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D-Dimer in Ischaemic Stroke Subtypes

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Abstract:

Background: *Stroke is the third most common cause of death in the developed world after cancer and ischaemic heart disease, and is the most common cause of severe physical disability. Although, there are many patients in Bangladesh suffering from these disorders, systematic research on them, especially serum biological markers of ischaemic stroke are yet to be evaluated. So the objectives of the present study are to see the serum d-dimer among acute ischaemic stroke patient.* ***Methods:*** *This is a hospital based cross sectional study conducted in neurology department of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Total 162 cases of acute ischaemic stroke irrespective of their gender were included who were admitted in BSMMU during the period from October, 2012 to October, 2013. Blood sample was taken for d-dimer measurement from each patient and d-dimer was estimated in department of haematology, BSMMU.* ***Results:*** *Among 162 patients with acute ischaemic stroke, it showed that d-dimer level mean was 1.0862 mg/L ± SD 0.9844 with maximum 4.4 and minimum 0.01. D-dimer was highly raised (n=75, 46%; mean 1.6519 ± SD 1.1396; min 0.11 and max 4.4) in early days of 1st week with a descending manner and almost reached normal level (n=87, 54%; mean 0.5986 ± SD 0.4210; min 0.01 and max 2.25) in later half of 2nd week.* ***Conclusion****: The study showed that raised d-dimer (DD) was significantly associated total anterior circulation infarction (TACI) & partial anterior circulation infarction PACI and raised DD was significant differentiating point TACI or PACI from lacunar infarction (LACI) & posterior circulation infarction (POCI) and raised D-Dimer might not differentiate between TACI & PACI.*

Key word: *Stroke, D-dimer, Ischemia etc.*

Introduction:

Acute stroke is characterized by the rapid appearance (usually over minutes) of a non-convulsive, non-traumatic focal deficit of brain function, most commonly a hemiplegia with or without signs of focal higher cerebral dysfunction (such as aphasia), hemi sensory loss and visual field defect or brain-stem deficit. Confusion, memory or balance disturbance are more often due to causes other than stroke. The neurovascular syndromes enable the physician to localize the lesion so precisely that even the affected arterial branch can be specified[2]. It is the abruptness with which the neurologic deficit develops, usually a matter of seconds that stamps the disorder as vascular. Most embolic strokes characteristically occur suddenly, and the deficit reaches its peak almost at once. Thrombotic strokes may have an abrupt onset, but they evolve somewhat more slowly over a period of several minutes or hours and occasionally days; in the latter case, the stroke usually progresses in a saltatory fashion, i.e., in a series of steps rather than smoothly. In hypertensive cerebral hemorrhage, also abrupt in onset, the deficit may be virtually static or steadily progressive over a period of minutes or hours, while subarachnoid haemorrhage is almost instantaneous[3]. Another important aspect of the temporal profile is the arrest and then some regression of the neurologic deficit in almost all except the fatal strokes.

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At one extreme of rapid regression is a focal syndrome that reverses itself entirely and dramatically over a period of minutes or up to an hour; this defines the "transient ischaemic attack" (TIA). Often, an extensive deficit from embolism partially reverses itself within a few hours or days. There are, however, many exceptions, such as the additive effects of multiple vascular occlusions and the progression of a focal deficit that is caused by secondary brain edema surrounding large infarctions and cerebral hemorrhages[2,3].

All acute occlusions occur because of the occlusion of an artery either by local atherosclerotic/ atherothrombotic or by a thrombus stemming from a proximal artery or the heart. When coagulation occurs in an artery or vein, D-dimer levels increase. D-dimer is a reaction product of intravascular thrombus formation, and thrombolysis can be a clue for hypercoagulation in the patient. For patients considered as having ischaemic stroke, the threshold value of D-dimer is an important diagnostic issue[4].

Methods:

This was a hospital based, cross sectional study carried out on the patientswith the diagnosis of first attack of ischaemic stroke within 2 weeks of onset in indoor and outdoor of Neurology department of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. The period of study spans from October, 2012 to October, 2013. Age 18 or more, diagnosed patient of ischemic stroke (IS) within first 2 weeks of presentation and diagnosed by combined Oxfordshire Community Stroke Project classification (OCSP) criteria and confirmed by Neuroimaging, patient or patient's attendance willing to participate were included in the study and haemorrhagic stroke, venous stroke, recent or prior history of disseminated intravascular coagulation (DIC), venous thromboembolism (VTE), Pulmonary embolism (PE), cardiac thrombus, sickle cell crisis, major surgery and trauma, Systemic infection/Septicaemia, snake bites, Myocardial infarction in last 3 months and during study period, SGPT > 88 IU/L, serum creatinine > 1.5 mg/dl, recent bleeding diathesis or disorder (GI bleeding or haematuria), pregnancy & or its complication, treated with or on anticoagulant or fibrinolytic or by antithrombotic, recent or prior IM injection or IV cannula within 48 hours and recent Blood transfusion within 1 month were excluded. With the use of a sample size formulae 162 patients were taken as proportion of occurring Ischaemic Stroke among all Strokes = 88%. Between October 2012 to October 2013 in Neurology department, 162 patients of age 18 or more than 18 with first ever IS presented within first 2 wks of symptoms at any age, was included in this study after taking informed written consent. Detailed history and clinical examination was carried for each patient in presence of a consultant neurologist.

The medical records and demographic, clinical, laboratory and radiological records of each patient was examined.All relevant baseline investigations (e.g.- complete blood count, urine R/M/E etc.) were performed. Eachpatientwas diagnosed as IS subtype as total anterior circulation infarcts (TACI), partial anterior circulation infarcts (PACI), lacunar infarcts (LACI), and posterior circulation infarcts (POCI) by Oxfordshire Community Stroke Project classification (OCSP) criteria and was confirmed by neuroimaging (brain CT & or MRI) with excluding hemorrhagic stroke or other intracerebral diseases. Blood sample was taken for d-dimer measurement from each patient within the first 2 weeks of presentation of IS symptoms and before the anticoagulant or t-PA treatment was started and plasma sample was stored at "32°C till estimation in department of haematology, BSMMU.All the data was checked and edited after collection. It was expressed as Mean ± SD. Qualitative data was analyzed by chi-square test and ANOVAand quantitative data was analyzed by t-test and Post Hoc analysis was done by Fisher's least significant difference (LSD) test. p value of < 0.05 was considered statistically significant. Statistical analysis was done using SPSS (Statistical package for social sciences) win version 12 software programme.

Results:

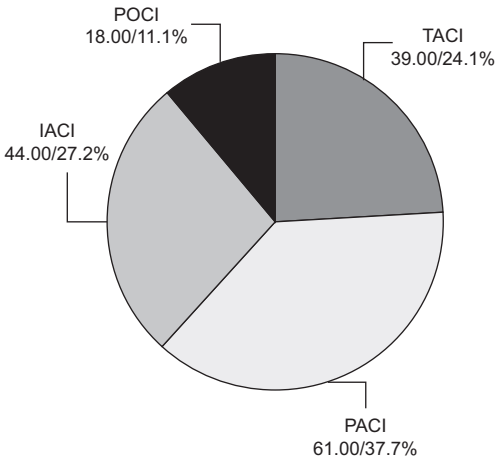

Fig.-1: *Distribution of acute ischaemic stroke patients according to OCSP Ischaemic stroke subtypes (n=162).*

It shows total anterior circulation infarction (TACI) was 39 (24.1%), partial anterior circulation infarction (PACI) was 61 (37.7%), lacunar infarction (LACI) was 44 (27.2%) and posterior circulation infarction (POCI) was 18 (11.1%).

Table-I

Distribution of serum d dimer of acute ischaemic stroke patients (n=162).

D-dimer	N	Min	Max	Mean	Std. deviation (SD)
	162	.01	4.40	1.0862	.9844

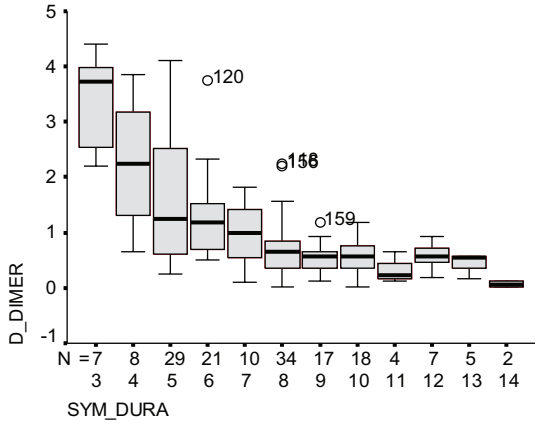

Fig.-2: *Distribution of serum d dimer in each in admission day of acute ischemic stroke patients (n=162)*

Table-I shows serum d-dimer level mean was 1.0862 mg/L ± SD 0.9844 with maximum 4.4 and minimum 0.01.

Figure-2 shows serum d-dimer level value maximum 4.4 and minimum 0.01 with high 1st week level and almost reach normal level at latter part of 2nd weeks with a descending manner.

Table-II

Distribution of serum d-dimer on weekly basis among acute ischemic stroke patients (n=162).

Symptom Duration	Mean	N	SD	Min	Max
First week	1.6519	75	1.1396	.11	4.40
Secondweek	.5986	87	.4210	.01	2.25
Total	1.0862	162	.9844	.01	4.40

Table-II shows serum d-dimer was highly raised (n=75, 46%; mean 1.6519 ± SD 1.1396; min 0.11 and max 4.4) in early days of 1st week with a descending manner and almost reached normal level (n=87, 54%; mean 0.5986 ± SD 0.4210; min 0.01 and max 2.25) in later half of 2nd week.

Table-III

Distribution of serum d dimer of acute ischemic stroke patients according to OCSP Subtypes (n=162).

OCSP Class	Mean	N	SD	Min	Max
TACI	2.1687	39	1.2319	.35	4.40
PACI	1.0995	61	.5781	.35	2.54
LACI	.3659	44	.1996	.01	.70
POCI	.4567	18	.2329	.11	.75
Total	1.0862	162	.9844	.01	4.40

Table-III shows among the 39 patients with TACI, it shows serum d-dimer level mean was 2.1687 mg/L ± SD 1.2319 with maximum 4.4 and minimum 0.35. Among the 61 patients with PACI, it shows serum d-dimer level mean was 1.0995 mg/L ± SD 0.5781 with maximum 2.54 and minimum 0.35. Among the 44 patients with LACI, it shows serum d-dimer level mean was 0.3659 mg/L ± SD 0.1996 with maximum 0.70and minimum 0.01 and it also shows among the 18 patients with POCI, it shows

serum d-dimer level mean was 0.4567 mg/L ± SD 0.2329 with maximum 0.75 and minimum 0.11.

Table-IV

Distribution of serum d dimer on weekly basis in TACI (n=39)

Symptom Duration	Mean	N	SD	Min	Max
First Week	2.8056	25	1.0162	1.18	4.40
Second Week	1.0314	14	.5970	.35	2.25
Total	2.1687	39	1.2319	.35	4.40

Table-IV shows serum d-dimer was highly raised (n=25, 64%; mean 2.8056 ± SD 1.0162; min 1.18 and max 4.4) in 1st week and it also remained above normal level (n=14, 36%; mean 1.0314 ± SD 0.5970; min 0.35 and max 2.25) in of 2nd weeks.

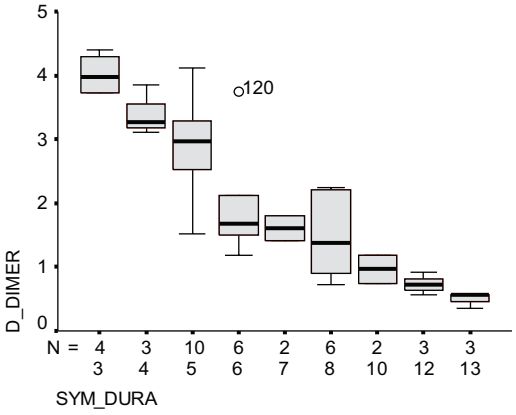

Fig.-3: *Distribution of serum d-dimer in each in admission day of TACI (n=39)*

Figure-3 shows serum d-dimer was highly raised (mean 2.8056; min 1.18 and max 4.4) in early days of 1st week with a descending manner and it also remained above normal level (mean 1.0314; min 0.35 and max 2.25) in whole part of 2nd week with a descending manner.

Table-V

Distribution of serum d dimer on weekly basis in PACI (n=61)

Symptom Duration	Mean	N	SD	Min	Max
First Week	1.6175	24	.5648	.85	2.54
Second Week	.7635	37	.2414	.35	1.55
Total	1.0995	61	.5781	.35	2.54

Table-V shows serum d-dimer was moderately raised (n=24, 39%; mean 1.6175 ± SD 0.5648; min 0.85 and max 2.54) 1st week and it also remained above normal level (n=37, 61%; mean 0.7635± SD 0.2414; min 0.35 and max 1.55) in 2nd week.

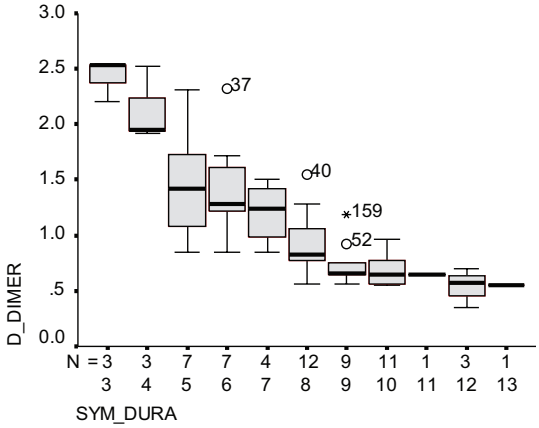

Fig.-4: *Distribution of serum d-dimer in each in admission day of PACI (n=61)*

Figure-4 shows serum d-dimer was moderately raised (mean 1.6175; min 0.85 and max 2.54) in early days of 1st week with a descending manner and it also remained above normal level (mean 0.7635; min 0.35 and max 1.55) in initial half and almost reaches normal in later half of 2nd week.

Table-VI

Distribution of serum d dimer on weekly basis in LACI (n=44)

Symptom Duration	Mean	N	SD	Min	Max
First Week	.5929	14	4.615E-02	.55	.70
Second Week	.2600	30	.1471	.01	.56
Total	.3659	44	.1996	.01	.70

Table-VI shows serum d-dimer was almost normal (n=14, 32%; mean 0.5929; min 0.55 and max 0.70) in 1st week and it also remained within normal level (n=30, 68%; mean 0.26 ± SD 0.1471; min 0.01and max 0.56) in 2nd week.

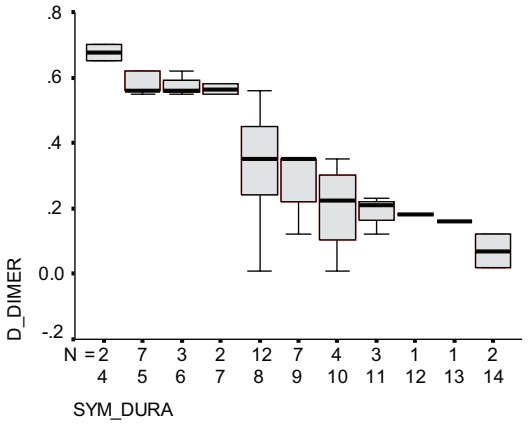

Fig.-5: *Distribution of serum d dimer in each in admission day of LACI (n=44).*

Figure-5 shows serum d-dimer was almost normal with early little rise (mean 0.5929; min 0.55 and max 0.70) in 1st week with a descending manner and it also remained within normal level (mean 0.26; min 0.01and max 0.56) throughout the 2nd week.

Table-VII

Distribution of serum d dimer on weekly basis in POCI (n=18)

Symptom Duration	Mean	N	SD	Min	Max
First Week	.5929	14	4.615E-02	.55	.70
Second Week	.2600	30	.1471	.01	.56
Total	.3659	44	.1996	.01	.70

Table-VII shows serum d-dimer was almost normal (n=12, 67%; mean 0.5525 ± SD 0.2004; min 0.11 and max 0.75) in 1st week and it also remained within normal level (n=6, 33%; mean 0.2650± SD 0.1732; min 0.12 and max 0.59) in the 2nd week.

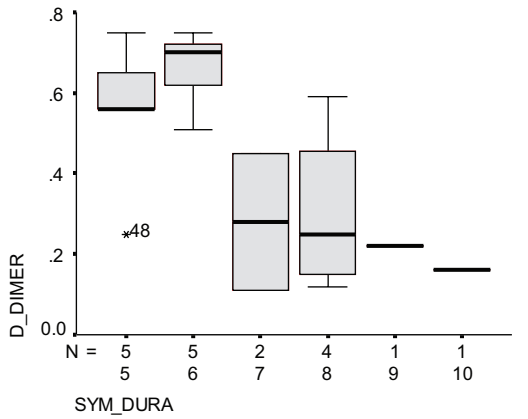

Fig.-6: *Distribution of serum d dimer in each in admission day of POCI (n=18).*

Figure-6 shows serum d-dimer was almost normal with early little raise (mean 0.5525; min 0.11 and max 0.75) in 1st week with a descending manner and it also remained within normal level (mean 0.2650; min 0.12 and max 0.59) throughout the 2nd week.

Discussion:

In Bangladesh prevalence of stroke above age of 40 is 370 per 1,00,000. About 350 patients admitted with stroke in BSMMU in Neurology unit per year[5]. In this study, among the 162 patients with acute ischaemic stroke, total anterior circulation infarction (TACI) was found 39 (24.1%), partial anterior circulation infarction (PACI) were reveals 61 (37.7%), lacunar infarction (LACI) were found 44 (27.2%) and posterior circulation infarction (POCI) were found 18 (11.1%). According to Dewan et al., out of 151 patients with ischaemic stroke admitted in Dhaka medical college from September 2010 to august 2011, reported as non-lacunar stroke (79.47%) was predominant than lacunar stroke (20.52%)[6]. In a study at the National Hospital Abuja, Nigeria between January 2010 and June 2012 where all patients presenting with acute stroke were prospectively recruited and among 163 patients with ischemic stroke, stroke subtypes composed of TACI (19.9%), PACI (43.5%), POCI (17.4%) and LACI (19.3%)[7]. In the study at Sun Yat-Sen University, Zhuhai and at Zhuhai People's Hospital, China from July 2002 to July 2009 where young patients (aged between 18 and 45 years) with first-ever acute stroke who were hospitalized, were included and findings of cases of IS using the OCSP criteria were 15 (6.7%) total anterior circulation infarcts (TACI), 117 (52.0%) partial anterior circulation infarcts (PACI), 61 (27.1%) lacunar infarcts (LACI), and 32 (14.2%) posterior circulation infarcts (POCI) [8].

In this study, among the 162 patients with acute ischaemic stroke, it shows serum d-dimer level mean was 1.0862 mg/L ± SD 0.9844 with maximum 4.4 and minimum 0.01 and normal serum d-dimer level is < 0.55 mg/L. Serum d-dimer was highly

raised (n=75, 46%; mean 1.6519 ± SD 1.1396; min 0.11 and max 4.4) in early days of 1st week with a descending manner and almost reached normal level (n=87, 54%; mean 0.5986 ± SD 0.4210; min 0.01 and max 2.25) in later half of 2nd week. In this study, among the 39 patients with TACI, it shows serum d-dimer level mean was 2.1687 mg/L ± SD 1.2319 with maximum 4.4 and minimum 0.35. Serum d-dimer was highly raised (n=25, 64%; mean 2.8056 ± SD 1.0162; min 1.18 and max 4.4) in early days of 1st week with a descending manner and it also remained above normal level (n=14, 36%; mean 1.0314 ± SD 0.5970; min 0.35 and max 2.25) in whole part of 2nd week with a descending manner. In this study, among the 61 patients with PACI, it shows serum d-dimer level mean was 1.0995 mg/L ± SD 0.5781 with maximum 2.54 and minimum 0.35. Serum d-dimer was moderately raised (n=24, 39%; mean 1.6175 ± SD 0.5648; min 0.85 and max 2.54) in early days of 1st week with a descending manner and it also remained above normal level (n=37, 61%; mean 0.7635± SD 0.2414; min 0.35 and max 1.55) in initial half and almost reach normal in later half of 2nd week. In this study, among the 44 patients with LACI, it shows serum d-dimer level mean was 0.3659 mg/L ± SD 0.1996 with maximum 0.70and minimum 0.01. Serum d-dimer was almost normal with early little rise (n=14, 32%; mean 0.5929; min 0.55 and max 0.70) in 1st week with a descending manner and it also remained within normal level (n=30, 68%; mean 0.26 ± SD 0.1471; min 0.01and max 0.56) throughout the 2nd week.

In this study, among the 18 patients with POCI, it shows serum d-dimer level mean was 0.4567 mg/L ± SD 0.2329 with maximum 0.75 and minimum 0.11. Serum d-dimer was almost normal with early little raise (n=12, 67%; mean 0.5525 ± SD 0.2004; min 0.11 and max 0.75) in 1st week with a descending manner and it also remained within normal level (n=6, 33%; mean 0.2650± SD 0.1732; min 0.12 and max 0.59) throughout the 2nd week.

In 2011 Young-Woo Park et al. mean D-dimer level at admission was 626.6 µg/L (range, 77-4,752 µg/L) and the mean level measured after seven days of treatment was 238.3 µg/L (range, 50-924 µg/L). Mean D-dimer level at admission was 215.3 µg/L in patients with focal infarctions, 385.7 µg/L in patients with multiple embolic infarctions, 566.2 µg/L in those with 1-19 cc infarctions, 668.8 µg/L in 20-49 cc infarctions, 702.5 µg/L in 50-199 cc infarctions, and 844.0 µg/L in >200 cc infarctions (p=0.044). On the 7th day of treatment, the D-dimer levels had fallen to 201.0 µg/L, 293.2 µg/L, 272.0 µg/L, 232.8 µg/L, 336.6 µg/L, and 180.0 µg/L, respectively (p=0.530) [9]. In 2010 SkoloudÃ-k D et al. a significant increase in the D-dimer levels was detected in patients with strokes of cardioembolic and atherothrombotic etiologies, and patients with occlusion of cervical or large intracranial arteries (P < 0.05) [10].

In 1993, Yamazaki M et al. in patients with cardioembolic stroke, D-dimer and alpha 2-antiplasmin-plasmin complex levels were higher during the acute and subacute phases, while thrombin-antithrombin III complex levels were higher during the acute phase than in patients with lacunar stroke and controls. In contrast, only D-dimer levels were higher in atherothrombotic stroke patients than controls during the acute and chronic phases and no significant alterations in these markers were observed in the patients with lacunar stroke[11].

In 2012 Tomoyuki Kono et al. concluded in acute ischaemic stroke, the cancer-associated ischaemic stroke is associated with elevated d-dimer and fibrin degradation products (n = 111, ÷2 = 67.21, P < 0.0001), even after controlling hypertension, hyperlipidaemia and advanced cancer (clinical stage IV) [12]. According to CotherHajatet al., risk factors between lacunar and non-lacunar infarct subtypes, atrial fibrillation was less prevalent in the lacunar (47 [17.5%]) than the non-lacunar group (139 [25.2%]) (P=0.01), and hypertension was more prevalent in the lacunar (173 [65.5%]) than the non-lacunar group (310 [57.4%]) (P=0.03). Risk factor profiles were similar between individual Bamford subtypes[13].In the multivariate model for non-lacunar versus lacunar infarct and individual Bamford subtypes versus lacunar infarct, Atrial fibrillation was independently associated with non-lacunar infarct (odds ratio [OR]=1.64; 95% CI, 1.08 to 2.50; P=0.02). When they compared individual Bamford subtypes versus lacunar infarct, atrial fibrillation was significantly more prevalent for TACI (OR=2.08; 95% CI, 1.22 to 3.54; P=0.007) and PACI (OR=1.83; 95% CI, 1.12 to 3.00; P=0.02) but not for POCI (OR=0.92; 95% CI, 0.48 to 1.74; P=0.9) [13]. In a study of 350 patients with first-ever acute ischemic stroke within 24 hours of their

symptoms, which lasted until entry, and who were admitted to the Department of Neurology, Toda Central General Hospital, from May 1994 to August 1999, it revealed no difference in background characteristics and risk factors among 4 clinical categories i. e. LACI, TACI, PACI, and POCI[14]. After multivariate analysis, higher D-dimer levels remained independently associated with cardioembolic stroke (p = 0.022) [15]. According to Tsuneaki et al., it showed that high D-dimer levels, CHF and history of stroke were associated with increased thromboembolic events[16].

Conclusion:

In this study, it shows that raised d-dimer (DD) was significantly associated total anterior circulation infarction (TACI) & partial anterior circulation infarction PACI and raised DD was significant differentiating point TACI or PACI from lacunar infarction (LACI) & posterior circulation infarction (POCI) (p value <0.0001) and raised D-Dimer might not differentiate between TACI & PACI (p value = 0.890).

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