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Platelet distribution width as an early prognostic marker in acute ischemic stroke: An observational study

Nikhil Kumar[1], Polok Das[1], Mehjabin Lashkar[1], Satakshi Shome Purkayastha[1,*] and Arnab Purkayastha[1]

[1]Department of General Medicine, Silchar Medical College and Hospital, Ghungoor, Masimpur, Silchar, Assam 788014, India

Abstract

Introduction: Stroke remains one of the leading causes of disability and death worldwide, with ischemic stroke accounting for the majority of cases. Platelet activation plays a crucial role in the pathogenesis of atherosclerosis and thromboembolism. Platelet Distribution Width (PDW), a measure of platelet size variability, reflects platelet activity and function. Elevated PDW levels have been implicated as a risk factor in thromboembolic diseases. This study aimed to evaluate PDW levels in patients with acute ischemic stroke and to determine its prognostic value for risk stratification in the acute setting.

Material and methods: This hospital-based observational study was conducted in the Department of General Medicine, Silchar Medical College and Hospital, from June 2021 to May 2022. A total of 100 patients with acute ischemic stroke presenting within 48 hours of symptom onset were included, along with 100 age- and sex-matched healthy controls. Blood samples were collected, and PDW was analysed within 2 hours of admission. Stroke severity was assessed using the Modified Rankin Scale (mRS).

Results: PDW values were significantly higher in stroke patients (14.33 ± 2.78 fL) compared with controls (12.87 ± 1.86 fL; $p < 0.001$). Increased PDW was positively correlated with higher mRS scores ($p < 0.00001$), longer hospital stays, higher Intensive care unit (ICU) admissions, poorer functional outcomes at day 7, and increased in-hospital mortality.

Conclusion: Elevated PDW is associated with greater stroke severity and poorer outcomes. As an inexpensive and readily available parameter, PDW may serve as a useful prognostic biomarker in acute ischemic stroke, especially in resource-limited settings.

Keywords: acute ischemic stroke; platelet distribution width; modified Rankin scale; prognosis; mortality

Introduction

A stroke, or cerebrovascular accident, is defined as "an abrupt onset of neurological deficit that is attributable to a focal vascular cause." Thus, the definition of stroke is primarily clinical, with laboratory investigations and neuroimaging used to support the diagnosis. Stroke is diagnosed when the symptoms and signs persist for more than 24 hours or when there is radiological evidence of infarction or haemorrhage. The definition excludes transient ischemic attack (TIA), which presents with focal neurological symptoms lasting less than 24 hours and without evidence of infarction.

According to the World Health Organization, approximately 15 million strokes occur globally each year, of which 5 million results in death and another 5 million leads to permanent disability. In India, stroke remains a leading cause of mortality and disability, with an estimated age-adjusted prevalence rate of 84–262 per 100,000 in rural areas and 334–424 per 100,000 in urban areas, while the incidence rate ranges from 119 to 145 per 100,000 [1]. Stroke may result from cerebrovascular occlusion or haemorrhage, with ischemic strokes accounting for nearly 85% of all cases. Commonly used scoring systems for assessing stroke severity include the NIH Stroke Scale and the Modified Rankin Scale (mRS).

***Corresponding author:** Dr. Satakshi Shome Purkayastha, Department of General Medicine, Silchar Medical College and Hospital, Ghungoor, Masimpur, Silchar, Assam 788014, India. Email: satakshi7@gmail.com

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Platelets are small (1–4 μm in diameter), discoid, non-nucleated structures formed by the fragmentation of megakaryocytes. Beyond their role as acute-phase reactants, platelet morphology and function are influenced by an individual's health and nutritional status. Approximately 65% of platelets are smooth and disc-shaped, while 10–35% are spherical. Platelet morphology plays a crucial role in determining platelet function and in measuring their size [2]. Counterflow centrifugation is used to separate platelets based on differences in platelet volume, which correlates with density, dense body content, lactate dehydrogenase activity, aggregation to adenosine diphosphate (ADP), and serotonin uptake and release. These findings support mean platelet volume (MPV) as an indicator of platelet function [3].

Platelet Distribution Width (PDW) represents the variability in platelet size (anisocytosis) and is derived from the distribution curve of individual platelet volumes. The normal range of PDW is 9–14 fL, indicating that most platelets are of similar size. Elevated PDW values suggest greater variability in platelet size, a marker of platelet activation, and have been associated with vascular disorders. PDW is considered a more specific indicator of platelet activation than MPV, as it is not affected by simple platelet swelling [4]. Some studies recommend measuring both MPV and PDW to obtain more reliable information [5], although the predictive utility of PDW remains underexplored and its role as a platelet activation marker is still debated [6].

Platelet volume is influenced by various intrinsic and extrinsic factors. Disruption of the megakaryocyte–platelet haemostatic axis (MPHA) can lead to the formation of hyper-functional platelets, resulting in increased platelet size variability (higher PDW). This alteration may contribute to vascular diseases or acute thrombotic events such as ischemic stroke or myocardial infarction [7]. Larger platelets are metabolically and enzymatically more active, producing greater quantities of prothrombotic factors, showing increased aggregation in response to ADP, collagen, and adrenaline, and releasing higher levels of thromboxane A_2 (TxA_2) [8]. Increased platelet size has also been observed in patients with vascular risk factors such as diabetes [9], hypercholesterolemia, metabolic syndrome, stroke [10], and acute myocardial infarction.

Advances in haematology analysers have enhanced the understanding of platelet function in thrombosis, inflammation, immunity, and angiogenesis. Consequently, platelet activation has emerged as a key therapeutic target in atherothrombosis, hypertension, and diabetes.

The association between PDW and cerebrovascular accidents, as well as its prognostic significance, remains under investigation. Previous studies have demonstrated elevated MPV levels in various subtypes of stroke, both during the acute phase and in later stages [11]. However, there is limited data from India evaluating the role of PDW during acute ischemic events. Hence, the present study aims to assess the correlation of PDW with risk factors of ischemic stroke and to determine its prognostic significance in predicting stroke severity and outcome in the acute setting.

Materials and methods

This hospital-based observational study was conducted in the Department of General Medicine, Silchar Medical College and Hospital, Silchar, Assam, over a period of one year, from June 2021 to May 2022. A total of 276 stroke patients admitted to the hospital were screened, out of which 100 patients with acute ischemic stroke, who met the inclusion and exclusion criteria, along with an equal number of age- and sex-matched controls (n = 100), were enrolled in the study. The diagnosis of stroke was made clinically according to the World Health Organization criteria and was confirmed by neuroimaging using non-contrast computed tomography (NCCT) or magnetic resonance imaging (MRI) of the brain when indicated.

Venous blood samples were collected under aseptic precautions, and PDW was analysed in Automated Haematology analyser (SYSMEX XN 550), within two hours of venepuncture to minimize the time-dependent platelet swelling that occurs in EDTA vials. The severity of ischemic stroke was assessed using the Modified Rankin Scale (mRS) [12]. The mean PDW values of stroke patients were calculated and compared with those of the control group. Further analysis was performed to evaluate the correlation between PDW and various risk factors, as well as the clinical outcomes of stroke. The primary outcome of interest was the functional status of patients, determined using the mRS score. Secondary outcomes included the duration of hospital stay, the need for intensive care unit (ICU) admission, and in-hospital mortality.

Patients included in the study were those presenting with the first episode of acute ischemic stroke confirmed by neuroimaging (NCCT or MRI brain) and who reported to the hospital within 48 hours of symptom onset. Only adults above 18 years of age were included. Patients were excluded if they had a previous history of cerebrovascular accident or transient ischemic attack, intracranial haemorrhage (subarachnoid, subdural, extradural, or intracerebral), degenerative neurological or other central nervous system diseases, or presented more than 48 hours after symptom onset.

Individuals on antiplatelet or anticoagulant therapy, immunosuppressants, or those who had received blood transfusion within six weeks were excluded. Patients with peripheral vascular disease, acute infections, hepatic or renal disorders, malignancies, hereditary platelet disorders, or other haematological abnormalities were also excluded from the study.

The control group consisted of 100 age- and sex-matched apparently healthy individuals, primarily attendants of patients admitted to the hospital. All controls voluntarily participated after providing written informed consent.

The Modified Rankin Scale (mRS) [12] was used to assess the degree of disability or dependence in the daily activities of stroke patients. The scale scores range from 0 to 6, where 0 indicates no symptoms; 1, no significant disability despite symptoms; 2, slight disability but independent in daily affairs; 3, moderate disability requiring some help but ambulatory; 4, moderately severe disability with dependence for bodily needs and ambulation; 5, severe disability with bedridden status requiring constant care; and 6, death.

Statistical analysis

Statistical analysis was performed using Microsoft Excel and the Statistical Package for the Social Sciences (SPSS for Windows, version 20.0, Chicago, SPSS Inc.). Continuous variables were expressed as mean ± standard deviation (SD), and comparisons between groups were made using appropriate statistical tests. A p-value < 0.05 was considered statistically significant. The study protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to inclusion.

Results

Demographic profile

The mean age of the study population was 63.25 ± 14.19 years among stroke cases and 62.84 ± 13.71 years among controls. The majority of stroke cases (29%) were within the age group of 61–70 years, followed by 23% in the 51–60 years age group. Of the 100 stroke patients, 66 (66%) were males and 34 (34%) were females. Female patients with stroke were significantly older (67.06 ± 12.55 years) than male patients (61.29 ± 14.67 years) (p = 0.043).

Hypertension was the most common comorbidity and was more prevalent among cases (61%) compared to controls (42%) (p = 0.007). A history of smoking was also more frequent in stroke cases (42%) than in controls (27%) (p = 0.025). Other risk factors such as dyslipidaemia, diabetes mellitus, and alcoholism were observed more commonly among cases than controls, although the differences were not statistically significant.

The mean PDW was significantly higher in stroke patients (14.33 ± 2.78 fL) compared to controls (12.87 ± 1.86 fL) (p = 0.000021), indicating a strong association between elevated PDW and acute ischemic stroke. (Table 1)

Table 1: Baseline characteristics of the study population.

Characteristic	*Cases (n=100)*	*Controls (n=100)*	*P value*
Males	66 (66%)	66 (66%)	1.00
Females	34 (34%)	34 (34%)	1.00
Mean age (years)	63.25 ± 14.19	62.84 ± 13.71	0.84
Hypertension	61 (61%)	42 (42%)	0.007*
Diabetes mellitus	34 (34%)	23 (23%)	0.084
Dyslipidaemia	29 (29%)	19 (19%)	0.097
Smoker	42 (42%)	27 (27%)	0.025*
Alcoholic	21 (21%)	12 (12%)	0.086
Platelet distribution width (PDW)(fL)	14.33 ± 2.78 (9.0-25.9)	12.87 ± 1.86 (9.2-18.6)	0.000021*

Note: *significant

Relation of PDW with stroke risk factors

The mean PDW among stroke patients aged <60 years was 13.95 ± 2.55 fL, whereas in those aged ≥60 years it was 14.56 ± 2.91 fL; however, this difference was not statistically significant. PDW values were significantly higher among hypertensive patients compared to non-hypertensives (p = 0.008). Although the mean PDW was higher in diabetic patients compared to non-diabetics, the difference did not reach statistical significance.

No significant difference in mean PDW values was observed between male and female patients, suggesting that gender has no apparent influence on platelet indices. Similarly, comparison of PDW across other vascular risk factors, including dyslipidaemia versus normal lipid profile, smokers versus non-smokers, and alcoholics versus non-alcoholics, did not reveal any statistically significant differences (Table 2).

PDW and severity of acute ischemic stroke (at Presentation)

The Modified Rankin Scale (mRS) was used to assess the clinical severity of stroke at the time of presentation, where a score of 0 denotes “no symptoms” and a score of 6 denotes “death.” Among the 100 stroke patients

studied, 26% had a moderately severe disability (mRS score 4), 24% had moderate disability (score 3), 20% had severe disability (score 5), 18% had slight disability (score 2), and 12% had no significant disability (score 1).

Table 2: Relationship between platelet distribution width (PDW) and risk factors in patients with acute ischemic stroke.

Risk factors(cases)	*n(%)*	*PDW(fL)*	*P value*
Gender			
Male	66(66%)	14.26 ± 2.97	0.71
Female	34(34%)	14.47 ± 2.40	
Age (years)			
<60	38(38%)	13.95 ± 2.55	0.29
≥60	62(62%)	14.56 ± 2.91	
Hypertension			
Hypertensive	61(61%)	14.84 ± 3.24	0.008*
Non-hypertensive	39(39%)	13.52 ± 1.57	
Diabetes Mellitus			
Diabetic	34(34%)	14.67 ± 2.66	0.37
Non-Diabetic	66(66%)	14.15 ± 2.84	
Dyslipidaemia			
Yes	29(29%)	14.70 ± 4.03	0.51
No	71(71%)	14.19 ± 2.08	
Smoking			
Smoker	42(42%)	14.73 ± 3.07	0.235
Non-smoker	58(58%)	14.04 ± 2.53	
Alcoholism			
Alcoholic	21(21%)	14.92 ± 3.23	0.337
Non-alcoholic	79(79%)	17.17 ± 2.65	

Note: *significant

A strong positive correlation was observed between PDW and stroke severity based on the mRS score ($p < 0.00001$). Higher PDW values were consistently associated with more severe forms of stroke at presentation, indicating that elevated PDW may serve as an early prognostic indicator of stroke severity (Table 3).

The correlation between PDW and the mRS score was assessed using the Pearson correlation coefficient. A positive correlation was observed with an r value of 0.5824, which was statistically significant ($p < 0.00001$), as illustrated in Figure 2.

Table 3: Relationship between platelet distribution width (PDW) and stroke severity.

MRS score (0-6) (on admission)	*n(100)*	*PDW(fL)*	*P value (One way ANNOVA)*
Score 1 (No Disability)	12(12%)	12.28 ± 1.78	< 0.00001*
Score 2 (Slight Disability)	18(18%)	12.47 ± 1.35	
Score 3 (Moderate Disability)	24(24%)	13.77 ± 1.93	
Score 4 (Moderately Severe Disability)	26(26%)	15.01 ± 2.18	
Score 5 (Severe Disability)	20(20%)	17.02 ± 3.39	

Note: *significant

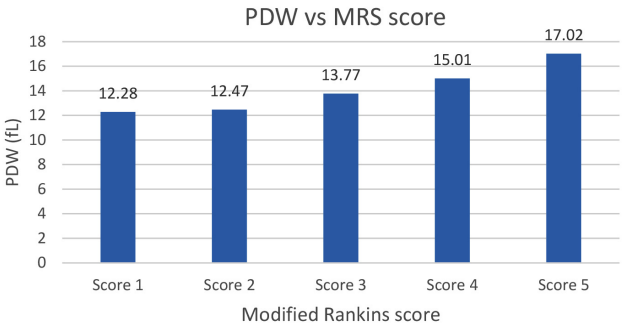

Figure 1: Bar diagram showing the comparison of platelet distribution width (PDW) with Modified Rankin Scale (mRS) scores.

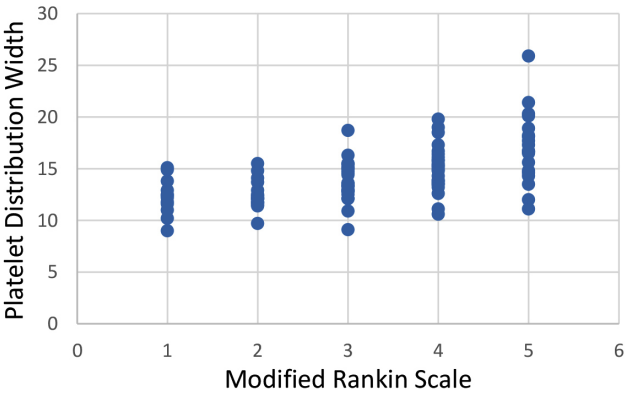

Figure 2: Scatter plot showing the correlation between platelet distribution width (PDW) and modified Rankin scale (mRS) scores.

PDW and outcomes of stroke

The primary outcome of the study was the functional status of patients at discharge or on Day 7 of admission, whichever occurred earlier, assessed using the mRS. An mRS score of 0–2 was categorized as a good outcome, whereas a score of 3–6 was considered a poor outcome. Secondary outcomes included the need for intensive

care unit (ICU) admission, in-hospital mortality, and duration of hospital stay.

The mean duration of hospitalization among stroke patients was 5.71 days (≈6 days). A prolonged hospital stay was defined as more than 6 days, which was observed in 43% of patients. Additionally, 31% of patients required ICU admission, and 12% succumbed during hospitalization.

At Day 7, the mRS score was re-evaluated and compared with the PDW values recorded at admission. A total of 36% of patients demonstrated a good functional outcome (mRS 0–2), while 64% had a poor outcome (mRS 3–6), including 12% who died. The mean PDW among patients with poor functional outcome at Day 7 was significantly higher than those with good outcome (15.35 ± 2.82 fL vs. 12.51 ± 1.47 fL; $p < 0.001$). Thus, higher PDW values at admission were strongly associated with poor short-term functional recovery.

The mean PDW among patients who required ICU admission was significantly higher compared to those who did not ($p = 0.001$). Similarly, patients with prolonged hospital stays (>6 days) had significantly higher PDW values than those with shorter stays (≤6 days) ($p < 0.001$). Among the 100 stroke patients studied, 12 deaths occurred during hospitalization. The mean PDW in non-survivors was 16.48 ± 2.04 fL, whereas in survivors it was 14.03 ± 2.75 fL ($p = 0.002$).

Higher PDW values were therefore consistently associated with longer hospital stays, increased need for ICU care, poorer functional outcomes, and greater in-hospital mortality.

Table 4: Relationship between platelet distribution width (PDW) and outcomes of acute ischemic stroke.

OUTCOME	*n(%)*	*PDW*	*P value*
Functional outcome (at Day 7)			
MRS< 2 (Good outcome)	36 (36%)	12.51 ± 1.47	< 0.001*
MRS ≥ 3 (Poor outcome)	64 (64%)	15.35 ± 2.82	
Hospital stay			
≤ 6 days	57 (57%)	13.39 ± 2.25	<0.001*
> 6 days	43 (43%)	15.57 ± 2.95	
ICU admissions			
Yes	31 (31%)	15.99 ± 3.29	0.001*
No	69 (69%)	13.58 ± 2.19	
Mortality			
Yes	12 (12%)	16.48 ± 2.04	0.002*
No	88 (88%)	14.03 ± 2.75	

Note: *significant

Stroke patients were categorized into four groups based on their PDW values at the time of admission. For each group, the mean duration of hospital stay was calculated and compared. It was observed that patients with higher PDW values at admission had a significantly longer duration of hospitalization. This association was statistically significant with a p value of 0.023, as presented in Table 5.

Table 5: Comparison of platelet distribution width (PDW) with length of hospital stay among patients with acute ischemic stroke.

PDW (fL)	*N(number)*	*Days of hospital stay (in days)*	*P-value*
≤12 fL	17(17%)	5.6	0.023*
12.1-14.0 fL	35(35%)	5.5	
14.1-16.0 fL	26(26%)	6.6	
>16 fL	22(22%)	7.4	

Note: *significant

Discussion

Stroke remains a major global health problem and is among the leading causes of death and disability worldwide. India is currently experiencing a stroke epidemic, with a rapidly increasing burden of disease. Recent advances in clinical laboratory techniques have opened new avenues for understanding the role of platelets in thrombosis, immunity, inflammation, and angiogenesis. PDW, a marker of platelet function, is a physiological variable of haemostatic significance and is being recognized as an emerging risk factor for atherothrombosis.

In the present study, the mean age of stroke cases was 63.25 ± 14.1 years, with the majority of cases (29%) in the 61–70-year age group. This finding is comparable with Indian studies by Patil et al. [13] (62.51 ± 12.72 years) and Ot et al. [14] (61.22 ± 11.9 years), but lower than that reported in Western studies such as O'Malley et al. [8] (79.5 ± 6.5 years) and Pikija et al. [15] (76 years). The disparity may be attributed to higher life expectancy in Western countries compared to developing nations like India. Stroke was more prevalent in males (66%) than in females (34%). Males are more prone to ischemic stroke due to a higher prevalence of risk factors such as smoking, hypertension, and diabetes mellitus, whereas genetic factors and the protective effects of estrogen on cerebral circulation may reduce stroke risk in females.

Hypertension (61%) was the most prevalent risk factor among stroke patients, followed by smoking (42%), both being significantly higher than in the control group. These findings are consistent with previous studies by Muscari et al. [16] (84.7%) and Ot et al. [14] (64%),

which also identified hypertension as the predominant risk factor. Arterial hypertension predisposes individuals to ischemic stroke by promoting atherosclerosis, while cigarette smoking reduces blood oxygen-carrying capacity, increases blood coagulability, triggers arterial spasm, and predisposes to thrombosis.

In the present study, PDW was significantly higher among hypertensives as compared to normotensives, in both the case and control groups. This observation aligns with findings from Lit et al. [17], who reported PDW as a potential risk factor for isolated systolic hypertension (ISH). The proposed mechanisms include platelet activation secondary to sympathetic and renin–angiotensin system stimulation, shear stress, oxidative stress, altered calcium signalling, endothelial dysfunction, and reduced nitric oxide bioavailability. Elevated PDW may reflect an increased proportion of large, reticulated platelets that are more reactive and release greater amounts of thromboxane A2, while expressing more glycoprotein IIb/IIIa receptors crucial for coagulation. Thus, variations in platelet size as reflected by PDW may indicate hypertensive status and predict thrombotic risk. However, in this study, no significant correlation was found between PDW and other risk factors such as age, gender, diabetes mellitus, dyslipidaemia, smoking, or alcohol intake.

PDW values were found to be significantly higher among stroke patients as compared to controls (14.33 ± 2.78 fL vs 12.87 ± 1.86 fL; $p = 0.000021$). PDW thus had an independent association with acute ischemic stroke, which was consistent with study done by Shah et al. [18]. A strong correlation was also observed between PDW and stroke severity (based on the Modified Rankin Scale), with higher PDW values being associated with more severe disability. This supports the prognostic role of PDW in stroke outcomes, consistent with studies by Sarkar et al. [19], Sundari et al. [20], and Lyu et al. [21].

Higher PDW values at admission were associated with poor functional outcomes (MRS 3–6) at day 7, confirming an inverse relationship between PDW and recovery. Similar findings have been reported by Shahet al. [18], Xie et al. [22], and Li et al. [23], who demonstrated that elevated PDW at presentation independently predicted poor functional outcome at 1 week and 3 months post-stroke. Additionally, higher PDW values correlated significantly with secondary outcomes such as prolonged hospital stay, increased need for ICU admission, and higher in-hospital mortality. This indicates that elevated PDW at admission may serve as a marker of poor prognosis and increased morbidity and mortality in stroke patients.

The exact mechanism linking elevated PDW with cerebrovascular accidents remains incompletely understood, but several explanations have been proposed. Platelet activity increases in ischemic stroke, as evidenced by elevated soluble platelet P-selectin and thromboxane A2 levels, both of which are atherogenic factors. Secondly, cytokines such as interleukin-3 and interleukin-6, which play key roles in ischemic stroke pathophysiology, also influence megakaryocyte ploidy, leading to the production of larger, more reactive platelets and consequently elevated PDW. This proinflammatory state may predispose individuals to a prothrombotic condition. Thirdly, larger platelets may also result from altered secretion and metabolism of biologically active substances during aging, diabetes, hypertension, and obesity, which further increase the risk of ischemic stroke. The findings of this study suggest that larger, heterogeneous platelets (reflected by higher PDW) contribute to the pathogenesis of cerebral thrombosis.

One could argue that increased PDW and platelet reactivity are simply markers for a more severe stroke and a more pronounced acute-phase reaction. This study included patients who presented within 48 hours of the onset of their stroke, and a sample for PDW was taken immediately upon admission. Because platelets have a life span of 8–10 days, the increased PDW at admission suggests that the increase must have occurred prior to the stroke. Over 90% of the platelet population whose distribution was assessed following a stroke, was already in circulation prior to the vascular occlusion. These observations, thus likely represent changes occurring during thrombopoiesis, where increased platelet size may precede the thrombotic event rather than result from it. Overall, our findings support the hypothesis that patients who later develop more severe strokes already have higher PDW levels, reflecting increased platelet reactivity prior to the ischemic event.

Recent evidence indicates that elevated PDW may reflect heightened platelet reactivity and a pro-thrombotic state that contributes to worse outcomes after acute ischemic stroke. A 2022 meta-analysis by Zheng et al. found limited predictive value of PDW alone [24]. However, a 2023 Mendelian-randomisation study by Li et al. suggested a potential causal association between platelet indices and stroke risk [25]. Most recently in 2024, Shen et al. demonstrated a statistically significant causal link between genetically determined PDW and poor 3-month functional outcome after stroke (OR 1.48) [26]. Together, these findings strengthen the rationale for PDW as a cost-effective, early prognostic biomarker in stroke.

Limitations: This single-centre study with a relatively small sample size and short duration may limit the

generalizability of results. Controls were matched only for age and sex, not for other vascular risk factors. A larger, multicentric study including diverse populations and comprehensive risk factor matching would better validate the prognostic role of PDW in stroke.

Conclusion

The present study demonstrates that elevated Platelet Distribution Width (PDW) is independently associated with acute ischemic stroke, reflecting platelet activation and its role in cerebral thrombosis. Higher PDW values also correlate with greater stroke severity, longer hospital stays, higher ICU admission rates, and increased mortality. As a simple, cost-effective, and readily available haematological parameter, PDW may serve as a valuable early prognostic biomarker in acute ischemic stroke. Larger multicentric studies with diverse populations are warranted to further validate its predictive accuracy and clinical utility in stroke management and prevention.

Conflicts of interest

Authors declare no conflict of interest.

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