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Comparative Analysis of Platelet Parameters and Serum Thrombopoietin Levels in Patients with Acute Coronary Syndrome and Acute Ischemic Stroke

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Abstract: **Background:** Platelet activity has an important role in the thrombotic mechanisms that occur following acute coronary syndrome (ACS) and acute ischemic stroke (AIS). Platelet size and activation status are inferred from mean platelet volume (MPV) and platelet distribution width (PDW), respectively, while platelet production via megakaryopoiesis involves the modulation by thrombopoietin (TPO). **Objective:** The purpose of this study was to evaluate the diagnostic value of serum TPO, platelet count, MPV and PDW in distinguishing AIS patients and ACS patients from normal subjects. **Methods:** A single center case-control study was performed on 25 AIS patients, 25 ACS patients and 20 healthy controls. Platelet indices and serum TPO was tested, followed by multivariable regression with adjustment for covariates (age, gender, hypertension, diabetes and smoking). The discriminative value of MPV, PDW and TPO were analyzed by receiver operating characteristic (ROC) curves. **Results:** MPV and PDW were significantly increased in both AIS and ACS than those of the control group ($P < 0.01$). Levels of serum TPO were significantly higher in AIS (129.0 ± 88.2 pg/mL) and ASC (179.4 ± 197.3 pg/mL) than in the controls (33.7 ± 7.9 pg/mL). Cases also had higher platelet counts. Receiver operating characteristic (ROC) analysis revealed that MPV had the greatest discriminatory power for AIS (area under the curve [AUC]: 0.834), and PDW was best for ACS (AUC: 0.783). **Conclusion:** The remarkable increase of MPV, PDW, and TPO in AIS and ACS provides evidences for them as promising diagnostic biomarkers that showed the increased platelet activation status and thrombopoietic activity. Larger quality-controlled multicenter studies are needed to confirm these findings and to investigate their prognosis implications.

Keywords: Acute ischemic stroke, acute coronary syndrome, Platelets, Mean Platelet Volume, thrombopoietin, Thrombosis.

INTRODUCTION

Acute coronary syndrome (ACS) that encompasses unstable angina, non-ST segment elevation myocardial infarction (NSTEMI), and ST-segment elevation MI (STEMI) develops due to sudden thrombotic occlusion of the coronaries [Abbott, A. L. *et al*., 2017; Al-Obeidi, S. R. H. *et al*., 2013].; Acute coronary syndrome (ACS), which encompasses unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI), develops as a result of sudden thrombotic occlusion of the coronary arteries [Abbott, A. L. *et al*., 2017; Al-Obeidi, S. R. H. *et al*., 2013]. Acute ischemic stroke (AIS), which mainly originates from partial or whole obstruction of the cerebral circulation, accounts for about 80% of all strokes [Arévalo-Lorido, J. C. *et al*., 2012]. Atherosclerosis, a systemic arterial disease is the basic pathophysiology of both heart and brain ischemic disease might cause either cardiac or cerebral ischemic events [Balcik, Ö. S. *et al*., 2013]. Atherosclerosis is a systemic chronic inflammatory process that causes thickening of arterial walls and can lead to the vessel's vulnerability for plaque development, luminal narrowing, or thrombotic occlusion disease [Boos, C. J. *et al*., 2008]. TPO also binds to c-Mpl receptors on megakaryocytes, promoting proliferation and maturation with resultant increase in platelet production. In the setting of acute atherothrombotic events, this process is significantly amplified by inflammatory cytokines (most notably IL-6), leading to generation of enriched and more active platelets. This inflammatory-mediated thrombopoiesis is responsible for the simultaneous increase of TPO, MPV and PDW in both AIS and ACS population [Bröijersén, A. *et al*., 1998; Vasse, M. *et al*., 2012; Chu, S. G. *et al*., 2010; Cohen-Solal, K. *et al*., 1996]. Platelets, larger in size, are more highly metabolically active and correspondingly are main players in vascular injury responses [D'erasmo, E. *et al*., 1990]. The mean platelet volume is elevated in hypertensive and diabetic patients; predisposing the later to vascular pathology [Dogan, N. O. *et al*., 2013]. Large platelets, characterized by an increased MPV, are metabolically and enzymatically more active and contain more prothrombotic factors, and therefore have higher aggregability. Although MPV has been identified as a potentially useful inflammatory marker with negative prognostic implications concerning

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cardiovascular outcome, its exact relative role in the early stages of ACS and AIS continues to be investigated [Ferrari, R. *et al*., 1990]. MPV has been reported to be associated with several cardiovascular risk states; its role as an additional marker in ACS and AIS is still uncertain. The authors goal was to compare TPO, platelet count, MPV and PDW between AIS, ACS and controls and particularly to compare the differences of their levels in patients with ACS and patients with AIS from identical center during identical period in order to illustrate common or different characteristics related systemic atherothrombosis [Galan, A. M. *et al*., 2011].

METHODS

Design and setting: A single-centre case control study in Baghdad Teaching Hospital (January 15 – April 15, 2017). The study was approved by the ethics committee; oral consent of the participants was obtained.

Participants

All patients (n=25) with AIS (imaging confirmed, aged >50 years) and ACS (chest pain >30 min, age >50 years, ECG changes and/or cardiac troponin I elevation [>0.1 ng/mL]) were included before intervention. Controls were 20 healthy volunteers of the same age and sex. Exclusions were hemorrhagic stroke, transient ischemic attack, head injury, admitted with AIS >24 hours from symptom onset arrival and bleeding diathesis/myeloproliferative disease or previous anticoagulant/antiplatelet use.

Measurements

Venous blood (3 mL) was drawn into K3-EDTA tubes for the full CBC count (including platelet count, MPV, PDW, Neutron and Hemoglobin), by a Sysmex automated haematology analyser (Japan) within 2 hours. Pre-analytical conditions were standardized. Serum TPO was assayed by sandwich ELISA (Quantikine Human TPO kit); samples were tested in duplicate and read at 450 nm (with correction at 540 nm).

Statistical Analysis

Statistical analysis was undertaken using SPSS software (version 17.0; Chicago, IL, USA). Normality was tested using Shapiro-Wilk test and Q-Q plots. Groups were compared using ANOVA or Kruskal–Wallis test. Multivariable linear regression models were used to control for age, sex, hypertension, diabetes and smoking. For TPO, the analyses were performed for log10-transformed values as a result of skewness. Receiver Operating Characteristic (ROC) analyses were conducted to estimate the discriminatory power of MPV, PDW and TPO with area under the curve (AUC), 95% CI sensitivity/specificity and the cut-off providing the greatest sum of sensitivity and specificity on Youden's index. $P < 0.05$ (two-sided) were considered significant. Post-hoc power analysis revealed approximately 80% power in detecting MPV variations.

RESULTS

The mean age was 67.16 ± 9.46 years for AIS; 59.48 ± 6.07 years for ACS; and 61.10 ± 5.88 for controls. AIS was older than controls ($P = 0.008$). Baseline participant characteristics are summarized in Table 1. While rates of diabetes and smoking were similar across groups, the prevalence of hypertension was significantly higher in the patient cohorts compared to controls ($P = 0.024$). Neutrophil counts were notably higher in AIS (7.9 ± 3.0 x10⁹/l; $P < 0.001$ vs controls 4.4 ± 1.1 x10⁹/l) and consistent with acute-phase responses (Table 2). Hemoglobin levels were similar across groups ($P > 0.05$).

Table 1: Participant characteristics

Risk Factors	Group	Yes (%)	No (%)	P -Value
Diabetes	Stroke	54	46	0.778
Diabetes	ACS	46	54	0.778
Hypertension	Stroke	60	40	0.024
Hypertension	ACS	40	60	0.024
Smoking	Stroke	46	54	0.778
Smoking	ACS	54	46	0.778

Note: Hypertension was significantly more prevalent among patient groups compared with controls ($P = 0.024$)

Whereas the ischaemic stroke group had 13.5±2.1 and the ACS group had 13.4±1.6, the mean haemoglobin level in the control group was 13.7±1.3 g/dl. Hemoglobin was similar across groups. With P=0.005 and P=0.001, the mean neutrophil count in the control group was 4.4 ± 1.1

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× 10^9/l; the ischaemic stroke and ACS groups had notably higher levels (7.9 ± 3.0 and 7.7 ± 3.9 × 10^9/l, respectively). Neutrophil counts were higher in AIS and ACS versus controls (P < 0.01), consistent with acute-phase responses. As shown in Table 2.

Table 2: Comparison of Mean Hemoglobin level and neutrophil count.

Parameter	Control (N=20) Mean ± SD (Min-Max)	ACS (N=25) Mean ± SD (Min-Max)	P (ACS vs Control)	Stroke (N=25) Mean ± SD (Min-Max)	P (Stroke vs Control)
Neutrophil (×10^9/L)	4.4 ± 1.1 (2.9-6.3)	7.7 ± 3.9 (3.5-19.4)	0.001	7.9 ± 3.0 (2.4-14.4)	0.005
Hemoglobin (g/dL)	13.7 ± 1.3 (11.6-16.7)	13.4 ± 1.6 (9.0-15.4)	0.486	13.5 ± 2.1 (8.4-17.3)	0.612

Platelet counts are controls 208 ± 59 ×10^9/L; AIS 246 ± 69 ×10^9/L; ACS 300 ± 76 ×10^9/L. Higher counts in AIS (P = 0.007) and ACS (P = 0.05) versus controls. As detailed in Table 3, both patient cohorts demonstrated significantly elevated platelet counts compared to controls (ACS: 300 ± 76 vs. 208 ± 59 ×10⁹/L, P=0.05; AIS: 246 ± 69 vs. 208 ± 59 ×10⁹/L, P=0.007). No significant differences in these parameters were observed by gender, diabetes, hypertension, or smoking (Tables 4–7). The mean MPV level in the control group was 6.3 ± 0.7 fl, while in the ischemic stroke group the mean level was 8.6 ± 0.7 fl, and ACS was 8.0 ± 0.6 fl. The increase of MPV in ischemic stroke and ACS groups was significantly different from control ((P < 0.01 vs controls). PDW level in the control group was 16.0 ± 0.4 fl, while in the ischemic stroke group the mean level was 17.4 ± 0.9 fl, and in ACS was 17.6 ± 0.5 fl. The PDW in ischemic stroke and ACS groups was significantly different from control (P < 0.01 vs controls) (Table 3). TPO controls 33.7 ± 7.9 pg/mL; AIS 129.0 ± 88.2 pg/mL (P = 0.017); ACS 179.4 ± 197.3 pg/mL (P = 0.005).There was insignificant difference in MPV and PDW according to gender, DM, HT, and smoking (P>0.05) in both groups (Tables 6 and 7).

Table 3: Comparison Analysis of TPO, Platelet Count, MPV, and PDW (Unadjusted) vs. Controls. Comparison of hematological parameters between acute coronary syndrome (ACS), acute ischemic stroke (AIS), and healthy controls. Data are presented as mean ± standard deviation (SD) with minimum-maximum (Min-Max) ranges. Statistical significance was determined using ANOVA with P < 0.05 considered statistically significant.

Parameter	Control Mean ± SD (Min-Max)	ACS Mean ± SD (Min-Max)	P -Value (ACS vs Control)	AIS Mean ± SD (Min-Max)	P-Value (AIS vs Control)
TPO (pg/ml)	33.7 ± 7.9 (21.4-46.9)	179.4 ± 197.3 (43.9-1023.0)	0.005 **	129.0 ± 88.2 (51.0-378.0)	0.017 *
Platelets count (×10⁹/L)	208 ± 59 (180-301)	300 ± 76 (170-454)	0.05 *	246 ± 69 (137-397.0)	0.007 **
MPV (fl)	6.3 ± 0.7 (5.4-8.4)	8.0 ± 0.6 (6.5-9.3)	0.005 **	8.6 ± 0.7 (7.2-10.0)	0.007 **
PDW (fl)	16.0 ± 0.4 (15.3-16.5)	17.6 ± 0.5 (16.7-18.9)	0.005 **	17.4 ± 0.9 (15.4-19.5)	0.005 **

Notes: Data were analyzed using one-way analysis of variance (ANOVA). Asterisks denote statistical significance levels: P < 0.05 (significant), P < 0.01 (highly significant). Both ACS and AIS patient groups demonstrated significantly elevated thrombopoietin levels, platelet counts, mean platelet volume, and platelet distribution width compared to healthy controls. The most pronounced elevations were observed in mean platelet volume (AIS: 36.5% increase; ACS: 27.0% increase relative to controls) and thrombopoietin (AIS: 282.8% increase; ACS: 432.3% increase relative to controls).

Table 4: Stratified Analysis of Thrombopoietin and Platelet Count by Risk Factors in Acute Ischemic Stroke Patients Subgroup analysis of hematological parameters in acute ischemic stroke patients (n = 25) stratified by presence or absence of diabetes mellitus (DM), hypertension (HT), and smoking status. Data are presented as mean ± SD with range (Min-Max). P values were calculated using independent samples t-tests

Risk Factor	N	TPO Mean ± SD (Min-Max)	Platelets count Mean ± SD	P Value (TPO)	P Value (Platelets)

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			(Min-Max)		
Diabetes Mellitus (Yes)	14	120.6 ± 101.0 (51.0-378.0)	259 ± 69 (167-397)	0.603**(NS)**	0.321**(NS**)
Diabetes Mellitus (No)	11	139.6 ± 72.2 (53.7-332.8)	230 ± 70 (137-349)	0.603	0.321
Hypertension (Yes)	22	121.4 ± 82.0 (51.0-378.0)	249 ± 71 (137-397)	0.253**(NS)**	0.522**(NS)**
Hypertension (No)	3	184.6 ± 132.6 (77.0-332.8)	221 ± 63 (167-290)	0.253	0.522
Smoking Status (Yes)	12	101.8 ± 34.1 (57.9-144.0)	248 ± 71 (137-358)	0.142(NS)	0.876(**NS**)
Smoking Status (No)	13	154.1 ± 114.4 (51.0-378.0)	244 ± 71 (161-397)	0.142	0.876

Notes: NS = not statistically significant (P > 0.05). No significant differences in thrombopoietin or platelet count were observed when stratified by diabetes mellitus, hypertension, or smoking status in AIS patients.

Table 5: Stratified Analysis of Thrombopoietin and Platelet Count by Risk Factors in Acute Coronary Syndrome Patients**.**

Risk Factor	N	TPO Mean ± SD (Min-Max)	Platelets count Mean ± SD (Min-Max)	P Value (TPO)	P Value (Platelets)
Gender (Male)	15	150.3 ± 95.8 (43.9-325.0)	295 ± 85 (170-454)	0.378(NS)	0.653(NS)
Gender (Female)	10	223.0 ± 293.2 (53.7-1023.0)	309 ± 65 (200-362)	0.378	0.653
Diabetes Mellitus (Yes)	12	240.0 ± 265.1 (43.9-1023.0)	300 ± 99 (170-454)	0.143(NS)	0.983(NS)
Diabetes Mellitus (No)	13	123.4 ± 79.6 (52.3-325.0)	301 ± 52 (190-362)	0.143	0.983
Hypertension (Yes)	15	197.4 ± 247.9 (43.9-1023.0)	293 ± 81 (190-454)	0.587(NS)	0.561(NS)
Hypertension (No)	10	152.3 ± 82.8 (52.3-325.0)	312 ± 71 (170-424)	0.587	0.561
Smoking Status (Yes)	14	140.5 ± 81.1 (46.7-355.2)	293 ± 92 (170-454)	0.276(NS)	0.575(NS)
Smoking Status (No)	11	228.8 ± 282.9 (43.9-1023.0)	310 ± 53 (192-362)	0.276	0.575

Subgroup analysis of hematological parameters in acute coronary syndrome patients (n = 25) stratified by gender, diabetes mellitus (DM), hypertension (HT), and smoking status. Data are presented as mean ± SD with range (Min-Max). P values were calculated using independent samples t-tests.

Notes: NS = not statistically significant (P > 0.05). No significant differences in thrombopoietin or platelet count were identified across gender, diabetes mellitus, hypertension, or smoking status in ACS patients.

Table 6: Stratified Analysis of Mean Platelet Volume and Platelet Distribution Width by Risk Factors in Acute Ischemic Stroke Patients

Risk Factor	N	MPV Mean ± SD (Min-Max)	PDW Mean ± SD (Min-Max)	P Value (MPV)	P Value (PDW)
Gender (Male)	12	8.7 ± 0.7 (7.4-10.0)	17.5 ± 0.8 (16.4-18.9)	0.629(NS)	0.486(NS)
Gender (Female)	13	8.5 ± 0.7 (7.2-9.7)	17.3 ± 1.1 (15.4-19.5)	0.629	0.486
Diabetes Mellitus (Yes)	14	8.8 ± 0.6 (8.0-9.7)	17.6 ± 0.9 (16.3-19.5)	0.058(NS)	0.162(NS)
Diabetes Mellitus (No)	11	8.3 ± 0.8 (7.2-10.0)	17.1 ± 0.9 (15.4-18.9)	0.058	0.162

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Hypertension (Yes)	22	8.6 ± 0.7 (7.2-10.0)	17.4 ± 1.0 (15.4-19.5)	0.952(NS)	0.955(NS)
Hypertension (No)	3	8.6 ± 0.9 (7.6-9.1)	17.4 ± 0.4 (17.1-17.8)	0.952	0.955
Smoking Status (Yes)	12	8.7 ± 0.6 (8.0-10.0)	17.6 ± 0.7 (16.4-18.9)	0.517(NS)	0.278(NS)
Smoking Status (No)	13	8.5 ± 0.8 (7.2-9.7)	17.2 ± 1.1 (15.4-19.5)	0.517	0.278

Subgroup analysis of platelet indices in acute ischemic stroke patients (n = 25) stratified by gender, diabetes mellitus (DM), hypertension (HT), and smoking status. Data are presented as mean ± SD with range (Min-Max). P values were calculated using independent samples t-tests.

Notes: NS = not statistically significant (P > 0.05); MPV = mean platelet volume; PDW = platelet distribution width. Notably, no significant differences in mean platelet volume or platelet distribution width were observed when stratified by gender, diabetes mellitus, hypertension, or smoking status in AIS patients.

Table 7: Stratified Analysis of Mean Platelet Volume and Platelet Distribution Width by Risk Factors in Acute Coronary Syndrome Patients

Risk Factor	N	MPV Mean ± SD (Min-Max)	PDW Mean ± SD (Min-Max)	P Value (MPV)	P Value (PDW)
Gender (Male)	15	8.0 ± 0.8 (6.5-9.3)	17.5 ± 0.4 (16.7-17.9)	0.514 (NS)	0.52 (NS)
Gender (Female)	10	8.2 ± 0.4 (7.6-8.9)	17.7 ± 0.6 (16.9-18.9)	0.514	0.52
Diabetes Mellitus (Yes)	12	8.0 ± 0.5 (6.7-8.9)	17.6 ± 0.6 (16.7-18.9)	0.535 (NS)	0.614 (NS)
Diabetes Mellitus (No)	13	8.1 ± 0.7 (6.5-9.3)	17.5 ± 0.4 (17.0-18.1)	0.535	0.614
Hypertension (Yes)	15	8.2 ± 0.5 (7.6-9.3)	17.7 ± 0.5 (16.9-18.9)	0.203 (NS)	0.055 (NS)
Hypertension (No)	10	7.8 ± 0.8 (6.5-9.1)	17.4 ± 0.5 (16.7-17.9)	0.203	0.055
Smoking Status (Yes)	14	8.1 ± 0.8 (6.5-9.3)	17.5 ± 0.6 (16.7-18.9)	0.911 (NS)	0.568 (NS)
Smoking Status (No)	11	8.0 ± 0.4 (7.6-9.1)	17.6 ± 0.3 (17.0-18.1)	0.911	0.568

Subgroup analysis of platelet indices in acute coronary syndrome patients (n = 25) stratified by gender, diabetes mellitus (DM), hypertension (HT), and smoking status. Data are presented as mean ± SD with range (Min-Max). P values were calculated using independent samples t-tests.

Note: All P-values exceed the conventional significance threshold (P < 0.05), indicating no statistically significant differences in platelet parameters across risk factor subgroups. The hypertension–PDW relationship (P = 0.055) represents a borderline non-significant finding deserving cautious interpretation and future validation

DISCUSSION

Platelets exert an important role in the pathogenesis of AIS and ACS [Gasparyan, A. Y. *et al*., 2019]. The main cytokine that controls megakaryopoiesis and platelet production is TPO [Giles, H. *et al*., 1994]. High MPV is reported in conditions of enhanced platelet turnover e.g. Bernard-Seulier syndrome and inherited platelet disorders [Gongora-Rivera, F. *et al*., 2007]. There was no significant change in hemoglobin between groups, whereas neutrophil count increased, indicating an acute inflammatory response induced by catecholamines and cortisol. [Grech, E. D. *et al*., 2003; Ha, S. I. *et al*., 2011; He, S. *et al*., 2024]. In our investigation patients in the AIS and ACS groups had higher platelet counts. This is in contrast with cases of infarcted-related consumption thrombocytopenia, yet it supports the view that systemic inflammation in the context of acute atherosubthrombotic events can lead to reactive thrombocytosis [Kaushansky, K. 2006]. This inflammatogenic bloom could result in fresh blood release of younger, larger and more reactive platelets as indexed by the concurrently increased MPV and PDW [20]. MPV was markedly

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increased in the ischemic stroke group, as previously reported [Galan, A. M. *et al*., 2011; Kuter, D. J., & Gernsheimer, T. B. 2009]. Bigger platelets are functionally and enzymatically more active corresponding to significantly higher MPV, they also carry higher number of prothrombotic materials and showing greater aggregability [Long, M.W. *et al*., 2000]. This prothrombotic phenotype may promote further vascular occlusion. Although some studies did not find a significant association between MPV and ischemic stroke [Mackman, N. 2008], reduction in MPV was reported as a result of selective loss of larger, more active platelets in others [artin, J. F. *et al*., 1991]. A study by Balcik *et al*. [Galan, A. M. *et al*., 2011] demonstrated significantly increased MPV in ischemic stroke. Arevalo *et al*. 776 reported a relation between high MPV and severity of carotid atherosclerosis. In the present study, we noticed a significantly high PDW in both patient groups. PDW, an index of platelet volume heterogeneity, is currently regarded by some as a more specific parameter of reactive de-novo thrombopoiesis than MPV [Massberg, S. *et al*., 2002]. The PDW was the most potent predictor for ACS in our ROC curve analysis, and it is a critical part of an activated platelet population. Platelet count was significantly greater in the ACS group compared to controls as previously described [Mithra, M. *et al*., 2017]. In some studies there were no significant elevations [Grech, E. D. *et al*., 2003], whereas other reported a decreasing because of the consumption after myocardial infarction (MI) [McCabe, D. J. *et al*., 2004]. Patients Presented with ACS had significantly higher MPV than controls [Grech, E. D. *et al*., 2003]. The increased MPV in ACS may be due to either swelling of platelets, longer life span or enhanced activity of mega-karyocytes [Nadar, S. K. *et al*., 2004]. Chu *et al*. [Pathansali, R. *et al*., 2001] observed an elevated MPV in MI patients and restenosis following angioplasty. Al-Obeidi *et al*. [Renoy, A. 2009] in ACS patients, and this was more remarkable in unstable angina and MI. Urs *et al*. [Ross, R. 1993] but also higher MPV and PDW levels in ACS, specifically in STEMI individuals.

The increased TPO concentrations in both patients groups are evidence for a direct connection between inflammation and platelet elevation [Şenaran, H. *et al*., 2001; Threatte, G. A. *et al*., 1984]. Thrombopoietin (TPO) is the most important megakaryopoiesis-regulating cytokine, and its rise indicates a very strongly expressed systemic wish to generate more platelets. This may be a response to the increased platelet turnover and removal in the narrowed, turbulent arteries of atherosclerosis. Results are in agreement with Balcik *et al*., indicating a hypersensitive state of megakaryopoiesis in acute thrombotic disorders [Bellucci, S. *et al*., 1996; Yetkin, E. 2008]. These figures help to be tension in the narrative from my findings. Mean and standard deviation are shown in figure 1 for each age, TPO, platelet count MPV, and PDW across groups with their tendencies towards an increase in patient groups (MPV is higher for AIS) Figure 1. Figure 2 The ROC curves for AIS, the goodness of diagnostic accuracy was demonstrated in MPV (AUC=0.834), and an appropriate cutoff point for prediction of 8.15 fL (with sensitivity 80%and specificity 77.8%), indicating potential as a biomarker. Figure 3 presents ROC curves of ACS, among which PDW is found to be the best predictor (AUC=0.783) at the value of 17.3 fL (sensitivity 72%, specificity 68.9%), with possible clinical application for risk stratification. The distributions of risk factor in Figure 4 showed that hypertension was significantly more prevalent (P=0.024) among groups, which may confound platelet parameters. Sensitivity-specificity trade-offs at optimal thresholds showing balanced diagnostic measurements at Figure 5 are provided. Figure 6 compares means showing older age of AIS patients (P=0.001–0.008) and different hematological profile arguing age as a covariate in multivariable models. Taken together, these data support the results of our study but are restricted due to small case numbers and a case-control design that does not permit drawing conclusions on causality. Longitudinal designs and larger sample sizes should be included in any further research. Our study also has a number of limitations, one being the relatively small size and single-center nature of the study which may restrict generalizability. An obvious limitation of the cross-sectional design is that no causal relations can be implied.

CONCLUSION

In summary, the present respective study shows that AIS and ACS patients have the same hematological profile consisting of increased MPV, PDW, TPO levels reflecting general-inflammation and increased but mitochondrial-thrombopoiesis activation. Conclusions: These potentially easily accessible and low-cost biomarkers, MPV in AIS and PDW in ACS especially, are very promising as diagnostic

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adjuncts. In future studies (preferentially large, multi-center prospective trials), these diagnostic thresholds for diagnosis of ACS should be validated. Moreover, exploration of the prognostic significance of these markers in predicting short- and long-term outcomes, such as stroke recurrence or MACE, is an essential direction to be further pursued.

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