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Original research article

Analyzing mean platelet volume and its relationship to the occurrence of acute ischemic stroke

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Abstract

Background and objective: The purpose of the study was to investigate if there is a significant association with both MPV and ischemic stroke incidence, as well as if there is a significant association among stroke severity and mean platelet volume. The goal of this study is to determine if MPV is linked to the occurrence of ischemic stroke. The goal of this study is to determine if there is a statistically significant correlation between subtypes of stroke and MPV. The purpose of this study is to examine whether or not MPV is associated with the severity of ischemic stroke.

Methods: From August 2021 to July 2022, at Department of General Medicine, Siddartha Medical College, Vijaywada, Andhra Pradesh, India, conducted a Case Control study. Fifty acute ischemic stroke patients who met the criteria presented within 48 hours of symptom onset. The Modified Rankin's scale assessed stroke severity. Automated analyzers measured mean platelet volume in EDTA and Citrate samples. MPV was assessed in 50 age- and gender-matched controls.

Results: Acute ischemic stroke had higher mean platelet volume. With p = 0.005, mean platelet volume (MPV) is associated with ischemic stroke, with individuals experiencing 7.35+/-0.81 MPV and controls 6.94+/-0.59. MPV (EDTA) is 7.86+/-0.82 in cases and 7.58+/-0.70 in controls, but the variability is small (p value =0.074). Cases have a mean 2.56+/-0.58 (1.43-4.40) platelet count, while controls have 2.69+/-0.83 (1.60-5.40). This difference is not statistically significant (p value =0.380). Platelet mass, calculated from platelet count and MPV, is stable. Clinical stroke severity was not associated with MPV in this study (p value =0.281).

Conclusion: This study found higher MPV in acute ischemic stroke. A rise in MPV independently predicts ischemic stroke after multivariate regression analysis. The findings suggest thrombopoiesis changes cause cerebral thrombosis with larger platelets. Platelet volume's role in stroke pathogenesis and outcome, especially in stroke-risk patients, needs further study.

Keywords: Ischemic stroke, platelets, Mean platelet volume

Introduction

Stroke caused by ischemia occurs when a thrombus blocks an artery that has been damaged by atherosclerosis. The platelets in the blood have a major part in the development and pathophysiology of both atherosclerosis and thrombosis. Ischemic stroke, a type of cerebro-vascular disease, is a major public health concern in India and elsewhere. Asian and middle/eastern European countries have notably higher stroke rates. The increased prevalence of cerebrovascular stroke as a result of increased life expectancy in developing and less developed countries is a major public health concern worldwide [1, 2, 3]. In India, the prevalence of stroke is estimated to be around 470 per 1,00,000 people, and cerebrovascular stroke is responsible for 3.9% of all hospital admissions, 4.5% of all medical cases, and roughly 33% of all neurological cases. Stroke of the brain or brain stem is the second leading cause of death after cardiovascular disease. The effects of a cerebro-vascular stroke on a patient's abilities and prospects for future employment or reintegration into the family dynamic are often devastating. Because of this, stroke may be viewed as a family illness. Resulting Stroke-related disabilities typically necessitate the involvement of a spouse or other family member, either temporarily or permanently [3, 4]. After dementia, cerebrovascular stroke is the leading cause of neurological disability and prolonged institutionalization. The cognitive impairments caused by degenerative neurological disorders are made worse by recurrent attacks of cerebro-vascular stroke, which in turn leads to dementia. As a result, cerebral infarction and its aftereffects are major concerns in the realm of healthcare policy and planning. Prevention of cerebro-vascular stroke will be a worthwhile cost-efficient strategy due to the high cost of treatment and the severe economic consequences of lost efficiency and productivity [4, 5].

A cerebrovascular stroke is characterized by a sudden onset of neurological symptoms and/or signs of focal, and/or global loss of cerebral function, which persist for more than 24 hours, result in disability, or cause death, and have no other obvious cause. Consequently, the diagnosis and definition of a

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cerebrovascular stroke depend on clinical evidence. Due to the complexity of the brain and its vasculature, the clinical manifestations of a stroke can be very different from one patient to the next. Larger platelets are more likely to aggregate, to be highly reactive, and to generate a greater quantity of pro thrombotic factors. Platelet volume may affect thrombotic blood vessel occlusion, as suggested by the observation of large platelets and altered platelet size parameters in patients with ischemic cerebro-vascular stroke. As far as we could tell, there are only a small number of published studies in India comparing MPV and the risk of ischemic cerebrovascular stroke. Consequently, the goal of this research is to determine whether or not MPV is linked to ischemic cerebro-vascular stroke [7, 8].

Material and Methods

This study looked back at data collected by Department of General Medicine, Siddartha Medical College, Vijaywada, Andhra Pradesh, India, between August 2021 to July 2022. Fifty patients with acute ischemic cerebro-vascular stroke who presented to the hospital within 48 hours of symptom onset were included in the study. Fifty controls of the same age and sex as the cases were also enrolled.

1. Stroke is defined as a neurological deficit in one area of the brain that lasts for more than 24 hours and for which no other cause can be determined.
2. Adult men and women over the age of 18 were enlisted for the study.
3. There was no evidence of economic bias in the selection of cases and controls.

Exclusion criteria

1. Thrombocytopenic patients.
2. History of Known Inherited Platelet Disorders
3. Medications that alter platelet parameters, including hydroxyurea, anticancer drugs, and drugs that inhibit platelet surface proteins like integrin IIb3.
4. Patients presented with symptoms of hemorrhagic stroke.
5. Patients who are unable to respond because they suffered a severe stroke, or who initially presented with aphasia and/or delirium.
6. Patients who seek care more than 48 hours after the onset of symptoms. Peripheral blood smears demonstrating platelet clumps in 7 patients
7. Cases and controls were matched based on age (+/- 5 years) and gender.

Inclusion criteria

1. A close friend or family member of a patient currently staying in any hospital unit.
2. Any Patient's Unrelated Visitors
3. Patients who visit the ER or OPD for reasons unrelated to their initial medical problem.

Exclusion criteria

People who have had a previous stroke are

1. In this category.
2. People whose platelet counts are abnormal
3. Clumps of platelets can be seen on a peripheral smear.

All patients who presented to the hospital with a diagnosis of stroke during the aforementioned time frame were analyzed. Every patient who suffered a stroke was meticulously documented. Patients meeting the study's criteria were recruited after informed consent was obtained. The information was recorded and collected in accordance with the template. Upon enrolling in the study, each participant was given a special identification number. The severity of each patient's condition was measured using a modified Rankin's Scale. EDTA and citrate vacutainers were used to store the blood that was taken from the antecubital vein. After transporting the samples to the lab, we used an Automated ABX pentra analyzer to measure the mean platelet volume by means of electrical impedance, somewhere between 2 and 4 hours after collection. When the analysis was complete, the same sample was sent to the for a peripheral smear to look for platelet aggregates. Patients were withdrawn from studies where platelet aggregates were found. The same method was used to collect samples for the Controls, which were then placed in vacutainers (containing EDTA and citrate) before being run through an automatic analyzer (ABX pentra). Participants were disqualified from the study if their peripheral smear showed the presence of aggregates.

Results

Demographic data

A total of 230 patients of ischemic cerebro-vascular stroke were sifted to obtain fifty cases.

Table 1: Stroke Log

Total patients screened	230
Patients recruited	50
patients barred	180

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Table 2: Age distribution cases and controls

Age in years	Cases		Controls	
	No	%	No	%
21-30	2	4.0	2	4.0
31-40	4	8.0	4	8.0
41-50	6	12.0	6	12.0
51-60	18	38.0	18	36.0
61-70	13	28.0	13	28.0
71-80	6	12.0	8	12.0
>80	I	2.0	1	2.0
Total	50	100.0	50	100.0
Mean±SD	58.10-113.67		57.50+13.82	
Male	55.86E14.38		55.24±14.27	
Female	64.46±9.12		63.92±9.78	
P value	0.050*		0.049*	

Samples arc age matched with P=0.828

The maximum No of cases fall in the age group between 51-60yrs, followed by 61-70 yrs group.

Table 3: Gender wise distribution

Gender	Cases		Controls	
	No	%	No	%
Male	37	74.0	37	74.0
Female	13	26.0	13	26.0
Total	50	100.0	50	100.0

Samples are gender matched with P=1.000

The distribution of cases as per gender was denoted in the table and fig below. Females patients (64.46±9.122) were significantly older than males patients (55.86±14.381) with a p value- 0.050*

Table 4: Profile of risk factors

Past History	Cases (n= 50)		Controls (n= 50)		P value
	No	%	No	%	
Previous Stroke	4	8.0	0	0.0	0.411
Hypertension	26	52.0	17	34.0	0.069+
DM	14	28.0	12	24.0	0.648
Smoking	7	14.0	4	8.0	0.338
Alcohol	4	8.0	4	8.0	1.000
Angina/MI	9	18.0	2	4.0	0.026*
AF	1	2.0	0	0.0	1.000
Metabolic syndrome	37	74.0	24	48.0	0.008**

Metabolic syndrome turned out to be the most predominant risk factor for stroke in our research group, totalling 74 percent of cases and 48 percent of controls, and a significant p value of 0.008. With calculated p of 0.069, hypertension came second with 52 percent of cases and 34 percent of controls. Only angina /MI exhibited a significant correlation of 0.026, whereas DM and Angina /MI were ranked third and fourth, respectively.

Table 5: Clinical profile of cases

Clinical Manifestations	No	%
Hemiplegia (motor), facial palsy	18	36
Hemiplegia (sensory+motor)	17	34
Hemiplegia, facial palsy, aphasia, homonymous hemianopia	3	6
Monoplegia	4	8
Cerebellar signs	6	12
Acute Memory Loss	1	2
UMN Facial palsy	1	2

Table 6: Classification of stroke syndromes as per oxfordshire community stroke project

Stroke sub type	Number (n=50)
LACS (Lacunar syndromes)	35

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POCS (Posterior circulation syndromes)	6
PACS (Partial anterior circulation syndromes)	6
TACS (Total anterior circulation syndromes)	3

Strokes were divided into subtypes based on clinical criteria. Lacunar syndrome turned out to be most prevalent, accounting for 70% of all cases. POCS and PACS came in second and third, with 12 percent apiece, with TACS landing in fourth with 6 percent of the cases. This information was depicted in the table and graph below

Table 7: Blood indices - cases vs controls

Blood parameters	Cases	Controls	Significance
Hemoglobin (g/d1)	13.37±2.21 (7.10-18.60)	12.49±1.71 (8.30-16.00)	t=2.256:P=0.026*
Total count (%)	9728.00±2966.96 (4300-15900.0)	9638.00±3168.39 (4200-18000)	t=0.145:P=0.884
Platelet count (%)	2.56±0.58 (1.43-4.40)	2.69±0.83 (1.60-5.40)	t=0.881;P=0.380
Neutrophil (%)	71.14±12.02 (39-89)	75.86±9.79 (48-96)	t=2.152;P=0.034*
Lymphocyte (%)	23.98±9.98 (7-44)	20.74±11.11 (3-70)	t=1.535: P=0.128
Eosinophil (%)	3.20±2.72 (1-16)	2.51±1.79 (1-10)	t=1.422;P=0.159
Monocyte (%)	2.70±1.19 (1-6)	2.56±1.23 (1-5)	t =0.496:P=0.621

Although there was a tendency toward reduced platelet counts in patients, it was not statistically significant (p value =.380). The platelet counts in patients ranged 2.56±0.581 (1.43-4.40) compared to 2.69±0.833(1.60-5.40) of controls.

Table 8: Arterial territory of the infarct

Territory	Number (n=50)	%
ACA	2	4.0
MCA	36	72.0
ACA+MCA	5	10.0
VBA	7	14.0

In 72 percent of the patients, MCA territory was affected while14 percent of the cases revealed involvement of the vertebra basilar artery (posterior circulation).The combined contribution of ACA and MCA accounted for 10% of the cases. In 4% of instances, the ACA territory was directly involved.

Table 9: Severity of stroke

Modified Rankin's Score	Number (n=50)	%
Score 1: No significant disability	14	28.0
Score 2: Slight disability	9	18.0
Score 3: Moderate disability	6	12.0
Score 4: Moderately severe disability	11	22.0
Score 5: Severe disability	10	20.0

The Modified Rankin's scale was used to assess the clinical severity of the stroke at the time of presentation, and significant impairment was found in 20% of the patients. In 28% of the instances, there was no major impairment.

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Table 10: MPV in cases vs controls

MPV (fL)	Cases	Controls	Significance
MPV(EDTA)	7.86±0.82 (6.50-10.00)	7.58±0.70 (5.90-9.00)	t=1.834;P=0.074+
MPV(CITRATE)	7.35±0.81 (6.10-9.60)	6.94±0.59 (5.80-8.20)	t=2.894;P=0.005**

Results are presented as Mean ± SD (Min-Max)

With a P value of 0.005, MPV (citrate) exhibits a statistically significant association with acute Ischemic stroke. Despite the fact that MPV (EDTA) has a substantial trend in cases and controls (7.86±0.824 and 7.58±0.701, respectively), the difference isn't really significant statistically.

Table 11: Platelet mass- cases vs controls

Platelet Mass	Cases	Controls	Significance
PLATELET MASS (EDTA)	20.09±4.60 (10.87-31.68)	20.19±5.79 (11.52-42.12)	t=0.316;P=0.919
PLATELET MASS (CITRATE)	18.85±4.67 (10.30-31.68)	18.52±5.31 (9.76-36.72)	t=0.330;P=0.742

Results are presented as Mean ± SD (Min-Max)

Platelet mass is virtually constant as a function of platelet count and mean platelet volume. The readings in cases and controls across both EDTA (20.09±4.603, 20.19±5.791) and citrate (18.85±4.675, 18.52±5.312) samples demonstrate this. There is, however, no statistical relationship between platelet mass and ischemic stroke.

Table 12: Mpv (edta)- cases vs controls as per risk factors

Risk factors	Levels	Cases	Controls	P value
Hypertension	Absent	8.11±0.93	7.53±0.72	0.144
	Present	7.77±0.78	7.67±0.67	0.623
	P value	0.230	0.508	-
DM	Absent	7.73±0.83	7.57±0.73	0.394
	Present	8.08±0.79	7.61±0.64	0.086+
	P value	0.138	0.665	-
Smoking	Absent	7.81±0.82	7.58±0.69	0.156
	Present	8.14±0.78	7.55±0.88	0.278
	P value	0.328	0.935	-
Alcohol	Absent	7.83±0.80	7.55±0.72	0.087+
	Present	7.32±0.99	7.90±0.33	0.560
	P value	0.357	0.343	-

The stroke risk variables were analyzed with MPV (EDTA) to find out a positive relationship, but no statistically meaningful association was observed, presumably due to the limited sample size.

Table 13: MPV (citrate) - cases vs controls as per risk factors

Risk factors	Levels	Cases	Controls	P value
Hypertension	Absent	7.64±0.82	6.91±0.57	0.053*
	Present	7.26±0.79	7.02±0.62	0.256
	P value	0.161	0.584	-
DM	Absent	7.26±0.82	6.96±0.61	0.078+
	Present	7.51±0.78	6.90±0.54	0.025*
	P value	0.309	0.769	-
Smoking	Absent	7.30±0.79	6.95±0.61	0.022*
	Present	7.69±0.89	6.85±0.37	0.116
	P value	0.248	0.742	-
Alcohol	Absent	7.32±0.78	6.95±0.61	0.012*
	Present	7.75±1.08	6.95±0.23	0.200
	P value	0.314	0.983	-

Similarly, risk variables for stroke were evaluated to MPV (citrate) to find out a positive association, but no significant association was identified, but diabetes did have a modest correlation with high MPV

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Table 14: MPV (Edta/Citrate) As Per Other Risk Factors

Risk factors	Levels	MPV(EDTA)	MPV(CITRATE)
Age in years	<60	7.84±0.80	7.34±0.73
	60 and above	7.88±0.85	7.36±0.89
	P value	0.932	0.932
Gender	Male	7.72±0.74	7.21±0.77
	Female	8.25±0.95	7.76±0.82
	P value	0.047*	0.034*
WHR[57, 58, 59.]	Normal	7.43±1.08	7.00±0.82
	Abnormal	7.88±0.81	7.38±0.88
	P value	0.361	0.441
HDL(mg/dl)	Normal	7.99±0.97	7.41±0.96
	Abnormal	7.83±0.79	7.34±0.78
	P value	0.575	0.810
LDL (mg/dl)	Normal	7.77±0.77	7.32±0.79
	Abnormal	8.18±0.96	7.48±0.90
	P value	0.141	0.560
Triglycerides (mg/dl)	Normal	8.10±0.83	7.63±0.83
	Abnormal	7.51±0.67	6.99±0.62
	P value	0.012*	0.004**
Total cholesterol (mg/dl)	Normal	7.79±0.79	7.34±0.82
	Abnormal	8.06±0.89	7.41±0.82
	P value	0.304	0.785
Metabolic syndrome	Absent	7.90±0.96	7.48±0.93
	Present	7.84±0.78	7.31±0.78
	P value	0.803	0.531

Median age of cases studied is 59 years

Other risk factors such as age, sex, WHR, lipid levels, and metabolic syndrome were compared to MPV. Females had greater MPV compared to men, as found to be a positive association. Only high triglyceride group demonstrated a significant connection with a p value of 0.004.
The infarct arterial territory and MPV were evaluated, but no significant association found.

Table 15: Arterial teritory and mpv (edta and citrate)

Stroke sub type	Number (n=50)	MPV(EDTA)	MPV(CITRATE)
LACS	35	7.83±0.90	7.34±0.84
POCS	6	7.78±0.66	7.40±0.98
PACS	6	7.78±0.59	7.12±0.59
TACS	3	8.47±0.21	7.87±0.31
Significance		F=0.579; P=0.632	F=0.563; P=0.642

Table 16: Stroke syndromes and correlation with mpv

Territory	Number (n=50)	MPV (EDTA)	MPV (Citrate)
ACA	2	7.30±0.57	6.85±0.64
MCA	36	7.88±0.81	7.39±0.70
ACA+MCA	5	8.20±1.14	7.50±1.33
VBA	7	7.64±0.73	7.17±1.02
P value		0.525	0.720

In each of these clinical categories of stroke, no statistically significant difference was noted in MPV.

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Table 17: Stroke severity score and MPV

Modified Rankin's Score	Number (n=50)	MPV(EDTA)	MPV(CITRATE)
Score 1: No significant disability	14	8.00±0.69	7.44±0.58
Score 2: Slight disability	9	8.00±1.06	7.81±0.97
Score 3: Moderate disability	6	7.18±0.67	6.70±0.66
Score 4: Moderately severe disability	11	7.80±0.89	7.51±1.02
Score 5: Severe disability	10	8.00±0.68	7.49±0.69
Significance	P value	0.283	0.318

The Modified Rankin's score was compared to the matching mean's of MPV in each category to establish the relationship between MPV and stroke severity. However, there was no significant association found by analyzing the Modified Rankin's score and corresponding mean of platelet mass in each group, the relationship between platelet mass and stroke severity was identified. There was, however, no significant relationship.

Table 18: Platelet mass in cases based on severity score

Platelet mass	Severity of disease based on Modified Rankin's Score					P value
	No significant disability	Slight disability	Moderate disability	Moderately severe disability	Severe disability	
PLATELET MASS (EDTA)	20.22±4.6	17.43±3.3	20.77±6.4	21.11±4.55	20.75±4.5	0.434
PLATELET MASS (CITRATE)	18.83±4.4	15.88±2.7	19.54±6.77	20.37±4.87	19.47±4.53	0.281

The MPV value was sorted into quintiles and correlated with stroke severity score, which was split into two groups. Group 1 is the least severe, with a score 0-2; group 2 is the most severe, with score between 3-6. In EDTA samples, the p value obtained is 0.654 and has little to nil statistical significance.

Table 19: Quintiles of MPV (EDTA) and stroke severity

MPV (EDTA) Quintiles	Stroke severity score		P value
	Score: 0-2	Score: 3-6	
6.50-7.20	5 (21.7%)	10 (37.1%)	0.654
7.21-7.60	3 (13.1%)	4 (14.8%)	
7.61-7.86	5 (21.7%)	3 (11.1%)	
7.87-8.60	8 (34.8%)	6 (22.2%)	
8.61-10.00	4 (17.4%)	4 (14.8%)	
Total	23 (100.0%)	27 (100.0%)	-

Table 20: Quintiles of MPV (Citrate) and stroke severity

MPV (Citrate) Quintiles	Stroke severity score		P value
	Score: 0-2	Score: 3-6	
6.10-6.70	4 (17.4%)	8 (29.6%)	0.490
6.71-7.08	4 (17.4%)	4 (14.8%)	
7.09-7.36	7 (30.4%)	3 (11.1%)	
7.37-8.18	4 (17.4%)	7 (25.9%)	
8.18-9.60	4 (17.4%)	5 (18.5%)	
Total	23 (100.0%)	27 (100.0%)	-

MPV sorted into quintiles was compared to the stroke severity score, which was split into two groups. Group 1 is the least severe, with score 0-2; group 2 is the most severe, with score between 3-6. In citrate samples, the p value obtained is 0.490, which has little significance.

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Table 21: Prediction of stroke with multi-variate logistic regression analysis

Variables	Logit co-efficient	P value	Adj. OR
Age in years	-0.03	0.099+	0.97
Female	-0.02	0.977	0.98
H/O Hypertension	2.31	<0.001**	10.10
H/O DM	-0.12	0.833	0.89
Smoking	0.08	0.951	1.08
Alcohol	-0.10	0.944	0.91
MPV CITRATE	2.09	0.009**	8.10
MPV EDTA	-1.02	0.156	0.36

Multiple logistic regression analysis suggests that MPV is one of the most significant risk factors for stroke, with a p value of 0.009 and an adjusted odds ratio - 8.10. It is next to hypertension, which has a p value - 0.001 and an adjusted OR of 10.10.

Discussion

Cerebrovascular diseases have many platelet abnormalities. Circulating platelet aggregates, platelet aggregation, and platelet-specific granuleprotein release suggest platelet activation. Other investigations show that platelet aggregation occurs days after the trauma, not in the acute period. Platelet intake during the event may have prevented acute activation. These studies may differ due on specimen processing and technique [8, 9].

Pre-Analytic

In healthy individuals, EDTA, citrate, and time delay were tested on MPV measurements. Five participants gave EDTA-citrate blood samples. The graph shows that each sample was examined at periodic intervals from 1 to 48 hrs. Citrate MPV was 1 flower than EDTA. MPV likewise varies highest in the first 2 hours and least in the last 4. MPV is stable between two and four hours and beyond 24 hours. According to western studies, platelet swelling is greatest in the first two hours and settles after 24 hours with EDTA. This study ran samples between 2 and 4 hours, when they were reasonably steady. EDTA was used in addition to citrate, the global norm. Because EDTA is the most common anticoagulant used for regular complete blood counts, it might be utilized to preemptively target patients if MPV and stroke are found in it [9, 10].

Demographics

The study removed 180 stroke victims. A 48-hour delay in hospital arrival resulted in 46% exclusion. Hemorrhagic stroke (16.1%), recruitment delays (16.71%), informed consent refusal (15.10%), and peripheral smear platelet aggregates (5.89%) were further exclusion factors. Controls were (57.509 ± 13.823) and patients were (58.101 ± 13.674) yrs old onaverage. This study found the most cases in 51-60-year-olds and 61-70-year-olds. Males averaged 55.86 ±14.38 years old, while controls averaged 55.24 ±14.27. Controls were 63.92±9.78, while females were 64.46±9.12. Both groups had older women than men. 74% of cases and controls were male, 26% female. Our study's mean age was 58.102±13.674, lower than earlier research. Male stroke patients dominated this study. Except for Pikija and O'Malley, all other research found equivalent tendencies [10, 11].

Risks

In this study, 52% of cases and 34% of controls have hypertension, the most common stroke risk factor. 28 cases and 24 controls rated diabetes second. As indicated below, hypertension was the main risk factor in other trials. Pikija *et al*. scored 82.7 percent, whereas A. Muscari scored 84.7 percent. Compared to western research, only 8% of studies considered prior strokes a risk factor. Bath *et al* had a stunning 72% of prior strokes since this trial was a substudy on ACE inhibitors (perindopril) and stroke prevention. When compared to the literature, the population's age-matched 2% atrial fibrillation rate was surprising. The average patient age in this study was 58.103±13.67, much younger than in other worldwide studies. Elderly people have atrial fibrillation. Because stroke is commonly thrombotic, this research group's MPV findings are more credible. Compared to other study, 28% of patients had diabetesmellitus. Metabolic syndrome was the most common in this study, with 74 patients and 48 controls with a p value of 0.008.

MPV is compared to stroke risk factors. Multiple logistic regression of baseline risk variables showed that HTN was the most prevalent stroke risk factor with p = 0.001 and an adjusted OR of 10.10. Ischemic stroke and DM patients had higher MPV than controls (p = 0.021). Females showed a higher MPV than

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men in citrated samples (p = 0.033). Comparing age, WHR, lipid indicators, metabolic syndrome, smoking, and alcohol use [11, 12].

Drugs history

16 percent of cases were on antiplatelet medication due to strokes or ischemic heart disease. It was unclear if utilizing antiplatelets simultaneously would alter the MPV studied. Aspirin and MPV did not interact *in vitro* or *in vivo* in previous studies. Antiplatelet drugs differ. All trial participants took 150 milligrams of aspirin and no other antiplatelet medicines. 32% of hypertensives took calcium channel blockers, followed by beta-blockers and diuretics [13, 14].

MPV, mass, and count were examined. MPV was examined first. With a p value of 0.005, MPV (citrate) is statistically associated with ischemic stroke, with an average MPV of (7.35±0.813) in cases and (6.94 ± 0.592) in controls. MPV (EDTA) shows a variation in cases and controls of 7.86 ± 0.824 and 7.86 ± 0.821, however it is not statistically significant. Only Butterworth *et al*. tested MPV with EDTA and citrate, and the results were 8.04 ±1.044 (7.69±0.831) for EDTA and 7.35 ±1.050 (7.09±0.743) for citrate. The results were consistent across varied groups. The other trials used EDTA as an anticoagulant, although the collection, analysis, specimen transportation, and storage procedures varied. After 24 hours at room temperature, O'Malley research tested it [15, 16]. Most studies tested after two hours. This investigation's sample materials. Only Tohji *et al*. discovered lower MPV in patients than controls. This trial did not specify the testing time or preservation temperature after venipuncture. Patients' platelet counts decreased by 2.56±0.582 (1.43-4.401) compared to controls' 2.69±0.834. (1.60-5.403). This trend is insignificant. All case control studies, including Butterworth, Tohji, and O'Malley, showed this tendency. Platelet mass is almost independent of platelet count and meanplateletvolume. Cases and controls in EDTA (20.09±4.60, 20.19±5.79) and citrate (18.85±4.67, 18.52±5.31) samples show this. Platelet mass does not predict ischemic stroke statistically. Western research hasn't investigated this. Multiple logistic regression study showed that MPV is second only to hypertension as a risk factor for ischemic stroke, with a p value of 0.009 and an OR of 8.10 [16, 17].

Although platelet production is unknown, MPV and platelet count may be maintained by distinct hormonal regulation. Il-6, Il-3, thrombopoietin, and colony-stimulating factors may affect. Platelet volume and count are regulated at thrombopoiesis, 8 and these findings may indicate fundamental bone marrow (megakaryocyte) changes, as in ischemic heart disease. MPV increases megakaryocyte ploidy and size. Ischemic stroke may be caused by megakaryocyte activation, as shown by MPV increase [17, 18].

Stroke subtypes

The Oxfordshire community stroke study classified strokes as lacunar or non-lacunar syndromes (PACS, POCSand TACS). Two trials indicated a substantial increase in MPV in non-lacunar strokes compared to lacunar strokes. O' Malley*et al*. showed no correlation. This study revealed no significant connection. Ischemic stroke patients had larger platelets, which is consistent with recent investigations showing increased platelet activity in cerebral infarction, such as elevated b-thromboglobulin (b-TG) and TxA2 levels in blood and urine. We cannot verify that cortical ischemic stroke causes thrombomegaly. Cortical events generated by athero-thromboembolic heart, aorta, carotid, or major intracranial artery events may activate platelets. Tinyvessel lipohyalinosis, a platelet-free pathology, may cause several devastating white matter lacunar strokes [18, 19].

Stroke subtypes

72% involved MCA territory. 14% implicated the vertebrobasilar artery. 10% had both ACA and MCA. 4% of cases affected only the ACA territory. MPV and vascular territory were compared. MPV citrate has 0.722 and MPV-EDTA 0.523. These were insignificant statistically. Western research hasn't publicized this [19].

Stroke severity

At presentation, 20% had significant stroke disability according to the modified Rankin's assessment. 28% of patients were unimpaired. 18% had minor disabilities, 13% had intermediate disabilities, and 22% had severe disabilities. Cases are nonfatal. MPV correlated with stroke severity when compared to modified Rankin's score in each group. MPV – EDTA and MPV – citrate revealed nonsignificant p values of 0.289 and 0.318, respectively. O' Malley performed homogenous experiments and classed the outcomes as independent, dependent, or dead. MPV was insignificant [20, 21]. According to Butterworth *et al*., patients who died or became dependent by 3 months had significantly higher platelet volumes and lower platelet counts. Statistical significance was absent. MPV was separated into quintiles and compared to the stroke severity score, which was divided into two classes, despite the small sample size. Group 1 scores 0-2 and Group 2 scores 3-6. EDTA and citrate sample p values are 0.653 and 0.491, respectively. Griesenegger *et al*. observed that acute ischemic cerebrovascular episode patients with a higher MPV had a worse outcome. The highest quintile of MPV had a 2-fold higher risk of a major stroke than the lowest quartile. After correcting for confounders, high MPV and severe stroke remained significant. P=0.002 in this study. Since critically ill patients were not included in our analysis, there was

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a bias. Indirect data suggests MPV and platelet count changes occurred before the vascular event and were not caused by platelet consumption at the infarct site. Platelets have a typical lifespan of 8 days, hence the high MPV detected within 48 hours of a stroke is likely platelets released before infarction [22, 23].

Isolated localized thrombosis also does not alter peripheral venous platelet parameter estimations. MPV did not differ between massive cortical strokes and lacunar infarctions, supporting this approach. Big platelets may cause thrombosis in sensitive people, and a rise in MPV may have caused the stroke rather than being a result of the acute event. Thus, acute ischemic cerebrovascular stroke increased MPV and decreased platelet count. After adjusting for other factors, MPV increases are uniquely associated with ischemic stroke in multivariate regression. Bigger platelets may play a role in cerebral vascular thrombosis and thrombopoiesis. Platelet volume in ischemic cerebrovascular stroke pathogenesis and outcome, especially in at-risk patients, needs further study [24].

Conclusion

Our study found an increase in MPV in the first few hours after an ischemic cerebrovascular stroke. Multivariate regression analysis using this association found a significant association between an increase in MPV and stroke after controlling for other potential confounding factors. The results suggest a role for larger platelets in the progression of vascular thrombosis and are likely to represent changes at thrombopoiesis. We need more research on the role of platelet volume in the pathophysiology and outcome of stroke, especially in people who are at high risk for stroke.

There was a noticeable uniformity in the platelet mass measurements. That is to say, this study showed that even though there was no statistically significant connection between MPV and early-stage ischemic stroke, there was still a small drop in platelet count. The study did not find a statistically significant correlation between clinical stroke severity and platelet volume.

In this study, vascular regions were used to clinically categorize different types of ischemic strokes. However, no statistically significant association was found when MPV was compared to the various subtypes of stroke. Thrombomegaly is linked to lacunar syndromes and cortical ischemic stroke.

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