0.66 before treatment and 15 ± 0.32 after treatment ($p=0.050$). MPV was 9.39 ± 1.41 before treatment and 9.67 ± 1.21 after treatment ($p=0.12$). H. pylori infection severity was not correlated with platelet count, MPV, PDW.

DISCUSSION

H. Pylori infection has an effect on platelet count and bone marrow. Studies have shown a decrease in platelet count after H. Pylori eradication in non-ITP patients (10). Peripheral platelet counts have been shown to increase rapidly in H. Pylori positive patients with bone marrow transplantation. It has also been shown that H. Pylori infection increases IL-6 levels and this then has a positive effect on bone marrow platelet production (11). In the last decade, platelets have been reported to act as immune cells. Platelets increase cytokines IL-6 and TNFalpha release against H. Pylori IL-6 causes an increase in the number of platelets, while TNFalpha has an adverse effect on the number of platelets. This dual effect prevents an excessive decrease in platelet number after H. Pylori eradication (12). In this study, a decrease in the number of platelets was observed after eradication of H. Pylori but it was not statistically significant.

Mean platelet volume (MPV), a widely used platelet function marker, is influenced by production, activation, and finally sequestration. which is a marker of platelet function is influenced by platelet activation, production and sequestration. MPV is reported to have been involved in many diseases. As an important component of the immune system, MPV is known to associate with inflammatory conditions. Platelets with high MVP have been proven to be more active and more susceptible to aggregation. Additionally, MPV is considered to be higher in H.pylori positive patients than compared to H.pylori negative patients (2, 13, 14). There was no significant difference found in the study. PDW is a measure of platelet size variability. It shows heterogeneity in platelet morphology and increases with platelet activation. PDW is also considered to be a more specific indicator of platelet reactivity than MPV (15). In this study, a significant decrease in PDW was observed with eradication of H.pylori, indicating that platelet activation decreased.

The results from this study indicate that there is a relationship between platelet indices and H. Pylori infection. The relationship between H. Pylori infection and MPV platelet count was demonstrated in previous studies. However, this study provided new information about the impact of H. Pylori infection on PDW, which is another platelet function parameter. In the future, it is possible that platelet parameters may be used to determine eradication success of H.pylori infection.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Umraniye Research and Training Hospital (18.12.2019/B.10.I.TKH.4.34.H.GP.01/237).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – E.P, E.E.; Design – E.P, E.E.; Supervision – E.P, E.E.; Resources – E.E, E.P; Materials – E.E, E.P; Data Collection and/

Main Points:

- Bone marrow can be affected by inflammatory conditions.
- Changes in platelet parameters (platelet count, mean platelet volume and platelet distribution width) in H.pylori infection is to be expected.
- Platelet parameters can be used for the evaluation of H. pylori infection eradication.

or Processing – E.E, E.P.; Analysis and/or Interpretation – E.E.; Literature Search – E.P, E.E.; Writing Manuscript – E.P, E.E.; Critical Review – E.P, E.E.

Conflict of Interest: Authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Talebi Bezmin Abadi A. Diagnosis of Helicobacter pylori Using Invasive and Noninvasive Approaches. *J Pathog* 2018; 2018: 9064952. [\[Crossref\]](#)
2. Umit H, Umit EG. Helicobacter pylori and mean platelet volume: a relation waybefore immune thrombocytopenia? *Eur Rev Med Pharmacol Sci* 2015; 19(15): 2818-23.
3. Kamboj AK, Cotter TG, Oxentenko AS. Helicobacter pylori: The Past, Present, and Future in Management. *Mayo Clin Proc* 2017; 92(4): 599-604. [\[Crossref\]](#)
4. Hooi JKY, Lai WY, Ng WK, Suen MMY, Underwood FE, Tanyingoh D, et al. Global Prevalence of Helicobacter pylori Infection: Systematic Review and Meta-Analysis. *Gastroenterology* 2017; 153(2): 420-9. [\[Crossref\]](#)
5. Zamani M, Ebrahimitabar F, Zamani V, Miller WH, Alizadeh-Navaei R, Shokri-Shirvani J, et al. Systematic review with meta-analysis: the worldwide prevalence of Helicobacter pylori infection. *Aliment Pharmacol Ther* 2018; 47(7): 868-76. [\[Crossref\]](#)
6. Jones NL, Koletzko S, Goodman K, Bontems P, Cadranel S, Casswall T, et al. Joint ESPGHAN/NASPGHAN Guidelines for the Management of Helicobacter pylori in Children and Adolescents (Update 2016). *J Pediatr Gastroenterol Nutr* 2017; 64(6): 991-1003. [\[Crossref\]](#)
7. Gravina AG, Zagari RM, De Musis C, Romano L, Loguercio C, Romano M. Helicobacter pylori and extragastric diseases: A review. *World J Gastroenterol* 2018; 24(29): 3204-21. [\[Crossref\]](#)
8. Khandekar MM, Khurana AS, Deshmukh SD, Kakrani AL, Katdare AD, Inamdar AK. Platelet volume indices in patients with coronary artery disease and acute myocardial infarction: an Indian scenario. *J Clin Pathol* 2006; 59: 146-9. [\[Crossref\]](#)
9. Aljarad S, Alhamid A, Tarabishi S, Tarabishi AS, Suliman A, Aljarad Z. The impact of Helicobacter pylori eradication on platelet counts of adult patients with idiopathic thrombocytopenic purpura. *BMC Hematol* 2018; 18: 28. [\[Crossref\]](#)
10. Matsukawa Y, Iwamoto M, Kato K, Mizuno S, Gon Y, Hemmi A, et al. Long term changes in platelet counts after H. pylori eradication in non-ITP patients. *Platelets* 2010; 21(8): 628-31. [\[Crossref\]](#)
11. Wakae T, Takatsuka H, Mori A, Okada M, Fujimori Y, Okamoto T, et al. Influence of Helicobacter pylori on platelets after bone marrow transplantation from unrelated donors. *Bone Marrow Transplant* 2003; 31(6): 493-6. [\[Crossref\]](#)
12. Scott T, Owens MD. Thrombocytes respond to lipopolysaccharide through Toll-like receptor-4, and MAP kinase and NF-kappaB pathways leading to expression of interleukin-6 and cyclooxygenase-2 with production of prostaglandin E2. *Mol Immunol* 2008; 45(4): 1001-8. [\[Crossref\]](#)
13. Baxendell K, Waleign S, Tesfaye M, Wordofa M, Abera D, Mesfin A, et al. Association between infection with Helicobacter pylori and platelet indices among school-aged children in central Ethiopia: a cross-sectional study. *BMJ Open* 2019; 9(4): e027748. [\[Crossref\]](#)
14. Topal F, Karaman K, Akbulut S, Dincer N, Dölek Y, Cosgun Y, et al. The relationship between mean platelet volume levels and the inflammation in Helicobacter pylori gastritis. *J Natl Med Assoc* 2010; 102(8): 726-30. [\[Crossref\]](#)
15. Vagdatli E, Gounari E, Lazaridou E, Katsibourlia E, Tsikopoulou F, Labrianou I. Platelet distribution width: A simple, practical and specific marker of activation of coagulation. *Hippokratia* 2010; 14(1): 28-32.