

Risk of Cerebrovascular Stroke with Primary Percutaneous Coronary Intervention Using Thrombus Aspiration Device in STEMI with High Thrombus Burden Lesions, A Single Center Experience.

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Abstract:

Background; Early restoration of coronary flow in STEMI reduces mortality, high thrombus burden has a higher incidence of no-reflow, stent thrombosis, LV dysfunction and mortality, thrombus aspiration can reduce thrombus burden, but recent data suggest higher stroke incidence.

Objectives; We aimed in the study to determine the safety of the thrombus aspiration in high thrombus burden lesions in primary PCI for STEMI patients.

Methods : One hundred patients were divided into two groups: Group 1: Aspiration catheter was used in 46 patients with high thrombus burden, Group 2: 54 Patients with low thrombus burden, was managed with standard PCI and thrombus aspiration as bailout only. Group A: patients who developed cerebrovascular stroke, Group B: who didn't develop stroke. Procedural complications and MACE data were obtained and follow up for stroke incidence for 30 days.

Results: There were no differences between group-1 and group-2, or between group-A and group-B regards ST segment resolution, intra-procedural complications, re-infarction. No differences between group-1 and group-2 regards death and stroke. Group-1 had lower age, higher DM and smoking prevalence, lower EF, and higher wall motion score index (48.76vs53.68, P-value0.003)(29.6%vs54%, p-value0.012)(58%vs33%, p-value0.011)(47%vs51%, p-value0.018)(1.35vs1.21, p-value0.0029). Group-A had lower EF, TIMI III flow, higher WMSI, higher no-reflow, and had higher mortality than group-B (34%vs50%, p-value0.0001)(40%vs81.3%, p-value0.0165)(1.65vs1.26, p-value0.00015)(60%vs3%, p-value0.00001)(40%vs2.1%, p-value0.011) respectively.

Conclusions: Thrombus aspiration didn't cause a significant increase in stroke rate at 30 days. LV systolic dysfunction is an important risk factor for stroke during and after pPCI irrespective of the method of revascularization. The stroke had higher mortality in pPCI patients.

Introduction:

The mechanism of acute ST-segment elevation myocardial infarction (STEMI) is the sudden thrombotic obstruction of the coronary flow. Early restoration of blood flow plays a critical role in improving patient prognosis,[1] regardless of the method of reperfusion.[2],[3].

Direct stenting strategy is assumed as an effective method to limit microvascular dysfunction in comparison to conventional stenting after using balloon dilatation with better myocardial perfusion and ECG ST resolution [4][5]. Manual thrombus Aspiration has been considered to decrease the risk of distal embolization and to improve myocardial perfusion.[6]

Coronary thrombi while performing PPCI is a bad prognostic factor with a higher incidence of adverse cardiac events.[7] AMI patients with high thrombus burden (HTB) have a higher incidence of MACE, cardiac rupture, malignant arrhythmia, and LV systolic dysfunction due to inadequate myocardial perfusion.[8]

Furthermore, long-term data from the Study TAPAS [9] and the meta-analyses [10][11] suggested that the aspiration devices had good results regards the indices of myocardial perfusion which may lead to better clinical outcomes. Jolly et al. founded that in HTB, routine thrombus aspiration led to a significant reduction in 30-day all-

cause mortality. [12]Of note, grade 3 myocardial blush was achieved only in few patients in whom TIMI 3 flow was achieved with standard PPCI.[13]

However, the TOTAL Trial found that thrombus aspiration was associated with a higher risk of CV stroke inSTEMI patients who were routinely managedwith thrombus aspiration plus primary PCI versus PPCI alone.[14]Possible explanations include catheter-relatedembolization, aggressive guide catheter manipulation which dislodges aortic atheroma, and longer procedure times.[15] But we haven't other reasonable explanations for the higher stroke rate even between 3 and 6 months and higher hemorrhagic stroke rate with thrombus aspiration. [16]

Aim of the study:

We aimed in this study to determine the safety of the thrombus aspiration device in a special group of STEMI patients with high thrombus burden containing lesions during PPCI, during the hospital course, and outpatient period for 30 days with special concern to cerebrovascular stroke.

Methods:

One hundred patients presented to the ER with acute STEMI were referred to primary PCI (PPCI) and enrolled in this study.The patients were divided into two groups according to thrombus burden;

Group 1: included46 patients with high thrombus burden (TIMI thrombus grade ≥ 4) before lesion stenting. An aspiration catheter was routinely used duringPPCI.

Group 2:included 54 patients with lower thrombus burden (TIMI thrombus grade < 4) who were managed with direct lesion stenting or balloon pre-dilatation with stenting, but aspiration catheter was used only as a bailout.

Thrombus grading was assessed after wiring the culprit lesion.

Then the patients were subdivided into 2 groups regards the development of cerebrovascular stroke.

Group A: included the patients who developed cerebrovascular stroke.

Group B: included the patients who didn't develop cerebrovascular stroke.

For each enrolled patient, we collected the following data:

Clinical data: All the patients were subjected to a complete clinical assessment (full history and clinical examination), and the following data were reported:

1.Cardiac risk factors for CAD: **DM:** Defined as HbA1c $\geq 6.5\%$, or random plasma glucose ≥ 200 mg/dl and symptoms of diabetes, or fasting plasma glucose ≥ 126 mg/dl, or patient on anti-diabetic treatment [17].

Hypertension(HTN)was defined as systolic/diastolic blood pressure $\geq 140/90$ mmHg or patients having a history of hypertension and current use of any antihypertensive medications [18].**A family history**of premature CAD was defined as CAD in males less than 55 years or female less than 60 years [19].**Smoking** was recognized as a lifetime history of >100 cigarettes in their entire life and had continued smoking in the last 6 months was considered a positive smoking history), while Ex-smokers (were defined as those who had a history of smoking at least 100 cigarettes in their entire life and had completely stopped smoking for at least 6 months) [20]. **Dyslipidemia** was defined by total cholesterol ≥ 220 mg/dl, triglycerides ≥ 150 mg/dl, low-density lipoproteins (LDL cholesterol) ≥ 140 mg/dl, high-density lipoprotein (HDL cholesterol) ≤ 40 mg dl, or current use of an anti-hyperlipidemia drug [19].

2.ECG

Resting standard 12 leads electrocardiogram was done routinely at ER for each patient to detect ECG findings consistent with myocardial ischemia, arrhythmias, or chamber abnormalities.

3.Echocardiography

M-mode, two-dimensional echocardiography, and Doppler examination were performed for all patients in the left lateral decubitus position during normal respiration according to the recommendations of the American Society of Echocardiography to assess ventricular dimensions, systolic function by area length method at apical 4 and 2 chambers views, valvular morphology and flow, and to detect any wall motion abnormalities and assess wall motion score (WMS) and score index(WMSI), and to detect ischemic complications (VSR, myocardial rupture, LV aneurysm, LV thrombus).[21]

4. Laboratory data

Routine labs included complete blood count, coagulation profile, kidney functions, serum cardiac biomarkers that included cardiac troponin I (CTnI), the myocardial band of creatine kinase (CK-MB), and creatine kinase (CK).

5. Procedure-related data

Coronary artery anatomy was assessed by one single experienced cardiac catheterization physician, and the coronary artery disease extension was quantified and categorized. From the categorical viewpoint, coronary artery anatomy was defined as follows, obstructive coronary artery disease (any obstruction > 70%, or > 50% if in the left main coronary artery or proximal LAD).

a. PCI procedure: All Patients were pretreated with 300 mg of acetylsalicylic acid, 600 mg of clopidogrel, and intravenous heparin 10000 IU boluses administered during the procedure. All pPCI procedures were performed by experienced interventional cardiologists through a femoral approach.

b. Assessment of the thrombus score was done in all patients. The decision to limit the procedure to direct stenting or thrombus aspiration before stenting was taken according to Thrombolysis In Myocardial Infarction (TIMI) thrombus grade classification. If thrombus burden ≥ 4 the decision was aspiration device use before stenting, if thrombus burden was <4, PCI without thrombus aspiration was the procedure of choice. The use of Glycoprotein IIb/IIIa whether during the procedure or post-procedural was left to the operator's decision.

TIMI Thrombus score; Figure (1)

1. TIMI thrombus **grade 0**: no cine-angiographic characteristics of thrombus are present.
2. TIMI thrombus **grade 1**: possible thrombus is present, with such angiography characteristics as reduced contrast density, haziness, irregular lesion contour, or a smooth convex "meniscus" at the site of total occlusion suggestive but not diagnostic of thrombus.
3. TIMI thrombus **grade 2** (small thrombus): there is a definite thrombus, with the greatest dimensions $\leq 1/2$ the vessel diameter.
4. TIMI thrombus **grade 3** (moderate thrombus): there is a definite thrombus but with the greatest linear dimension $> 1/2$ but < 2 vessel diameters.
5. TIMI thrombus **grade 4** (large-sized thrombus): there is a definite thrombus, with the largest dimension ≥ 2 vessel diameters.
6. TIMI thrombus **grade 5**: there is total occlusion [22]

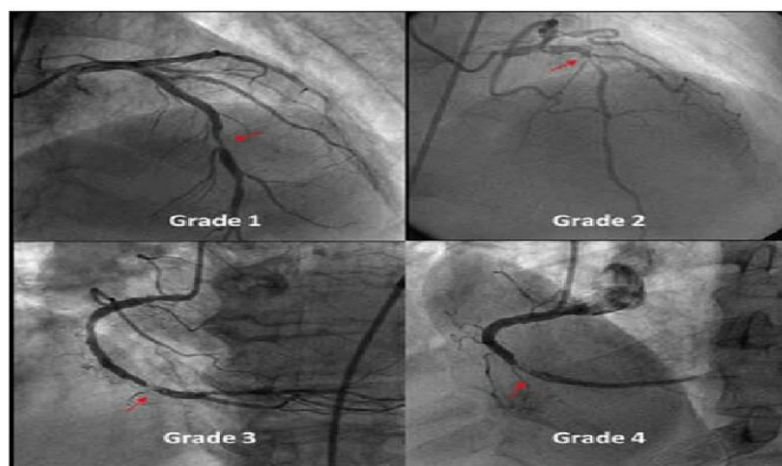


Figure (1): TIMI thrombus grading. grade 1: possible thrombus is present, with such angiography characteristics as reduced contrast density, haziness, irregular lesion contour, or a smooth convex "meniscus" at the site of total occlusion suggestive but not diagnostic of thrombus. Grade 2 there is definite thrombus, with greatest dimensions $\leq 1/2$ the vessel diameter, grade 3: there is definite thrombus but with greatest linear dimension $> 1/2$ but < 2 vessel diameters. Grade 4 there is a definite thrombus, with the largest dimension ≥ 2 vessel diameters

c. TIMI flow grade: This method estimates the epicardial coronary flow by evaluating the flow rate of the contrast material during angiography. Four grades (0–3) have been defined:

Grade 0 (no perfusion): There is no antegrade flow beyond the point of occlusion.

Grade 1 (penetration without perfusion): The contrast material passes beyond the area of obstruction but "hangs up" and fails to opacify the entire coronary bed distal to the obstruction for the duration of the filming sequence.

Grade 2 (partial perfusion): The contrast material passes across the obstruction and opacifies the coronary bed distal to the obstruction. However, the rate of entry of the contrast material or its clearance from the vessel distal to the obstruction (or both) is perceptibly slower than those in the opposite coronary artery or the coronary bed proximal to the obstruction.

Grade 3 (complete perfusion): Antegrade flow into the bed distal to the obstruction occurs as promptly as antegrade flow into the bed proximal to the obstruction, and clearance of contrast material from the involved bed is as clearance from an uninvolved bed in the same vessel or the opposite artery.[23]

Figure 2 showed a case of acute inferior STMI with RCA TIMI thrombus grade 5, after the wire passed the thrombus and fixed distally, and the artery after thrombus aspiration showed TIMI III flow with mild residual thrombi.

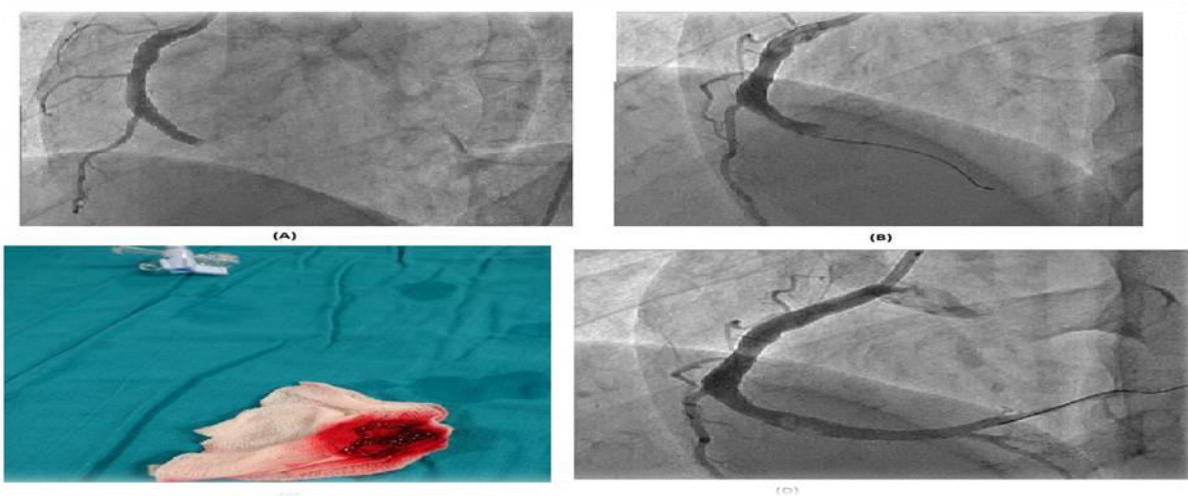


Figure (2): a case of acute inferior STMI with RCA TIMI thrombus grade 5, (a) RCA with distal total occlusion with TIMI thrombus grade 5, (b) thrombus grade didn't change after wiring the RCA distally, (c) thrombus extracted by aspiration device demonstrated on gauze, (d) RCA TIMI III flow after thrombus aspiration with residual thrombi.

6. Post-procedural:

a. ECG:

A 12-lead ECG was done immediately after PCI, 60 minutes after PCI, 3 to 6 hours later, and before discharge.

b. Follow up data

Follow up was carried out during the hospital stay and outpatient period for 30 days for assessment of:

1. Major adverse cardiac events (MACE) as a composite of:

- a. Death
- b. Recurrence of myocardial infarction (whether with the same vessel or other vessel affection).
- c. Occurrence of malignant arrhythmias.

d. Cerebrovascular nonfatal stroke.

2. Non-cardiac complications as hematoma to the puncture site, contrast-induced nephropathy, hospital-acquired infections.

Statistical analysis

Statistical analysis was carried out with SPSS 23.0 Package (IBM Inc., Chicago, Illinois, USA). Continuous variables were tested for normal distribution using Kolmogorov–Smirnov’s test. Normally distributed data were presented as mean±SD and were compared by Student’s t-test. Non-normally distributed data were presented as a median (first quartile, third quartile) and compared by Mann–Whitney U-test. Categorical variables were reported as a percentage and compared using χ^2 or Fisher’s exact test. A two-sided P value of less than 0.05 was considered statistically significant.

Results

There was a statistically significant difference between group 1 and group 2 regards age, DM, and smoking, where age was lower in group 1, while the prevalence of DM and smoking were higher in group 1. There were no statistically significant differences regards to sex, HTN, dyslipidemia, and family history of premature CAD. The male predominance was clear in both groups. (Table 1)

There were no statistically significant differences between group 1 and group 2 regards, heart rate, Killip class, SBP, DBP, CKMB, and troponin. But s. creatinine was statistically significantly lower in group 1. Table (1)

There were statistically significant differences between group 1 and group 2 regards EF and WMSI, where group 1 had lower EF and higher WMSI. There was no statistically significant difference between group 1 and group 2 regards LVEDd. There was no significant difference between group 1 and group 2 regards the incidence of LV systolic dysfunction (EF<0.5) Table (1)

There were no statistical differences between group 1 and group 2 regards the time of chest pain onset to FMC and door to balloon time. Table (2)

There were no statistically significant differences between group 1 and group 2 regards the affected coronary artery or affected coronary artery segment. (table 2)

There was no statistically significant difference between group 1 and group 2 regards ST-segment resolution after coronary intervention. Table(2). There was no significant difference between group 1 and group 2 regards no-reflow and perforation. Table(2)

As regard stroke, there was no significant difference between group 1 and group 2. There was no significant difference between group 1 and group 2 regards death and re-infarction. Table(3)

In our study all stroke cases occurred within the first week, four of them occurred within the first 48 hours while the 5th case occurred on the 6th day.

There were no statistically significant differences regards to age, sex, DM, HTN, dyslipidemia, and smoking between group A and group B. But there was a statistically higher prevalence of a family history of premature CAD in group A. (Table 1)

There were no statistically significant differences between group A and group B regards, heart rate, Killip class, SBP, DBP, CKMB, and troponin. But s. creatinine showed a trend to be lower in group A. Table (1)

There were statistically significant differences between group A and group B regards EF and WMSI, where group A had lower EF and higher WMSI. There was no statistically significant difference between group A and group B regards LVEDd. Table (1)

Table 1: Demographic data, clinical, laboratory, and echocardiographic data of group 1 and group 2, and between group A and group B

| | | Group 1 (no:46) | Group 2 (no:54) | P-value G1 vs G2 | Group A (no:5) | Group B (no:95) | P-value G: A vs G: B |
|---------------------------|-----|--------------------|--------------------|---------------------|-------------------|--------------------|----------------------------|
| Age (mean/years) | | 48.76 | 53.68 | 0.003 | 52.4 | 52.2 | 0.3 |
| Sex | M | 43 (93%) | 50 (92%) | 0.8 | 5 (100%) | 88 (92%) | 0.4 |
| | F | 3 (7%) | 4 (8%) | | 0 (0%) | 7 (8%) | |
| DM | | 25 (54%) | 16 (29.6%) | 0.012 | 2 (40%) | 39 (41%) | 0.6 |
| HTN | | 21 (54%) | 20 (37%) | 0.38 | 3 (60%) | 38 (40%) | 0.58 |
| Smoking | | 27 (58%) | 18 (33%) | 0.011 | 4 (80%) | 41 (43%) | 0.11 |
| Dyslipidemia | | 14 (30%) | 21 (39%) | 0.4 | 2 (40%) | 33 (35%) | 0.4 |
| F.H of PCAD | | 3 (7%) | 5 (9%) | 0.6 | 2 (40%) | 6 (6.3%) | 0.007 |
| Heart rate (mean) | | 94.01 | 96.4 | 0.3 | 101 | 94 | 0.16 |
| Killip Class no (%) | I | 28 (61%) | 34 (63%) | 0.7 | 2 (40%) | 60 (63%) | 0.37 |
| | II | 11 (24%) | 13 (25%) | | 1 (20%) | 23 (24%) | |
| | III | 5 (11%) | 3 (5%) | | 1 (20%) | 7 (7%) | |
| | IV | 2 (4%) | 4 (7%) | | 1 (20%) | 5 (5.2%) | |
| SBP (mean) | | 126.7 | 120 | 0.08 | 121.5 | 125 | 0.09 |
| DBP (mean) | | 78.1 | 75.6 | 0.25 | 77.3 | 76.6 | 0.3 |
| High CKMB | | 36 (78%) | 48 (85%) | 0.14 | 5 (100%) | 81 (85%) | 1 |
| High troponin | | 40 (86%) | 45 (83%) | 0.8 | 5 (100%) | 80 (85%) | 0.98 |
| S.creat (mean) mg/dl | | 1.1±0.3 | 1.26±0.4.1 | 0.0003 | 1.01±0.2 | 1.2±0.183 | 0.06 |
| LVEDd (CM) | | 5.76 | 5.5 | 0.25 | 6.0 | 5.7 | 0.1 |
| EF | | 47% | 51% | 0.018 | 34% | 50% | 0.0001 |
| WMSI | | 1.35 | 1.21 | 0.0029 | 1.65 | 1.26 | 0.00015 |
| LV EF <0.50 | | 26 (48%) | 28 (60%) | 0.2 | | | |

DM; diabetes mellitus, HTN; hypertension, FH of PCAD; family history of premature coronary artery disease. SBP; systolic BP, DBP; diastolic BP. S.creat; serum creatinine. LVEDd; left ventricular end-diastolic diameter at diastole. EF; ejection fraction. WMSI; wall motion score index.

There were no statistical differences between group A and group B regards the time of chest pain onset to FMC, and door to balloon time. Table (2)

There were no statistically significant differences between group A and group B regards affected coronary artery or affected artery segment. Table(2)

There was no statistically significant difference between group A and group B regards ST-segment resolution after coronary intervention. Group A had a lower TIMI III flow than group B. Table(2).

Table (2); Comparison of intervention findings between group 1 and group 2, and between-group a and group B.

| | | Group 1 (no:46) | Group 2 (no:54) | P-value G 1 vs G 2 | Group A (no:5) | Group B (no:95) | P-value G A vs G B |
|----------------------|----------|--------------------|--------------------|--------------------------|----------------------|--------------------|--------------------------|
| Onset to FMC (hr) | | 8.83 | 7.89 | 0.2 | 10.75 | 8.89 | 0.18 |
| Door to balloon (mn) | | 73.4 | 75.6 | 0.3 | 80.4 | 78.2 | 0.33 |
| Affected artery | LM | 1 | 0 | 0.8 | 0 | 1 | 0.08 |
| | LAD | 17 | 25 | | 3 | 39 | |
| | LCX | 13 | 12 | | 2 | 23 | |
| | RCA | 15 | 17 | | 0 | 32 | |
| Affected CA | proximal | 31 | 30 | 0.3 | 4 | 57 | 0.15 |

| | | | | | | | |
|---------------------------------|-------------|----------|---------|------|---------|------------|---------|
| segment | Mid | 7 | 14 | | 1 | 20 | |
| | distal | 8 | 10 | | 0 | 18 | |
| ST resolution post-PTCA | | 70% | 67% | 0.3 | 66.1% | 69.2% | 0.29 |
| post-PTCA TIMI III flow. No (%) | | 30 (62%) | 41(75%) | 0.24 | 2 (40%) | 79 (83.1%) | 0.0165 |
| Complications | Perforation | 0 | 1 | 1.0 | 0 | 1 | 1 |
| | Dissection | 2 | 0 | 0.2 | 0 | 2 | 1 |
| | No reflow | 0 | 3 | 0.2 | 3 | 3 | 0.00001 |

FMC; first medical contact. hr; hour. Mn; minute. LM; left-main. LAD; left anterior descending coronary artery. LCX; left circumflex coronary artery. RCA; right coronary artery. ST; ECG ST-segment. CA; coronary angiography.

There was no significant difference between group A and group B regards dissection and perforation. But no-reflow was statistically higher in group A than group B, Table (2). There was no significant difference between group A and group B regards re-infarction. There was a significant difference between group A and group B regards death where it was higher in the stroke group. Table (3)

Table (3): Hospital course complication of group 1 and group 2, and group A and group B.

| | Group 1 (no:46) | Group 2 (no:54) | P-value G 1 vs G 2 | Group A (no:5) | Group B (no:95) | P-value G A vs G B |
|---------------|-----------------|-----------------|--------------------|----------------|-----------------|--------------------|
| Death | 2 | 2 | 1 | 2 | 2 | 0.011 |
| Stroke | 4 | 1 | 0.17 | 0 | 2 | 1 |
| Re-infarction | 1 | 1 | 1 | | | |

There was no significant difference between group A and group B regards AF arrhythmia and coronary artery thrombus grade. Table (4)

Table (4): AF rhythm and CA thrombus grade in group A and group B.

| | | Group A (no:5) | Group B (no:95) | P-value |
|----------------|----|----------------|-----------------|---------|
| AF | | 1 | 3 | 0.19 |
| Thrombus grade | ≥4 | 4 | 42 | 0.17 |
| | <4 | 1 | 53 | |

AF; atrial fibrillation, CA; coronary artery grade.

Discussion:

This study was a single-center experience, retrospective study that was conducted on patients presenting with acute STEMI

The aim was to evaluate the safety of manual thrombus aspiration in acute STEMI patients presented with high thrombus burden in comparison to conventional stenting during periods of hospital admission and up to 30 days with special concern to cerebrovascular stroke.

Despite the promising results of the early trials TASTE, TAPAS, and INFUSE-AMI regards coronary artery thrombus aspiration catheter in STEMI patients where it showed improved outcomes, however, the TOTAL trial failed to show mortality benefits [16]. But in the scenario of patients with high thrombus burden containing lesions during PCI, aspiration catheter still has a reasonable role to minimize residual intracoronary thrombus. [24]

In our study there was no significant difference between group 1 and group 2 regards cerebrovascular stroke incidence at 30 days follow up, and this is in agreement with the TASTE trial and Göran K et. al [25], also Tung et al found the incidence rates of stroke during the index hospitalization were 1.1 and 2.34 per 100 person-months in the thrombectomy and PCI alone groups, respectively (SHR: 0.59; 95% CI: 0.22 to 1.59; P=.3) , or at 30 days (0.36

vs 0.84 per 100 person-months; SHR: 0.63; 95% CI: 0.27 to 1.45; P=.28) or even at 12 months follow-up (0.09 vs 0.15 per 100 person-months; SHR: 0.94, 95% CI: 0.53 to 1.69; P=.85), but Bekir S. et.al.[26] found similar data in their study during the hospital stay but, stroke was higher in the group that was managed with TA at 30 months follow up. In our study all stroke cases occurred within the first week, four of them occurred within the first 48 days while the 5th case occurred on the 6th day, and this was in agreement with Jeffrey T et.al.[27], study who found a median time to stroke is 6 days. All our stroke cases were non-hemorrhagic strokes (100%) and this agreed with Jeffrey T et.al., study where most cases were non-hemorrhagic stroke (88%). And as regard mortality, the stroke group had a higher rate of death than those without stroke (40% vs 2.1% respectively) and it is expected especially in our study as they were STEMI patients with LV systolic dysfunction and complicated with acute cerebrovascular stroke. And it was found in the study done by Shmuel Fuchs et.al.[28], that the mortality rate was higher during the hospital stay and up to one year in stroke patients, also in agreement with Jeffrey T et.al., 2013[27] study where the stroke was associated with an increased risk of 30-day death (HR, 8.0; 95% CI, 4.2–12.8; P<0.001). Stroke in our study had a higher incidence than most known reports, and actually, all of them occurred in patients with LV systolic dysfunction, even the single case of group 2 had low EF which may suggest an embolic nature of the stroke more than procedure-related, which is in agreement with the study done by Jeffrey T et.al.[27], who found a higher incidence of congestive heart failure with stroke group vs those without stroke (7% vs 2% respectively). And Shmuel Fuchs et.al.[28] also found that LV was a source of stroke in 4 patients. We think the high incidence of LV systolic dysfunction (48% in group 1 and 60% in group 2) and the high incidence of stroke (4%) was a reflection of the long onset to balloon time which was about 10 hours while it was about 2-3 hours in the previous studies[1],[16],[38].

We didn't find a difference in total death rate at 30 days between group 1 and group 2, although group 1 had a lower systolic function and higher WMSI at presentation, and this may denote a potentiality for TA to offer a protective action in the patients with high thrombus burden. Bekir S. et.al.[26], found similar data in their study, also this was mentioned in the analysis done by Göran K et.al.[25], for TASTE trial, and in the TOTAL trial where there was no significant difference regards the mortality, also Tung et al who found the incidence rates of all-cause mortality for the thrombectomy group and the PCI alone group at 30 days were 5.32 and 7.76 per 100 person-months, respectively (HR: 0.99; 95% CI: 0.78 to 1.25, P=.91) Furthermore, the post-discharge mortality rate at 1 year was found to be lower in the subgroup of patients treated with aspiration thrombectomy by physicians with a high PPCI volume (HR: 0.47; 95% CI: 0.24 to 0.94; P=.03 [29]. Carlos M et.al.[30], also found that patients with thrombus containing lesions tend to have lower systolic function than those without thrombi (56.8±11.9 versus 54.7±11.9; P=0.052).

The present study showed that males represented 93% of the total study population affected with STEMI while females represented only 7%, and this male predominance doesn't reflect the nature of the disease but it reflects the same pattern of employees gender distribution in the company covered by the health center.

The mean age of the studied groups is 52.4 years, and this relatively young age may reflect the aggressive nature of CAD in these patients, this premature coronary artery disease may be due to the high incidence CAD risk factors (HTN 41%, DM 41%, smoking 45%, dyslipidemia 35%) and this is in agreeing with (Yunyun et al., 2014)[31], who stated that STMI in the young patients' group is explained by their lifestyles which is characterized by high work stress, fast pace, overwork, smoking, drinking alcohol and overeating, that likely cause a disturbance in the internal environment, such as coronary atherosclerosis, that increase the incidence of AMI. Also we found The age of patients with higher thrombus burden was lower than those with lower thrombus burden, it may be due to the higher prevalence of DM and smoking in this group, and this is in agreement with Carlos M et.al.,[30] who found patients with thrombus containing lesions had lower age than those without thrombi, but this was against the findings in the study done by Ibrahim Halil et.al.,[32] on 109 patients with STEMI to detect the determinants of angiographic thrombus, where he found that high thrombus burden group had higher age. The higher prevalence of DM and smoking in group 1 may explain the high thrombus burden of this group, as Hyperglycemia promotes thrombosis, also erythrocyte aggregation is higher in diabetic than in non-diabetic patients, and hyperglycemia augments the expression of proinflammatory cytokines by stimulating inflammatory reaction [33]. Autopsy studies found the platelet adhesion and aggregation are higher in diabetic patients than non-diabetic ones[34].

Smoking is independently associated with fatal coronary thrombosis[35]. Smoking causes damage to the vascular endothelium through oxidative stress, vasoconstriction, and fibrinogen elevation that enhance thrombosis. Smoking also promotes platelet aggregation, blood viscosity, and activation of centrosomes[36], which induces the expression of intercellular adhesion molecule-1, which initiates immune-inflammatory responses, with subsequent coronary spasm and thrombosis[37].

There were no differences between high thrombus and low thrombus groups regards, gender, hypertension, and dyslipidemia, also, there were no differences between both groups in clinical findings as systolic BP, diastolic BP, heart rate, and Killip class and this was in agreement with Ibrahim Halil et. al[32] and Ole Fröbert et. al[38]. Also there were no differences between high thrombus and low thrombus groups regards cardiac biomarkers (CKMB and troponin), while, Ibrahim Halil et.al found a significant difference in these biomarkers, this may be because we had qualitative assessment (positive or negative at the time of presentation) of these markers,while Ibrahim Halil et.al study[32]used a quantitative assessment (at presentation and the peak). Regards s.creat,. It was higher in patients with higher thrombus burdenand this goes along with Dragu et. al[39] and BarisSensoy et. al[40] who found that baseline renal impairment was associated with high thrombus burden.

In our study, there was no significant difference in the incidence of no-reflow between group 1 and group 2 and this comes in concomitant with Bekir S[26], but Ibrahim Halil et. al[32] found a significant difference between both groups, we explained that in our study by the use of TA device in high thrombus burden group while Ibrahim Halil et.al didn't use TA.

There was no significant difference between group 1 and group 2 regards AF (as history or recently discovered at presentation or after MI) and this was in agreement with Bekir S et.al.,[26] who also found no difference in AF history or even in AF incidence after MI.

The present study showed no significant difference in complications(perforation, coronary dissection) and re-infarction between group 1 and group 2 in the period of hospital admission, and this comes in concomitant with Bekir S[26] andJamil et al., 2013[41] where there was no significant MACE recorded in the period of hospitalization and four weeks after discharge.And in agreement with the TOTAL trial[16], there was no difference in time of chest pain onset to FMC, door to balloon time, location of STEMI, affected vessel, or affected coronary artery segment between group 1 and group 2.

There was no difference between the group who developed CV stroke and those without stroke in age, history of DM,smoker, history of hypertension, dyslipidemia, systolic BP, diastolic BP, heart rate, CKMB, s. creatinine, MI location, time from onset FMC and door to balloon time, and this was similar to the findings in the study done by Jeffrey T et.al. 2013[27], except the history of DM which was higher in the stroke group. Also there was no difference between the group A and group B regards Killip class and incidence of AF but, in the study done byTeffery T et.al.[27],they found a higher incidence of AF, higher Killip class, and higher age in the stroke group. Group A had a higher incidence of no-reflow and lower incidence of TIMI III flow, and this was explained by the presence of LV systolic dysfunction in all the patients in this group which was found also in Govindan N et.al study [42]

Conclusion

Left ventricular systolic dysfunction is an important risk factor for stroke during and after pPCI irrespective of the method of revascularization. Stroke leads to a higher mortality rate in patients with pPCI. The thrombus aspiration device didn't cause a significant increase in stroke at 30 days and safe to be used in patients with high thrombus burden. Although the high thrombus burden group had lower LVsystolic function and higher WMSI they didn't show higher MACE which suggestive a protective effect of thrombus aspiration in such high-risk category.

Keywords

CAD, STEMI, Stroke, PCI, Aspiration Catheter, Primary PCI, Direct Stent, Thrombus Burden.

Limitations:

To judge the safety of TA we compared two groups with different TIMI thrombus grades. The follow-up period was 30 days and the stroke risk may persist up to 180days in some previous studies. CKMB and troponin were qualitative only. All the procedures were done with clopidogrel only and only through the femoral approach. The studied sample wasn't equally sex distributed.

Acknowledgments

“Not applicable”.

List of Abbreviations:

DM; diabetes mellitus,
HTN; hypertension,
FH of PCAD; family history of premature coronary artery disease.
BP; blood pressure.
SBP; systolic BP,
DBP; diastolic BP.
S.creat; serum creatinine.
LVEDd; left ventricular end-diastolic diameter at diastole.
EF; ejection fraction.
WMSI; wall motion score index
FMC; first medical contact.
Hr; hour.
Mn; minute
LM; left-main.
LAD; left anterior descending coronary artery.
LCX; left circumflex coronary artery.
RCA; right coronary artery
ST; ECG ST-segment.
CA; coronary angiography
AF; atrial fibrillation,
CA; coronary artery grade.
STEMI; ST-segment elevation myocardial infarction
PCI; percutaneous coronary intervention.
PPCI; primary PCI
TIMI; thrombolysis in myocardial infarction.
AMI; acute myocardial infarction.
CAD; coronary artery disease
VSR; ventricular septal rupture
CTnI; cardiac troponin I
MACE; Major adverse cardiac events.
TA; Thrombus aspiration
MI; myocardial infarction
CV; cerebrovascular.

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