

Original Research Paper Medicine

MEAN PLATELET VOLUME (MPV) IN NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD) PATIENTS

Nallathambi N	Assistant Professor, Department of Medicine, Trichy SRM Medical College Hospital & Research Centre, Irungalur, Trichy.	
Sivakumar K*	Associate Professor, Department of Medicine, Trichy SRM Medical College Hospital & Research Centre, Irungalur, Trichy.*Corresponding Author	
Jibin joshua	MBBS student, Trichy SRM Medical College Hospital & Research Centre, Irungalur, Trichy.	
K. Ambedkar Raj	Professor, Department of Pathology, Trichy SRM Medical College Hospital & Research Centre, Irungalur, Trichy.	
Thamarai R	Associate Professor, Department of Biochemistry, Trichy SRM Medical College Hospital & Research Centre, Irungalur, Trichy.	

ABSTRACT Introduction

Increased mean platelet volume (MPV), a marker of platelet activity. There is a strong correlation between increased MPV and NAFLD patients.

Objectives

The objectives of this study were to assess the mean platelet volume in patients with NAFLD, and to compare the MPV of patients with NAFLD and of the individuals without fatty liver.

Materials and Methods

Fifty NAFLD male patients and 50 age matched individuals without fatty liver were involved in this study as cases and controls respectively. Complete blood count was done in those patients to assess the MPV. Ultrasound Abdomen was done to diagnose NAFLD. All analyses were performed using SPSS software version 21.

Results

The mean age of cases and controls was 47.8 ± 4.5 and 46.3 ± 3.8 respectively. MPV was significantly higher in NAFLD than non-fatty liver disease group (p = 0.01).

Conclusion

MPV strongly correlates with NAFLD patients. Increased MPV indicates enhanced platelet activity and this may be used as a simple surrogate marker for the prediction of NAFLD.

KEYWORDS : Complete blood count, Mean Platelet Volume, Non-alcoholic fatty liver disease, platelet activity.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD)is nothing but the deposition of fat in the absence of alcohol consumption1. NAFLD is also associated with steatosis, hepatitis, fibrosis, cirrhosis, and in later stage may progressed to hepatocellular carcinoma (HCC)2,3. NAFLD is comprised of non-alcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH)2,3. Most of the NAFLD patients remain asymptomatic and it may be diagnosed during routine checking of Ultrasound Abdomen. The aetiology of NAFLD remains inadequately described, and the previous research clearly depicted that this disease is progressed to cirrhosis and HCC3. The unpleasant truth is that the prevalence of NAFLD is rising globally with every year, and the reason behind this is the changing the trends in dietary habits and preponderance of sedentary lifestyle2,4. NAFLD has been proposed as the main reason of liver associated morbidity and mortality as well as a foremost indication for liver transplantation4,5. The incidence of NAFLD is more common in diabetes and insulin resistance6. A two-hit model of NAFLD occurrence has been elucidated with the first hit comprises of hepatic lipid accumulation, high fat diet, sedentary lifestyle, obesity, and insulin resistance 2,7. The second hit activates an inflammatory event with associated fibrogenesis2,8. Associations have been observed between sedentary behaviour and the risk of developing NAFLD and NASH; the severity of NAFLD also aggravates with decreased physical activity6. Mean platelet volume (MPV) measures the thrombocyte volume9. MPV is an instrument - computed measurement of average platelet size, commonly comes under the complete blood count test. MPV ranges from 7.2 femtoliters (fL) to 11.7 fL in healthy subjects9,10. In MPV, thrombocyte volume is estimated by analysing the platelet distribution curve, which is determined from a log transformation of the platelet volume distribution curve, to yield a geometric mean by using impedance technology systems9,11. The platelet volume is closely related to cytokines like interleukin-6, interleukin-3, thrombopoietin and which regulate megakaryocyte ploidy and platelet number and ends in the formation of larger sized platelets9,12. Young platelets become bigger and MPV levels increased when platelet production is decreased. Increased MPV represents increased platelet diameter and can be used as a marker of production rate and platelet activation9. Few studies have explored the relationship between MPV and NAFLD. Increased MPV is the risk factor for thromboembolic events and associated with mortality. To the best of our knowledge, no studies have focused on the association between MPV and NAFLD in rural area of southern TamilNadu. In few studies, it was postulated that increased MPV was associated with NAFLD. There are studies which showed no significant difference among the MPV and NAFLD. Therefore, this study was designed to to assess the mean platelet volume in patients with NAFLD, and to compare the MPV of patients with NAFLD and of the individuals without fatty liver.

Materials and methods

This was the case control study conducted in the tertiary care teaching hospital, Trichy. Fifty patients with NAFLD were enrolled as cases. Based on the medical records, the patients excluded were thrombocytopenic purpura, malignancy, history of Myocardial infarction, stroke, chronic liver disease, and those who had the habit of alcohol consumption, patients on drugs which cause fatty liver (amiodarone, valproic acid, antiretroviral drugs) and on drugs that interact with normal platelet functions (aspirin). Fifty apparently

VOLUME-7, ISSUE-11, NOVEMBER-2018 • PRINT ISSN No 2277 - 8160

healthy age matched individuals without fatty liver were involved in this study as cases and controls respectively. The study group which consists of age group more than 20 years of males. The study duration was 3 months. After getting informed written consent from the study population, under strict aseptic precautions, 3ml of venous blood in fasting state was taken. Complete blood count to assess MPV was done. Body Mass Index (BMI) and Waist circumference was calculated among the study group. WC was measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest, using a stretch-resistant tape13. The measurements were taken with the individuals in light clothes and when they were breathing quietly at the end of their expirations. The BMI is defined as the body mass divided by the square of the body height and is universally expressed in units of kg/m2 resulting from weight in kilograms and height in metres13. Blood glucose estimation was done using Glucose oxidase -Peroxidase method. Liver enzymes like Alanine Transaminase, Aspartate Transaminase was also estimated. Lipid profile which includes Total Cholesterol, Low-density lipoprotein (LDL), Highdensity lipoprotein [HDL] and triglycerides (TGL) were estimated. Ultrasound Abdomen was used to diagnose NAFLD. Ethical clearance was obtained from Institutional ethical committee. All analyses were performed using SPSS software version 21. Categorical variables were expressed as percentages. Continuous parameters were presented as mean ± standard deviation and compared using the independent samples t-test or the Mann-Whitney U test. Association of variables were done using logistic regression analysis. P value < 0.05 was considered as statistically significant.

RESULTS

Table 1 shows the demographic profile of the study population. The data in table 2 are depicted as the mean \pm SD or median (minimum–maximum). The mean age of cases and controls was 47.8 \pm 4.5 and 46.3 \pm 3.8 respectively. MPV was significantly higher in NAFLD than non-fatty liver disease group (p = 0.01). A statistically significant increase in MPV values was observed in NAFLD patients (10.76 \pm 2.23 fL) compared to healthy controls (7.01 \pm 0.81 fL) (P < 0.001). Body mass index and waist circumference levels were higher in the NAFLD group when compared to the controls as shown in Table 2. Comparison of lipid profile among cases and controls were given in Table 3. OR (logistic regression analysis) was given in table 4.

Table 1 Demograp	hic profile o	f the study po	pulation
------------------	---------------	----------------	----------

Variables	Cases (N)	Controls (N)
Age (Years)		
20-39	11	12
40-59	30	28
> 60	9	10
Diabetes	28	7
Non-Diabetes	22	43
Obese	29	17
Non-Obese	21	33

n-number

Table 2 Comparison of variables among cases and controls

Variables	Cases	Controls	P value
Age (years)ª	47.8 ± 4.5	46.3 ± 3.8	0.15
WC (cm)ª	85.8 ± 2.9	79.1 ± 2.5	< 0.01
BMI (kg/m2)ª	27.5 ± 3.3	22.3 ± 2.1	< 0.01
RBS (mg/dL)ª	116.4 ± 4.5	86.8 ± 3.4	< 0.01
ALT (IU/L) ^b	79 (39-194)	18 (10-35)	< 0.001
AST (IU/L) ^b	39 (23-124)	19 (12-37)	< 0.01
MPV (fL) ^b	10.76 ± 2.23	7.01 ± 0.81	< 0.01

a-Mann-Whitney test; b-independent 't' test; WC- Waist circumference; BMI - Body mass Index; RBS - Random Blood Sugar; ALT-Alanine Transaminase; AST- Aspartate transaminase; MPV-

Mean platelet volume.

т	ahle	30	omnarison	of linid pro	ofile among	cases and	controls

-		-	
Variables	Cases	Controls	P value
TC (mg/dL)	189 ± 4.5	159 ± 2.6	< 0.01
LDL (mg/dL)	118 ± 3.8	110 ± 3.2	0.55
HDL (mg/dL)	36± 3.5	38 ± 3.6	0.64
TGL (mg/dL)	174 ± 5.2	119 ± 7.2	< 0.01

TC-Total Cholesterol; LDL-Low-density lipoprotein; HDL- High-density lipoprotein; TGL-Triglycerides. Data are given as mean \pm Standard deviation using independent 't' test.

Table 4 Association of variables with fatty liver using logistic regression analysis

Variables	OR	95%CI
Age (years)a	1.25	0.93 - 1.106
WC (cm)a	1.04	0.95-1.09
BMI (kg/m2)a	1.05	0.95 - 1.11
MPV (fL)b	0.95	0.86 - 1.05

WC- Waist circumference; BMI - Body mass Index; MPV-Mean platelet volume.

Mean values of (TC 189 \pm 4.5 vs. 159 \pm 2.6; p <0.01), and TGL (174 \pm 5.2 vs. 119 \pm 7.2; p <0.01) was significantly higher in NAFLD group than in non-NAFLD group. On the other hand, LDL and HDL were statistically not significant between NAFLD and non-NAFLD cases (Table 2). BMI (odds ratio [OR] = 1.05, confidence interval [CI] = 0.93 - 1.106), MPV (OR = 0.95, CI = 0.86 - 1.05), were significantly associated with the risk of NAFLD occurrence using logistic regression analysis.

DISCUSSION

This study deals with the MPV in NAFLD patients and non-fatty liver patients. The mean age of the NAFLD cases was 47.8 ± 4.5 . Among the NAFLD cases, 28 had diabetes and 29 were obese. The mean MPV of NAFLD and non-NAFLD were 10.76 ± 2.23 and 7.01 ± 0.81 respectively. Platelet volume heterogeneity occurs during its formation and increases MPV elucidating that bone marrow produces platelets and releases into the blood stream. ALT and AST levels were increased in cases than controls with significant p value. Serum total cholesterol and triglycerides were significantly increased in NAFLD individuals than non-fatty liver group. Ozhan et al. elucidated that lower PC and higher MPV are independent predictors of NAFLD14. Another study in korea had also proved that significant association was observed between the presence of NAFLD and higher MPV values in 628 obese individuals15. In our study, the OR of MPV was 0.95 and this shows the positive association between NAFLD and controls. Obesity also play an imperative role in the occurrence of fatty liver. In our study there was significant link between increased BMI and WC with NAFLD with OR 1.05 and 1.04 respectively (table 4). Diet has also act as an independent risk variable for the occurrence of NAFLD, particularly a diet rich in fats. Restriction of carbohydrates, fat, or enrichment with monounsaturated fatty acids, that dietary modifications can reduce metabolic syndrome. In this study, among NAFLD, 28 individuals were diabetics. Insulin resistance that act as an imperative role in the occurrence of steatosis/NASH, which results in hepatic de novo lipogenesis and reduction of adipose tissue lipolysis, with a resultant increase of fatty acids in the liver2. Variations in the formation and secretion of adipokines and inflammatory cytokines are result of dysfunction of adipose tissue which is due to insulin resistance2. To our knowledge the MPV is not included as a part of the test to investigate fatty liver. This simple test may be helpful to identify the fatty liver in combination with other biochemical parameters. This test is a quick, simple, cost effective and potentially useful test and may provide a clue to the inflammation that takes place in liver.

CONCLUSION

MPV and ALT seems to be a predominant laboratory marker which increases significantly in NAFLD patients. MPV is found to be a potential risk factor for atherosclerosis, this marker may be helpful in follow-up of NAFLD patients. Further studies are needed to elucidate the associations between MPV and NAFLD patients. More studies are needed to determine the prognostic value of these laboratory parameters.

Limitations

This study is single centred with small sample size and this study includes only males. The diagnosis of NAFLD was not confirmed by biopsy of liver.

Financial support and sponsorship Nil.

.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

- 1. Non-alcoholic fatty liver disease in 2015. World J Hepatol. 2015;7(11):1450-9.
- Benedict M, Zhang X. Non-alcoholic fatty liver disease: An expanded review. World J Hepatol. 2017;9(16):715-732.
- Sayiner M, Koenig A, Henry L, Younossi ZM. Epidemiology of Nonalcoholic Fatty Liver Disease and Nonalcoholic Steatohepatitis in the United States and the Rest of the World. Clin Liver Dis. 2016 May; 20(2):205-14.
- Abd El-Kader SM, El-Den Ashmawy EM. Non-alcoholic fatty liver disease: The diagnosis and management. World J Hepatol. 2015;7(6):846-58.
- Calzadilla Bertot L, Adams LA. The Natural Course of Non-Alcoholic Fatty Liver Disease. Int J Mol Sci. 2016 May 20; 17(5).
- Wei H, Qu H, Wang H, Deng H. Associations between sitting time and non-alcoholic fatty liver diseases in Chinese male workers: a cross-sectional study. BMJ Open. 2016;6(9):e011939.
- Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of non-alcoholic fatty liver disease (NAFLD). Metabolism. 2016 Aug; 65(8):1038-48.
- Peverill W, Powell LW, Skoien R. Evolving concepts in the pathogenesis of NASH: beyond steatosis and inflammation. Int J Mol Sci. 2014;15:8591–8638.
- Budak YU, Polat M, Huysal K. The use of platelet indices, plateletcrit, mean platelet volume and platelet distribution width in emergency non-traumatic abdominal surgery: a systematic review. Biochem Med (Zagreb). 2016;26(2):178-93.
- Demirin H, Ozhan H, Ucgun T, Celer A, Bulur S, Cil H et al. Normal range of mean platelet volume in healthy subjects: Insight from a large epidemiologic study. Thromb Res. 2011 Oct; 128(4):358-60.
- 11. Senaran H, Ileri M, Altinbaş A, Koşar A, Yetkin E, Oztürk M et al. Thrombopoietin and mean platelet volume in coronary artery disease. Clin Cardiol. 2001 May; 24(5):405-8.
- Larsen SB, Grove EL, Hvas AM, Kristensen SD. Platelet turnover in stable coronary artery disease - influence of thrombopoietin and low-grade inflammation. PLoS One. 2014;9(1):e85566.
- Petkeviciene J, Klumbiene J, Kriaucioniene V, Raskiliene A, Sakyte E, Ceponiene I. Anthropometric measurements in childhood and prediction of cardiovascular risk factors in adulthood: Kaunas cardiovascular risk cohort study. BMC Public Health. 2015; 15:218.
- 14. Ozhan H, Aydin M, Yazici M, Yazgan O, Basar C, Gungor A et al. Mean platelet volume in patients with non-alcoholic fatty liver disease. Platelets. 2010; 21(1):29-32.
- Shin W.-Y., Jung D.-H., Shim J.-Y., Lee H.-R. The association between non-alcoholic hepatic steatosis and mean platelet volume in an obese Korean population. Platelets. 2011;22(6):442–446.