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Platelet to Lymphocyte Percentage Ratio as a Marker of Arterial Stiffness in Hemodialysis Patients

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Authors' contributions

This work was carried out in collaboration among all authors. Author HHAlG conceptualization, Methodology, Resources, Investigation, Writing - original draft, Formal analysis, Writing - review and editing. Author AMEl-B formal analysis, investigation, writing – review and editing. Author TAEl-B conceptualization, methodology, formal analysis, writing - review and editing. Author GFEl-N conceptualization, methodology, formal analysis, writing - review and editing. All authors read and approved the final manuscript.

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ABSTRACT

Aim of the Work: To evaluate platelet to lymphocyte percentage ratio as a marker of arterial stiffness in hemodialysis
Study Design: Cross sectional.
Place and Duration of Study: Tanta University Hospitals; Hemodialysis Units, from June 2019 till October 2020.
Methodology: The study included 80 end stage renal disease patients (40 males and 40 females) on regular hemodialysis for at least 3months. Laboratory investigations included complete blood counts (CBC), lipid profile, serum albumin, calcium, phosphorus, parathormone hormone, uric acid and C-reactive protein (CRP). Ankle brachial index (ABI) was measured using a hand held Doppler**.** Data obtained was statistically analyzed.
Results: In our study, abnormal ankle-brachial index was found to be associated with high neutrophil %, high platelet count, high platelet lymphocyte percentage ratio (PL%R) and platelet

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lymphocyte ratio (PLR), elevated cholesterol and low density lipoprotein levels, and presence of cerebrovascular and coronary artery diseases. In multivariate analysis, PL%R and PLR were independently related to abnormal ABI in hemodialysis patients with *P* value 0.03 and 0.04 respectively. PL%R had sensitivity 92% and specificity 83% while PLR had sensitivity 81% and specificity 60%. There was a positive correlation between PL%R and CRP as a marker of inflammation.

Conclusion: Increased platelet-to-lymphocyte percentage ratio was independently associated with increased arterial stiffness in hemodialysis patients.

Keywords: Haemodialysis; ankle brachial index; arterial stiffness; platelet to lymphocyte percentage ratio.

1. INTRODUCTION

Hemodialysis (HD) was applied to maintain lives of patients with end-stage renal disease. Now, about 1.4 million patients throughout the world undergo regular HD sessions with the annual incidence rate growing up to 8 % [1].

Myocardial infarction and cerebrovascular events represent major causes of death in these patients. Also, Patients with chronic kidney disease (CKD) have a higher prevalence of peripheral artery disease (PAD) compared to the general population [2].

The ankle-brachial index (ABI), defined as the ratio of ankle and brachial systolic blood pressure in the supine position, is used for diagnosis of PAD. CKD is associated with both high and low ABI. ABI of ≤ 0.9 is commonly indicates presence of PAD and related generalized atherosclerosis. Both low and high ABI were found to be a predictor of mortality in hemodialysis patients either caused by cardiovascular disease or any other cause [3].

Inflammation, under normal conditions, is considered a physiological response to various harmful stimuli with protective effects. However, in many chronic disorders, as in chronic kidney disease, inflammation becomes harmful. It becomes systemic and persistent resulting in development of the uremic phenotype including cardiovascular diseases, protein energy wasting, frailty, osteoporosis and depression [4]. Atherosclerosis as a systemic inflammatory disease is characterized by the presence of many types of inflammatory cells within the intima of the wall of the large arteries. Some of these inflammatory cells are monocytes/ macrophages, neutrophils, lymphocytes, and natural killer T-cell. [5].

Platelets are an important linkage between the process of inflammation, thrombosis, and development of atherosclerosis. There are complex interactions among platelets, leukocytes, and endothelial cells. These interactions result in recruitment of leukocytes into the wall of the blood vessel. Chronic inflammatory processes induced by platelets at the vascular wall end in formation of atherosclerotic lesions and thrombosis [5].

Elevated platelet counts were found to be strongly related to poor cardiovascular outcomes. Besides, lower lymphocyte counts were found to be significantly and independently related to increased cardiovascular morbidity and mortality. When both parameters are combined, the platelet to lymphocyte ratio (PLR) has been recently presented as a potential marker of inflammation and a predictor of major adverse outcomes in many cardiovascular diseases and malignancies [5].

2. METHODOLOGY

2.1 Study Approval and Ethics

Permission was obtained from the Research Ethics Committee as a part of Quality Assurance Unit in Faculty of Medicine at Tanta University before carrying out this study and using the facilities in the hospital. Informed written consent was taken from all patients after full explanation of expected benefits and risks of the study. Privacy of all patients' data was guaranteed by using a special code number for each patient file that included all investigations.

2.2 Study Design and PATIENTS

This was a cross sectional study, carried out in Tanta University Hospitals, Hemodialysis Unit, The study included 80 end stage renal disease (ESRD) patients on regular hemodialysis, three times weekly (each session lasted for 4 hours with a dialyzer using a blood flow rate of 250 to 300 mL per minute and dialysate flow of 500 mL/min) using Fresenius dialysis machine and

high flux Allmed filters. Patients were on hemodialysis for at least 3 months.

2.3 Inclusion Criteria

Patients with ESRD on regular hemodialysis for at least 3 months.

2.4 Exclusion Criteria

Patients on hemodialysis less than 3 months, patients with hematological disorders affecting platelet or lymphocyte counts, patients with atrial fibrillation or those who were hospitalized or prescribed with antibiotics in the last 4 weeks (patients with acute or chronic infection).

2.5 Methods

History taking included age, sex, past medical history, any previous medical treatment if present, period of HD, place of HD and number of sessions per week. Symptoms of PAD such as claudication, paraesthesia, numbness, paralysis or weakness were also recorded.

General clinical examination included blood pressure measurement, body mass index (BMI), signs of peripheral vascular disease such as pulselessness of dorsalis pedis and posterior tibial, and pallor of distal extremities.

Laboratory investigations including complete blood counts (CBC)**,** total lipid profile including total cholesterol, triglycerides, high density lipoproteins (HDL) and low density lipoproteins (LDL), serum albumin **,** fasting blood glucose, serum cre atinine and blood urea, total calcium, Phosphorous, parathormone hormone level**,** urea reduction ratio**,** serum uric acid**,** high sensitive c-reactive protein (CRP).

PL%R was calculated for each patient by dividing platelet count by lymphocytic percentage Also, PLR was calculated by dividing platelet count by lymphocytic count**.**

2.5.1 Blood sampling and processing

10 ml venous blood sample was collected just before hemodialysis session in plain vacutainer tubes after 6 hours fasting; 2 ml were added to EDTA for CBC and serum was separated from the other 8 ml blood for all specimens using fine centrifugation at 3000 rpm for 15 min. Other samples were taken for assessment of lipid profile after 12 hours fasting. Serum samples were sent to the lab within 2 hours of collection for analysis.

2.5.2 Radiological assay

A handheld Doppler was used for Measurement of Ankle Brachial Index (ABI).The ABI was performed by measuring the systolic blood pressure in the brachial artery and from both the dorsalispedis and posterior tibial arteries of each side after the patient had been at rest in the supine position for at least 10 minutes. An ABI was calculated for each leg separately. Then the ABI value was determined by taking the higher pressure of the 2 arteries at the ankle, divided by the brachial arterial systolic pressure. In calculating the ABI, the higher value of the two brachial systolic pressure measurements was used. The normal range for the ABI is between 0.90 and 1.30.

2.6 Statistical Analysis of the Data

Statistical analysis of the data obtained in the present study was conducted, using the mean;± standard deviation, standard student "t test", chi-square test by SPSS V.22, linear correlation coefficient [r], ROC-curve and univariate and multivariate analysis.

3. RESULTS

Our study included a total of 80 patients with 40 males (50%). Their ages ranged from 17 to 77 years with a mean age (51.6 ± 13.92) years. Their BMI ranged from 16 to 45 with mean value 27.19 ± 5.05. Hypertension, diabetes, CAD and cerebrovascular disease were found respectively in 76.3% (n=61), 26.3% (n=21) patients, 31.2 (n=25) and 8.8 (n=7).

ABI ranged from 0.5 to 2, fifty three patients were in the accepted range (from 0.9 to 1.4) with percentage of 66.25%,while 27 patients had abnormally low or high ABI (less than 0.9 or more than 1.4) with percentage of 33.75%.

In our study, abnormal ABI was found to be significantly related to high neutrophil, low lymphocytic count, high platelet-to-lymphocyte ratio and high platelet-to-lymphocyte percentage ratio. Also, elevated triglycerides, cholesterol, elevated low density lipoprotein and low high density lipoprotein were found to have significant relation with abnormal ABI.

In the univariate regression analysis, abnormal ABI was found to be associated with high neutrophil %, low lymphocyte%, high platelets,

high neutrophil-to-lymphocyte ratio, high platelet-to-lymphocyte percentage ratio, high cholesterol, high low density lipoprotein, presence of cerebrovascular and coronary artery disease and high c-reactive protein. In the multivariate stepwise analysis, high platelet-to-lymphocyte percentage and high platelet-to-lymphocyte ratio were independently associated with an abnormal ankle-brachial index.

There was significant difference between normal and abnormal ABI as regard presence of diabetes mellitus. Patients with abnormal ABI had more diabetic patient percentage.

There was a positive correlation between CRP and PLR in the study group.

There was a positive correlation between CRP and PL%R in the study group.

PL%R was found to have more sensitivity and specificity than PLR regarding their relation with abnormal ABI.

4. DISCUSSION

In the present study, we evaluated the association between some hematological parameters that can be calculated from routine CBC and abnormal ABI in HD patients and found that increased PL%R and PLR were independently associated with an abnormal ABI and PL%R had more sensitivity and specificity.

Cardiovascular disease is the leading cause of morbidity and mortality in patients with hemodialysis, presumably due to accumulation of risk factors for atherosclerosis. In this study we used the ABI as a good marker for atherosclerosis. The prevalence of ischemic heart disease among our patients was found to be as high as 31.3% and this is consistent with a study done by Levin [6] that found that at least 35% of patients with CKD have evidence of an ischemic event myocardial infarction or angina.

Ishi, et al [7] have found that ABI has a predictive value for mortality due to CVD and all-cause mortality risk in HD patients, in combination with geriatric nutritional risk index and high levels of CRP. Thus, a relationship among malnutrition, inflammation, and atherosclerosis might manifest in this high-risk population. In the present study, we evaluated the possible risk factors for abnormal ABI in HD patients such as low serum albumin level, elevated triglycerides, cholesterol, LDL low level of HDL, elevated serum uric acid, fasting blood glucose, hyperparathyroidism, elevated calcium-phosphorus product, presence of diabetes mellitus and cardiovascular disease. From these factors, abnormal ABI was found to be related to elevated cholesterol, triglycerides and LDL and low HDL. Besides, it was related to presence of diabetes mellitus and cardiovascular disease.

Table 1. Demographic data of the studied group

	Range	**Mean± S. D**	
Age (years)	17 – 77	51.6 ± 13.92	
Body mass index	16-45	27.19 ± 5.05	
	Type	**Number**	**Percentage**
Sex	Male	40	50
	Female	40	50
Hypertension	Yes	61	76.3
	No	19	23.7
Diabetes mellitus	Yes	21	26.3
	No	59	73.7
Coronary artery disease	Yes	25	31.3
	No	55	68.7
Cerebrovascular disease	Yes	7	8.8
	No	73	91.2

Table 2. Ankle-brachial index among patients of the study

		Range	**Mean ± SD**
ABI		0.5 – 2	1.06 ± 0.30
		No	**Percentage**
ABI	Abnormal (<0.9 or >1.4)	27	33.75%
	Normal (0.9-1.4)	53	66.25%

ABI: ankle brachial index

Table 3. Determinants of Ankle brachial index of the patients

Variant	Ankle-brachial index	Range			Mean	±	S. D	p value
Age (years)	Normal	17	–	69	49.92	±	15.34	0.154
	Abnormal	33	–	77	54.55	±	10.61	
duration of dialysis (years)	Normal	0.5	–	18	6.01	±	4.36	0.633
	Abnormal	1	–	13	5.55	±	3.62	
Body mass index	Normal	16	–	42	26.37	±	5.98	0.111
	Abnormal	20	–	45	28.62	±	6.02	
Hemoglobin (gm/dL)	Normal	7	–	15	10.34	±	2.06	0.431
	Abnormal	6.8	–	14	9.99	±	1.66	
White blood cells(×109 cells/L)	Normal	2.3	–	11	5.48	±	2.09	0.229
	Abnormal	1.7	–	12	6.05	±	1.94	
Neutrophil %	Normal	35	–	84	62.78	±	8.90	0.001*
	Abnormal	58	–	80	69.41	±	5.34	
Lymphocyte %	Normal	10	–	56	31.76	±	8.19	0.001*
	Abnormal	17	–	34	25.41	±	4.42	
Platelets(×109 cells/L)	Normal	90	–	302	188.43	±	54.59	0.001*
	Abnormal	163	–	398	282.59	±	62.88	
Neutrophil-to-lymphocyte ratio	Normal	0.5	–	8.2	2.22	±	1.15	0.007*
	Abnormal	1.7	–	5	2.90	±	0.85	
Platelet-to-lymphocyte ratio	Normal	34	–	251	126.82	±	54.75	0.001*
	Abnormal	118	–	542	202.31	±	84.45	
Platelet-to-lymphocyte percentage ratio	Normal	2.4	–	16	6.30	±	2.40	0.001*
	Abnormal	5.8	–	19.4	11.43	±	2.96	
Serum albumin (g/dL)	Normal	2.7	–	4.4	3.64	±	0.38	0.230
	Abnormal	2	–	4.4	3.51	±	0.56	
Fasting blood glucose (mg/dL)	Normal	68	–	331	125.08	±	56.12	0.571
	Abnormal	61	–	217	118.24	±	42.34	
Triglycerides (mg/dL)	Normal	44	–	272	131.45	±	60.47	0.004*
	Abnormal	62	–	522	186.62	±	106.41	
Total cholesterol (mg/dL)	Normal	81	–	303	156.69	±	37.43	0.001*
	Abnormal	83	–	250	186.76	±	33.15	
High density lipoproteins (mg/dL)	Normal	25	–	65	45.71	±	8.39	0.001*
	Abnormal	20	–	55	38.17	±	8.24	
Low density lipoproteins (mg/dL)	Normal	40	–	220	105.90	±	34.34	0.001*
	Abnormal	50	–	190	141.86	±	31.10	
Serum creatinine (mg/dL)	Normal	4.5	–	11	7.56	±	1.62	0.439
	Abnormal	3.8	–	12	7.25	±	1.90	
Total calcium (mg/dL)	Normal	6.9	–	9	8.11	±	0.51	0.267
	Abnormal	7	–	10	8.25	±	0.60	
Phosphorus (mg/dL)	Normal	2.3	–	6.5	4.94	±	1.09	0.475
	Abnormal	2.1	–	7	4.75	±	1.13	
Calcium phosphorus product	Normal	18	–	55	39.67	±	9.57	0.417
	Abnormal	3	–	56	37.73	±	11.24	
Serum uric acid (mg/dL)	Normal	2	–	7.3	3.89	±	1.41	0.262
	Abnormal	2.1	–	9	4.30	±	1.72	
Parathormone hormone (pg/mL)	Normal	3.2	–	1036	336.53	±	293.57	0.078
	Abnormal	47	–	1960	492.79	±	490.46	
C-Reactive protein (mg/L)	Normal	6	–	24	12.57	±	7.86	0.899
	Abnormal	5	–	48	13.00	±	10.61	
Urea reduction ratio	Normal	50	–	93	70.33	±	14.43	0.116
	Abnormal	54	–	92	75.38	±	12.18	

Table 4. Univariate and multivariate analysis of determinant of ankle-brachial index in the study

	Univariate analysis		Multivariate analysis	
	Odds ratio (95% Confidence Interval)	**P value**	**Odds ratio (95% Confidence Interval)**	**P value**
Neutrophil%	3.521 (1.694 – 8.317)	0.021*	2.205 (0.715 – 6.803)	0.169
Lymphocyte	0.521 (0.189 – 0.739)	0.018*	0.902 (0.419 – 1.945)	0.793
Platelets	3.687 (2.631 – 12.3247)	0.003*	1.012 (0.928 – 1.104)	0.786
Neutrophil-to-lymphocyte ratio	0.387 (0.064 – 0.697)	0.027*	0.132 (0.012– 2.348)	0.079
Platelet-to-lymphocyte ratio	5.621 (1.754 – 13.684)	0.005*	3.066 (1.995 – 7.141)	0.041*
Platelet-to-lymphocyte percentage ratio	8.521 (2.647 – 18.512)	0.001*	4.999 (2.230 – 10.779)	0.034*
Triglycerides	1.024 (0.979 – 1.072)	0.293		
Cholesterol	2.654 (1.035 – 5.631)	0.028*	0.717 (0.481 – 1.070)	0.104
High density lipoprotein	1.141 (0.783 – 1.664)	0.492		
Low density lipoprotein	3.258 (1.589 – 8.452)	0.029*	1.472 (0.951 – 2.278)	0.083
Diabetes mellitus	0.439 (0.136 – 1.416)	0.168		
Coronary artery disease	0.414 (0.145 – 1.182)	0.099		
Cerebrovascular disease	0.352 (2.589 – 7.521)	0.031*	0.151 (0.015 – 1.523)	0.109
C-reactive protein	0.624 (0.238 – 8.652)	0.038*	0.358 (0.189 – 2.314)	0.139

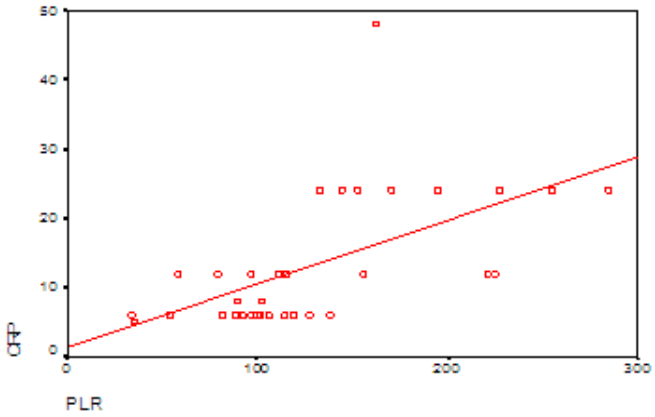

Fig. 1. Correlation between platelet-to-lymphocyte ratio and c-Reactive protein as a marker of inflammation

Table 5. Comparison between patients with normal and abnormal ABI regarding diabetes mellitus

Diabetes mellitus			**ABI type**	
			Normal	**Abnormal**
Yes		N	9	12
		%	17.6%	41.4%
No		N	42	20
		%	82.4%	58.6%
Chi-square	X^2	5.379		
	P-value	0.020*		

Table 6. Correlation between platelet-to-lymphocyte ratio and c-Reactive protein as a marker of inflammation

	Platelet-to-lymphocyte ratio	
	R	**P**
c-Reactive protein	0.586	0.001*

Table 7. Correlation between platelet-to-lymphocyte percentage ratio and C-reactive protein as a marker of inflammation

	Platelet-to-lymphocyte percentage ratio	
	R	**P**
C-reactive protein	0.751	0.001*

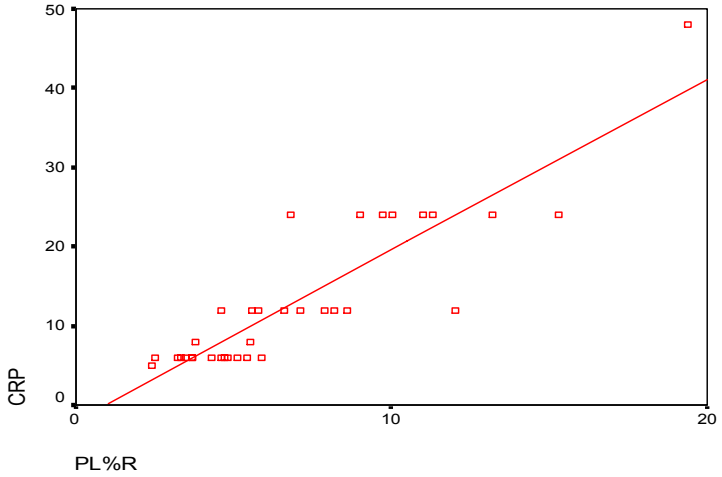

Fig. 2. Correlation between platelet-to-lymphocyte percentage ratio and C-reactive protein as a marker of inflammation

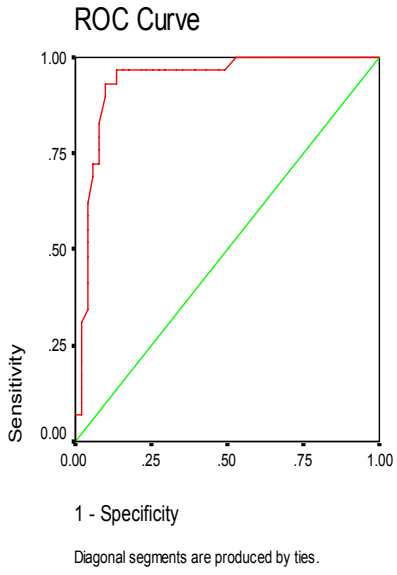

Fig. 3. ROC Curve for specificity and sensitivity regarding platelet-to-lymphocyte percentage ratio

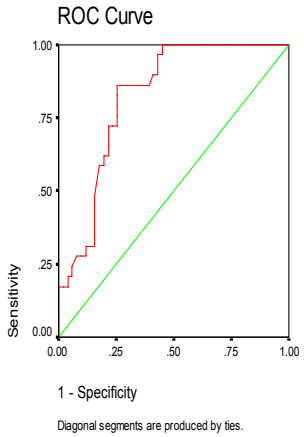

Fig. 4. ROC Curve for specificity and sensitivity regarding platelet-to-lymphocyte ratio

Table 8. PPV: positive predictive value, NPV: negative predictive value, AUC: area under curve, J: Youden index

	Cutoff	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy	J
platelet-to-lymphocyte percentage ratio	9	0.938	92	83	90	86	89	0.73
platelet-to-lymphocyte ratio	175	0.823	81	60	77	63	73	0.41

Several studies have evaluated these factors including a study done by Chang, et al [8] in which abnormal ABI was independently associated with decreased serum albumin level and significantly associated with diabetes mellitus while dyslipidemia and secondary hyperparathyroidism were not significantly related. This study agreed with our study considering the significant relation between diabetes mellitus and abnormal ABI and the non-significant relation with increased PTH. However, decreased serum albumin was not significantly related to abnormal ABI in our study.

Dyslipidemia is a well-established atherogenic factor in hemodialysis patients. This was noticed in the significant relation between abnormal lipid profile and abnormal ABI. Consistent with this, a study done by Tschöpe, et al [9] proved that high cholesterol is a potent predictor of cardiac death in uremic diabetics treated by maintenance hemodialysis. However, some studies have reported that dyslipidemia is not a predictor of mortality in hemodialysis patients. Fleischmann, et al [10] have found that hyperlipidemia did not correlate to patients' two-year mortality and suggested that conventional risk factors do not readily account for the higher mortality of patients on hemodialysis.

Secondary hyperparathyroidism and the effect of calcium, phosphorous and the calcium-phosphorous product have been reported to contribute to endothelial dysfunction and vascular calcification in patients with chronic renal failure in many studies for example Nishizawa, et al [11] have reported that deranged calcium-phosphate homeostasis and secondary hyperparathyroidism promote atherosclerosis in uremia, at least partly by affecting lipoprotein metabolism. These findings did not agree with our study because these parameters did not seem to be associated with abnormal ABI.

Hypoalbuminemia has been considered as a marker of malnutrition. Besides, it has been studied as a cardiovascular risk factor by Malatino, et al [12] and was found to be a significant and independent predictor of the number of atherosclerotic plaques in hemodialysis patients. While in the present study, its relation with ABI was not significant. We also investigated serum uric acid as a risk factor for atherosclerosis and the results of its

relation with ABI were not significant. These results did not agree with many studies including that one done by Krishnan, et al [13] which showed that hyperuricemia was an independent risk factor for subclinical atherosclerosis in adults.There was no significant difference between both sexes regarding ABI.

A large body of literature supports the idea that inflammation plays a major role in all phases of atherosclerosis. In our study we used CRP as a marker of inflammation and it was significantly related to abnormal ABI. Supporting these results, Carmine, et al [14] have found that in patients on chronic dialysis treatment CRP is independently associated with carotid atherosclerosis. CRP was found to be elevated in 43.3% of our study group. This is also agreed with a study done by Rahmati, et al [15] where CRP level was elevated in 41% of the study group of chronic hemodialysis patients.

Univariate analysis was done and found significant association between abnormal ABI and high neutrophil%, low lymphocyte percentage, high platelet count, high NLR, high PL%R, high cholesterol, high LDL, presence of cerebrovascular disease and diabetes mellitus. Multivariate stepwise analysis found that high PL%R and high PLR were independently associated with abnormal ABI. This is consistent with a study done by Chin, et al [16] which found that PL%R was independently associated with arterial stiffness using brachial ankle pulse wave velocity in HD patients.

Another study done by Bal, et al [17] has used other hematological parameters like neutrophil to lymphocyte ratio (NLR), red blood cell distribution width (RDW), and mean platelet volume (MPV), as measures of systemic inflammation and atherosclerosis in patients with end-stage renal disease (ESRD) and found that these parameters are independent predictors of the extent of coronary artery disease in patients with ESRD. These results partially agreed with our results where NLR was found to be significantly related to abnormal ABI in patients with ESRD.

Also, a study done by Cai, et al [18] has found that NLR is independently associated with arterial stiffness (assessed by brachial-ankle pulse wave velocity) in patients on peritoneal dialysis. In the same context, Mozos, et al [19] have studied inflammatory markers for arterial stiffness in CVD and reported that NLR may be used as a cost-effective biomarker of inflammation, atherosclerotic progression and systemic predictor of cardiovascular complications.

The present study also showed a statistically significant correlation between PLR and CRP and significant association between PLR and ABI. This goes in agreement with a study done by Akboga, et al [20] which showed a positive correlation between PLR and Gensini score assessing the risk of CVD and positive correlation between PLR and CRP in patients with CVD.

A comparison was done between PLR and NLR considering their relation with many inflammatory markers in patients with ESRD on HD by Turkmen, et al [21] and they found that PLR can predict inflammation better than NLR in this population which agreed with our results. Calculation of PLR and NLR are quite simple and cheap methods when compared with other inflammatory cytokines including IL-6, IL-1β, and TNF-α. This study confirmed that PLR and NLR can predict inflammation in ESRD patients. Therefore, these simple, relatively inexpensive and universally available methods can be used by internists, nephrologists, and other health care staff for the first evaluation of inflammation in ESRD patients before applying other expensive and invasive procedures.

PLR and NLR were established to show the poor prognosis and mortality in some diseases, such as cardiovascular diseases and malignancies. A study done by Yabark, et al [22] has shown that both parameters were associated with all-cause mortality in prevalent HD patients, however, only PLR could independently predict all-cause mortality in these populations. This is consistent with our study where PL%R and PLR were found to be independently related to abnormal ABI in the multivariate analysis while NLR was not.

Moreover, previous studies by Zouridakis, et al [23] have also evidenced that low lymphocyte count has been significantly related to adverse events in patients with coronary artery disease. Also, Ommen, et al [24] have reported a decrease in total and relative number of circulating lymphocytes during various cardiovascular diseases like acute myocardial infarction and congestive heart failure. This is consistent with our results where ABI was found to be significantly related to low lymphocytic count.

Our study has some limitations. First, it was retrospective; we did not perform an analysis of the prognostic value of PL%R in progression of CVD in HD patients. It was single-center study and we used a spot PL%R value for our analysis rather than several values at different time intervals. And, we used only the ABI to evaluate atherosclerosis we had no data regarding plaque characteristics by using other imaging techniques like computerized tomography angiography and intravascular ultrasonography. Also, we only used CRP as an inflammatory marker to correlate with PL%R. We did not evaluate other cytokines or inflammatory markers.

5. CONCLUSION

Our results demonstrated that an increased PL%R and PLR were independently associated with abnormal ABI in hemodialysis patients and both were also correlated with inflammation marker CR. PL%R had more sensitivity and specificity. Therefore, PL%R can be used as a simple, relatively inexpensive, and universally available method of identifying patients with increased arterial stiffness in HD patients.

CONSENT AND ETHICAL APPROVAL

Permission was obtained from the Research Ethics Committee as a part of Quality Assurance Unit in Faculty of Medicine at Tanta University before carrying out this study and using the facilities in the hospital. Informed written consent was taken from all patients after full explanation of expected benefits and risks of the study.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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