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Study of Correlation of Plasma D-Dimer Levels with the Severity of Acute Ischemic Stroke at a Tertiary Care Hospital

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ABSTRACT
Stroke of all types rank second as a cause of death and is the leading cause of disability in adults. Elevated D Dimer levels is a reflection of ongoing thrombus formation in blood vessels. Present study was aimed study correlation of plasma d-dimer levels with the severity of acute ischemic stroke at a tertiary care hospital. Present study was cross-sectional descriptive study, conducted in patients with sudden onset of focal neurological deficit with or without radiological evidence of ischemic stroke. Patient was subjected to relevant blood and radiological investigations. Sample for D-dimer levels was drawn at admission. D-dimer levels were correlated with the severity of stroke. Severity of stroke was assessed using the NIHSS scale. Out of 75 subjects, 14(18.70%) suffered from Minor Stroke, 42(56.00%) suffered from Moderate Stroke, 4(5.30%) suffered from Moderate to Severe Stroke, and 15(20.00%) from Severe Stroke. There was a significant difference in the mean age among the groups, as $p<0.05$. 22(29.30%) were females and 53(70.70%) were males. There was significant association between presence of comorbidities and severity of stroke. It was observed that the mean D-Dimer increased significantly with severity of stroke. It was observed that the mean NIHSS increased significantly with severity of stroke. Significant, highly strong, positive correlation was observed between D-Dimer and HBA1C, $r=0.928$, $p<0.05$. Significant, strong, positive correlation was observed between D-Dimer and RBS, $r=0.789$, $p<0.05$. Significant, moderate, positive correlation was observed between D-Dimer and Triglycerides, $r=0.512$, $p<0.05$. Significant, moderate, positive correlation was observed between D-Dimer and Total Cholesterol, $r=0.544$, $p<0.05$. Significant, moderate, positive correlation was observed between D-Dimer and LDL, $r=0.466$, $p<0.05$.Significant, weak, negative correlation was observed between D-Dimer and HDL, $r=0.260$, $p<0.05$. D-dimer levels can be measured in acute ischemic stroke to understand its severity and to predict its functional outcome.

INTRODUCTION

The term stroke also referred to as cerebrovascular accident (CVA), is the rapid loss of brain function due to disturbance in the blood supply to the brain. This can be due to ischemia (lack of blood flow) caused by blockage (thrombosis, arterial embolism) or a haemorrhage[1].

Stroke of all types rank second as a cause of death and is the leading cause of disability in adults[2-4]. Ischemic CVA account for approximately 80% of all strokes. D-dimer represents the activation of coagulation and fibrinolysis. The D-dimer levels were found to be higher in many disorders in which the coagulation system is activated such as acute ischemic stroke.

Elevated D Dimer levels is a reflection of ongoing thrombus formation in blood vessels. The D-Dimer levels are known to be directly proportional to the infarct volume and clot size. Measurement of d-dimer levels is a simpler, faster, non-invasive tool which would help in faster decision-making regarding initiation of reperfusion therapies. Present study was aimed study correlation of plasma d-dimer levels with the severity of acute ischemic stroke at a tertiary care hospital.

MATERIALS AND METHODS

study was cross-sectional descriptive study, conducted in department of General Medicine, at Mandya Institute of Medical Sciences, Mandya, India. Study duration was of 1 years (July 2021 to June 2022). Study was approved by institutional ethical committee.

Inclusion Criteria:

- Sudden onset of focal neurological deficit with or without radiological evidence of ischemic stroke, patients/patient's attenders giving informed consent.

Exclusion Criteria:

Patients with:

- Any focal neurological deficit with radiological evidence of intracranial hemorrhage, intracranial tumor.
- Other conditions mimicking stroke like seizure, metabolic encephalopathy.
- Patients with other reasons for elevation of D Dimer like Inflammation, Malignancy, Pregnancy, Immobility, Liver diseases, Recent surgery.

Study was explained to patients/patient's attenders in local language and written informed consent was taken. On admission detailed history was taken and clinical examination was done. Patients presenting with focal neurological deficit, with no radiological evidence of hemorrhage were included in the study. Plasma D Dimer levels was tested.

Information was collected through a pre tested and structured proforma for each patient. In the first part, data regarding identifier details like name, age, phone number, inpatient number, date of admission, etc., was obtained. In the second part, information about the chief complaints, history of presenting illness, past history, was obtained. Details regarding the general condition of the patient were obtained. In the third part, General physical examination and relevant systemic examination was undertaken. In the fourth part, patient was subjected to relevant blood and radiological investigations. Sample for D-dimer levels was drawn at admission. D-dimer levels were correlated with the severity of stroke. Severity of stroke was assessed using the NIHSS scale.

The data was entered in excel sheet and presented in the form of percentages, frequencies and figures such as tables, charts and graphs. Data was analyzed using SPSS software. Descriptive statistics like percentage, proportion, central tendency, SD and inferential statistics like chi-square test to know the association and t-test to know the difference between two means and other suitable tests was applied and depicted via bar charts, pie charts. Correlation between plasma D-dimer levels and severity of acute ischemic stroke using NIHSS scale was done.

RESULTS AND DISCUSSIONS

Out of 75 subjects, 14(18.70%) suffered from Minor Stroke, 42(56.00%) suffered from Moderate Stroke, 4(5.30%) suffered from Moderate to Severe Stroke, and 15(20.00%) from Severe Stroke. Table 1.

2(2.67%) subjects were of a group 30-39 years., 13(17.33%) were of 40-49 years., 12(16.00%) were of 50-59 years., 31(41.33%) were of 60-69 years., 15(20.00%) were of 70-79 years., 2 (2.67%) were of 80-89 years. The mean age of the subjects with minor stoke was 47±10 years. The mean age of the subjects with moderate stoke was 62±9 years. The mean age of the subjects with moderate to severe stoke was 61±11 years. The mean age of the subjects with severe stoke was 70±9 years. There was a significant difference in the mean age among the groups, as $p<0.05$. Table 2.

Out of 75 subjects, 22(29.30%) were females and 53(70.70%) were males. There was no significant association between sex of the subjects and the severity of stroke, as $p>0.05$. Table 3.

It was observed that 15(20%) subjects did not have any comorbidities. Diabetes mellitus along with hypertension was the most prevalent comorbidity, 26(34.70%), followed by only DM, 15(20%) and only Hypertension, 14(18.70%). There was significant association between presence of comorbidities and severity of stroke, as $p<0.05$. Table 4.

The mean D-Dimer of the subjects with minor stoke was 0.51±0.12. The mean D-Dimer of the subjects with moderate stoke was 0.998±0.31. The mean D-Dimer of the subjects with moderate to severe stoke was 2.965±0.88. The mean D-Dimer of the subjects with

Table 1: NIHSS Category Distribution

NIHSS Category	Number	Percentage
Minor Stroke	14	18.7%
Moderate Stroke	42	56.0%
Moderate to Severe Stroke	4	5.3%
Severe Stroke	15	20.0%
Total	75	100.0%

Table 2: Age-wise distribution

Age (years)	Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total	
	N	%	N	%	N	%	N	%	N	%
30-39	2	14.29%	0	0.00%	0	0.00%	0	0.00%	2	2.67%
40-49	7	50.00%	5	11.90%	1	25.00%	0	0.00%	13	17.33%
50-59	3	21.43%	7	16.67%	0	0.00%	2	13.33%	12	16.00%
60-69	1	7.14%	23	54.76%	2	50.00%	5	33.33%	31	41.33%
70-79	1	7.14%	7	16.67%	1	25.00%	6	40.01%	15	20.00%
80-89	0	0.00%	0	0.00%	0	0.00%	2	13.33%	2	2.67%
Mean age (years)	47±10		62 ± 9		61 ± 11		70 ± 9		61 ± 12	

p-value 0.009

Table 3: Gender-Wise NIHSS Category

Gender	NIHSS category										
	Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total		
	N	%	N	%	N	%	N	%	N	%	P-value
Female	10	71.43%	8	19.05%	2	50.00%	2	13.33%	22	29.30%	0.135
Male	4	28.57%	34	80.95%	2	50.00%	13	86.67%	53	70.70%	

Table 4: Comorbidity with respect to NIHSS category

Comorbidity	NIHSS category										
	Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total		
	N	%	N	%	N	%	N	%	N	%	P-value
COPD HTN	0	0.00%	1	2.40%	0	0.00%	0	0.00%	1	1.30%	0.0001
DM	1	7.10%	12	28.60%	2	50.00%	0	0.00%	15	20.00%	
DM HTN	0	0.00%	13	31.00%	1	25.00%	12	80.00%	26	34.70%	
DM HTN IHD	0	0.00%	0	0.00%	0	0.00%	3	20.00%	3	4.00%	
HTN	3	21.40%	10	23.80%	1	25.00%	0	0.00%	14	18.70%	
HTN IHD	0	0.00%	1	2.40%	0	0.00%	0	0.00%	1	1.30%	
Nil	10	71.40%	5	11.90%	0	0.00%	0	0.00%	15	20.00%	

Table 5: D-Dimer with Respect to NIHSS Category

NIHSS Category											
Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total			
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p-value	
0.51	0.12	0.998	0.31	2.965	0.88	7.89	0.17	2.39	2.83	0.001	

Table 6: Mean NIHSS

NIHSS Category										
Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total		
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p-value
3	0.1	11	2	17	1	39	1	15	13	0.001

Table 7: HbA1c with respect to NIHSS category

NIHSS Category										
Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total		
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p-value
5.8	0.5	7.4	1.1	9.2	2	13.7	1.3	8.4	3	0.001

Table 8: RBS with respect to NIHSS category

NIHSS Category										
Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total		
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p-value
120	18	193	60	260	57	341	46	213	89	0.047

Table 9: Lipid Profile

Lipid profile	NIHSS Category										
	Minor Stroke		Moderate Stroke		Moderate to Severe Stroke		Severe Stroke		Total		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	p-value
Triglycerides	160	22	195	29	217	40	229	30	196	36	0.003
LDL	107	33	126	28	157	44	159	32	131	35	0.001
HDL	45	7	45	5	42	5	41	7	44	6	0.001
TC	137	27	166	36	187	33	208	23	170	39	0.001

Table 10: Correlation of D-dimer

Correlation	Age	NIHSS	HbA1c	RBS	Triglycerides	TC	LDL	HDL
Pearson Correlation	.466**	.981**	.928**	.789**	.512**	.544**	.466**	-.260*
Sig.(2-tailed)	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.024
N	75	75	75	75	75	75	75	75

severe stoke was 7.89± 0.17. It was observed that the mean D-Dimer increased significantly with severity of stroke, as p<0.05. Table 5.

The mean NIHSS of the subjects with minor stoke was 3±0.1. The mean NIHSS of the subjects with moderate stoke was 11±2. The mean NIHSS of the subjects with moderate to severe stoke was 17±1. The mean NIHSS of the subjects with severe stoke was 39±1. It was observed that the mean NIHSS increased significantly with severity of stroke, as p<0.05. Table 6.

The mean HbA1c of the subjects with minor stoke was 5.8±0.5. The mean HbA1c of the subjects with moderate stoke was 7.4±1.1. The mean HbA1c of the subjects with moderate to severe stoke was 9.2±2. The mean HbA1c of the subjects with severe stoke was 13.7±1.3. It was observed that the mean HbA1c increased significantly with severity of stroke, as p<0.05. Table 7.

The mean RBS of the subjects with minor stoke was 120±18. The mean RBS of the subjects with moderate stoke was 193±60. The mean RBS of the subjects with moderate to severe stoke was 260±57. The mean RBS of the subjects with severe stoke was 341±46. It was observed that the mean RBS increased significantly with severity of stroke, as p<0.05. Table 8.

It was observed that the mean Lipid Profile (Triglycerides, LDL, HDL, Total Cholesterol) increased significantly with severity of stroke, as p<0.05. Table 9 Significant, moderate, positive correlation was observed between D-Dimer and Age, r=0.466, p<0.05. Significant, highly strong, positive correlation was observed between D-Dimer and NIHSS, r=0.981, p<0.05. Significant, highly strong, positive correlation was observed between D-Dimer and HBA1C, r=0.928, p<0.05. Significant, strong, positive correlation was observed between D-Dimer and RBS, r=0.789, p<0.05. Significant, moderate, positive correlation was observed between D-Dimer and Triglycerides, r=0.512, p<0.05. Significant, moderate, positive correlation was observed between D-Dimer and Total Cholesterol, r=0.544, p<0.05. Significant, moderate, positive correlation was observed between D-Dimer and LDL, r=0.466, p<0.05.Significant, weak, negative correlation was observed between D-Dimer and HDL, r=0.260, p<0.05. Table 10.

Stroke is a leading cause of death and disability in the world. Prevention and treatment of risk factors can reduce the occurrence of stroke. Early diagnosis and treatment of stroke is vital in preventing and limiting neurological disabilities in stroke.

In this study, mean age in our study is 61±12 years. Aiyar[5] study showed mean age of affected person was 55.39 years. Our subjects had a slight increase in age compared to studies done by Gauri[6] (19%), Chitrambalam[7] (20%). Out of 75 subjects, 22(29.30%) were females and 53(70.70%) were males. There was no significant association between sex of the subjects and the severity of stroke, as p>0.05.

In the above study males were most commonly affected by stroke which is supported by the studies conducted in western countries[7-9] It also correlates with study done by Aiyar[5] Pinhero[8] and Eapen[9] who found the incidence of stroke is more common in males than females.

However, the ratio of 2.4:1 seen in this study which is higher than the studies conducted elsewhere. It could be due to lack of proper care to women and late admission in hospitals after it became severe illness.

In above study 53.4% of stroke patients had hypertension. This result correlates with Banerjee[10] study which was conducted in Calcutta on urban population in which systemic hyper tension emerged as single most important risk factor.

Diabetes mellitus is one well known, studied risk factor causing macrovascular complications. When compared with non-diabetic patient stroke risk doubles in diabetes[11]. The above study reveals 54.4% stroke patients had diabetes. In Framingham study 10% to 14% persons with stroke had diabetes. The higher prevalence seen in our study may be due to higher prevalence of diabetes in southern India from where most of the population under study hails. The data is in agreement with several other Indian studies[12].

In this study, it was observed that the mean D-Dimer increased significantly with severity of stroke. Pearson correlation analysis revealed significant, highly strong,

positive correlation between D-Dimer and NIHSS, r=0.981, p<0.05.

D-dimer can be elevated in any case with deep venous thrombosis, pulmonary thromboembolism, myocardial infarction, disseminated intravascular coagulation, surgery, trauma, or stroke[13-18]. The report by Laskowitz[18] suggests that a biomarker panel may add valuable and time-sensitive diagnostic information to early stroke evaluation and rapid identification of patients with suspected stroke, which would expand the availability of time-limited treatment strategies. Laskowitz[18] also demonstrated that, for the evaluation of early ischemia, a strategy incorporating the current biomarker test in conjunction with non-contrast CT has significantly greater sensitivity than CT alone possesses. They have demonstrated the usefulness of some serologic markers, such as D-dimer, brain natriuretic peptide, matrix metalloproteinase-9 and protein S100-beta, for detecting cerebral ischemic stroke.

Skoloudík[19] found that the D-dimer levels increase within 6 hours after stroke onset and is greater in patients with large artery occlusion and in patients with cardioembolic stroke than it is in patients with lacunar stroke or in patients without arterial occlusion. Barber[20] showed D-dimer can help physicians target interventions for preventing early neurological deterioration after acute ischemic stroke. However, some studies postulated that D-dimer assessment cannot be used as an AIS index, with the exception of the cardioembolic subtype[17,18].

Lövblad[21] provided evidence that infarction volume may be predictive of clinical severity and outcome. Also, infarction volume has shown significant correlations with NIHSS and brain injury scores[22,23] Baird[22] reported a high correlation between volume change and change in NIHSS score.

In the present study, it was observed that the mean Lipid Profile (Triglycerides, LDL, HDL, Total Cholesterol) increased significantly with severity of stroke, as p<0.05. Significant, moderate, positive correlation was observed between D-Dimer and Triglycerides, r=0.512, p<0.05. Significant, moderate, positive correlation was observed between D-Dimer and Total Cholesterol, r=0.544, p<0.05. Significant, moderate, positive correlation was observed between D-Dimer and LDL, r=0.466, p<0.05. Significant, weak, negative correlation was observed between D-Dimer and HDL, r=0.260, p<0.05.

Shahar[24] Bowman[25] reported the lack of association between lipids and stroke. Study conducted by Bowman[25] of 296 stroke patients and the same number of controls, found that levels neither of total cholesterol, triglycerides nor HDL were associated with risk of ischemic stroke, although a high total cholesterol/ HDL ratio was found to increase the risk. However, in their study, due to inability to differentiate between hemorrhagic and ischemic stroke, serum cholesterol's positive association with ischemic stroke may have been concealed by a negative relationship with hemorrhagic stroke.

Okumara[26] Irribarren[27] Tirschwell[28] in their studies observed that hypercholesterolemia is a protective factor against ischemic stroke and that low blood cholesterol increases risk for ischemic stroke with an inverse relationship existing between serum cholesterol and incidence of ischemic stroke.

Bang[29] postulated that cholesterol is known to have effects on the vasculature and is essential for normal membrane fluidity, and adequate cholesterol levels may be important for maintaining the integrity of vessels and their resistance to rupture.

CONCLUSION

data suggest that D-dimer which is a reflection of ongoing thrombus formation and its fibrinolysis. The levels of D-dimer are proportional to the size of the clot and the infarct volume. Measurement of infarct volume and clot size require extensive neuroimaging involving CT angiography and MRI. D-dimer measurement is a simple, noninvasive tool to estimate and understand the severity of stroke. This allows us to make faster decisions regarding reperfusion and recanalization strategies. Rapid recanalization improves functional outcomes of ischemic stroke.

In conclusion D-dimer levels can be measured in acute ischemic stroke to understand its severity and to predict its functional outcome. Patients with elevated D-dimer can be given faster medical care thereby reducing the functional disability and improving the outcome of strokes.

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