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Histopathological Study of Thrombi Retrieved from Cerebral Arteries following Mechanical Thrombectomy and Correlation with Radiographic, Clinical Outcomes and Origin of the Thrombus

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Abstract. Background: Advancements in endovascular methods for addressing strokes resulting from major cerebral artery occlusions have facilitated the examination of recovered thrombus material, offering a significant avenue to improve the determination of stroke causes. This study aimed to investigate the immuno-histopathological features of clots retrieved through mechanical thrombectomy in individuals with acute ischemic stroke (AIS).
Methods: A total of 22 consecutive patients, aged 18 years or older, were included in this prospective study. All had experienced an acute ischemic stroke caused by large vessel occlusion (LVO) and underwent endovascular thrombectomy at our facility between September 2021 and June 2023. The thrombi extracted during the procedure were subsequently subjected to detailed histopathological analysis.
Results: There were significant differences found between clot composition (WBC, fibrin, and RBC groups) and several factors including ischemic heart disease (IHD), stroke cause, ASPECT score, collateral score, NIHSS immediately post-procedure, mRS at discharge, procedure duration, eTICI, number of passes, and brain edema (P<0.05). The ASPECT and collateral scores were higher in the WBC group compared to the fibrin and RBC groups (P<0.05). Additionally, clot composition influenced immediate post-procedural NIHSS, mRS at discharge, brain edema, and hemorrhage (P<0.05). The fibrin group had a significantly longer procedure duration, and more passes compared to the RBC and WBC groups (P<0.05).
Conclusions: Clot composition was linked to hemorrhage, mRS, and discharge outcomes. Higher CD3 and CD31 levels were associated with better outcomes and higher ASPECT scores. Post-intervention NIHSS and procedure parameters (duration, eTICI) were also associated with CD3.

Keywords: Thrombi, Cerebral Arteries, Thrombectomy, Radiography

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Introduction

Ischemic stroke remains a major health concern, frequently resulting in prolonged neurological deficits (1). With the promising outcomes of recent studies showing that mechanical thrombectomy can successfully eliminate emboli in the majority of emergent large vessel occlusion (LVO) cases, there is an emerging opportunity to employ precise and targeted assays on the recovered clots to determine the underlying cause of stroke (2).

Beyond their therapeutic advantages, endovascular interventions have facilitated the examination of thrombotic material responsible for large vessel occlusions. Despite technically successful mechanical thrombectomies (MTs), approximately 30% result in futile recanalization, where full reperfusion is achieved but does not lead to a positive clinical outcome. The factors contributing to the clinical ineffectiveness of these procedures in acute ischemic stroke (AIS) remain poorly understood, owing to gaps in our comprehension of AIS pathophysiology (3).

Analyzing the histopathologic characteristics of intracranial thrombi could carry significant clinical importance. Firstly, a substantial proportion of strokes—around 30%—remain cryptogenic despite extensive diagnostic assessments (4). Secondly, the composition of clots can influence the effectiveness of fibrinolytic treatments or the success of endovascular procedures. Given that secondary prevention strategies are tailored according to stroke etiology, it is probable that many patients receive only empiric medications, which may not align precisely with their specific underlying cause (5).

Emerging research has highlighted a connection between the outcomes of endovascular thrombectomy and pre-treatment imaging results. For example, the hyperdense vessel sign detected on computed tomography (CT) scans, which is believed to suggest a thrombus with a high erythrocyte content, has been identified as a significant factor in predicting the success of these interventions (6).

Sporns et al. (7) carried out one of the largest studies to date investigating the relationships between clot characteristics and stroke subtypes, as classified by the Trial of ORG 10172 in Acute Stroke Treatment (TOAST). The study utilized an extensive histopathologic approach, incorporating immunohistochemical methods to evaluate the levels of lymphocyte and macrophage activity within the thrombi. Notably, the research revealed that clots originating from cardiac sources exhibited different compositions compared to those of noncardiac origin (8).

A key advantage of immunochemical analysis is its ability to facilitate detailed examination of white cell subtypes, which have been recognized as potential biomarkers for atheromatous vulnerable lesions. Importantly, research has shown that T-cells play a substantial role in the composition of vulnerable atherosclerotic carotid lesions (9).

This study sought to examine the histopathological makeup of clots and investigate the relationships between clot composition, stroke etiology, clinical features, and radiological findings in patients treated with mechanical thrombectomy using stent retrievers or aspiration techniques for acute ischemic cerebrovascular stroke. Furthermore, the research aimed to quantify the CD3+ cell content within intracranial thrombi to assist in identifying atherothrombotic origins of stroke. The study also focused on analyzing retrieved thrombi from acutely occluded vessels to detect denuded endothelial cells and subendothelial connective tissue.

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Patients and Methods

This prospective study was done between September 2021 and June 2023, it included 22 consecutive patients with AIS caused by LVO who had endovascular thrombectomy at our hospital were studied. A National Institutes of Health Stroke Scale (NIHSS) score of ≥6 (10), a pre-stroke modified Rankin Scale (mRS) score of 0 to 1 (11), and LVO influencing the anterior or posterior circulation qualified participants of both sexes aged 18 years or older. Initially computed tomographic angiography (12), confirmed LVO as a visible occlusion of the common carotid artery, internal carotid artery (ICA), first division middle cerebral artery (MCA, M1), second division MCA (P2), first division anterior cerebral artery (A1), second division anterior cerebral artery (A2), basilar artery (BA), intracranial vertebral artery (VA), first division posterior cerebral artery (P1), or second division posterior cerebral artery (P2). Participants had to additionally show with a clear neurological impairment lasting fewer than 24 hours (10). The Ethical Committee of Alexandria University Hospitals, Alexandria, Egypt, approved the research (No: 0201477); all subjects gave informed written permission.
If an alternative diagnosis on admission non-contrast CT might explain the neurological impairments, including tumor, subarachnoid hemorrhage, or intracerebral hemorrhage, or if the stroke was ascribed to small artery disease, patients were not included in the research. Every qualified participant had an extensive evaluation including sophisticated radiological scans, physical examination, and a complete medical history. To enable thorough study, these tests included non-contrast CT, multiphase CT angiography (mCTA), collateral imaging, and advanced CT perfusion imaging.

Stroke severity using the national institute of health stroke scale (NIHSS) (13)

Comprising eleven items, the NIHSS covers level of consciousness, best gaze, visual fields, facial palsy, motor function for both arms and legs, limb ataxia, sensory function, best language, dysarthria, and extinction or inattention. The scores go from 0 to 42; 0 denotes no signs of a stroke and 42 denotes a severe stroke. Higher scores match more neurological damage. Clinically significant is an NIHSS score over 7 as it is linked to poor prognosis, increased mortality risk, and possibility of severe impairment. On the other hand, a score less than three usually corresponds with good functional recovery, which helps patients to restore their independence and engage in social events.
Designed to separate LVO from less severe strokes in prehospital environments, the Los Angeles Motor Scale (LAMS) examines three important components: face droop, arm drift, and grip strength, therefore producing a total score of up to 5. On the LAMS, a score of 4 or above points to an LVO (14).
Using mRS, functional outcome: 0: No symptoms at all. 1: Not disabled in any substantial way despite symptoms; able to do all routine tasks and activities. 2: Slight impairment; incapable of doing all past occupations, but able of looking after personal affairs on his own initiative without help. 3: Moderate handicap; able to walk without assistance but needing some aid. 4: Moderately severe impairment; unable to go about daily tasks without help and attention to physical demands. 5: Severe impairment;

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bedridden, incontinent; needing continuous nursing care and attention. 6: Dead (15).
The etiological factor was classified into five subtypes using the TOAST system (16) : large artery atherosclerosis (LAA), cardio embolism, small-vessel occlusion, stroke of another determined pathogenesis, and stroke of undetermined pathogenesis. This was done as part of the trial of ORG 10172 for acute stroke treatment.
The imaging requirements for thrombectomy comprised standardized post-processing procedures, sophisticated CT perfusion imaging, and non-contrast CT, as well as mCTA collateral imaging. In our study, we utilized a range of imaging criteria for selection, including the Alberta stroke program early CT score (ASPECTS) 6-10, moderate-to-good collateral status on magnetic resonance angiography (mCTA) (>50% MCA territory), small core infarct volumes (<50-70 mL), and a significant penumbra to core mismatch on perfusion imaging (17).
As a main treatment option, utilizing stent retrievers and/or aspiration technique, mechanical thrombectomy was performed on certain patients with AIS within 6 to 16 hours of their last known normal, or on patients who met the eligibility criteria for DAWN or DEFUSE 3 (10).
A biplanar DSA system (Allura Xper FD20; Philips Healthcare, Best, the Netherlands) was used to execute mechanical thrombectomy using an aspiration catheter and/or a stent retriever while the patient was given general anesthesia (18). Using the Seldinger method, a guiding catheter was placed trans-femorally. Then, the major artery proximal to the suspected blockage was probed. In order to do selective DSA, Ultravist 300 (iopromide; Bayer HealthCare, Berlin, Germany) was used as a contrast agent (19). A distal access or aspiration catheter was inserted coaxially after the arterial occlusion was identified. The catheters used were the 5F Sofia distal access catheter or 6F Sofia Plus aspiration catheter from Micro-Vent of Aliso Viejo, California, or the 3MAX/4MAX/5MAX or ACE 64 reperfusion catheter from Penumbra of Alameda, California (20) the use of a microwire (Stryker's Transend EX; MicroVention's Traxcess14) (21) the thrombus, and docked there if it was feasible. Afterwards, a VacLok syringe (Merit Medical, South Jordan, Utah) was used for direct contact thrombus aspiration.
Should recanalization be insufficient, the aspiration technique was repeated or, alternatively, a stent retrieval (Solitaire FR, Medtronic, Minneapolis, Minnesota; Trevo XP Provue System, Stryker) (22) was placed in the thrombus, positioned, and then under continuous aspiration one or many stent-retrieving movements were executed. The thrombi were gathered under sterile conditions, and those that fit for histopathologic examination were straight away stored in formalin. After every experiment, visible clots caught within or within the tip of the retrieval devices were classified as clot retrieval (23). Following every trial, the recanalization status was assessed and categorized according to the arterial occlusive lesion (AOL) recanalization score (24) : 0: no recanalization of the main lesion of obstructive nature. 1: partial or incomplete recanalization absent distal flow. 2: any distal flow imperfect or partial recanalization. With any distal flow, 3: full recanalization. Following the last retrieval trial for every patient, the final reperfusion status under the distal arterial beds of the

originally blocked artery was calculated.
thrombolysis in cerebral infarction (TICI) scale grades the ultimate reperfusion state as follows (25): 0: zero perfusion. 1: minimum perfusion penetration. 2a: partial filling with only partial perfusion. 2b: partial perfusion with full but delayed distal filling. 3: totally perfusion. The definition of the last successful reperfusion came from TICI grades 2b or 3 being reached (17).

After hematoxylin and eosin (H&E) staining all tissue slices, they were sliced into 3-4 μm sections in histological evaluation. To find the major components of each clot—fibrin/platelets, red blood cell, white blood cell—two representative sections for each clot were stained with Martius Scarlett blue (MSB). Under ×20 magnification, stained slides underwent entire slide scanning (Leica, Germany). Using the gross picture of the clot, MSB quantification of clot components was done to calculate total extracted clot area (ECA) of every clot.

Immunohistochemical staining and interpretation

CD3+ cells were identified and quantified by immunohistochemistry testing for CD3. Every immunohistochemistry slide underwent the same magnification (x200). We selected the slice with the most CD3+ cells among all the ones seen on a specific slide. Before the high-density slice was selected, every slice that showed up on each slide was noted. When a cell satisfied three criteria—that of successful staining with clearly marked cells, suitable size, and visible nucleus—it was deemed positive for CD3+ marking. After that, CD3+ T-cells were manually counted using the plugin cell-counter (26). Every thrombi specimen will be immunohistochemically examined for CD31 to identify and count endothelial cells. The CD34 tests were performed backwards. Three levels of underlying intimal injury were distinguished: Absence of endothelial cells: intimal damage improbable; solitary or clustered endothelial cells: small intimal damage cannot be excluded and endothelial cells and subendothelial connective tissue: relevant intralogical damage confirmed (27). The correlation between collected radiological, clinical and clot compositional data and outcomes expressed as mRS score and perfusion success.

The main outcome was the modified mTICI scale-based mRS score, which ranges from zero (no symptoms) to 6 (death) after three months and reflects success in reperfusion.

Statistical analysis

SPSS version 27 (IBM ©, Chicago, IL, USA) was used for statistical analysis. Histograms and the Shapiro-Wilks test helped one to evaluate the data distribution's normalcy. Reported as mean and standard deviation (SD), quantitative parametric data were examined using the ANOVA (F) test then subjected to pairwise comparisons using Tukey's post hoc test. Using the Mann-Whitney U test for group comparisons, quantitative non-parametric data were reported as median and interquartile range (IQR) and subjected to Kruskal-Wallis test. Analyzed using the Chi-square test, qualitative variables—presented as frequency and percentage (%)—were the association between a dependent variable and a single independent variable was found by means of univariate regression analysis. Additionally used to evaluate the connection between a dependent variable and many independent factors was multivariate regression analysis. A two-tailed P value less than 0.05 was judged to show statistical relevance.

Results

Demographic, medical characteristics, medical history, clinical pre procedure, baseline radiological data, NIHSS, clinical outcome, intra-procedural parameters, complication, types of complications, component of thrombi, CD 3 and CD 31 were enumerated in Table

Table 1 - Demographic, medical characteristics, medical history, clinical pre procedure, baseline radiological data, NIHSS, clinical outcome, intra-procedural parameters, complication, types of complications, component of thrombi, CD 3 and CD 31 of the studied patients

		N=22
Age (years)		67.0±13.6
<80		10(45.5%)
≥80		12(54.5%)
Sex	**Male**	10(45.5%)
	Female	12(54.5%)
Medical characteristics and history		
DM		11(50.0%)
HTN		16(72.7%)
Hyperlipidaemia		12(54.5%)
Smoking		7(31.8%)
IHD		8(36.4%)
AF		3(13.6%)
Thrombophilia		22(100.0%)
Previous TIA		11(50.0%)
Previous stroke		5(22.7%)
Clinical pre procedure data		
Admission systolic BP		160.5 ± 38.1
Admission diastolic BP		93.6 ± 19.4
Pre procedural systolic BP		146.4 ± 22.8
Admission random glucose level		206.9 ± 93.1
HR		86.2 ± 15.3
Stroke symptom onset	**Unwitnessed**	4 (18.2%)
	Wake up stroke	3 (13.6%)
	Witnessed	15 (68.2%)
Last seen well		4.9 ± 1.8
Presumed stroke cause (According to Trial of ORG 10172 in Acute Stroke Treatment classification)	**Cardioembolism**	2 (9.1%)
	LAA	14 (63.6%)
	Other determined etiology	5 (22.7%)
	Undetermined etiology	1(4.5%)
Baseline radiological data	**HU**	40.0±7.1
	MCA ratio	1.3±0.2
	HMCAS	6(27.3%)
	ASPECT	7.64±1.05

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	CBS	4.0 (1.0–5.75)
	Volumetric mismatch*	39.8±12.2
	Clot pervasiveness	15 (68.2%)
	mCTA collateral score	1.86 ± 0.89
NIHSS		
Baseline NIHSS		15.5±4.0
NIHSS immediate post procedural		9.5±4.5
NIHSS (score change)		6.5(4.0–8.75)
NIHSS (score change)	**Improved**	18(81.8%)
	Not improved	4(18.2%)
Clinical outcome		
Pre-stroke mRS	**0**	17 (77.3%)
	1	5 (22.7%)
mRS (at discharge)		2.0 (1.0 – 3.75)
mRS (at discharge)	**Bad outcome**	9 (40.9%)
	Good outcome	13 (59.1%)
Intra-procedural parameters		
Onset to puncture time (hours)		6.1±2.0
Procedure duration (min)		96.1±33.1
Procedure TICI		2.67±0.48
eTICI	**2b**	8(36.4%)
	2c-3	14(63.6%)
First-line MT strategy	**Manual Aspiration**	2(9.1%)
	Combined	19(86.4%)
	Stent retriever	1(4.5%)
Site occlusion	**Carotid T-top**	4 (18.2%)
	Extracranial ICA	2 (9.1%)
	M1	10 (45.5%)
	Tandem	6 (27.3%)
Number of passes		5.0 (3.0 – 6.0)
Rescue stenting		3 (13.6%)
Complication		16 (72.7%)
Types of complications		
Access		1 (4.5%)
Brain edema		13 (59.1%)
Intracranial haemorrhage	**Yes**	6 (27.3%)
	Yes, and symptomatic	1 (4.5%)
Seizures		3 (13.6%)
Neurological deterioration		1 (4.5%)
Distal embolization		3 (13.6%)
Death (cause)	**Neurologically related death**	1 (4.5%)
Component of thrombi	**Fibrin predominant**	6 (27.2%)
	RBC predominant	5 (22.7%)
	WBC predominant	11 (50%)
CD 3		27.4±12.9
CD 31		8.3(2.0–9.85)

Data is presented as mean ± SD or frequency (%) or median (IQR). *Ratio of total volume of perfusion defect: core volume. DM: diabetes mellitus, HTN: hypertension, IHD: ischemic heart disease, AF: atrial fibrillation, TIA: transient ischemic attack, BP: blood pressure, HR: heart rate, LAA: large artery atherosclerosis, HU: Hounsfield unit, HMCAS: hyperdense middle cerebral artery sign, ASPECT: Alberta stroke program early computed tomography score, CBS: clot burden score, mCTA: multiphase computed tomography angiography, NIHSS: national institutes of health stroke scale, mRS: modified Rankin scale, eTICI: expanded thrombolysis in cerebral infarction, ICA: internal carotid artery, M1: middle cerebral artery.

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There was no significant difference between CD 3 and demographic data, DM, HTN, hyperlipidaemia, smoking, AF, previous TIA, previous stroke, clinical pre procedural data, last seen well, HU, MCA ratio, HMCAS, CBS, clot pervasiveness, baseline NIHSS, NIHSS (change score), pre stroke mRS, onset to puncture, eTICI (2b and 2c-3) , first-line MT strategy, site occlusion, rescue stenting, access, haemorrhage, seizures, neurological deterioration, distal embolization and death. There was a significant difference between CD 3 and IHD, presumed stroke cause, ASPECT, volumetric mismatch, mCTA collateral score, NIHSS immediate post procedural, mRS at discharge, procedure duration, eTICI, number of passes and brain edema (P<0.05) (Table 2).

Table 2 - Comparison between CD 3 and demographic data, medical characteristics, medical history, clinical pre procedural data, baseline radiological data, NIHSS, mRS, intra-procedure parameters and types of complications of the studied patients

		CD 3	P
Age (years)		31.0(20.7-39.2)	0.98
<80 ≥80		31.0(24.4-36.9) 30.7(17.2-39.7)	0.598
Sex	**Male**	26.8(10.2-36.0)	0.176
	Female	37.3(22.2-39.6)	
Medical characteristics and history			
DM		20.7(12.6-38.0)	0.189
HTN		29.3(15.5-38.8)	0.319
Hyperlipidaemia		32.8(20.1-39.4)	0.621
Smoking		24.2(13.7-31.0)	0.139
IHD		14.7(5.7-26.1)	**0.012***
AF		24.2(13.8-31.9)	0.599
Previous TIA		24.2(20.7-39.4)	0.974
Previous stroke		22.7(5.8-36.0)	0.196
Clinical pre procedural data			
Admission Systolic		31.0 (20.7 - 39.2)	0.809
Admission Diastolic		31.0 (20.7 - 39.2)	0.802
Admission random Blood glucose level		31.0 (20.7 - 39.2)	0.212
Admission HR		31.0 (20.7 - 39.2)	0.368
Stroke symptom Onset	**Unwitnessed**	30.0 (16.4 - 39.5)	0.971
	Wake up stroke	29.5 (26.1 - 34.5)	
	Witnessed	32.5 (19.6 - 38.0)	
Last seen well		31.0 (20.7 - 39.2)	0.592
Presumed stroke cause	**Cardioembolic**	24.6 (24.4 - 24.8)	**0.026***
	LAA	38.0 (26.0 - 39.6)	
	Other	5.8 (5.3 - 6.7)	
	Yes	22.8 (19.0 - 34.2)	
Baseline radiological data	**HU**	31.0 (20.7 - 39.2)	0.89
	MCA ratio	31.0 (20.7 - 39.2)	0.182
	HMCAS	23.4 (10.7 - 24.8)	0.184
	ASPECT	31.0 (20.7- 39.7)	**<0.001***
	CBS	31.0 (20.7 - 39.2)	0.099

	Volumetric mismatch	31.0 (20.7 - 39.2)	**0.038***
	Clot Pervasiveness	32.5 (20.7 - 39.0)	0.99
	mCTA Collateral score	31.0 (20.7 - 39.2)	**0.002***
NIHSS			
Baseline NIHSS		31.0(20.7-39.2)	0.059
NIHSS immediate post procedural		31.0(20.7-39.2)	**0.01***
NIHSS (change score)		31.0(20.7-39.2)	0.515
NIHSS (change score)	**Improved (≥4)**	34.2(21.2-39.2)	0.469
	Not improved (<4)	22.8(16.4-28.7)	
mRS			
Pre stroke mRS	**0**	32.5(20.7-39.5)	0.196
	1	22.7(5.8-36.0)	
mRS at discharge		31.0(20.7-39.2)	**<0.001***
mRS at discharge	**Bad outcome**	18.5 (5.8 - 20.7)	**<0.001***
	Good outcome	38.7 (32.5 - 39.7)	
Intra-procedure parameters			
Onset to puncture (hours)		31.0(20.7-39.2)	0.656
Procedure duration (min)		31.0(20.7-39.2)	**0.024***
eTICI		31.0(20.7-39.2)	**0.033***
eTICI	**2b**	20.7(6.5-28.4)	0.076
	2c-3	36.6(25.5-39.5)	
First-line MT strategy	**Aspiration**	38.0(37.7-38.3)	0.623
	Both	29.5(19.6-39.4)	
Number of passes		31.0(20.7-39.2)	**0.006***
2 -3		37.2 (31.0 - 39.8)	
Site occlusion	**Carotid T**	28.9 (16.4 - 37.8)	0.585
	ICA	22.0 (14.3 - 29.7)	
	M1	34.2 (25.5 - 39.8)	
	Tandum	22.8 (19.0 - 35.2)	
Rescue stenting		37.3 (29.0 - 38.3)	0.702
Types of complications	**Access**	29.5 (20.7 - 39.3)	0.478
	Brain edema	20.7 (6.7 - 29.5)	**0.002***
	Haemorrhage	22.8 (19.0 - 34.2)	0.13
	Seizures	24.2 (15.4 - 32.1)	0.99
	Neurological deterioration	32.5 (20.7 - 39.3)	0.098
	Distal embolization	29.5 (20.7 - 38.0)	0.363
	Death	40.0 (21.8 - 40.0)	0.098

Data is presented as median (IQR). * Significant P value <0.05. DM: diabetes mellitus, HTN: hypertension, IHD: ischemic heart disease, AF: atrial fibrillation, TIA: transient ischemic attack, BP: blood pressure, HR: heart rate, LAA: large artery atherosclerosis, HU: Hounsfield unit, HMCAS: hyperdense middle cerebral artery sign, ASPECT: Alberta stroke program early computed tomography score, CBS: clot burden score, mCTA: multiphase computed tomography angiography, NIHSS: national institutes of health stroke scale, mRS: modified Rankin scale, eTICI: expanded thrombolysis in cerebral infarction, ICA: internal carotid artery, M1: middle cerebral artery.

There was no significant difference between CD 31 and demographic data, DM, HTN, hyperlipidaemia, smoking, AF, previous TIA, previous stroke, clinical pre procedural data, stroke symptom onset, last seen well, presumed stroke cause, baseline radiological data, NIHSS, pre stroke mRS, site occlusion, intra-procedural parameters and types of complications. There was a significant difference between CD 31 and IHD and mRS at discharge (P<0.05) (Table 3).

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Table 3 - Comparison between CD31 and demographic data, medical characteristics, medical history, clinical pre procedural data, baseline radiological data, NIHSS, mRS, site occlusion, intra-procedure parameters and types of complications of the studied patients

		CD 31	P
Age (years)		8.3(2.0-9.8)	0.122
<80 ≥80		8.3(6.8-10.6) 5.3(1.0-9.1)	0.198
Sex	**Male**	7.1(2.1-9.8)	0.792
	Female	8.7(1.9-9.6)	
Medical characteristics and history			
DM		2.4(1.8-9.4)	0.411
HTN		5.0(1.4-8.8)	0.065
Hyperlipidaemia		7.3(1.9-0.0)	0.766
Smoking		6.6(2.0-8.1)	0.358
IHD		2.2(1.0-7.1)	**0.047***
AF		10.2(6.3-10.6)	0.231
Previous TIA		8.8(2.0-10.5)	0.469
Previous stroke		2.4(2.0-8.0)	0.255
Clinical pre procedural data			
Admission Systolic		8.3 (2.0 - 9.8)	0.563
Admission Diastolic		8.3 (2.0 - 9.8)	0.262
Pre procedure Systolic		8.3 (2.0 - 9.8)	0.287
Admission Blood glucose level		8.3 (2.0 - 9.8)	0.71
Admission HR		8.3 (2.0 - 9.8)	0.201
Stroke symptom Onset	**Unwitnessed**	6.2 (2.3 - 10.4)	0.67
	Wake up stroke	6.6 (3.8 - 7.7)	
	Witnessed	8.6 (1.8 - 9.7)	
Last seen well		8.3 (2.0 -9.8)	0.701
Presumed stroke cause	**Cardioembolic stroke**	10.2 (9.8 - 10.6)	0.107
	LAA	8.8 (8.2 -9.8)	
	Others	2.0 (1.0 - 2.4)	
Baseline radiological data	**HU**	8.3 (2.0 - 9.8)	0.342
	MCA ratio	8.3 (2.0 - 9.8)	0.909
	HMCAS	5.9 (1.4 - 10.0)	0.99
	ASPECT	8.3 (2.0 - 9.8)	0.06
	CBS	8.3 (2.0 - 9.8)	0.291
	Volumetric mismatch	8.3 (2.0 - 9.8)	0.164
	Pervasiveness	8.0 (1.8 - 9.4)	0.502
	mCTA Collateral score	8.3 (2.0 - 9.8)	0.202
NIHSS			
Baseline NIHSS		8.3(2.0-9.8)	0.552
NIHSS immediate post procedural		8.3(2.0-9.8)	0.585
NIHSS change	**Improved (≥4)**	8.3(1.7-10.0)	0.798
	Not improved (<4)	5.5(2.3-8.8)	
mRS			

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Pre stroke mRS	**0**	8.6(2.0-10.0)	0.255
	1	2.4(2.0-8.0)	
mRS at discharge		8.3(2.0-9.8)	**0.011***
mRS at discharge	**Bad outcome**	1.6 (1.0 - 2.0)	**<0.001***
	Good outcome	9.4 (8.6 - 10.2)	
Site occlusion	**Carotid T**	5.0(2.3-8.2)	0.968
	ICA	6.7(3.8-9.6)	
	M1	8.6(6.9-9.7)	
	Tandum	5.4(1.7-9.2)	
Intra-procedural parameters			
Onset to puncture time (hours)		8.3(2.0-9.8)	0.536
Procedure duration(min)		8.3(2.0-9.8)	0.151
eTICI	**2b**	2.2(1.8-9.0)	0.231
	2c-3	8.6(6.8-10.0)	
First line MT strategy	**Aspiration**	10.6(9.7-11.5)	0.105
	Both	7.6(1.8-9.1)	
Site occlusion	**Carotid T**	5.0 (2.3 - 8.2)	0.968
	ICA	6.7 (3.8 - 9.6)	
	M1	8.6 (6.9 - 9.7)	
	Tandum	5.4 (1.7 - 9.2)	
Rescue Stenting		11.6 (6.1 - 12.0)	0.362
Types of complications	**Access**	8.0 (2.0 - 10.0)	0.636
	Brain edema	2.4 (1.6 - 9.4)	0.216
	Hemorrhage	5.4 (1.7 - 9.2)	0.837
	Seizures	10.0 (5.5 - 10.5)	0.565
	Neurological deterioration	8.6 (2.0 - 10.0)	0.58
	Distal embolization	10.0 (6.2 - 10.0)	0.415
	Death	8.6 (2.0 - 10.0)	0.58

Data is presented as median (IQR). * Significant P value <0.05. DM: diabetes mellitus, HTN: hypertension, IHD: ischemic heart disease, AF: atrial fibrillation, TIA: transient ischemic attack, BP: blood pressure, HR: heart rate, LAA: large artery atherosclerosis, HU: Hounsfield unit, HMCAS: hyperdense middle cerebral artery sign, ASPECT: Alberta stroke program early computed tomography score, CBS: clot burden score, mCTA: multiphase computed tomography angiography, NIHSS: national institutes of health stroke scale, mRS: modified Rankin scale, eTICI: expanded thrombolysis in cerebral infarction, ICA: internal carotid artery, M1: middle cerebral artery.

There was no significant difference between type of clot and demographic data, medical characteristics, history, clinical pre procedural data, HU, MCA ratio, HMCAS, CBS, volumetric mismatch, pervasiveness, admission NIHSS, NIHSS change, pre-stroke mRS, site occlusion, onset to puncture, eTICI, procedure technique, rescue stenting, access, seizures, neurological deterioration, distal embolization and death. ASPECT and collateral scores were significantly higher in WBC group than fibrin group and RBC group (P<0.05). There was a significant difference between type of clot and NIHSS immediate post-procedural, mRS at discharge, brain edema and haemorrhage (P<0.05). Procedure duration and no passes were significantly higher in fibrin group than RBC group and WBC group (P<0.05) (Table 4).

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Table 4 - Comparison between type of clot and demographic data, medical characteristics, medical history, clinical pre-procedural data, type, baseline radiological data, NIHSS, mRS, site occlusion, intra-procedure parameters and types of complications of the studied patients

		Fibrin	RBC	WBC	P
Age (years)		72.5(69.8-74.5)	63.0(46.0 - 75.0)	70.0(62.0-73.5)	0.696
<80 ≥80		2(33.3%) 4(66.7%)	3(60.0%) 2(40.0%)	5(45.5%) 6(54.5%)	0.676
Sex	**Male**	4(66.7%)	2(40.0%)	4(36.4%)	0.469
	Female	2(33.3%)	3(60.0%)	7(63.6%)	
Medical characteristics and history					
DM		5(83.3%)	2(40.0%)	4(36.4%)	0.158
HTN		6(100.0%)	3(60.0%)	7(63.6%)	0.21
Hyperlipidaemia		4(66.7%)	1(20.0%)	7(63.6%)	0.209
Smoking		2(33.3%)	2(40.0%)	3(27.3%)	0.876
IHD		4(66.7%)	2(40.0%)	2(18.2%)	0.137
AF		1(16.7%)	1(20.0%)	1(9.1%)	0.814
Previous TIA		4(66.7%)	3(60.0%)	4(36.4%)	0.431
Previous stroke		3(50.0%)	0(0.0%)	2(18.2%)	0.126
Clinical pre procedural data					
Admission systolic		165.0(132.5-197.5)	150.0(150.0-170.0)	150.0(135.0-195.0)	0.948
Admission diastolic		95.0(90.0-107.5)	90.0(80.0-100.0)	90.0(80.0-115.0)	0.683
Procedure Systolic		150.0 (132.5-167.5)	140.0(140.0-160.0)	150.0(120.0- 170.0)	0.956
Per-procedure diastolic		90.0 (82.5-90.0)	80.0 (80.0-90.0)	90.0 (75.0-90.0)	0.679
Admission Blood glucose level		248.5(187.5-341.8)	187.0(162.0-215.0)	168.0(124.0-216.0)	0.328
HR		73.5(70.0-77.0)	98.0(79.0-100.0)	88.0(81.0-95.0)	0.158
Stroke symptoms onset	**Unwitnessed**	1(16.7%)	1(20.0%)	2(18.2%)	0.903
	Wake up stroke	1(16.7%)	0(0.0%)	2(18.2%)	
	Witnessed	4(66.7%)	4(80.0%)	7(63.6%)	
Last seen well		5.4(3.9-6.5)	4.6(4.1-5.6)	4.0(3.4-4.8)	0.559
Presumed Stroke cause	**Cardiogenic stroke**	0(0.0%)	2(40.0%)	0(0.0%)	0.065
	LAA	3(50.0%)	2(40.0%)	9(81.8%)	
	Others	3(50.0%)	1(20.0%)	1(9.1%)	
Type and baseline radiological data	**HU**	42.5 (37.5 - 46.8)	40.0 (32.0 - 45.0)	39.0 (37.0 - 42.5)	0.675
	MCA ratio	1.4 (1.1 - 1.5)	1.3 (1.2 - 1.6)	1.1 (1.1 - 1.2)	0.092
	HMCAS	3 (50.0%)	2 (40.0%)	1 (9.1%)	0.149
	ASPECT	7.0 (7.0 - 7.0)	7.0 (6.0 - 7.0)	9.0 (8.0 - 9.0)	**0.005***
		P1= 0.78, **P2= 0.02***, **P3= 0.02***			
	CBS	2.0 (1.0 - 3.8)	4.0 (2.0 - 4.0)	4.0 (2.0 - 6.0)	0.264
	Volumetric mismatch	35.5 (31.8 - 37.8)	31.0 (28.0 - 44.0)	48.0 (35.5 - 53.0)	0.125
	Pervasiveness	4 (66.7%)	3 (60.0%)	8 (72.7%)	0.876
	mCTA Collateral score	1.0 (1.0 - 1.8)	1.0 (1.0 - 1.0)	3.0 (2.0 - 3.0)	**0.03***
		P1= 0.96, P2= 0.07, P3= 0.08			
NIHSS					
Admission NIHSS		19.0(18.2-19.0)	16.0(15.0-17.0)	14.0(11.0-16.0)	0.106
NIHSS immediate post-procedural		11.0(9.5-11.8)	12.0(10.0-14.0)	6.0(5.0-9.0)	**0.017***
NIHSS Change		7.5(5.5-8.8)	4.0(3.0-5.0)	7.0(4.5-9.0)	0.122
NIHSS change	**Improved (≥4)**	5(83.3%)	3(60.0%)	10(90.9%)	0.329
	Not improved (<4)	1(16.7%)	2(40.0%)	1(9.1%)	

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mRS					
Pre-stroke mRS		0.5(0.0-1.0)	0.0(0.0-0.0)	0.0(0.0-0.0)	0.139
mRS at discharge		4.0(3.2-4.0)	2.0(2.0-3.0)	1.0(1.0-2.0)	**0.002***
mRS at discharge	**Bad outcome**	6 (100.0%)	2 (40.0%)	1 (9.1%)	**0.001***
	Good outcome	0 (0.0%)	3 (60.0%)	10 (90.9%)	
Site occlusion	**Carotid T**	1(16.7%)	1(20.0%)	2(18.2%)	0.476
	ICA	1(16.7%)	1(20.0%)	0(0.0%)	
	M1	1(16.7%)	2(40.0%)	7(63.6%)	
	Tandum	3(50.0%)	1(20.0%)	2(18.2%)	
Intra-procedural data					
Onset to puncture (hours)		6.9(4.8-8.4)	5.6(5.5-6.0)	4.8(4.5-6.0)	0.584
Procedure duration (min)		125.0(112.5-137.5)	80.0(70.0-145.0)	70.0(62.5-95.0)	**0.028***
		P1= 0.72, **P2= 0.02***, P3= 0.71			
eTICI	**2b**	4(66.7%)	2(40.0%)	2(18.2%)	0.137
	2c-3	2(33.3%)	3(60.0%)	9(81.8%)	
Procedure technique	**Aspiration**	0(0.0%)	1(20.0%)	1(9.1%)	0.271
	Both	6(100.0%)	3(60.0%)	10(90.9%)	
	Stent retriever	0(0.0%)	1(20.0%)	0(0.0%)	
No passes		6.0(6.0-6.8)	5.0(3.0-6.0)	3.0(3.0-4.5)	**0.027***
		P1= 0.24, **P2= 0.02***, P3= 0.99			
Rescue Stenting		1 (16.7%)	1 (20.0%)	1 (9.1%)	0.814
Types of complications					
Access		0 (0.0%)	0 (0.0%)	1 (9.1%)	0.592
Brain edema		5 (83.3%)	5 (100.0%)	3 (27.3%)	**0.009***
Haemorrhage		2 (33.4%)	4 (80.0%)	1 (9.1%)	**0.018***
Seizures		1 (16.7%)	1 (20.0%)	1 (9.1%)	0.814
Neurological deterioration		1 (16.7%)	0 (0.0%)	0 (0.0%)	0.247
Distal embolization		1 (16.7%)	0 (0.0%)	2 (18.2%)	0.598
Death	**Neurologically related death**	1 (16.7%)	0 (0.0%)	0 (0.0%)	0.247

Data are presented as median (IQR) or frequency (%). * Significant P value <0.05. P1: significant between Fibrin and RBC, P2: significant between Fibrin and WBC, P3: significant between RBC and WBC DM: diabetes mellitus, HTN: hypertension, IHD: ischemic heart disease, AF: atrial fibrillation, TIA: transient ischemic attack, BP: blood pressure, HR: heart rate, LAA: large artery atherosclerosis, HU: Hounsfield unit, HMCAS: hyperdense middle cerebral artery sign, ASPECT: Alberta stroke program early computed tomography score, CBS: clot burden score, mCTA: multiphase computed tomography angiography, NIHSS: national institutes of health stroke scale, mRS: modified Rankin scale, eTICI: expanded thrombolysis in cerebral infarction, ICA: internal carotid artery, M1: middle cerebral artery, RBC: red blood cell, WBC: white blood cell.

Good outcome was also associated with a significantly higher CD 3 and CD 31 level compared to a bad outcome respectively, with a coefficient of 20.05 and 7.33 ($P < 0.001$ and < 0.001).

Additionally, when one unit increase in mRS at discharge would significantly decrease CD3 and CD31 level respectively by (coefficient = -6.73 and-2.00, $P < 0.001$ and < 0.001), while one unit ASPECT scores increased would significantly increase CD 3 and CD 31 level respectively by (coefficient = 7.89 and1.91, P = 0.001 and 0.022). One unit in number of passes increased, were also associated negatively with CD 3 and CD 31 level respectively by (coefficient = -3.44 and-1.11, P = 0.005 and 0.004). In the multivariable analysis, only the ASPECT score retained significance, which meant that one unit increased in ASPECT score would increase the CD 3 level by (coefficient = 4.03, P = 0.049) while controlling the other variables. In the multivariable analysis, only the

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outcome retained significance, which meant that good outcome was also associated with a significantly higher CD 31 level compared to a bad outcome, with a coefficient of 10.34 (P = 0.001) while controlling the other variables (Table 5).

Table 5 - Univariate and multivariate linear regression of CD3 and CD31 and some parameters as predicators

		Coefficient (univariable)	P	Coefficient (multivariable)	P
CD3					
Smoking		-7.38(-19.53-4.77)	0.220	-6.58(-14.15-1.00)	0.083
Outcome	**Bad**	--	--	--	--
	Good	20.05(12.59-27.51)	**<0.001***	14.48(-6.32-35.28)	0.155
NIHSS immediate postprocedural	**<10**	--	--	--	--
	10-20	-3.42(-14.60-7.76)	0.530	0.01(-9.75-9.77)	0.998
mRS at discharge		-6.73(-9.65- -3.81)	**<0.001***	2.00(-9.09-13.09)	0.702
ASPECT		7.89(3.48-12.29)	**0.001***	4.03(0.03-8.03)	**0.049***
Number of passes		-3.44(-5.70- -1.18)	**0.005***	-0.93(-4.23-2.37)	0.549
CD31					
Outcome	**Bad**	--	--	--	--
	Good	7.33(5.62-9.04)	**<0.001***	10.34(5.19-15.48)	**0.001***
NIHSS immediate postprocedural	**<10**	--	--	--	--
	10-20	-0.39(-4.25-3.47)	0.834	0.10(-2.32-2.51)	0.932
mRS at discharge		-2.00(-3.01- -1.00)	**<0.001***	1.74(-1.00-4.48)	0.193
ASPECT		1.91(0.30-3.51)	**0.022***	0.30(-0.69-1.29)	0.525
No passes		-1.11(-1.83- -0.39)	**0.004***	0.04(-0.78-0.85)	0.922

NIHSS: national institutes of health stroke scale. * Significant P value <0.05. mRS: modified Rankin scale, ASPECT: Alberta stroke program early computed tomography score.

Discussion

Common cerebrovascular diseases linked with high incidence, disability rate, and death rate include AIS (28). This work offers methodical histological examination of thrombi obtained from 22 consecutive patients receiving mechanical thrombectomy with stent-retriever AIS.

Our findings show the significance of the thrombus recovered after mechanical thrombectomy's CD3+ cell count. Thrombi from proven atherosclerotic causes revealed far more CD3+ cells than those from "cardioembolism" group and "other causes “. There was a significant difference was observed in those with cardiac conditions, who had a median CD3 of 14.7, and cardioembolic stroke was significantly associated with a lower median CD3 level of 24.6, whereas LAA showed a higher median of 38.0. The stroke classifications differed statistically significantly with relation to CD3. But research by Dargazanli et al. (26) revealed that thrombi of atherothrombotic origin had far more CD3+ T cells than cardioembolic ones. Sujijantarat et al. (29) revealed that there was a significant correlation between the proportions of clot histological components and causes of stroke.

The current study revealed that regarding the comparison between CD3 and

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radiological parameters, it was found that there was no significant difference in HU, MCA ratio and HMCAS regarding CD3. The relation of CD3 levels to clinical outcome showed a significant difference. This agreed with Zhou et al. (30) illustrated that noted higher percentages of circulating T cells and higher levels of CD3+ T-cells in individuals with good prognosis.

In the present study, CD3 levels were analysed in relation to NIHSS scores at presentation and immediate post-intervention. Post-procedural NIHSS levels had a median of 31.0 and were statistically significant associated with CD3. While there was no statistically significant difference with CD3 and the other NIHSS parameters. In agreement with our findings, Valeria Guglielmi et al. (31) showed baseline NIHSS scores for individuals with cervical carotid atherosclerotic and cardioembolic strokes were not different.

The current study revealed that CD3 levels in relation to pre stroke mRS and at discharge demonstrated significant difference in mRS at discharge. Although, post-mRS at discharge revealed significant associations. In the same manner, Zhou et al. (30) illustrated that patients in the mRS score ≤2 group had higher CD3+ T-cell count than those in the mRS score >2 group 1–3 days after stroke.

The comparison between ASPECT and CD 3 level showed a significant correlation. The relationship between CD3 levels and various clinical factors revealed a significant association with volumetric mismatch with median 31.0 and collaterals level with median CD3 levels 31.0. The comparison between CD3 and intra-procedural factors showed significant differences, especially for 31.0 as procedure duration with CD3 median as well as procedure TICI with CD3 median 31.0. Additionally, the number of procedural passes was statistically significant with CD3 levels with a median 31.0.

In the current study, the current study revealed that the comparison between CD3 and types of complications showed significant difference in brain edema regarding CD3. Where patients without brain edema had a median CD3 level of 39.5, compared to a significantly lower median of 20.7 in those with brain edema.

There was a significant difference in those with cardiac conditions, who had a median CD31 of 2.2, and LAA was significantly associated with a higher median CD31 level of 8.8, whereas other mechanisms showed a lower median of 2.0. There was a significant difference between the stroke causes regarding CD 31+.

The current study also revealed that the relation of CD31 levels to clinical outcome showed a significant difference. This is supported by Boeckh-Behrens et al. (32) who showed that The density of CD31+ cells was positively related to early stoke patient improvement.

In the present study, CD31 levels in relation to mRS before and after stroke intervention demonstrated significant difference in discharge mRS. Although, discharge mRS outcomes revealed significant associations. The comparison between CD31 and intraprocedural factors showed significant difference, especially for the number of procedures passes with median 8.3.

For HU, the median was 42.5 for fibrin, 40.0 for RBC, and 39.0 for WBC. The MCA ratio yielded median 1.4 for fibrin, 1.3 for RBC, and 1.1 for WBC. Regarding HMCAS, 50.0%

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of patients without HMCAS had fibrin compared to 50.0% with HMCAS, but this difference was not significant. The comparison between outcomes and types of thrombus components showed significant difference. However, all patients with a bad outcome had elevated fibrin levels (100.0%). 40.0% of patients with a bad outcome had RBC levels, while 60.0% of those with a good outcome with high RBC levels. Only 9.1% of patients with a bad outcome had WBC levels compared to 90.9% of those with a good outcome. Hernández-Fernández et al. (33) reported that the multivariate analysis showed that the absence of HMCA was predictor of fibrin-predominant clot. In the same manner, Jolugbo et al. (34) demonstrated that RBC-rich thrombi are typically associated with favourable outcomes in AIS patients.

The current study revealed that the comparison between NIHSS and types of thrombus components showed a significant in NIHSS post scores, where patients with fibrin, RBC, and WBC levels had median scores of (11.0, 12.0, and 6.0, respectively). Also, there was a significant difference between type of blood components and ASPECT. However, ASPECT median was elevated in WBC group (9.0) compared to fibrin group (7.0) and RBC group (7.0). Consistent with this, Zhang et al. (35) demonstrated that the erythrocyte-rich group had a much higher mean NIHSS score at the 30-day follow-up compared to the fibrin-rich group. The comparison between the types of thrombus components and mRS indicated a significant difference in discharge mRS scores among the fibrin, RBC, and WBC groups, with higher scores in the fibrin group compared to the RBC and WBC groups. Additionally, there was a significant association between the type of thrombus components and mRS outcome: the fibrin group had a 100% rate of bad outcomes, while good outcomes were more common in the RBC (60%) and WBC (90.9%) groups. In this context, Zhang et al. (35) found that functional results (mRS scores of 0-2) were better for patients with erythrocyte-rich thrombi at the 90-day follow-up than for those with fibrin-rich thrombi. Also, Maekawa et al. (36) showed that the clinical outcomes for patients with erythrocyterich thrombi were generally better, with mRS scores of 0-2 or no change at 90 days.

The comparison between types of thrombus components and intraprocedural factors showed that significant differences were observed in procedure duration and the number of passes.

Regarding the procedure duration, the WBC group had a shorter median procedure duration (70.0 minutes) compared to the Fibrin group (125.0 minutes). Regarding the number of passes, the WBC group required fewer passes compared to the Fibrin group. Weafer et al. (37) showed that there is less clot integration into the thrombectomy device in fibrin-rich thrombi, which makes mechanical removal of these thrombi more difficult. Also, Maekawa et al. (36) showed found the procedure time was lower for individuals with erythrocyte-rich thrombi compared to those with fibrin-rich thrombi.

Findings from this research show that thrombus component types and the types of complications revealed a significant association for brain edema and haemorrhage. Brain edema was more frequent in the fibrin (83.3%) and RBC (100.0%) groups compared to the WBC group (27.3%). While haemorrhage occurred more often in the RBC group (80.0%) and Fibrin group (33.4%) than in the WBC group (9.1%). In the same line, Lee et al. (38) showed that the fibrin clot led to edema formation even in the absence of mass effect provided by the microspheres.

In the univariable analysis, good outcome was also associated with a significantly higher CD 3 level compared to a bad outcome, with a coefficient of 20.05. A good outcome being associated with significantly higher CD3 levels likely reflects the protective role of an effective adaptive immune response. Higher CD3 levels may indicate a robust immune activity (39) capable of mitigating post-stroke inflammation, reducing secondary damage, and promoting recovery processes such as neuroprotection and repair. Additionally, when one unit increase in mRS at discharge scores would statistically decrease CD3 level, while one unit ASPECT scores increase would statistically increase CD3 level. one unit in number of passes increased, were also associated negatively CD 3 level. In the multivariable analysis, only the ASPECT score retained significance, which meant that one unit increased in ASPECT score would increase the CD3 level while controlling the other variables.

A good outcome was also associated with a significantly higher CD31 level compared to a bad outcome, with a coefficient of 7.33. Additionally, when one unit increased in discharge mRS scores would statistically decrease CD31 level, while one unit ASPECT scores increased would statistically increase CD31 level. one unit in number of passes increased, were also associated negatively CD31 level. In the multivariable analysis, only the outcome retained significance, which meant that good outcome was also associated with a significantly higher CD 31 level compared to a bad outcome, while controlling the other variables. Boeckh-Behrens et al. (32) showed that there was a stable relation between CD31+ cells and early clinical improvement of patients with AIS.

Relationship between CD31 levels and outcomes can vary depending on the disease or condition being studied. As found in Silva et al. (40) study CD31 expression was significantly associated with greater disease-free survival.

First, our research was a single center study with possible results different from those of others. Second, the sample size was somewhat limited. Furthermore, considered as a benefit to our research and a strong feature is the multicenter source of our samples as it offers a greater portrayal of variety.

One of the strengths of our study was incorporating procedural and clinical criteria. Further clarification of the therapeutic relevance of the reported structural characteristics depends on continuous investigation of bigger sets of stroke thrombi.

Conclusions:

Clot composition was significantly associated with the incidence of hemorrhage (among patients without hemorrhage, 66.7% had fibrin compared to 33.4% with hemorrhage), and clinical outcome (measured by mRS) (with higher scores in the fibrin group compared to the RBC and WBC groups). Brain edema was more frequent in the fibrin and RBC groups compared to the WBC group. Good outcome was associated with higher CD 3 level and CD31 level, also, CD3 level and CD31 level had a significant relation with ASPECT score. Immediate NIHSS (post intervention) levels were significantly associated with CD3. Also, CD3 significantly associated with procedural parameters as procedure duration as well as procedure TICI. Additionally, the number of procedural passes was significantly associated with CD3 levels. Lower CD3 is significantly associated with cardioembolic stroke. Emerging insights on thrombus composition provide valuable information that can stimulate the development of alternative and better strategies to efficiently remove the occluding thrombus.

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