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ORIGINAL ARTICLE

Evaluation of Platelet Parameters in HBsAg positive somalian patients.

Oznur Sari[1], Mehmet Polat[2]

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ABSTRACT... Objective: To evaluate platelet parameters in HBsAg-positive Somalian patients. **Study Design:** Cross-sectional study. **Setting:** Recep Tayyip Erdogan Hospital in Mogadishu, Somalia. **Period:** January 2017 to November 2020. **Methods:** Three hundred sixty five HBsAg-positive patients and 544 HBsAg-negative healthy individuals included in this study. HBsAg-positive patients were divided into two groups according to their HBV-DNA levels. Complete blood count results of patients and healthy controllers were obtained retrospectively from the information processing system in the hospital. **Results:** The average platelet count in HBsAg-positive patients and healthy controls was 246.8±76.0x103/mm3 and 283.1±67.7 x103/mm3, respectively and the difference was statistically significant ($p<0.001$). There was a statistically significant difference in mean platelet volume values between the two groups ($p<0.001$). There was statistically significant difference between platelet distribution width values of HBsAg-positive patients and HBsAg-negative group ($p < 0.001$). **Conclusion:** In this study, we found significant decrease in platelet count and mean platelet volume level. We also found significant increase in platelet distribution width level in HBsAg-positive Somalian patients.

Key words: Hepatitis B, Mean Platelet Volume, Platelet Distribution Width.

INTRODUCTİON

Hepatitis B virus (HBV) infection is a serious health problem that affects approximately 350-400 million people worldwide, is the leading cause of progressive liver fibrosis, liver cirrhosis and hepatocellular carcinoma.[1,2] The incidence in sub-Saharan Africa ranges between 9% and 20%.[3] HBV infection can manifest as different clinical types, including infection with Hepatitis B surface antigen (HBsAg) positivity and non-clinical symptoms, chronic Hepatitis B, cirrhosis and hepatocellular carcinoma.[4,5] It is recognized that inflammation plays an important role in the development and progression of liver disease.

It has been suggested that platelets have an important role in the pathogenesis of local and systemic inflammation-related disorders.[6] Mean platelet volume (MPV) and platelet distribution width (PDW) are some parameters in complete blood count (CBC), which define thrombocyte size and the extent of diversity in thrombocyte size. MPV is a simple, inexpensive, and readily available measure in hospital and outpatient settings. MPV is a potentially useful marker as an indicator reflecting platelet function and activity. Additionally MPV had been shown to be an markers of systemic inflammation.[7-10] Peripheral artery disease, acute coronary syndromes, autoimmune disorders, thyroid functional abnormalities, local or systemic infections, inflammatory diseases may affect MPV levels.

The clinical significance of platelets in HBV-related liver diseases has been shown in many studies. Clinical experience suggests that platelet parameters may be useful in the assessment of liver inflammation and fibrosis. Some researchers have investigated the value of MPV in predicting the severity, fibrosis, and inflammation in several hepatic diseases, such as steatosis, cirrhosis and hepatitis.[11-13] Higher MPV has been demonstrated among HBV infected cases.[14]

1. MD, Associate Professor, Republic of Turkey Ministry of Health, General Directorate of Turkish Public Hospitals, Ankara-Turkey.
2. MD, Specialist, Karabuk Training and Research Hospital.

Correspondence Address:
Öznur Sarı
Üniversiteler Mh., Dumlupınar Bulvarı, 6001 Caddesi No:9, 06490 Çankaya, Ankara, Turkey.
oznurure@yahoo.com

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It has been showed that MPV is higher in HBV infection patients than levels in healthy controls.[14-16] Minimal research has been conducted to investigate the relationship between MPV and HBsAg positive patients. Therefore, in the present study, we investigated the MPV and PDW in HBsAg positive Somalian patients.

METHODS

This cross-sectional study was conducted from January 2017 to November 2020, at Turkey Recep Tayyip Erdogan Hospital, Mogadishu, Somalia. Ethics committee decision was taken from Recep Tayyip Erdogan Hospital for this study (MSTH/2613-25-11-2019). This study included 365 HBsAg-positive patients and 544 HBsAg-negative healthy individuals. Patients and healthy individuals were excluded if they were <18 years of age. The patients having underlying such as cardiac diseases, renal failure, hypertension, diabetes mellitus, atherosclerotic disease, chronic infections, malignancy, autoimmune disorders, hematological diseases, chronic obstructive lung diseases and coinfection with HCV, HDV, and HIV, as well as patients taking drugs such as aspirin, warfarin, heparin, oral antidiabetics, hyperlipidemics, and antihypertensives were excluded from the study. During the same time period, 544 HBsAg-negative healthy controls who had no specific illnesses requiring treatment, were evaluated.

Platelet parameters were evaluated using an automated haematology analyser (Sysmex XN-1000 Sysmex Corporation, Kobe, Japan). Venous blood samples were collected in a vacutainer containing di-potassium EDTA and processed within 4 hours of sample collection. Platelet parameters included platelet count, MPV and PDW. Platelet count of <150,000/μL was considered as thrombocytopenia. HBsAg concentrations were measured by use of an electro-chemiluminescence immunoassay (Cobas e411, Roche Diagnostics GmbH, Mannheim, Germany). HBV-DNA was determined by real-time polimerase chain reaction (PCR) with automated system according to manifacturer's protocols. CBC, HBsAg and HBV-DNA results of patients and healthy controllers were obtained retrospectively from the information processing system in the hospital.

In this study, 365 HBsAg-positive patients were divided into two groups according to their HBV-DNA levels. HbsAg-positive patients with HBV DNA level above 2000 IU/mL were considered as high HBV-DNA group and HbsAg-positive patients with HBV DNA level below 2000 IU/mL as low HBV-DNA group.

Statistical analysis was performed using SPSS 26 version. Data were presented as mean ± standard deviation. Between group comparisons of continuous variables were made using independent sample t-test. The chi-square test was used for categorical variables. Comparison between the three studied groups means were analyzed using analysis of variance test (ANOVA). P values <0.05 were considered to be statistically significant for all analyses.

RESULTS

In this study, platelet count and platelet indices (MPV and PDW) were evaluated in HBsAg-positive patients and compared with HBsAg-negative healthy individuals. A three hundred sixty five HBsAg-positive patients and 544 HBsAg-negative healthy individuals matched for age included in this study. HBsAg-positive patients age ranged between 18-76 years with mean age 37.43±12.36 years and healthy individuals age ranged between 18-92 with mean age 39.94±16.87 years. There was no significant differences between these two groups, in terms of age (p=0.191). The male female sex ratio was 1.66:1 for HBsAg-positive patients and 1.17:1 for healthy individuals. There was statistically significant difference between these two groups for sex ratio (p=0.01).

The average platelet count in HBsAg-positive patients and healthy controls was 246.8±76.0x103/mm3 and 283.1±67.7x103/mm3, respectively and the difference was statistically significant (p<0.001). Thrombocytopenia was found in 6.3% (n =23) of the HBsAg-positive patients and 2.4% (n=13) of healthy controls (p=0.003). MPV was significantly decreased in the HbsAg positive group (n = 365) compared to the control group

(n = 544), (9.73±1.32 in HbsAg positive group and 9.97±0.69 in control group; p=0.001). The PDW, a measure of variation in platelet size, was increased in the the HbsAg positive patients. There was statistically significant difference between PDW values of HbsAg positive patients and healthy individuals (14.8±2.37 vs 12.5±2.08, respectively p = 0.001). The demographics and characteristics of of HbsAg positive and HbsAg negative groups are illustrated in Table-I.

Of the 365 HBsAg-positive patients, 101 (27.7%) were high HBV-DNA group (≥2000 IU/mL) and 264 (72.3%) were low HBV-DNA group (<2000 IU/mL). There was no statistically significant difference between these two groups in terms of platelet count (p = 0.617), MPV (p = 0.471) and PDW (p = 0.197). Frequency of thrombocytopenia was 7.9% in the HBsAg-positive high HBV-DNA group and 5.7% in HBsAg-positive low HBV-DNA group (p=0.431). Alanine aminotransferase (ALT) and Aspartate aminotransferase (AST) level were significantly higher in high HBV-DNA group compared to low HBV-DNA group (127.9±367.4 U/L vs 60.6±202.9 U/L; p=0.027 for ALT and 101.4±301.1 U/L vs 30.5±16.3 U/L; p=0.001 for AST). Using ALT threshold of more than 2 × ULN, found 25 (24.8 %) patients in high HBV-DNA group and 28 (10.6%) patients in low HBV-DNA group to have high ALT level (p=0.001). This was for AST 18 (17.8%) patients in high HBV-DNA group and 6 (1.6%) patients in low HBV-DNA group. (p=0.028). The characteristics of HbsAg positive high and low HBV-DNA groups and control group are illustrated in Table-II.

DİSCUSSION

HBV infection is an important health problem worldwide. In Asia and sub-Saharan Africa, HBV infection is endemic and thought to be the main etiological factor more than 75% of the chronic liver diseases.[17] It has been well established that many haematological and biochemical abnormalities occur in HBV infection.[14,18] In the present study, out of 365 HBsAg positive patients and 544 HBsAg negative controls were included. We aimed to investigate whether exists a relationship between HBsAg status and platelet parameters.

We found that HBsAg positive patients had significantly lower MPVs compared with HBsAg negative controls. Additionally, we found a decrease in platelet count and increase in PDW level in HBsAg-positive patients. Low MPV level was not in accordance with other studies in the literature. For example, Turhan et al. reported that MPV was significantly higher in an inactive HBsAg carrier group compared with a control group.[12]

Characteristic	HBsAg Positive n=365	HBsAg Negative n=544	P-Value
Age (years) (mean±SD)	37.43±12.36	39.94±16.87	p=0.191
Sex (male/female)	228/137	293/251	p=0.01
Platelet count (×10³/μl)(mean±SD)	246.8±76.0	283.1±67.7	p<0.001
Platelet count <150×10³/μl	23/365 (6.3%)	13/544 (2.4%)	p=0.003
Mean platelet volume (fL) (mean±SD)	9.73±1.32	9.97±0.69	P=0.001
Platelet distribution width %	14.8±2.37	12.5±2.08	P=0.001

Table-I. Characteristics of HbsAg positive patients and HbsAg negative control group

Characteristic	High HBV DNA Group n=101	Low HBV DNA Group n=264	Control Group n=544	Anova P-Value
Age (years) (mean±SD)	37.43±13.75	39.03±11.78	39.94±16.87	p=0.283
Sex (male/female)	58/43	170/94	293/251	p=0.018
Platelet count (×10³/μl)(mean±SD)	243.6±79.7	248.1±74.7	283.1±67.7	P=0.001*
Platelet count <150×10³/μl	8/101 (7.9%)	15/264 (5.7%)	13/544 (2.4%)	p=0.008
Mean platelet volume (fL) (mean±SD)	9.80±1.38	9.69±1.29	9.97±0.69	P=0.001*
Platelet distribution width %	14.6±2.56	14.9±2.28	12.5±2.08	P=0.001*

Table-II. The characteristics of of HbsAg positive high and low HBV-DNA groups and control group

Hu et al. were reported that MPV has significantly increased in chronic HBV-infected patients and is associated with disease severity.[18] Additionally, it has been suggested that MPV values are higher in chronic hepatitis B patients, are associated with severity, and can be considered as independent predictors of liver fibrosis.[14,19] In a study demonstrated that elevated MPV might be an independent predictor of cirrhosis in patients with chronic HBV infections.[20] The difference between the results in our study and other studies may be related to the genotype of the virus. Genotype A predominates in northern Europe and America, genotypes B and C in Asia, genotype D in southern Europe and genotype E in Africa.[21]

PDW is an indicator of heterogeneity of platelet size. Our findings have shown that PDW increased in HBsAg-positive patients. This finding is in contrast to many studies (Turhan et al.,2010; Hu et al.,2014; Ekiz et al., 2011; Karagoz et al.,2014).

High replication of HBV DNA was a risk factor for the development of cirrhosis in chronic hepatitis B patients. Our study did not show significant difference between HbsAg positive high HBV DNA and low HBV DNA groups in terms of MPV and PDW. There was significant increase in AST and ALT in high HBV-DNA group compared to low HBV-DNA group (p=0.001).

CONCLUSION

The study are limited by the retrospective study design. In addition, MPV levels of the patients were not monitored during the course of disease. It is unclear whether the MPV is elevated when a patients disease progressively worsen. We need for further studies to comparing MPV with healthy controls and chronic hepatitis B patients and HBsAg-positive inactive carriers.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION

No.	Author(s) Full Name	Contribution to the paper	Author(s) Signature
1	Oznur Sari	Conception or deisgn, Data collection, Analysis, Interpretation, Literature search, Writing, Critical review.	
2	Mehmet Polat	Conception or design, Analysis, Interpretation, Literature search, Writing, Critical review.	