

# Complete blood cell parameters of Anti-HP IgG antibody positive and negative subjects

[Anti-HP IgG antikoru pozitif ve negatif bireylerde tam kan sayım parametreleri]

Tevfik Noyan

Ordu Üniversitesi, Tıp Fakültesi, Tıbbi Biyokimya  
Anabilim Dalı, Ordu

Yazışma Adresi  
[Correspondence Address]

Doç. Dr. Tevfik Noyan

Ordu Üniversitesi, Tıp Fakültesi, Tıbbi Biyokimya  
Anabilim Dalı, Ordu  
Tel. 04322150470/1242  
Fax.  
E-mail. tevfiknoyan@hotmail.com

## ABSTRACT

**Objective:** The aim of this study was to determine whether there is any difference in complete blood cell count parameters between anti-*Helicobacter pylori* (HP) IgG antibody (anti-IgG) positive and negative subjects.

**Methods:** In a hospital based survey, total of 125 people were included to the study which measured anti-HP IgG antibody. The total 85 subjects, titers of anti-HP IgG antibody > 1 was considered as IgG positive group and <1, total 40 subjects, as IgG negative group.

**Results:** There was no significant relationship between anti-HP IgG antibody positivity and gender. However, significantly decreased mean corpuscular volume and increased percent eosinophil values were found in IgG positive group (p=0.04).

**Conclusion:** In conclusion, IgG positive group characterized lower mean corpuscular volume and higher eosinophil values. These results might be sign of inflammatory changes associated to chronic gastritis.

**Key Words:** complete blood cell, anti-HP antibody, mean corpuscular volume

**Conflict of Interest:** No conflict of interest was declared by the authors.

## ÖZET

**Amaç:** Bu çalışmanın amacı, *Helicobacter pylori*'ye (HP) karşı IgG antikoru (anti-IgG) pozitif ve negatif olan bireylerde tam kan hücre parametreleri arasında farklılık olup olmadığının belirlenmesidir.

**Yöntemler:** Hasta bilgileri taranarak elde edilen bilgiler ışığında, anti-HP IgG antikoru ölçümü gerçekleştirilen toplam 125 birey çalışmaya alındı. Anti-HP IgG antikoru pozitif olan 85 birey IgG pozitif grup, anti-HP IgG antikoru negatif olan 40 birey ise IgG negatif grup olarak çalışmaya katıldı.

**Bulgular:** Anti-HP IgG antikoru pozitifliği ile cinsiyet arasında anlamlı bir ilişki bulunmadı. Bununla birlikte, IgG pozitif grupta, ortalama korpusküler hacim anlamlı derecede azalmış ve yüzde eozinofil değeri artmış olarak bulundu (p=0.04).

**Sonuç:** Çalışmamızın sonuçları, Anti-HP IgG antikoru pozitif grubun azalmış ortalama korpusküler hacim ve artmış eozinofil seviyeleriyle karakterize olduğunu göstermektedir. Bu sonuçlar kronik gastritteki inflamatuvar değişikliklerin belirtisi olabilir.

**Anahtar Kelimeler:** tam kan sayımı, anti-HP antikoru, ortalama korpusküler hacim

**Çıkar Çatışması:** Yazarların çıkar çatışması yoktur.

## Introduction

*Helicobacter pylori* (HP) is a gram-negative spiral-shaped microaerophilic bacterium, which colonized in the gastric mucosa of humans. HP infection is a worldwide pathogenic condition in the development of infection, causing different problems in the gastric mucosa [1]. Parent et al. [2] have been reported prevalence of HP is 56% in healthy individuals and there is not a significant relationship between HP infection and, lipid, haemostatic and inflammatory factors. Publications that support this work as well as [3], there are studies that report an association between HP infection, and ischemic heart disease, plasma fibrinogen, and some lipid parameters [4]. The changes in hemoglobin, white blood cell and platelet values may occur in acute and chronic infectious diseases. The development of anemia that complication of gastritis and peptic ulcers associated with HP infection is an expected complication. Indeed, Annibale et al. [5] have reported iron deficiency anemia improved after eradication of HP infection. However, studies are also available that reporting increase in platelet count after eradication of HP in patients with immune thrombocytopenic purpura [6, 7].

There was a little study that investigating the relationship between whole blood parameters and HP infection. Therefore, we aimed to investigate whether there is any difference in complete blood cell count parameters in anti HP Ig-G antibody positive and negative subjects in the present study.

## Material and Methods

Approval for the study was obtained from the Ethics Committee of Ordu University School of Medicine (2012-01). The ones who admitted to the various clinic of hospital because of dyspeptic complaints were enrolled into the study. Examination of the patients as a result of files, without any known systemic disease, glucose, urea and creatinine values within the normal range, and the people without a history of drug use were included in the study. Exclusion criteria were prior eradication therapy for HP, anti-ulcer drug use within past 1 month, gastrointestinal system and other organ malignancies, inflammatory and infectious diseases, and prior gastric surgery. The total of 125 people was included to the study which measured titers of anti-HP IgG antibody as serologically in the Ministry of Health Training and Research Hospital, Biochemistry Laboratory of the University. As a result of titers of anti-HP IgG antibody <1 those negative (-), and > 1 positive (+) was considered. As a result of the examination, total 85 subjects, testing for HP (+) was considered as IgG (+) group and test results for HP (-), total 40 subjects, as IgG (-) group.

The complete blood count, fasting blood glucose, urea, and creatinine results were evaluated in all study participants. The measurement of anti-HP IgG antibody was performed original Vidas kits that based

on measurement of HP infection was exclusively serum IgG response on Vidas analyzer. The biochemical measurement was performed with original Abbot reagents in the Abbot C-8000 autoanalyser. The complete blood count was performed in the Cell-Dyne 3700 analyzer.

## Statistical Analysis

Descriptive statistics were expressed as mean and standard deviation for the continuous variables. Student t test was used to compare means of patient and control groups for these variables. In addition, Pearson correlation analysis was carried out for determination of linear relationships among the variables. Statistical significance levels were considered as 5% for all statistical computations.

## Results

The anti-HP IgG antibody was positive in 68% of all subjects who including the study. As a result of the evaluation according to gender, there was no significant relationship between gender and anti-HP IgG antibody positivity ( $p > 0.05$ ).

The comparison of data that obtained from the groups is presented in Table 1. Significantly decreased mean corpuscular volume (MCV) and increased percent eosinophil (EOS %) values were obtained in IgG (+) group than in IgG (-) group (respectively,  $p=0.04$  and  $p=0.04$ ). There were no significant differences among the other parameters between the groups ( $p > 0.05$ ).

The correlation between HP, MCV and eosinophils (EOS) are presented in Table 2. There was no found significant correlation among HP, MCV and EOS in the each group ( $p > 0.05$ ).

## The Conclusion

There are invasive and non-invasive methods for determining presence of HP. Biopsy by endoscopy have traditionally been used to obtain gastric or duodenal tissue specimens for subsequent stain, culture, and/or direct urease detection. Invasive methods such as endoscopy involve patient discomfort, risk, and are costly to perform. Non-invasive methods include urea breath tests and serological methods. The VIDAS HP IgG qualitative test was used for determination HP serology in all subjects. In the present study, anti-HP IgG antibody was positive in 68% of all subjects who including the study. This study included the subjects that living in Black Sea region of Turkey. According to international epidemiological studies, industrializing countries have demonstrated a higher prevalence of HP infection than industrialized countries and a tendency was noted whereby the prevalence became higher concomitantly with ageing of the population, even in the same countries [8]. It is clear also that the environmental hygiene and socioeconomic status made differences in the prevalence [9, 10].

**Table 1.** Statistical analyses of groups. (WBC: white blood cell, RBC: red blood cell, Hb: hemoglobin, HCT; hematocrit, MCV; mean cell volume, MCH; mean cell hemoglobin, MCHC; mean cell hemoglobin concentration, PLT; platelet, MPV; mean platelet volume, RDW; red cell distribution width, LYM; lymphocyte, MONO; monocyte, NEU; neutrophil, EOS; eosinophil, BASO; basophil, BUN; blood urea nitrogen, Cr; creatinine)

	IgG (-) group n=40				IgG (+) group n=85				p
	Mean	SD	Minimum	Maximum	Mean	SD	Minimum	Maximum	
WBC, x10 <sup>3</sup> /μL	6.59	1.33	3.92	9.85	6.51	1.38	3.71	10.60	0.76
RBC, x10 <sup>6</sup> /μL	4.67	.43	3.77	5.58	4.78	.44	4.00	5.71	0.20
Hb, g/dL	13.55	1.46	10.80	17.20	13.62	1.58	9.87	16.90	0.81
HCT, %	41.04	4.23	33.60	53.00	40.92	4.15	31.30	49.0	0.88
MCV, fL	87.85	5.39	77.50	97.70	85.69	5.74	67.90	96.90	0.04
MCH, pg	29.02	2.04	24.40	32.90	28.52	2.20	21.60	32.50	0.22
MCHC, g/dL	33.02	.83	31.20	34.70	33.27	1.04	31.10	35.60	0.15
PLT, x10 <sup>3</sup> /μL	257.42	59.15	148.0	411.0	268.15	62.14	158.0	546.00	0.36
MPV, fL	8.45	1.15	6.80	11.80	8.41	1.04	6.37	10.90	0.85
RDW, %	13.6	1.67	11.20	18.60	14.12	1.52	11.70	17.70	0.08
LYM, x10 <sup>3</sup> /μL	2.13	.58	.84	3.64	2.02	.58	.63	3.57	0.32
MONO, x10 <sup>3</sup> /μL	.37	.13	.15	.77	.39	.14	.15	.74	0.56
NEU, x10 <sup>3</sup> /μL	3.83	1.21	1.92	6.77	3.79	1.14	1.54	7.54	0.87
EOS, x10 <sup>3</sup> /μL	.13	.10	.01	0.53	.17	.12	.00	.53	0.13
BASO, x10 <sup>3</sup> /μL	.05	.03	.01	.13	.07	.08	.00	.74	0.18
LYM, %	33.06	8.86	11.40	59.10	31.59	7.88	8.62	49.30	0.35
MONO, %	5.73	1.75	2.50	9.75	6.05	1.96	3.10	10.20	0.37
NEU, %	57.33	9.87	31.10	81.20	57.65	8.91	31.90	81.6	0.85
EOS, %	2.02	1.61	.11	9.17	2.81	2.17	.04	10.20	0.04
BASO, %	.78	.43	.20	1.66	0.90	0.43	.28	2.31	0.14
H.Pylori	.34	.29	.00	.90	3.48	1.76	1.03	8.09	0.00
Glucose, mg/dL	94.34	9.94	76.00	107.00	97.35	10.71	75.00	109.00	0.14
BUN, mg/dL	11.13	2.54	6.00	18.00	12.24	3.88	5.00	28.00	0.10
Cr, mg/dL	.69	.09	.53	.87	.72	.12	.40	1.22	0.21
Age (Year)	36.02	15.04	15.00	64.00	39.47	14.89	16.00	85.00	0.23

**Table 2.** The correlation among anti-HP IgG titers, MCV and EOS in HP(-) and HP(+) groups. (MCV; mean cell volume, EOS; eosinophil, HP; H.pylori)

Parameters	IgG (-) group		IgG (+) group	
	MCV	EOS	MCV	EOS
EOS	-.013	1	.128	1
Anti-HP IgG titers	.250	.203	-.018	.090

We did not find any association between gender and anti-HP IgG antibody response in this study. There were controversy reports in this issue. Some reports suggesting that the prevalence in males was higher than in females [10, 11]. And yet another reports stating that gender related differences did not exist [8, 12]. Recently Matsukawa et al [13] reported that females appeared more susceptible to HP infection than males and females with HP infection characterized manifested elevated platelet counts, and eradication of HP infection caused reduced peripheral platelets in both sexes. In contrast to these findings, we did not find any significant difference between groups regarding to platelet count. The different effects of HP infection on platelet count in two studies can be explained due to the fact that number of cases in the present study less than others. Therefore, we think that this aspect requires further investigation.

The measurement of the MCV, volume of the “average” red blood cell, is important in the differential diagnosis of anemia. The IgG positive group had characterized significant low MCV levels in the present study. However, the correlation between titers of anti-HP IgG antibody and MCV did not significant. Microcytosis, reflected in a low MCV, is due to defective hemoglobin production, either from ineffective heme or globin synthesis. A low MCV, suggesting small RBCs (microcytosis), occurs in several childhood disorders including iron deficiency anemia, beta-thalassemia trait, lead poisoning, anemia from chronic illness, and rarely, in sideroblastic anemia [14]. Recent epidemiologic studies have suggested an association between HP infection and iron deficiency [15, 16]. The current study has some limitations. The indicator of iron deficiency anemia that levels of serum iron, iron binding capacity, ferritin and transferrin were unknown of subjects who including the study. And also, there was no evaluation for upper gastrointestinal endoscopy of subjects. Infection with HP is recognized as a major risk factor in peptic ulcer disease and gastric cancer, in which lesions are likely to bleed either overtly or in an occult manner, eventually leading to iron deficiency anemia [5]. It has been suggested that infection with *H. pylori* may lead to iron deficiency or iron deficiency anemia by impairing iron uptake or increasing iron demand [16]. It has been shown that in patients with iron deficiency anemia and chronic HP related gastritis, cure of the infection leads to the reversal of the need of iron treatment, to normalization of hemoglobin levels after 6 months, and to long-lasting recovery from iron deficiency anemia [5]. We think that, although it is not observed a significant correlation between anti-HP IgG antibody and MCV, because of MCV values lower in the IgG positive group, therefore anti-HP IgG positive patients seem to be a need to investigate for iron deficiency anemia.

Eosinophils are involved in a broad range of diseases such as allergic, inflammatory, and malignant disorders [17, 18]. Several studies focused on the function of

eosinophils in gastrointestinal disease [19, 20]. Although the percent of blood EOS count is higher in IgG positive group than IgG negative group, the correlation between anti-HP IgG antibody titers and EOS count is not significant in the present study. There are several studies that support our results in HP infection [21, 22]. Activation of eosinophils seems to contribute to the pathophysiology of several inflammatory conditions [23]. The role of eosinophils in the pathogenesis of HP-associated gastritis and ulcer is not explained. One of the major constituents of the granule matrix is the eosinophil cationic protein [22]. Aydemir et al [22] reported that gastric mucosal eosinophil infiltration and the gastric juice ECP level were apparently greater in *H. pylori* infected subjects, however, serum ECP level no significant between *H. pylori* infected and non-infected groups. The presence of eosinophils is assumed as a protective mechanism of unspecific mucosal immunity response against bacteria and parasites [20, 24].

In accordance with these findings, the results of this study suggest a significant decreased mean corpuscular volume and increased values of percent eosinophil in *H. pylori* infection. The sample size was small; therefore further studies with larger groups of patients are needed to prove the efficacy of our results.

## Acknowledgements

The author would like to thank Associate Professor Siddık Keskin, Yuzuncu Yil University, Van-Turkey, for helping statistical analysis, and the clinics that used the clinical definition and data.

## Conflict of Interest

No conflict of interest was declared by the authors.

## References

- [1] Noyan T, Guducuoglu H, Ilhan M. A Study of Oxidative Stress Parameters in Anti Helicobacter Pylorus IgG Positive and Negative Gastric Cancer Patients. *Yonsei Med J* 2009; 50: 677–82.
- [2] Parente F, Imbesi V, Cucino C, MacOni G, Russo U, et al. Helicobacter pylori CagA seropositivity does not influence inflammatory parameters, lipid concentrations and haemostatic factors in healthy individuals. *J Intern Med* 2000; 247: 213-7.
- [3] Brenner H, Berg G, Froelich M, Boeing H, Koenig W. Chronic infection with Helicobacter pylori does not provoke major systemic inflammation in healthy adults: results from a large population-based study. *Atherosclerosis*. 1999; 147: 399–403.
- [4] Murray LJ, Bamford KB, O'Reilly DPJ, McCrum EE, Evans AE. Helicobacter pylori infection: relation with cardiovascular risk factors, ischaemic heart disease and social class. *Br Heart J* 1995; 74: 497-501.
- [5] Annibale B, Marignani M, Monarca B, Antonelli G, Marchegiano A, et al. Reversal of iron deficiency anemia after Helicobacter pylori eradication in patients with asymptomatic gastritis. *Ann Intern Med* 1999; 131: 668-72.
- [6] Franchini M, Veneri D. Helicobacter pylori infection and immune thrombocytopenic purpura: an update. *Helicobacter* 2004; 9: 342-6.

- [7] Fujimura K. Helicobacter pylori infection and immune thrombocytopenic purpura *Int J Hematol* 2005; 81: 113-8.
- [8] Matsuzaka M, Fukuda S, Yamai K, Tsuya R, Fukuoka Y, et al. Are individuals with lower neutrophil oxidative burst activity more prone to Helicobacter pylori infection? *Luminescence* 2008; 23:132-8.
- [9] Woodward M, Morrison C, McColl K. An investigation into factors associated with Helicobacter pylori infection. *J Clin Epidemiol* 2000; 53: 175-81.
- [10] Moayyedi P, Axon AT, Feltbower R, Duffett S, Crocombe W, et al. Relation of adult lifestyle and socioeconomic factors to the prevalence of Helicobacter pylori infection. *Int J Epidemiol* 2002; 31:624-31.
- [11] Murray LJ, McCrum EE, Evans AE, Bamford KB. Epidemiology of Helicobacter pylori infection among 4742 randomly selected subjects from Northern Ireland. *Int J Epidemiol* 1997; 26: 880-7.
- [12] The EUROGAST Study Group. Epidemiology of and risk factors for Helicobacter pylori infection among 3194 asymptomatic subjects in 17 populations. *Gut* 1993; 34: 1672-76.
- [13] Matsukawa Y, Kitamura N, Iwamoto M, Kato K, Mizuno S, et al. Helicobacter pylori upregulates peripheral counts mainly in female patients. *Acta Haematol* 2011; 126:172-5.
- [14] Hermiston ML, Mentzer WC. A practical approach to the anemic child. *Pediatr Clin North Amer* 2002; 49:877-91.
- [15] Milman N, Rosenstock SJ, Andersen LP, Jorgensen T, Bonnevie O. The relationship of Helicobacter pylori to iron status-serum ferritin and hemoglobin. A seroepidemiologic survey of 2794 Danes. *Ugeskr Laeger* 2000; 162: 1564-7.
- [16] Peach HG, Bath NE, Farish SJ. Helicobacter pylori infection: an added stressor on iron status of women in the community. *Med J Aust.* 1998; 169: 188-90.
- [17] Weller PF. The immunobiology of eosinophils. *N Engl J Med* 1991; 324: 1110-18
- [18] Rothenberg ME. Eosinophilia. *N Engl J Med* 1998; 338: 1592-600.
- [19] Levy AM, Kita K. The eosinophil in gut inflammation: effector or director? *Gastroenterology* 1996; 110: 768-74.
- [20] Bischoff SC, Mayer J, Nguyen QT, Stolte M, Manns MP. Immunohistological assessment of intestinal eosinophil activation in patients with eosinophilic gastroenteritis and inflammatory bowel disease. *Am J Gastroenterol* 1999; 94: 3521-9.
- [21] McGovern TW, Talley NJ, Kephart GM, Carpenter HA, Gleich GJ. Eosinophil infiltration and degranulation in Helicobacter pylori associated chronic gastritis. *Dig Dis Sci* 1991; 36:435-40.
- [22] Aydemir S, Tekin IO, Numanoğlu G, Borazan A, Ustundag Y. Eosinophil infiltration, gastric juice and serum eosinophil cationic protein levels in Helicobacter pylori-associated chronic gastritis and gastric ulcer. *Mediators Inflamm* 2004; 13: 369-72.
- [23] Giembycz MA, Lindsay MA. Pharmacology of the eosinophil. *Pharmacol Rev* 1999; 51: 213-340.
- [24] Winterkamp S, Raithel M, Hahn EG. Secretion and tissue content of eosinophil cationic protein in Chron's disease. *J Clin Gastroenterol* 2000; 30:170-5.