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Research Article

**A COMPARATIVE STUDY OF PLATELET COUNTS  
BETWEEN H. PYLORI INFECTED AND UNINFECTED  
INDIVIDUALS****Aniqa Liaquat, Zulquernain Zahid, Faizan Maqsood**  
Bahawal Victoria Hospital, Bahawalpur**Article Received:** April 2020**Accepted:** May 2020**Published:** June 2020**Abstract:**

**Introduction:** Immune thrombocytopenic purpura (ITP) can be associated with *Helicobacter Pylori* (*H. Pylori*) stomach infection, which in this case often leads to an improvement in platelet count, if not always. There are relatively few studies comparing platelet counts in infected *H. pylori* patients versus healthy individuals.

**Place and Duration:** In the Medicine department of Bahawal Victoria Hospital, Bahawalpur for one-year duration from March 2019 to February 2020.

**Methods:** We compared the platelet counts in 200 *H. Pylori* infected and 200 uninfected individuals during the upper part of the esophagus-gastroduodenoscopy in patients reporting to the gastrointestinal tract clinic with dyspepsia. *H. Pylori* positive and negative patients were comparable by age ( $35.89 \pm 10.73$  and  $34.93 \pm 11.31$ , respectively,  $P$  value: 0.3845). Twenty (10%) infected patients had platelets below Counts  $1.5 \times 10^6 / \text{mm}^3$  and 35 (17.5%) ranged between  $1.5$  and  $2 \times 10^6 / \text{mm}^3$  (1% and 5% respectively uninfected group,  $P$  value: 0.0001). In addition, all 20 patients with thrombocytopenia were over 45 years old age and persons with the lowest number were the oldest (in the sixth or seventh decade).

**Conclusion:** Patients with *H. pylori* gastric infection have lower platelet counts when compared with uninfected individuals after the exclusion of secondary causes of thrombocytopenia. Most patients with fewer platelets are in their five to seven decades.

**Key words:** *Helicobacter Pylori*, platelets, thrombocytopenia, ITP, advanced age.

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## INTRODUCTION:

The chronicity of many bacterial or viral infections is associated with the development of many erroneous immune phenomena (e.g., HIV, tuberculosis, subacute bacterial endocarditis)<sup>1-2</sup>. It is not surprising that the high incidence and chronicity of *H. pylori* infection occurs in most immunological diseases, especially in developing countries. Gasbarrini A et al. It was reported that in 1988 patients with immune thrombocytopenic purpura (ITP) were more likely to have *Helicobacter Pylori* (*H. Pylori*) infection in the gastric mucosa. After ITP treated with the regimen to eliminate *H. Pylori*, platelet counts did not change after 3H. *Pylori* infected patients who did not follow the treatment regimen. Since then, many studies around the world have reported an improvement in platelet counts in ITP patients treated with *H. pylori*, including refractory patients, while some studies have not shown a positive effect. Failure can be caused by different strains of *H. Pylori* in different geographical regions<sup>3-4</sup>. It is suggested that many factors associated with infectious organisms (e.g., Cag A Protein), variable distribution of these factors in different geographical locations, chronicity of infection, and variability of both host factors. Chronic occurrence of ITP in an infected host in response to elimination of infection. The question of whether any adequate platelet response in chronic ITP is due to eradication of *H. pylori* or the effects of drugs administered for eradication was raised in a meta-analysis of studies from different geographical regions<sup>5-6</sup>. It has been observed that patients with ITP and concomitant *H. pylori* infection are older than patients with uninfected ITP. An incorrect immune response may take many years. The answer to this question will be a long check-up of patients infected with ITP development. Such long-term follow-up in infected patients may raise ethical questions, although *H. pylori* eradication therapy is not indicated in asymptomatic infected patients<sup>7-8</sup>. We examined the comparison of platelet counts in infected and uninfected *H. pylori* patients arriving at the gastrointestinal endoscopy room (upper gastrointestinal endoscopy). We examined the incidence of asymptomatic thrombocytopenia in infected patients.

## METHODS:

This study was held in the Medicine department of Bahawal Victoria Hospital, Bahawalpur for one-year duration from March 2019 to February 2020. Study population: Two hundred more patients (infected and uninfected with *H. pylori*) were taken from each group who reported to the endoscopic room of our gastroenterological department for endoscopy of the upper gastrointestinal tract. research. Dyspepsia was the main complaint of all patients. The patient can be of any age and any sex over the age of 12 years. Exclusion criteria; 1)

gastrointestinal bleeding, 2) stomach or duodenal ulcers, 3) stomach and intestinal cancers, 4) portal hypertension, 5) chronic liver disease, 6) chronic kidney disease, 7) nonsteroidal anti-inflammatory drugs, patients with proton pump inhibitors or any cytotoxic treatment and 8) patient previously treated for *H. pylori* infection. The study was conducted in accordance with the guidelines of good clinical practice and was approved by the local Ethics Assessment Committee. All patients signed informed consent prior to participating in the study. Diagnosis of secondary thrombocytopenia: If a low platelet count is found, many etiologies should be considered and there is no confirmatory test for *H. pylori*, if any. Therefore, low platelet counts remain largely a diagnosis of exclusion in a patient infected with *H. pylori*. To investigate potential factors affecting platelet counts, all patients in both groups underwent detailed history, physical examination, and research for this purpose. Liver disease, abnormal liver function tests and portal hypertension (presence of acids and clinical presence of acids, presence of portal vein and esophageal varices), renal failure, sepsis and fever, fibrinogen degradation products (if indicated), prothrombin time, unexplained proteinuria, antinuclear and double chain antibodies (if indicated), HIV status (if indicated) are some of the important pre-induction assessments. Potentially any drug may be involved in thrombocytopenia. We do not exclude the patient from frequently prescribed drugs, i.e. antihypertensive and antidiabetic drugs. All medications currently or in the last month have been evaluated for potential thrombocytopenia induction based on literature review. Most drug-induced thrombocytopenia is acute and particular attention has been paid to drugs that started or were taken last month.

Diagnosis of *H. Pylori*: Two antral biopsy samples were taken and formalin was collected for histological examination, stained with Giemsa and evaluated by two pathologists who examined gastric biopsies daily and cut out surgical stomach samples. The sensitivity and specificity of antral specimens stained with Giemsa is 91% and 98% respectively, although admittedly, results are operator dependent. Complete blood counts were determined using automated Sysmex and Beckman Coulter LH 780 analyzers. Blood samples are taken into tubes containing EDTA for complete blood counts and are guaranteed to reach the laboratory within 150 minutes. All blood samples with platelets less than  $150 \times 10^9 / L$  were confirmed in citrate blood samples.

## RESULTS:

Table 1 shows comparisons of ages between H. Pylori positive and negative patients and between male patients (H. Pylori positive and negative) and female patients (H. Pylori positive and negative). None of these comparisons show a significant age difference. None of these comparison shows a significant age difference. Table 2 shows number and percentages of H. Pylori positive and negative patients in various ranges of platelet counts. As can

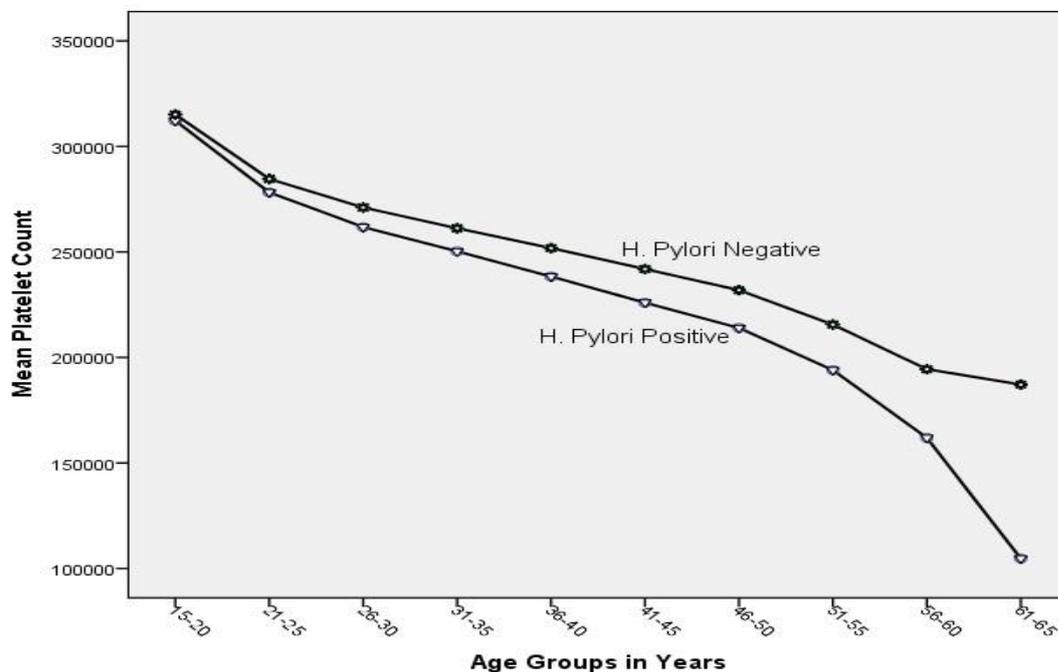
be seen, number of infected patients in lower ranges of platelet counts is more in comparison to non-infected patients and the difference is statistically significant. Graph 1 depicts differences in mean platelet counts between H. Pylori positive and negative patients divided into age groups of 5 years. The differences in platelet counts become wider with every 5 years of increasing age.

**Table 1: Age comparison two group of patients; H. Pylori positive and H. Pylori negative**

Characteristic	H. Pylori Positive	H. Pylori Negative	P Value
Age in years	35.89±10.73	34.93±11.31	0.3845
Age (males)	35.74±10.63	34.78±11.38	0.54
Age (females)	36.05±10.89	35.07±11.30	0.535

**Table 2: Number of H. Pylori Positive and Negative patients in various ranges of platelets counts**

Range of Platelet Count	H. Pylori Positive (n=200)	H. Pylori Negative (n=200)	p Value
Below $150 \times 10^9/L$	20(10%)	2(1%)	0.0001
150 - $200 \times 10^9/L$	35 (17.5%)	10 (5%)	
200 - $300 \times 10^9/L$	131 (65.5%)	171 (85.5%)	
Above $300 \times 10^9/L$	14(7%)	17 (8.5%)	



**Graph 1 Comparison of mean platelet counts between H. Pylori positive and negative patients divided in age groups of 5 years**

**DISCUSSION:**

The study shows that in patients with H. pylori stomach infections, the platelet count is generally lower than in the control group, which corresponds to the large number of infected patients, and the platelet count is below  $1.5 \times$  the normal lower limit. Studies combining  $109 / L$ . H. pylori infection with thrombocytopenia were mainly performed in patients diagnosed with ITP; The incidence of H. Pylori in ITP compared it with the general population in the same region, PTI, after H. pylori eradication therapy, a possible host and bacterial agents in thrombocytopenia. There are few studies on the incidence of thrombocytopenia in patients infected with H. pylori. Literature review investigating anti-H activity between 1950 and 2008. Studies of Pylori therapy in ITP patients concerned both H. Patients with Pylori positive and negative results performed 7 relevant tests (n = 222). Platelet counts were obtained in 65 (49.6%) of 131 infected patients and none of the 44 uninfected patients after H. pylori treatment. However, different definitions of 'success' limit the strength of this result, and most studies come from Japan, where the incidence of H. pylori infection is high. After excluding secondary causes of thrombocytopenia, we have found that age is an important determinant of platelet count. Patients with ITP and concomitant H. pylori infections were found to be older than uninfected patients. The authors claimed that the frequency of H. pylori infections increases with age in typical H. pylori regions. We also found that the lowest platelet count range was in the older population, mainly infected with H. pylori. Another possible factor may be a lower platelet count in healthy older people. We found an average difference of about  $100 \times 109 / L$  between individuals in the second and sixth years in platelet counts. An analysis of 12,517 residents of geographical isolates from geranium showed that a 10-year increase corresponds to a  $9 \times 109 / L$  decrease in platelet count. Very similar results were obtained in 7,266 inhabitants of the Moli-Sani Project cohort, including 24,318 people from five additional geographical isolates in different regions of Italy and 30 cities in Molise. Finally, the latest study collected all data from the participants in the three above-mentioned population studies and found that the age-related changes are actually quite large: the platelet count in old age has decreased. According to early childhood, 35% of men and 25% of women therefore there is no doubt that age is an important determinant of platelet count in healthy people.

The most suitable study for our study was conducted by H Ümit and EG Ümit. They compared the mean platelet count and mean platelet volume in patients with H. pylori positive and negative results. Data were collected retrospectively in 4,823 patients (H. Pylori positive = 1701, H. Pylori negative = 3122)

with dyspeptic complaints undergoing upper gastrointestinal tract endoscopy. The mean platelet count in positive and negative H. pylori patients was  $246381 \pm 92225 / mm^3$  and  $258135 \pm 89912 / mm^3$ , respectively (p <0.001). The mean platelet volume (MPV) was higher in the H. Pylori positive group ( $8.9 \pm 1.3$  vs.  $8.23 \pm 0.94$ , p <0.001). This difference was observed in both sexes. They concluded that a continuous and compensated platelet production process may be responsible for an increase in MPV in patients with H. pylori infection and normal platelet counts. There are many reasons for the increase in MPV, e.g. ischemic, renal artery stenosis, recognized by the authors as the role of the immune cells of younger and larger platelets (e.g. In ITP), possible innate condition, polymorphic nuclear leukocytes, impaired oxygenation and nutrition of tissues, diabetes, myocardial infarction cardiac shock and dilatation, hypertrophic cardiomyopathy etc. However, ignoring the existence of the above reasons, the authors may be the predecessors of open thrombocytopenia due to the increase in MPV in patients infected with H. pylori due to destruction of plaque-compensated gonging and increase in MPV. Next. However, the authors did not compare platelet counts in young and elderly patients, nor did they provide further information on platelet counts in the two groups as in our study.

**CONCLUSION:**

We conclude that patients infected with H. Pylori have lower platelets counts when compared to noninfected age matched controls, with significantly higher number of infected patients having platelet count below the normal range. The difference in platelet counts between the two groups is more prominent with increasing age.

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