Original Article

Correlation of mean platelet volume and red blood cell distribution width with obstructive sleep apnoea syndrome severity

Kyriaki Cholidou[1], Nektarios Anagnostopoulos[1], Konstantinos Bartziokas[2], Konstantinos Vafeiadis[1], Agamemnon Bakakos[1], Aggelos Vontetsianos[1], Vasiliki Gogou[1], Zoi Sotiropoulou[1], Christina Anagnostopoulou[1], Anna Papasarantou[1], Paschalis Steiropoulos[3], Petros Bakakos[1], Andriana I. Papaioannou[1]

[1]1st Respiratory Medicine Department, National and Kapodistrian University of Athens, Medical School, Sotiria Chest Diseases Hospital, Athens, Greece, [2]Private Sector, Trikala, Greece, [3]Department of Respiratory Medicine, Medical School, Democritus University of Thrace, Alexandroupolis, Greece

ABSTRACT

Introduction: Mean platelet volume (MPV) and red blood cll distribution width (RDW) have been assosiated with sleep apnea syndrome severity. **Objective:** To investigate the correlation of mean platelet volume and red blood cell distribution width with obesity sleep apnoea syndrome (OSAS) severity. **Methods:** Ninety patients underwent PSG. Patients with an apnoea-hypopnoea index (AHI) <5 were used as controls. Patients with AHI >5 were divided into mild: 5 ≤ AHI <15, moderate: 15≤ AHI <30 and severe OSAS: AHI ≥30. Patients >65 years, with body mass index (BMI) >40, central sleep apnoea syndrome, cardiovascular or other significant comorbidities were excluded. Blood sample collection occurred one day before polysomnography (PSG) **Results:** Sixty-four patients were included in our study. Fifty-seven (89.1%) had OSAS (16% mild, 25% moderate and 48.4% severe) while the remaining 7 (10.1%) were used as controls. MPV was similar among groups [8.1 (7.1, 9.2) vs 7.9 (6.8, 10.1) vs 8.5 (7.4, 9.1) vs 8.4 (7.6, 9.7), *P* = .930 for control, mild, moderate and severe OSAS, respectively]. RDW did not differ between OSAS patients and control [median (IQR) 14.4 (13.4, 15.3) vs 14.0 (13.5, 16.7), *P* = .950], while there was no significant difference among different stages of OSAS severity [14.0 (13.5, 16.7) vs 13.9 (11.4, 14.8) vs 14.4 (14.0, 15.3) vs 14.4 (13.3, 15.6), *P* = .517] for control, mild, moderate and severe OSAS, respectively. **Conclusion:** OSAS patients have elevated levels of RDW and MPV compared to controls; however, there was no association between OSAS severity and MPV or RDW.

KEY WORDS: Biomarkers, mean platelet volume, obstructive sleep apnoea syndrome, red blood cell distribution width, severity

Address for correspondence: Dr. Konstantinos Bartziokas, Pulmonologist, Private Sector, Trikala, Greece.
E-mail: bartziokas@gmail.com

Submitted: 30-Aug-2024 **Revised:** 05-Dec-2024 **Accepted:** 17-Dec-2024 **Published:** 29-Apr-2025

INTRODUCTION

Obstructive sleep apnoea syndrome (OSAS) is characterised by recurrent episodes of apnoeas and hypopnoeas during

Access this article online

Quick Response Code:

Website: https://journals.lww.com/lungindia

DOI: 10.4103/lungindia.lungindia_422_24

How to cite this article: Cholidou K, Anagnostopoulos N, Bartziokas K, Vafeiadis K, Bakakos A, Vontetsianos A, *et al*. Correlation of mean platelet volume and red blood cell distribution width with obstructive sleep apnoea syndrome severity. Lung India 2025;42:179-85.

sleep due to repetitive collapse of the upper airways.[1,2] Obstructive sleep apnoea is associated with an increased risk for cardiovascular diseases, such as hypertension, coronary artery disease and stroke, via the development of atherosclerosis.[2,3] The repetitive episodes of intermittent hypoxia and reoxygenation activate inflammatory processes and induce the production of pro-inflammatory cytokines resulting in increased oxidative stress, endothelial cell injury and dysfunction.

Mean platelet volume (MPV) and red blood cell distribution width (RDW) have been proposed as alternative markers to evaluate the burden of inflammation in OSAS. In recent years, several studies have focused on leukocyte subsets, red blood cell indexes, platelet indexes, neutrophils to lymphocytes ratio (N/L) and/or platelet to lymphocytes (P/L) ratio in patients with OSAS. MPV is an indicator of platelet activation and has an important role in the pathophysiology of cardiovascular diseases. RDW, a numerical measure of the size variability of circulating erythrocytes, is known as an inflammatory marker of cardiovascular diseases.

We designed this study to investigate the correlation of MPV and RDW values with the severity of OSAS defined by the apnoea–hypopnoea index (AHI) and other parameters from the polysomnography (PSG) in patients without any cardiovascular disease.

MATERIAL AND METHODS

Study population

From January 2023 to June 2023, 90 non-smoking patients, aged >20 years old, referred to our outpatient Sleep Clinic of the 1st Department of Respiratory Medicine, "Sotiria" Hospital, Athens University, were screened by PSG for OSAS. Demographic data, including age, gender, body mass index (BMI), and smoking status, as well as medical histories regarding sleep habits, were collected from patient records. Body mass index was calculated as weight in kilograms divided by height in meters squared.

Exclusion criteria were as follows: patients with a history of cardiovascular diseases (heart failure, coronary disease, angina, arrhythmias or pacemaker, and arterial hypertension), neurological diseases, renal failure, anaemia, metabolic disorders, thyroid diseases and diabetes mellitus. Patients diagnosed with obesity hypoventilation, overlap syndrome, complex sleep apnoea, central sleep apnoea, Cheyne-Stokes sleeping disorder, or REM-induced OSAS were excluded based on the PSG results because these diseases are often causing inflammation, hypoxia and oxidative stress. Furthermore, patients with inflammatory bowel disease, rheumatoid arthritis, juvenile idiopathic arthritis, psoriasis, systemic lupus erythematosus and current infections were excluded. Finally, we excluded patients with respiratory diseases other than OSAS (COPD, IPF, asthma, etc.), since these conditions are directly related to hypoxic events, oxidative stress, endothelial dysfunction and inflammation. According to these criteria, 64 out of 90 patients met our inclusion criteria and were further analysed [Figure 1].

According to the results of the PSG study, patients were classified as being in a control group or the OSAS group accordingly. The control group included patients with an AHI <5, while patients with AHI ≥5 were included in the OSAS group. Based on the AHI, patients were grouped into three OSAS severity categories: group 1 included patients with 5 ≤ AHI <15 (mild OSAS group), group 2 included patients with 15 ≤ AHI < 30 (moderate OSAS group) and group 3 included patients with AHI ≥30 (severe OSAS group). The study protocol was approved by the Ethics Committee of "Sotiria" Hospital (No. 14481), and all participants provided their written informed consent.

Study protocol

All candidates were asked to come in a fasting state to the Sleep Laboratory at 8:00 am one day before the PSG for blood sample collection to measure RDW and MPV. Afterwards, a 12-lead electrocardiogram (ECG), transthoracic echocardiogram and spirometry were performed. Arterial blood pressure was carefully measured based on the average of two seated BP readings with an electronic sphygmomanometer adapted to arm circumference.

Echocardiography

Transthoracic echocardiography (GE Logiq 9, General Electric, Ohio, USA) was performed and interpreted by the same experienced cardiologist. Ejection fraction was measured using end-diastolic and end-systolic left ventricle diameters from the short-axis views.

Spirometry

We performed spirometry before and after bronchodilation in the sitting position by the same technician for all patients using a portable spirometer (micro Sigma Medical, USA). Spirometry was performed according to the ERS/ATS guidelines by one experienced technician.[4] Forced expiratory volume in the 1st second (FEV1), forced vital capacity (FVC), and FEV1/FVC ratio were measured in each subject. Their absolute values and percent predicted were recorded. Patients with a 12% and 200 ml increase in FEV_1 after bronchodilation or patients with FEV_1/FVC ratio < 70% and FEV_1 < 80% of predicted were excluded from the study.

Polysomnography (PSG)

All participants underwent full overnight polysomnography (PSG) according to standard techniques, which included sleep staging by monitoring of central and occipital channels of electroencephalogram (C4-A1, C3-A2, O1-A2 and O2-A1), electro-oculogram and electromyograms (submental and anterior tibialis). Airflow was detected by monitoring of combined thermistor and nasal pressure transducer signals. Pulse oximetry using a

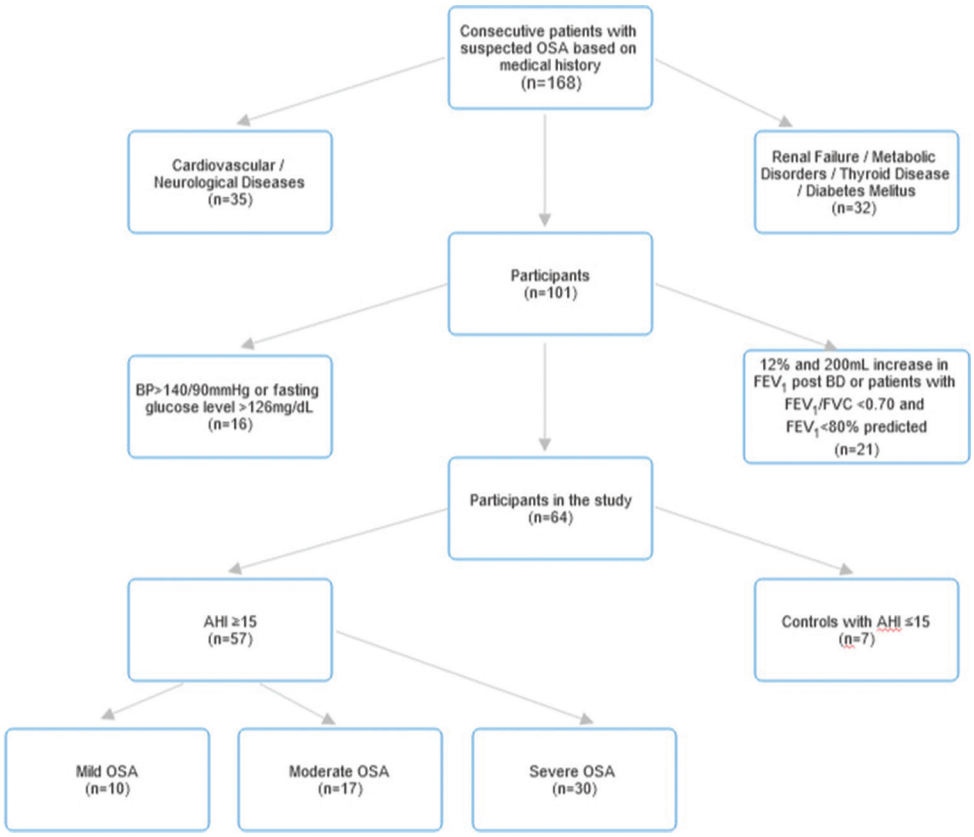

Figure 1: Study flow chart. OSAS: Obstructive sleep apnoea, BP: Blood pressure, FEV1: Forced expiratory volume in the 1st second, FVC: Forced vital capacity, BD: Bronchodilation

finger probe was continuously measuring arterial oxygen saturation (SaO_2). Standard limb leads were used for monitoring electrocardiogram and heart rate. Piezoelectric transducers were placed around the chest and abdomen, monitoring respiratory efforts. Changes of body position were detected by a body position sensor. A computerised system and software recorded all these variables (Alice 5, Philips Respironics, USA). The same physician, who was not informed about the patients' data, analysed manually all PSG recordings according to the current guidelines of the American Academy of Sleep Medicine (2018). Obstructive apnoea was defined as a reduction of airflow by ≥90% of baseline for at least 10s combined with the presence of an inspiratory effort. Hypopnoea was defined as a reduction of airflow by ≥50% lasting for at least 10 s and accompanied by a desaturation ≥4% or an arousal.

Laboratory analysis

The RDW, MPV and haemoglobin levels were determined using the Beckman Coulter LH-750 Hematology Analyzer (Beckman Coulter, Inc., Fullerton, CA, USA), as one part of a haemogram. The normal reference value for RDW in the laboratory of our hospital is between 7.2% and 11.1% and for MPV between 11.6% and 14.8%.

Statistical analysis

Normality of distributions was checked with *Kolmogorov-Smirnov test*. Categorical data were reposted as frequency counts and percentages, while numerical data were expressed as mean (standard deviation) (SD) or median (interquartile range) (IQR), for normal and skewed distribution, respectively. Associations were checked with Fisher's exact tests or Chi-squared tests for categorical data and unpaired *t*-test or Mann–Whitney U test for normally distributed and skewed data, respectively. Correlations were tested using Pearson's and Spearman's correlation coefficients for normally and skewed variables, respectively. *P* values <.05 were considered as statistically significant. Analysis was performed using the SPSS 23 statistical package (SPSS Inc., Chicago, IL, USA).

RESULTS

Study participants

Sixty-four patients, 43 (67.2%) males, age median (IQR) 59 (54-65) years, were included in the study. Of these patients, 57 (89.1%) were suffering from OSAS (AHI > 5) syndrome while the remaining 7 (10.1%) were used as control subjects. The study's flow chart is seen in Figure 1. Furthermore, 10 patients (16%) suffered from mild OSAS, 16 (25%) from moderate OSAS and 31 (48.4%) from severe OSAS. Demographic, laboratory and clinical characteristics of the study participants according to the presence or absence of sleep apnoea and according to AHI are presented in Table 1.

Mean platelet volume (MPV) and red blood cell distribution width (RDW) according to the presence and severity of sleep apnoea

MPV did not differ between patients suffering and not suffering from OSAS, median (IQR) 8.5 (7.4, 9.6) vs 8.1 (7.1, 9.2), $P = .705$, respectively. Furthermore, there was no significant difference between sleep apnoea severity 8.1 (7.1, 9.2) vs 7.9 (6.8, 10.1) vs 8.5 (7.4, 9.1) vs 8.4 (7.6, 9.7) for patients without OSAS, mild, moderate and severe OSAS, respectively, $P = .930$. The levels of MPV according to the presence of obstructive sleep apnoea syndrome and the severity of sleep apnoea are presented in Figure 2a and 2b.

RDW did not differ between patients suffering and those not suffering from sleep apnoea median (IQR) 14.4 (13.4, 15.3) vs 14.0 (13.5, 16.7), $P = .950$. Furthermore, there was no significant difference between sleep apnoea severity 14.0 (13.5, 16.7) vs 13.9 (11.4, 14.8) vs 14.4 (14.0, 15.3) vs 14.4 (13.3, 15.6), $P = .517$ for patients without OSAS, mild, moderate and severe OSAS, respectively, $P = .517$. The levels of RDW according to the presence of obstructive sleep apnoea syndrome and the severity of sleep apnoea are presented in Figure 3a and 3b.

Finally, the MPV/RDW ratio did not differ between patients suffering and those not suffering from sleep apnoea

Table 1: Demographic and laboratory characteristics of the study participants according to the presence of OSAS

Variables	All *n*=64	No OSAS *n*=7	OSAS *n*=54	*P*	Mild OSAS *n*=10	Moderate OSAS *n*=17	Severe OSAS *n*=30	*P*
Age (years)	59 (54, 65)	56 (54, 64)	59 (54, 65.5)	0.386	55 (60, 66)	62 (56, 65)	59 (54, 67.5)	0.463
Gender (males) *n* (%)	43 (67.2)	5 (71.4)	38 (66.7)	0.800	6 (60%)	9 (52.9)	23 (76.7)	0.380
BMI (kg/m^2)	37.7 (29.3, 35.5)	25.9 (25.5, 30.3)	32.9 (30.2, 36.2)	<.001	31.6 (29.4, 39.2)	32.7 (29, 33,6)	34 (31.4, 38.5)	0.001
AHI	26.7 (13.5, 63.7)	1.3 (1.0, 3.0)	33.6 (16.5, 69.2)	<.001	9 (7.7, 11.6)	19 (16.5, 23.5)	66.3 (41, 75.5)	<.001
Mean O_2 saturation (%)	92 (90, 94)	96 (94, 96)	91 (90, 93)	<.001	93 (91, 94)	93 (90.5, 93.5)	90.5 (89, 93)	<.001
Minimum O_2 saturation (%)	83 (77, 88)	93 (90, 95)	83 (77, 86)	<.001	88 (83, 88.5)	86 (81, 87.5)	77.5 (73.3, 83)	<.001
MPV (fl)	8.4 (7.4, 9.6)	8.1 (7.1, 9.2)	8.5 (7.4, 9.6)	0.705	7.9 (6.8, 10.1)	8.5 (7.4, 9.1)	8.4 (7.6, 9.7)	0.930
RDW (fl)	14.3 (13.4, 9.6)	14.0 (13.5, 16.7)	14.4 (13.4, 15.3)	0.950	13.9 (11.4, 14.8)	14.4 (14, 15.3)	14.4 (13.3, 15.6)	0.517
MPV/RDW ratio	0.59 (0.53, 0.64)	0.58 (0.55, 0.61)	0.59 (0.53, 0.64)	0.689	0.62, (0.52, 0.69)	0.59 (0.52, 0.63)	0.59 (0.54, 0.65)	0.798

Data are presented as *n* (%) or as median (IQR) unless otherwise indicated. BMI: Body mass index, AHI: Apnoea-hypopnoea index, MPV: Mean platelet volume, RDW: Red blood cell distribution width, OSAS: Obstructive sleep apnoea syndrome

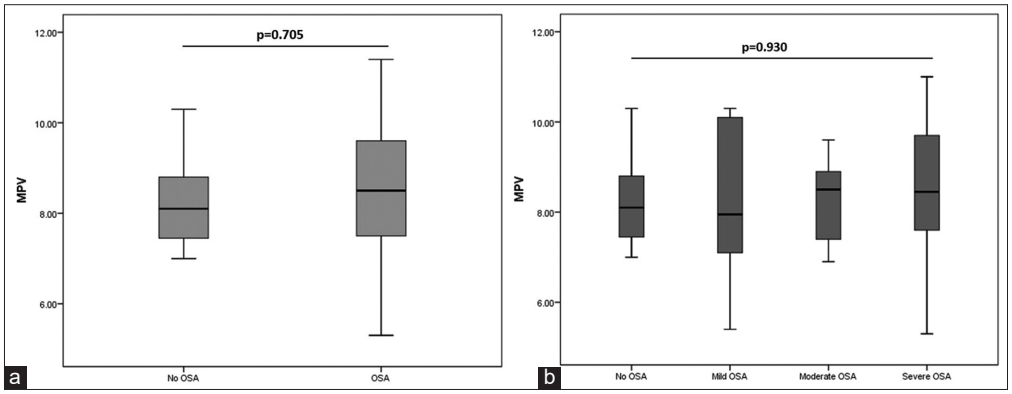

Figure 2: MPV levels according to (a) the presence of obstructive sleep apnoea syndrome and (b) the severity of sleep apnoea

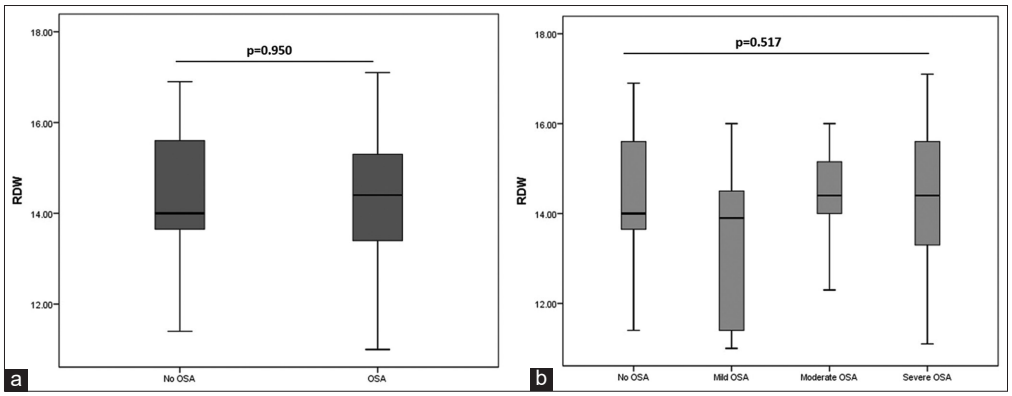

Figure 3: RDW levels according to (a) the presence of obstructive sleep apnoea syndrome and (b) the severity of sleep apnoea

median (IQR) 0.59 (0.53, 064) vs 0.58 (0.55, 0.61), $P = .689$, respectively. Furthermore, there was no significant difference between sleep apnoea severity 0.58 (0.55, 0.61) vs 0.62 (0.52, 069), vs 0.59 (0.52, 0.63) vs 0.59 (0.52, 0.63) vs 0.59 (0.54, 0.65) for patients without OSAS, mild, moderate and severe OSAS, respectively, $P = .798$. The MPV/RDW ratio according to the presence of obstructive sleep apnoea syndrome and the severity of sleep apnoea are presented in Figure 4a and 4b.

Correlations between MPV and RDW and sleep study variables

There was no significant correlation of MPV, RDW or the MPV/RDW ratio with BMI or sleep study parameters, such as AHI, minimum oxygen saturation and mean oxygen saturation, as shown in Table 2.

DISCUSSION

In the present study, we have shown that although OSAS patients have numerically elevated levels of RDW and MPV compared to controls this difference does not reach statistical significance. Furthermore, there was no significant association between the severity of OSAS and the values of RDW or MPV.

Red cell distribution width is an index of the variability in the size of circulating erythrocytes and is routinely obtained in standard complete blood counts. Anisocytosis, which can impair erythropoiesis and cause erythrocyte degradation in situations of chronic inflammation and severe oxidative stress, is thought to be indicated by a high RDW.[1] Recent evidence has shown that high RDW is associated with increased cardiovascular risk, while the levels of RDW are elevated in cardiovascular disorders, such as heart failure, coronary disease and acute coronary syndrome.[2,3] Inflammation is known to play an important role in the pathogenesis of OSAS, particularly in the presence of cardiovascular disease. For that purpose, we have included only a selected group of adult subjects with OSAS without co-morbidities known to be related to this inflammatory marker to test the existence of a significant association of this biomarker with OSAS in the absence of other factors that could affect the result.

Table 2: Correlations between MPV and RDW with sleep parameters

	AHI		Mean O_2 Saturation		Minimum O_2 Saturation	
	P	*r*	*P*	*r*	*P*	*r*
MPV	0.424	0.102	0.130	-0.191	0.217	-0.157
RDW	0.277	0.138	0.091	-0.213	0.217	-0.051
MPV/RDW	0.743	0.042	0.667	-0.055	0.163	-0.176

MPV: Mean platelet volume, RDW: Red blood cell distribution width

In our study, RDW levels were found to be numerically higher in patients with OSAS, but no association was found with the severity of OSAS according to AHI. Previous studies[5,6] have also shown higher RDW values in patients with OSAS compared to controls, but in those studies, RDW increased significantly as OSAS severity increased and was positively correlated with the AHI. Nevertheless, in these studies, the study population included patients with severe OSAS (AHI > 30), with concomitant cardiovascular diseases, while most of them were smokers and were younger compared to our study participants. Knowing that a high level of RDW in OSAS may be caused by numerous factors, including inflammation, age, and co-morbidities (e.g. anaemia, cardiovascular and metabolic diseases), we can hypothesise that the presence of those factors in the population of these studies might have affected the aforementioned findings. The exclusion of any concomitant disease except OSAS in our study population allows us to conclude that OSAS severity is not *per se* associated with RDW.

An essential element of the pathogenesis of OSAS is inflammation, especially when cardiovascular disease is present.[7] It was recently shown that OSAS severity is associated with the levels of inflammatory biomarkers, including hsCRP, RDW, interleukins and tumor necrosis factor-a (TNF-a).[8-11] In addition, in OSAS, it has been reported that intermittent hypoxia is associated with systemic oxidative stress, and higher RDW values were associated with intermittent hypoxic events, oxidative stress, endothelial dysfunction, up-regulation of pro-inflammatory

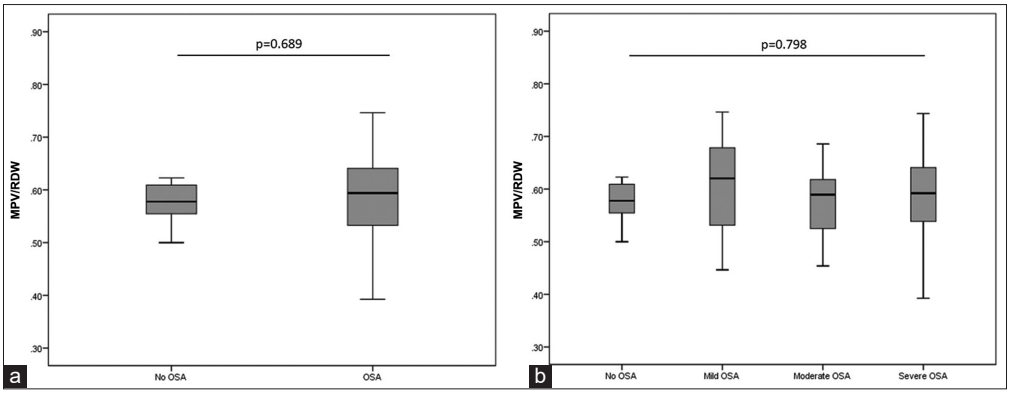

Figure 4: MPV/RDW ratio levels according to (a) the presence of obstructive sleep apnoea syndrome and (b) the severity of sleep apnoea

transcription factors and increase in inflammatory cells in OSAS patients.[5,12] Both inflammation and oxidative stress play major roles in the pathogenesis of OSAS through CRP, IL-6 and other inflammatory markers and oxidative stress products.[13,14] Although the mechanisms involved are not fully understood, oxidative stress and chronic systemic inflammation in OSAS patients play a critical role in the occurrence of adverse outcomes, mostly cardiovascular morbidity.[15] In addition, we should emphasise the fact that recent evidence has suggested that RDW levels are elevated in cardiovascular disorders.[2,3] Consequently, the lack of association between both MPV and RDW with OSAS severity, as defined by AHI, in our study population, may be the result of the exclusion of OSAS patients with CVD.

Mean platelet volume (MPV) is an indicator of platelet activation, and its levels has been found to be increased in patients with hypertension, hypercholesterolemia, diabetes mellitus, acute myocardial infarction and acute ischemic stroke.[16] While some studies reported that MPV levels increased in severe OSAS patients,[17,18] other studies showed that the MPV levels in patients with OSAS did not differ compared to the control group.[19,20] This assessment was also made among groups with different OSAS severity in our study, and it seems that although MPV values of patients with severe OSAS were significantly higher than those of the control group, there was no statistically significant correlation between MPV and AHI. MPV values are affected by too many internal and external causes, which might occasionally be altered resulting in changes in MPV values.[21] Consequently, it is necessary to understand under which circumstances MPV increases in patients with OSAS.

The association of platelet activation markers, including MPV and RDW in patients with OSAS has been evaluated thoroughly, especially during the last decade, and despite controversial results, none of the studies were designed to strictly exclude all co-morbidities and vascular risk factors. On the basis of this idea, we designed our study, and we found that serum MPV and RDW values in patients with severe OSAS were significantly higher than those of the control group, but none of these biomarkers correlated significantly with AHI. Our results suggest that patients with severe OSAS, without other cardiovascular risk factors, tend to have an increased platelet activation.

It is suggested that the hypoxic stress of OSAS is potentially a proximal mediator of systemic inflammation and platelet activation.[22] The mechanism of increased platelet activation in OSAS has been attributed to elevated levels of norepinephrine and adrenaline generated by hypoxemia and repetitive arousals,[23] and this is one of the proposed potential factors for the enhanced risk of cardiovascular events in these patients.[24] Our study showed no significant difference between the severity of OSAS and the RDW or MPV. Despite the weak statistical significance both RDW and MPV values were slightly higher in the severe stages of the OSAS, a fact that might be associated with acute and chronic hypoxia status and high oxygen desaturation levels in these stages. Finally, we should not forget that platelet activation is affected by obesity, and MPV was found to be higher in obese individuals.[25] In all three patient groups (mild, moderate and severe), the mean BMI was >30 kg/m^2 and there was no significant difference between the groups consistent with MPV and RDW.

The challenging findings regarding the association between MPV and RDW OSAS in previous studies most likely originate from patient selection. In 2012, Nena *et al*. showed that MPV and platelet distribution width (PDW) were higher in 606 non-diabetic patients with severe OSAS. In our study population, we also excluded patients with diabetes, but diabetes is by no means the only factor that could raise MPV. Finally, several studies have found that both passive and active smoking stimulate platelet function,[26,27] whereas others have indicated that MPV is higher in metabolic syndrome.[28,29] All these emphasise the importance of excluding all co-morbidities and vascular risk factors for an accurate analysis of the relationship between MPV and RDW and OSAS.

One limitation of our study may be the small sample size, which is mainly associated with the very strict inclusion and exclusion criteria. It is known that most patients with OSAS, are suffering by cardiovascular and metabolic co-morbidities which were among our exclusion criteria in our study while a great percentage of them are smokers often with concomitant respiratory disease. Another limitation of our study was the absence of a follow up and re-evaluation at some time after CPAP therapy. However, we aimed to examine the association of RDW and MPV with the presence and severity of OSAS. The alterations of these parameters after CPAP treatment was beyond the scope of our study. We also have not evaluated the possible role of environmental factors, such as diet, exercise frequency, work and place of residence. Furthermore, since the patients who have been included in the study represent the real population that has visited the sleep clinic, patients were not equally divided in groups and that could influence the results. Finally, since this was an observational study, we cannot discriminate causality from association with regard to the link between RDW, MPV and OSAS. However, in our study, elimination of all co-morbidities and vascular risk factors was a strength in comparison to other studies in the literature. To our opinion, it is crucial to rule out any other variables or factors that might have an impact on levels of RDW and MPV to come to safe conclusions on the value of measuring these biomarkers in patients with OSAS.

CONCLUSION

RDW and MPV are available and easily obtained biomarkers because a haemogram is routinely performed. These two simple biomarkers may potentially be used clinically for evaluating cardiovascular risk in OSAS patients. Future studies should also be designed to evaluate possible

alterations of these biomarkers after CPAP treatment and to determine whether their alterations are also related to alterations in cardiovascular risk.

Author's contribution and acknowledgments

Software, validation, analysis, and formal analysis: Cholidou Kyriaki, Anagnostopoulos Nektarios, Bartziokas Konstantinos, Bakakos Agamemnon, Vontetsianos Aggelos, Gogou Vasiliki, Sotiropoulou Zoi, Anagnostopoulou Christina, Papasarantou Anna, Papaioannou Andriana. Investigation, resources and data curation: Cholidou Kyriaki, Anagnostopoulos Nektarios, Bakakos Agamemnon, Vontetsianos Aggelos, Gogou Vasiliki, Sotiropoulou Zoi, Anagnostopoulou Christina, Papasarantou Anna. Writing - Original Draft: Cholidou Kyriaki, Bartziokas Konstantinos, Papaioannou Andriana. Visualization and supervision: Cholidou Kyriaki and Papaioannou Andriana I. Project administration: Cholidou Kyriaki, Papaioannou I. Andriana, Paschalis Steiropoulos and Petros Bakakos. All authors read and approved the final manuscript.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author upon reason able request.

Ethical approval

The study was approved by the ethics committees of "Sotiria" University Hospital (No. 14481) and was conducted in accordance with the Declaration of Helsinki.

Informed consent

Written informed consent was obtained from all participants.

Conflicts of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest in the present article.

REFERENCES

1. Ferrucci L, Guralnik JM, Woodman RC, Bandinelli S, Lauretani F, Corsi AM, *et al*. Proinflammatory state and circulating erythropoietin in persons with and without anemia. Am J Med 2005;118:1288.
2. Felker GM, Allen LA, Pocock SJ, Shaw LK, McMurray JJ, Pfeffer MA, *et al*. Red cell distribution width as a novel prognostic marker in heart failure: Data from the CHARM Program and the Duke Databank. J Am Coll Cardiol 2007;50:40-7.
3. Wang YL, Hua Q, Bai CR, Tang Q. Relationship between red cell distribution width and short-term outcomes in acute coronary syndrome in a Chinese population. Intern Med 2011;50:2941-5.
4. Graham BL, Steenbruggen I, Miller MR, Barjaktarevic IZ, Cooper BG, Hall GL, *et al*. Standardization of Spirometry 2019 Update. An Official American Thoracic Society and European Respiratory Society Technical Statement. Am J Respir Crit Care Med 2019;200:e70-88.
5. Ozsu S, Abul Y, Gulsoy A, Bulbul Y, Yaman S, Ozlu T. Red cell distribution width in patients with obstructive sleep apnea syndrome. Lung 2012;190:319-26.
6. Sökücü SN, Karasulu L, Dalar L, Seyhan EC, Altın S. Can red blood cell distribution width predict severity of obstructive sleep apnea syndrome? J Clin Sleep Med 2012;8:521-5.
7. Ryan S, Taylor CT, McNicholas WT. Systemic inflammation: A key factor in the pathogenesis of cardiovascular complications in obstructive sleep apnoea syndrome? Thora × 2009;64:631-6.
8. Lui MM, Lam JC, Mak HK, Xu A, Ooi C, Lam DC, *et al*. C-reactive protein is associated with obstructive sleep apnea independent of visceral obesity. Chest 2009;135:950-6.
9. Mehra R, Storfer-Isser A, Kirchner HL, Johnson N, Jenny N, Tracy RP, *et al*. Soluble interleukin 6 receptor: A novel marker of moderate to severe sleep-related breathing disorder. Arch Intern Med 2006;166:1725-31.
10. Minoguchi K, Tazaki T, Yokoe T, Minoguchi H, Watanabe Y, Yamamoto M, *et al*. Elevated production of tumor necrosis factor-alpha by monocytes in patients with obstructive sleep apnea syndrome. Chest 2004;126:1473-9.
11. Sunnetcioglu A, Gunbatar H, Yıldız H. Red cell distribution width and uric acid in patients with obstructive sleep apnea. Clin Respir J 2018;12:1046-52.
12. Stamerra CA, D'Elia E, Gori M, Roncali F, Cereda A, Gavazzi A, *et al*. Red cell distribution width (RDW) is correlated to time of oxygen desaturation < 90% and length of sleep apneas in patients with sleep disorder breathing (SDB) and acute heart failure with preserved ejection fraction (HFpEF). Front Cardiovasc Med 2023;10:1045702.
13. Carpagnano GE, Foschino-Barbaro MP, Resta O, Barnes PJ. Oxidative stress and cardiovascular complications in sleep apnea. Chest 2005;127:2294.
14. Jelic S, Padeletti M, Kawut SM, Higgins C, Canfield SM, Onat D, *et al*. Inflammation, oxidative stress, and repair capacity of the vascular endothelium in obstructive sleep apnea. Circulation 2008;117:2270-8.
15. Yamauchi M, Kimura H. Oxidative stress in obstructive sleep apnea: Putative pathways to the cardiovascular complications. Antioxid Redox Signal 2008;10:755-68.
16. Vizioli L, Muscari S, Muscari A. The relationship of mean platelet volume with the risk and prognosis of cardiovascular diseases. Int J Clin Pract 2009;63:1509-15.
17. Nena E, Papanas N, Steiropoulos P, Zikidou P, Zarogoulidis P, Pita E, *et al*. Mean platelet volume and platelet distribution width in non-diabetic subjects with obstructive sleep apnoea syndrome: New indices of severity? Platelets 2012;23:447-54.
18. Varol E, Ozturk O, Gonca T, Has M, Ozaydin M, Erdogan D, *et al*. Mean platelet volume is increased in patients with severe obstructive sleep apnea. Scand J Clin Lab Invest 2010;70:497-502.
19. Bülbül Y, Aydın Özgür E, Örem A. Platelet indices in obstructive sleep apnea: The role of mean platelet volume, platelet distribution widht and plateletcrit. Tuberk Toraks 2016;64:206-10.
20. Topçuoğlu Ö, Afşar G, Saraç S, Oruç Ö, Kuyucu T. In the absence of co-morbidities mean platelet volume is not a severity indicator in OSAS. Eur J Gen Med 2016;13:134-8.
21. Papanas N, Mikhailidis DP, Steiropoulos P. Obstructive sleep apnea and cardiovascular disease: Is mean platelet volume one of the links? Anatol J Cardiol 2016;16:223.
22. Minoguchi K, Yokoe T, Tazaki T, Minoguchi H, Oda N, Tanaka A, *et al*. Silent brain infarction and platelet activation in obstructive sleep apnea. Am J Respir Crit Care Med 2007;175:612-7.
23. Ziegler MG, Nelesen R, Mills P, Ancoli-Israel S, Kennedy B, Dimsdale JE. Sleep apnea, norepinephrine-release rate, and daytime hypertension. Sleep 1997;20:224-31.
24. Shamsuzzaman AS, Gersh BJ, Somers VK. Obstructive sleep apnea: Implications for cardiac and vascular disease. JAMA 2003;290:1906-14.
25. Coban E, Ozdogan M, Yazicioglu G, Akcit F. The mean platelet volume in patients with obesity. Int J Clin Pract 2005;59:981-2.
26. Davis JW, Hartman CR, Lewis HD Jr, Shelton L, Eigenberg DA, Hassanein KM, *et al*. Cigarette smoking--induced enhancement of platelet function: Lack of prevention by aspirin in men with coronary artery disease. J Lab Clin Med 1985;105:479-83.
27. Varol E, Icli A, Kocyigit S, Erdogan D, Ozaydin M, Dogan A. Effect of smoking cessation on mean platelet volume. Clin Appl Thromb Hemost 2013;19:315-9.
28. Park BJ, Shim JY, Lee HR, Jung DH, Lee JH, Lee YJ. The relationship of platelet count, mean platelet volume with metabolic syndrome according to the criteria of the American Association of Clinical Endocrinologists: A focus on gender differences. Platelets 2012;23:45-50.
29. Tavil Y, Sen N, Yazici HU, Hizal F, Abaci A, Cengel A. Mean platelet volume in patients with metabolic syndrome and its relationship with coronary artery disease. Thromb Res 2007;120:245-50.