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A Study on Association of Mean Platelet Volume and Ischemic Stroke

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Abstract: *Cerebrovascular diseases include some of the most common and devastating disorders. On the other hand, Mean platelet volume (MPV) is a physiological variable of haemostatic importance. Ischemic stroke is thought to occur as a result of thrombotic occlusion of a stenosed atherosclerotic blood vessel. Platelets are anucleate cells and are heterogeneous regarding their size, density &hemostatic potential. They play a crucial role in pathogenesis of atherosclerotic complications, contributing to thrombus formation. A morphological consideration of platelets has important implications for both interpreting the functional expression of platelets & experimental measurement of platelet size. Though there have been quite a few studies which have demonstrated an association between myocardial infarction and platelet size, very few studies have looked at the association between platelet size and ischemic stroke. This study was a prospective study carried out among fifty patients diagnosed with an acute ischemic stroke and presenting to the hospital within forty eight hours of onset of symptoms meeting our inclusion & exclusion criteria. Duration of study was between April 2016 to October 2017 at Department of Medicine, Rajendra Institute of Medical Sciences, Ranchi.*

The study has shown an elevation of MPV in acute phase of ischemic stroke. Within this relationship and adjusting for other significant variables in multivariate regression analysis, it can be stated that an increase in MPV is independently associated with stroke, suggesting a role for larger platelets in the genesis of cerebral thrombosis.

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I. Introduction

Cerebrovascular diseases include some of the most common and devastating disorders.The prevalence of stroke in India was estimated as 203 per 100,000 population above 20 years, amounting to a total of about 1 million cases. Stroke is one of the major causes of human morbidity and mortality. The most important risk factors for ischemic stroke are hypertension, heart disease, atrial fibrillation, diabetes mellitus, cigarette smoking, and hyperlipidemia.[11]Since this is such a huge public health problem, other risk factors and possible preventive measures need to be identified. It is in this context that this study has its significance.

Platelets play a crucial role in the pathogenesis of atherosclerotic complications, contributing to thrombus formation.[14] Platelets are anucleate cells and are heterogeneous regarding their size, density and haemostatic potential. Platelet size(mean platelet volume, MPV) is a marker and possibly determinant of platelet function, large platelets being potentially more reactive. In normal individuals the platelet count is inversely proportional to MPV; platelet mass (the product of MPV and platelet count) is a near constant. Also, there is evidence that platelet function is accentuated in acute ischemic stroke.[7]

Though there have been quite a few studies which have demonstrated an association between myocardial infarction and platelet size, very few studies has looked at the association between platelet size and ischemic stroke. Among them, there has been discrepancy regarding the sample size, methodology used and the final result. There are no documented studies in India comparing the association of mean Platelet volume with ischemic stroke; hence an attempt has been made to study the association if any between mean platelet volume and stroke in an Indian population.

II. Material And Methods

The study was a prospective study and data was collected from April 2016 to October 2017 at Department of Medicine, Rajendra Institute of Medical Sciences, Ranchi.The study was carried out among fifty patients diagnosed with an acute ischemic stroke and presenting to the hospital within forty eight hours of onset

of symptoms.Fifty age and sex matched controls were also recruited. The study protocol was approved by the Institutional Ethics Committee.

Study Design: Prospective case control study

Study Location: This was a tertiary care teaching hospital based study done in Department of Medicine, Rajendra Institute of Medical Sciences, Ranchi

Study Duration: April 2016 to October 2017

Sample size: 50 cases & 50 controls

Sample size calculation: This was done using the Power/Sample size calculator, our sample size came out be 45 (taking a confidence interval of 95%, power of study as 90%, prevalence of acute stroke as 119-142/lac population).The study was carried out among fifty patients diagnosed with an acute ischemic stroke and presenting to the hospital within forty eight hours of onset of symptoms. Fifty age and sex matched controls were also recruited.

Subjects & selection method

Cases:

Definition of stroke: Focal neurological deficit lasting more than 24 hours with no evidence of a non – vascular cause.

Inclusion criteria:

1. Gender: Males/Females
2. AgeRange: 18 years and above
3. Socioeconomic group: All socioeconomic groups were eligible
4. CT Scan to exclude hemorrhagic stroke.

Exclusion criteria:

1. Thrombocytopenia.
2. Known cases of hereditary disorders of large platelets.
3. Medications that can reduce the platelet count: hydroxyurea, antineoplastic agents, and inhibitors of the platelet integrin IIbβ3.
4. Haemorrhagic stroke.
5. Patients unable to communicate because of severe stroke, aphasia or dementia without a valid surrogate respondent.

(A valid surrogate respondent is considered a spouse or first degree relative that is living in the same home or is self- identified as aware of the participant‘s previous medical history and current therapies)

6. Patients presenting 48 hours after the onset of neurological symptoms.
7. Peripheral smear showing platelet aggregates.

Controls:

Controls were primarily hospital based. Each control was matched for sex and age (±5 years). There was at least one control for each case recruited.

Inclusion criteria

1. Relative of a patient from ward.
2. Unrelated Visitor of any patient.
3. Patients attending the hospital or outpatient clinic for other illness.

Exclusion criteria

1. Individuals with a previous history of stroke.
2. Thrombocytopenia
3. Peripheral smear showing platelet aggregates.

Procedure methodology

All stroke patients admitted to the hospital were screened during the time period described above. Each of them was entered into a stroke log. Patients fulfilling the criteria were enrolled into the study after obtaining an informed consent. Data was collected by the principal investigator and recorded, as per the proforma. Each patient was given a serial number and was formally included into the study as a case. Each patient was assessed and a modified Rankin‘s Scale assigned to them. A Blood sample was collected from the antecubital vein using a 5 cc syringe and transferred to an EDTA and citrate vacutainers. The samples were then taken to the laboratory between 2 hours and 4 hours of collection and analyzed using the ABX pentra automated analyzer using electrical impedance to measure the mean platelet volume. After the analysis the same sample was taken to the central laboratory and a peripheral smear was done to look for platelet aggregates. If platelet aggregates were found then such cases were excluded from the study. The same procedure was adopted in controls for taking the samples, and then transferred it to the vacutainers (EDTA and Citrate) and analyzed using the

automated analyzer (ABX pentra). Peripheral smears were done to look for aggregates and if present were excluded. Samples were also sent for routine blood work including complete blood count, lipid profile, RBS, CT scan/MRI for both cases & controls. Modified Rankin Scale was used for assessing severity of stroke

0 - No symptoms at all

1 - No significant disability despite symptoms; able to carry out all usual duties and activities

2 - Slight disability; unable to carry out all previous activities, but able to look after own affair without assistance

3 - Moderate disability; requiring some help, but able to walk without assistance

4 - Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance.

5 - Severe disability; bedridden, incontinent and requiring constant nursing care and attention

6 - Dead

III. Statistical Analysis

Sample size:

50 cases admitted in Rajendra Institute of Medical Sciences, Ranchi who gave informed consent and who met the inclusion criteria were recruited. 50 age and sex matched controls were also recruited from the hospital.

Data analysis:

The Statistical software namely SPSS ver20.0 was used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

***Statistical Methods*:**

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean±SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance. Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients, Student 't' test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups A Multivariate logistic regression analysis has been carried out to find the risk factors associated with stroke.[5,6,7.]

1. Analysis of Variance: F test for K Population means.[5,6]

Objective: To test the hypothesis that K samples from K Populations with the same mean.

Limitations: It is assumed that populations are normally distributed and have equal variance. It is also assumed that samples are independent of each other.

Method: Let the j^{th} sample contain nj elements (j=1, 2…K). Then the total number of elements is

$$N = \sum nj \qquad x.j = \sum \frac{xij}{nj}$$

$$S_1^{\,2} = \frac{\sum \sum_{i-1}^{n1} (x1 - \overline{x.j})^2}{N - K} \qquad S_2^{\,2} = \frac{\sum_{i-1}^{n1} nj(\overline{x.j} - \overline{x..})^2}{K - 1}$$

$F = S_2^{\,2}/S_1^{\,2}$ Which follows F distribution (K-1, N-K)

2. Student t test (Two tailed, independent)[63]

$$t = \frac{(\overline{x}_1 - \overline{x}_2) - (\mu_1 - \mu_2)}{\sqrt{s^2(1/n1 + 1/n2)}}$$

$$\text{Where } s^2 = \frac{(n1-1)\sum_{i-1}^{n1}(x1 - \overline{x1})^2 + (n2-1)\sum_{i-1}^{n2}(x2 - \overline{x2})^2}{n1 + n2 - 2}$$

3. Significant figures
+ Suggestive significance (p value: 0.05<p<0.10)
* Moderately significant (p value: 0.01 < p≤0.05)
** Strongly significant (p value: p≤0.01)

IV. Results

Table no 1 : Stroke log & Reasons for Exclusion

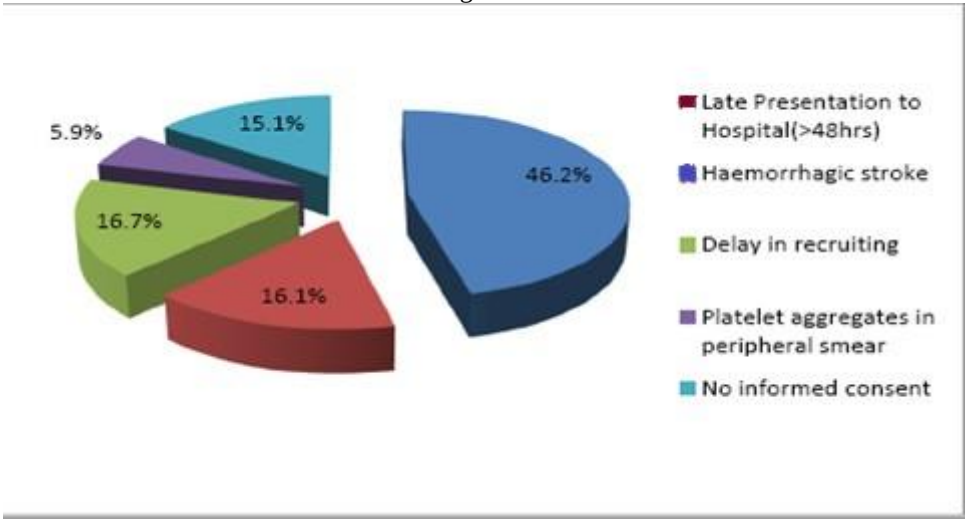
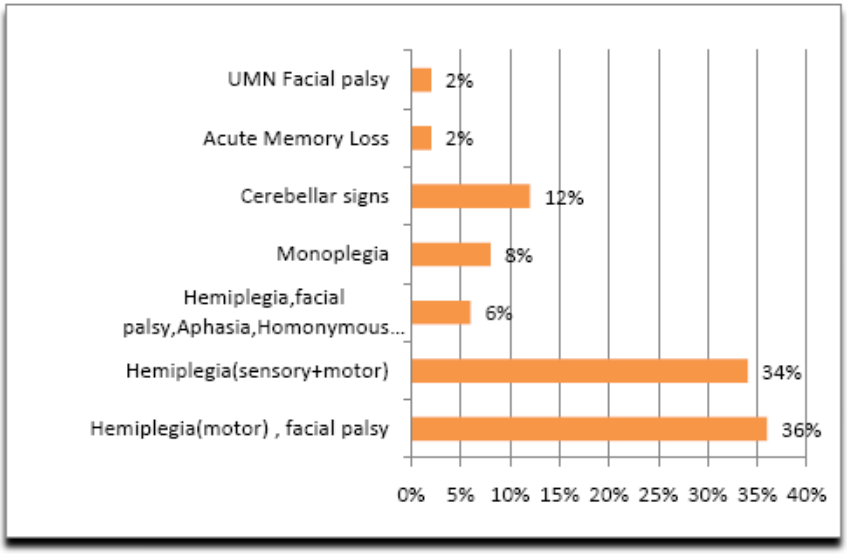


Table no 2: Clinical profile

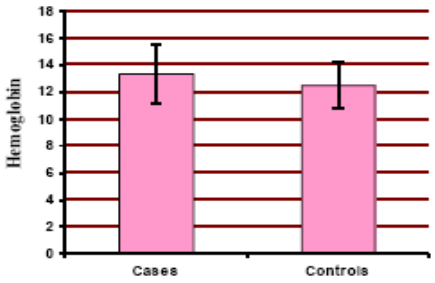
Clinical Manifestations	**No**	**%**
Hemiplegia (motor), facial palsy	18	36
Hemiplegia (sensory + motor)	17	34
Hemiplegia, facial palsy, aphasia, homonymous hemianopia	3	6
Monoplegia	4	8
Cerebellar signs	6	12
Acute Memory Loss	1	2
UMN Facial palsy	1	2

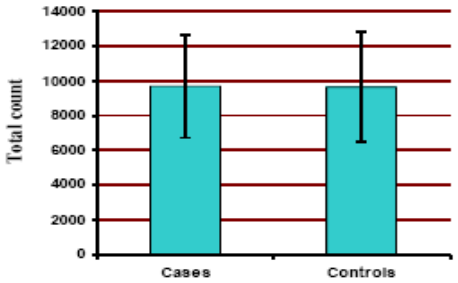


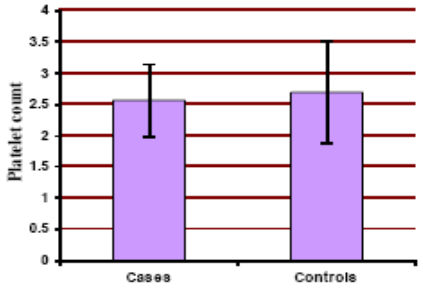
Results are presented as Mean ± SD (Min-Max)
There was a trend for lower platelet count in cases, but this was not significant (p value = 0.380) Thus the platelet counts in the cases averaged 2.56±0.58 (1.43-4.40) when compared to the controls in whom the average was 2.69±0.83 (1.60-5.40).

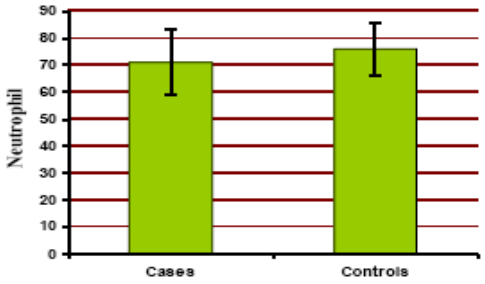
Table no 3 : Comparision of Blood Parameters in Cases & Controls

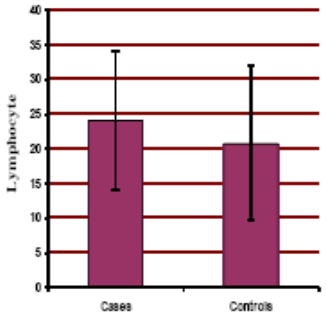
Blood parameters	**Cases**	**Controls**	**Significance**
Hemoglobin (g/dl)	13.37±2.21 (7.10-18.60)	12.49±1.71 (8.30-16.00)	t=2.256;P=0.026*
Total count (%)	9728.00±2966.96 (4300-15900.0)	9638.00±3168.39 (4200-18000)	t=0.145;P=0.884
Platelet count (%)	2.56±0.58 (1.43-4.40)	2.69±0.83 (1.60-5.40)	t=0.881;P=0.380
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

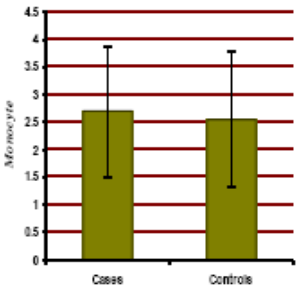












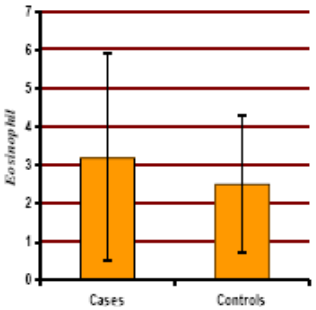


Table no4 Type of Infarct on basis of Imaging

The common patterns seen were parietal and temporo- parietal infarcts which comprised of 12% each of the cases. This was followed by Coronaradiata infarcts which comprised of 10% of the cases.

Type of Infarct	**Number (n=50)**	**%**
Parietal Infarct	6	12.0
Temporo parietal Infarct	6	12.0
Corona Radiata Infarct	5	10.0
Fronto parietal Infarct	4	8.0
Ganglio Capsular Infarct	4	8.0

Parasagittal Infarct	3	6.0
Basal ganglia Infarct	2	4.0
Cerebellar Infarct	2	4.0
Internal capsule Infarct	2	4.0
Pontine Infarct	2	4.0
Temporo parietal & Corona Radiata	2	4.0
B/L Cerebellar Infarct	1	2.0
Basal ganglia & Corona radiata	1	2.0
Cortical Infarct	1	2.0
Fronto Temporal Infarct	1	2.0
Medial Frontal Infarct	1	2.0
Medullary Infarct	1	2.0
Parietal & Thalamic Infarct	1	2.0
Perisylvian Infarct	1	2.0
Posterior Limb of internal capsule	1	2.0
Temporal Infarct	1	2.0
Temporo parietal & Occipital	1	2.0
Thalamic Infarct	1	2.0

Table no 5 Infarct Territory

MCA territory was involved in 72% of the patients. This was followed by involvement of the vertebra basilar artery (posterior circulation) with 14 % of the cases. Involvement of ACA and MCA together comprised of 10% of the cases. Pure ACA territory involvement was seen with 4% of cases.

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

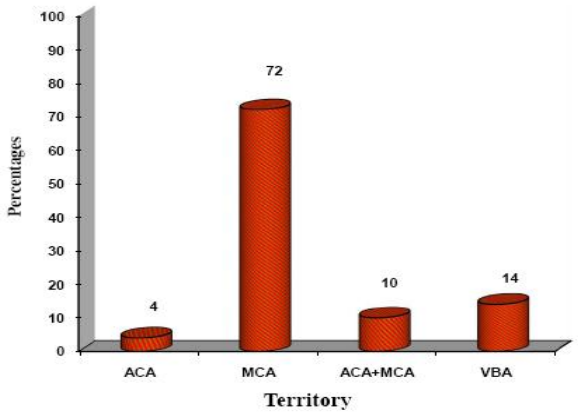


Table 6

The clinical severity of stroke at presentation was determined by the Modified Rankin's scale and severe disability was seen with 20% of the cases. 28% of the cases had no significant disability.

Stroke – Clinical Severity Score

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

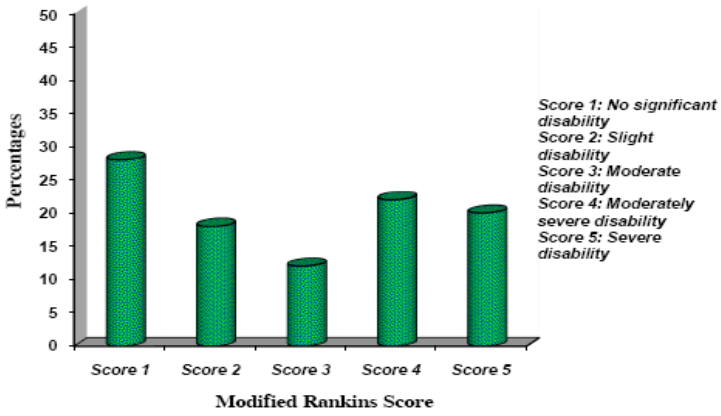


Table 7 Comparison of MPV in cases & controls

MPV (citrate) has got a statistically significant correlation with Ischemic stroke with a P value of 0.005 with an average MPV in cases being 7.35±0.81 compared to controls who average 6.94±0.59. Though MPV (EDTA) also shows a strong trend in cases and controls 7.86±0.82 and 7.86±0.82, the difference is not statistically significant.

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)

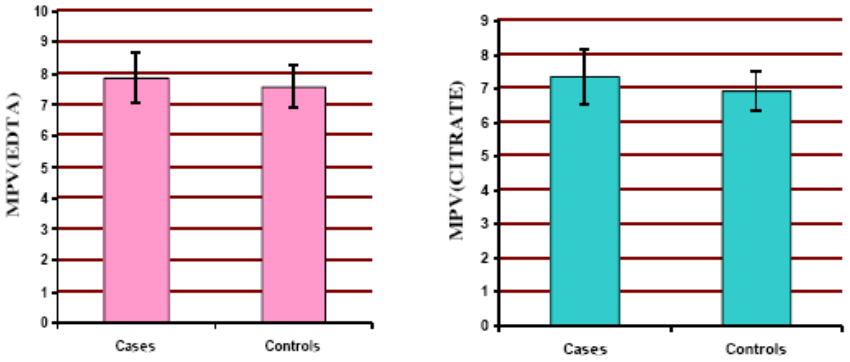


Table 8: Comparison of platelet mass in case & controls

The platelet mass being a product of platelet count and mean platelet volume is almost a constant. This is evident from the values in cases and controls in both EDTA (20.09±4.60, 20.19±5.79) and citrate (18.85±4.67, 18.52±5.31) samples. However there is no statistical correlation of platelet mass to Ischemic stroke.

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)

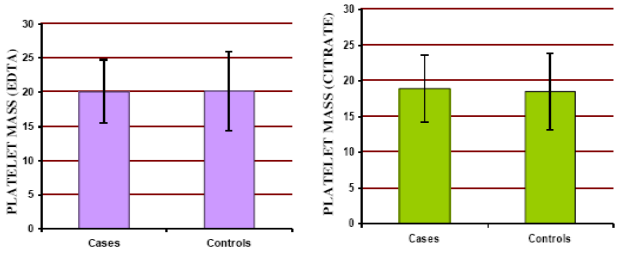


Table 9: Comparison of MPV in cases & controls according to risk factors.

The risk factors for stroke was compared with MPV(EDTA) to look for any positive correlation however no statistically significant correlation was found, possibly because of the small numbers.

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	-

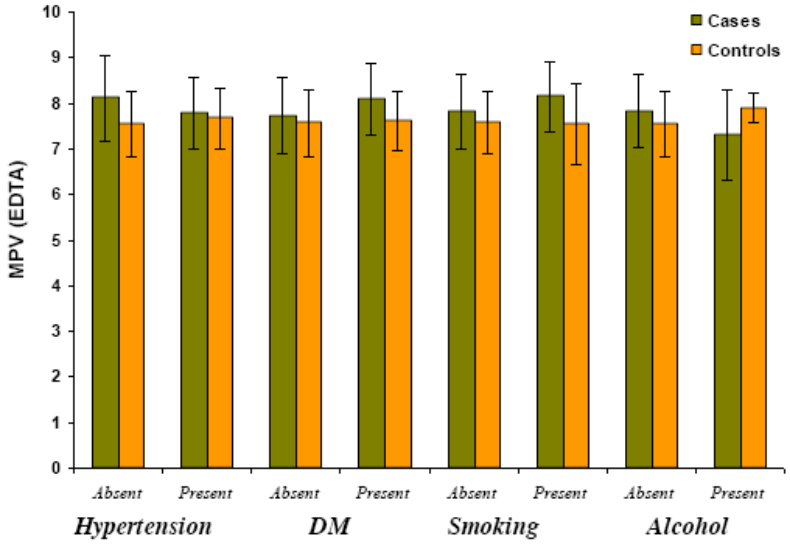


Table 10: Comparison of MPV in cases & controls according to other risk factors

MPV was compared to the other risk factors like age, gender, WHR, lipid profiles and metabolic syndrome. A positive correlation was seen with female gender having higher MPV than males. However a statistically significant correlation with a p value of .004 was seen only in high triglyceride group.

Median age of cases studied is 59 years

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

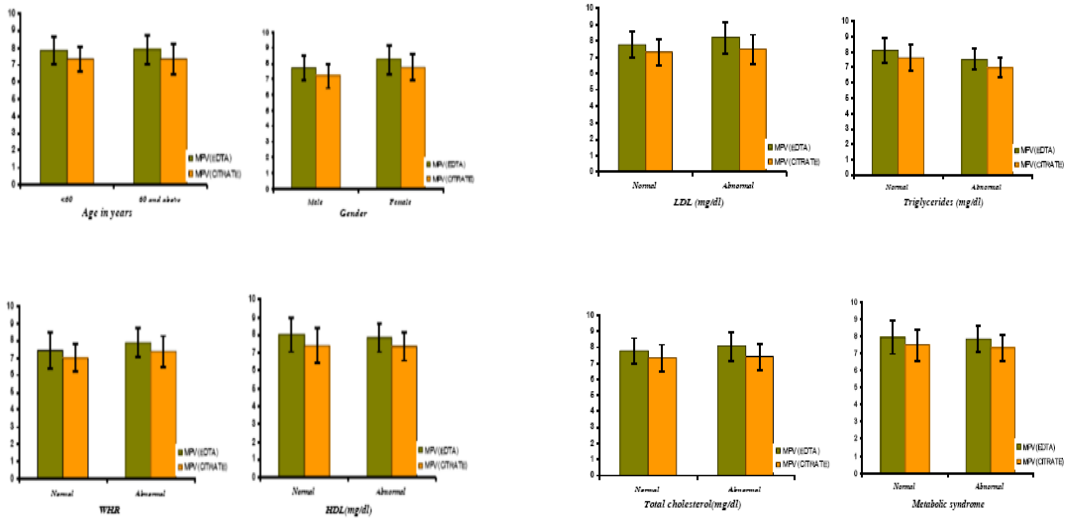


Table 11: Infarct Territory & MPV

The infarct territory and MPV (EDTA and Citrate) were compared to look for any correlation, but no statistical significance was obtained.

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

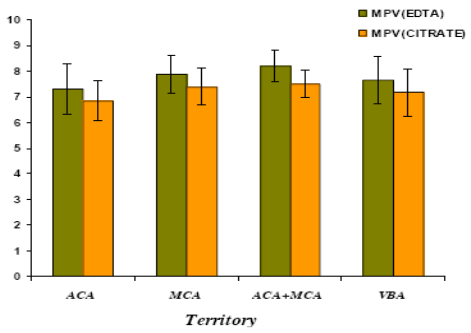


Table 12: Stroke- Clinical Severity Score & MPV

The association of MPV with severity of stroke was determined by comparing the Modified Rankin's score with corresponding mean values of MPV in each group. However no statistically significant correlation was obtained.

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

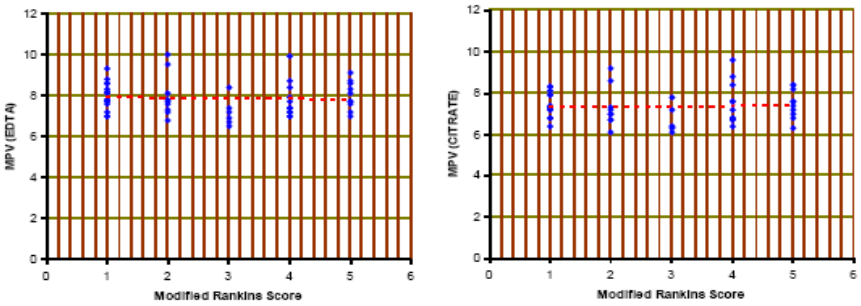


Table 13: Multivariate Logistic Regression Analysis to predict Stroke

It is demonstrated by multiple logistic regression analysis that MPV with a p value of 0.009 and an adjusted OR (Odds ratio) of 8.10, it is one of the most important risk factors associated with stroke only second to hypertension which had a p value of <0.001 and adjusted OR of 10.10.

Variables	**Logit co-fficient**	**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

V. Discussion

Cerebrovascular diseases include some of the most common and devastating disorders. They cause ~200,000 deaths each year in the United States and are a major cause of disability. The prevalence of stroke in India was estimated as 203 per 100,000 population above 20 years, amounting to a total of about 1 million cases. It is ranked as the sixth leading cause of disability-adjusted years (DALY; one DALY is one of the lost year of healthy life) in 1990 and is projected to rank fourth by the year 2020[13].

The platelet plays a major role in the pathogenesis of vascular disease, and mean platelet volume (MPV) is a physiological variable of hemostatic importance. Large platelets are more reactive, produce more prothrombotic factors[3 and 4] and aggregate more easily. They also contain more dense granules and release more serotonin and ß-thromboglobulin than do small platelets. Platelets have no nuclei, and their characteristics are determined by their progenitor cell, the bone marrow megakaryocyte. Ischemic stroke is thought to occur as a result of thrombotic occlusion of a stenosed atherosclerotic blood vessel. Initially platelets adhere to the damaged vessel, resulting in recruitment of further platelets, followed by aggregation, formation of a platelet plug and finally thrombotic occlusion. Thus, the detection of large platelets in patients would lend support to the idea that platelet volume influences thrombotic large vessel occlusion leading to ischemic stroke. Though there have been quite a few studies which have demonstrated an association between myocardial infarction and platelet size, very few studies has looked at the association between platelet size and ischemic stroke. Among them there has been discrepancy regarding the sample size, methodology used and the final results. There are no documented studies in India comparing the association of mean Platelet volume with Ischemic strokes. This was a prospective study and data collected from April 2016 to October 2017 at Rajendra Institute Of Medical Sciences, a tertiary care referral centre. The study was carried out among 50 patients diagnosed with an acute

ischemic stroke and presenting to the medicine wards in the hospital within forty eight hours of onset of symptoms and satisfying the inclusion and exclusion criteria. The data was compared to 50 age and sex matched controls.

Key findings included:

1) The main parameter studied was MPV. MPV(citrate) has got a statistically significant correlation with Ischemic stroke with a P value of 0.005 with an average MPV in cases being 7.35±0.81 compared to controls who average 6.94±0.59. Though MPV (EDTA) also shows a difference in cases and controls 7.86±0.82 and 7.58±0.70, the difference is not statistically significant. Probable theoretical reason for the same may be platelet swelling due to EDTA solution after 12 hrs. Thus the study has shown an elevation of MPV in acute phase of Ischemic stroke. Within this relationship and confounding for other significant variables in multivariate regression analysis, it can be stated that an increase in MPV is independently associated with stroke. The observations here suggest a role for larger platelets in the genesis of cerebral thrombosis and are likely to represent changes occurring at thrombopoiesis. Further research is required into the role of platelet volume in stroke pathology, outcome, and, most importantly, in individuals at risk for stroke.

2) The platelet count is lower in the cases with an average of 2.56±0.58 (1.43- 4.40) when compared to the controls in which the average was 2.69±0.83 (1.60-5.40). The platelet mass being a product of platelet count and mean platelet volume is almost a constant. This is evident from the values in cases and controls in both EDTA (20.09±4.60, 20.19±5.79) and citrate (18.85±4.67, 18.52±5.31) samples.

3) The clinical severity of stroke at presentation was determined by the modified Rankin‘s scale and severe disability was seen with 20% of the cases. 28% of the cases had no significant disability. Others included 18% with slight disability, 12% with moderate disability and 22% with moderately severe disability. There were no deaths recorded. The association of MPV with severity of stroke was determined by comparing the Modified Rankin‘s score with corresponding mean values of MPV‘s in each group. MPV – EDTA showed a p value of 0.028 and MPV –citrate a p value of 0.003, both of which were statistically significant.

In conclusion, this study has shown an elevation of MPV and reduction of platelet count in acute stroke. With this relationship and confounding for other significant variables in univariate analysis, an increase in MPV is independently associated with stroke & a poor functional outcome. The observations here suggest a role for larger platelets in the genesis of cerebral thrombosis and are likely to represent changes occurring at thrombopoesis. Further research is required into the role of platelet volume in stroke pathology, outcome, and most importantly in individuals at risk of stroke.

There were a few limitations in our study like small sample size & no evaluation of stroke volume.

VI. Conclusion

This study has shown an elevation of MPV in acute phase of ischemic stroke. Within this relationship and adjusting for other significant variables in multivariate regression analysis, it can be stated that an increase in MPV is independently associated with stroke. The observations here suggest a role for larger platelets in the genesis of cerebral thrombosis and are likely to represent changes occurring at thrombopoiesis.

Platelet mass was found to be more or less a constant .i.e. this study has shown not only an elevation of MPV but a slight reduction of platelet count in acute phase of ischemic stroke though no statistical correlation was established.

This study found a statistically significant correlation between clinical severity of stroke and mean platelet volume.

In this study strokes were sub typed clinically and based on the vascular territory on CT Brain. However no statistical correlation was obtained when MPV was compared to the various subtypes of stroke. The presence of Large platelets was restricted not just to patients with large territory cortical ischaemic stroke but also with lacunar syndromes.

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