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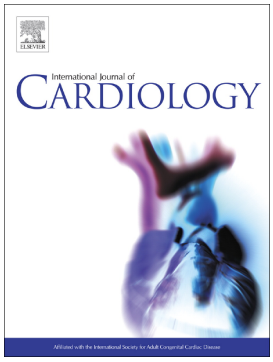

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PII: S0167-5273(26)00386-4

DOI: https://doi.org/10.1016/j.ijcard.2026.134537

Reference: IJCA 134537

To appear in: *International Journal of Cardiology*

Received date: 20 February 2026

Revised date: 25 April 2026

Accepted date: 29 April 2026

Please cite this article as: A. Del Torto, E. Ventura, F. Cannata, et al., Cardiac magnetic resonance in acute myocardial infarction undergoing thrombus aspiration, *International Journal of Cardiology* (2024), https://doi.org/10.1016/j.ijcard.2026.134537

Cardiac Magnetic resonance in Acute myocardial infarction undergoing THrombus aSpiration

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Abstract

Background: Microvascular obstruction (MVO) is a major prognostic determinant in STEMI. While thrombus aspiration (TA) during primary percutaneous coronary intervention (PCI) aims to reduce distal embolization and MVO, its impact on long-term myocardial scar and remodeling remains debated. The present study aimed to evaluate the impact of clinically indicated TA on myocardial scar and MVO using cardiac magnetic resonance (CMR) at baseline and 12-month follow-up in STEMI patients treated with PCI.

Methods: In this single-center observational cohort study, consecutive STEMI patients treated with primary PCI±TA who underwent CMR at baseline and at 12 months were enrolled. CMR parameters included left ventricular volumes, ejection fraction, global longitudinal strain, infarct size (late gadolinium enhancement) and MVO.

Results: Among 130 STEMI patients (84 PCI+TA, 46 PCI-alone) enrolled, the TA group had higher baseline thrombus burden (TIMI Thrombus Grade 5 [5;5] vs. 3 [2;5], p<0.001), higher MVO prevalence (44.6% vs. 25%, p=0.03) and larger infarct size [late gadolinium enhancement LGE: 24.2% vs 17.5% of left ventricle (LV) myocardial mass, p=0.001]. At follow-up CMR, PCI+TA

group experienced a greater reduction in myocardial scar (-5.0% [-21.8; 1.4] vs. -3.28% [-17.9; 4.6], p<0.05), particularly in patients with a high thrombus burden (Thrombus Grade >3) and baseline MVO (-10.3% [-19.8; -2.5] vs. -3.7% [-9.5; 1.2], p<0.05).

Conclusions: Despite worse baseline clinical and imaging characteristics, STEMI patients treated with TA showed more favorable myocardial tissue recovery at 12 months. These findings suggest that TA may optimize conditions for scar consolidation, particularly in high-risk patients with heavy thrombus burden.

Keywords

thrombus aspiration, CMR, infarct size, microvascular obstruction.

ABBREVIATIONS

CMR	cardiac magnetic resonance
IMH	intramyocardial hemorrhage
LGE	late gadolinium enhancement
LVEF	left ventricular ejection fraction
MVO	microvascular obstruction
PCI	primary percutaneous coronary intervention
SPECT	single photon emission computed tomography
STEMI	ST elevation myocardial infarction
TA	thrombus aspiration

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is a prevalent and prognostically impactful cardiovascular disease worldwide. The advent of primary percutaneous coronary intervention (PCI) has led to substantial improvements in the prognosis of patients with STEMI, resulting in notable reduction in both in-hospital and long-term mortality rates.[1] However, a significant proportion of patients experience a high residual risk of complications after reperfusion due to distal embolization of thrombotic material. This phenomenon, occurring spontaneously or as a consequence of PCI, is known as no-reflow and is associated with microvascular obstruction (MVO).[2]

The presence of MVO has been associated with an elevated risk of heart failure and long-term cardiovascular mortality.[2] A meta-analysis demonstrated that a high thrombotic burden (TIMI Thrombus Grade ≥3) was associated with a significantly increased risk of cardiovascular death,[3] and distal embolization during PCI has been linked to a more than threefold increase in cardiovascular mortality risk.[4]

Among the strategies proposed to limit distal embolization and reduce MVO incidence, coronary thrombus aspiration (TA) techniques have gained significant attention. However, due to conflicting data regarding the efficacy of TA, the 2023 ESC guidelines do not advocate for the routine use of TA in patients with STEMI undergoing primary PCI.[5]

To date, no studies have evaluated the impact of TA in addition to PCI by using cardiac magnetic resonance (CMR) at baseline and after a long-term follow-up in order to test the regression of myocardial damage as compared to PCI alone. The aim of the present study was to analyze patients with STEMI treated at Centro Cardiologico Monzino IRCCS, who underwent index and 12 months follow-up CMR, to assess acute myocardial tissue differences and long-term differences in left ventricular remodeling between patients who underwent primary PCI plus TA and those who underwent PCI alone.

MATERIALS AND METHODS

This study was conducted at the Centro Cardiologico Monzino IRCCS in Milan as a single-center observational cohort study. Between January 2011 and July 2019, all consecutive patients with STEMI who underwent PCI and a subsequent CMR scan during the index hospitalization (within 7 days of the acute event) as well as 12 months later were included.

Eligible patients were adults older than 18 years admitted for STEMI who underwent primary PCI, with or without manual thrombectomy, and who underwent CMR imaging during the index hospitalization and at 12-month follow-up. Exclusion criteria were STEMI treated with fibrinolytic

therapy prior to primary PCI (rescue PCI), re-infarction, target vessel re-occlusion, or severe restenosis during the 12-month follow-up period and any contraindication to perform CMR.

Furthermore, patients with a history of previous myocardial infarction were retained in the analysis only if the prior scar was located in a distinct, non-overlapping coronary territory compared to the index event, to ensure no confounding effect on the regional late gadolinium enhancement (LGE) quantification.

The study was approved by the Ethics Committee of Centro Cardiologico Monzino IRCCS (R1314/20-CCM 1382), and the handling of personal data complied with current privacy regulations and GDPR guidelines. All patients signed an informed consent.

Invasive coronary angiography and PCI Procedure:

Procedures were performed by experienced operators in accordance with international guidelines. The decision to perform thrombectomy was left to the operator's discretion, based on the patient's angiographic characteristics, such as the presence of a high thrombus burden.

Manual TA was performed using standard, CE-marked, 6F or 7F compatible monorail aspiration catheters. In all patients assigned to the TA group, suction was performed immediately after crossing the lesion with the guidewire, strictly avoiding any prior balloon pre-dilation. The procedure was carried out by advancing the aspiration catheter over the guidewire; suction was initiated proximal to the culprit lesion to capture loose thrombotic material and prevent distal embolization. The catheter was then slowly advanced across the lesion and withdrawn under continuous negative pressure. This aspiration sequence was repeated at least twice, or until the operator deemed the thrombotic burden sufficiently reduced to allow for stent implantation. Care was taken to maintain negative pressure until the catheter was fully removed from the guiding catheter to avoid systemic embolization. Subsequently, the TIMI Thrombus Grade was assessed, along with pre- and post-procedural TIMI flow grades, to evaluate the immediate angiographic success. The severity of non-culprit lesions was

assessed visually via angiography. In patients presenting with multivessel disease (MVD), complete revascularization was systematically achieved within 40 days of the index procedure.

CMR Protocol:

CMR scans were performed using a 1.5T scanner (Discovery MR450; GE Healthcare, Milwaukee, WI) with dedicated software, surface phased-array coils, and ECG gating. All studies included: balanced steady-state free precession cine sequences to assess left ventricular function, including end-diastolic volume, end-systolic volume, and ejection fraction (LVEF), triple inversion recovery T2-weighted spin-echo sequences to assess left ventricular myocardial edema, and LGE imaging, acquired 12–15 minutes after gadolinium administration, optimized to null normal myocardium using inversion recovery sequences. Myocardial scar was defined as an hyperintense region on LGE imaging. Myocardial scar was analyzed using the 17-segment American Heart Association model, assessing its absolute extent (grams) and relative extent (percentage of total left ventricular mass). Left ventricular strain analysis was performed with manual delineation of endocardial and epicardial borders at end-diastole in two-, three-, and four-chamber long-axis views, as well as in all short-axis cine slices. Following identification of the right ventricular insertion points, a fully automated algorithm generated global longitudinal strain (GLS) values of the left ventricle (Circle Cardiovascular Imaging: cvi42 version 6.3.1, Calgary, Canada).

Microvascular obstruction (MVO) was defined as a hypointense region within the infarcted myocardium on LGE images but not on T2-weighted images.

Intramyocardial hemorrhage (IMH) was defined as a hypointense region within the MVO region on LGE images and T2-weighted images.

Statistical Analysis:

Continuous variables were presented as means ± standard deviation or medians with interquartile range and compared using the Student t-test or Mann-Whitney U test, depending on data distribution. Categorical variables were reported as absolute numbers and percentages and compared using the chi-square test or Fisher's exact test. To analyze the correlation between clinical, angiographic, and imaging parameters, Spearman's correlation coefficient was used. Statistical significance was set at $p < 0.05$. All statistical analyses were performed using SPSS software, version 25 (IBM SPSS Statistics, IBM Corporation).

RESULTS

Population Characteristics

We included 130 STEMI patients (mean age: 59.2±9.3 years; 83% male), divided into two groups: 84 patients who underwent TA followed by PCI, and 46 patients who were treated with PCI alone. The index procedure ultimately involved drug-eluting stent (DES) implantation in all target vessels; in four cases (two per group), plain old balloon angioplasty (POBA) was initially performed to restore flow, followed by staged DES implantation during the same hospital stay. Patients in the TA group had higher Thrombus Grade score [5 (5;5) vs. 3 (2;5), $p < 0.001$] and a greater prevalence of pre-procedural TIMI 0 flow [67 (79.8%) vs. 19 (41.3%), $p < 0.001$]. Additionally, the TA group showed a higher prevalence of no-reflow [24 (28.6%) vs. 4 (8.7%), $p = 0.007$] and significantly higher peak high-sensitivity troponin levels [75,846.3 ng/L (22,285; 160.180.7) vs. 24,030.7 ng/L (6,107.6; 46,730), $p < 0.001$] (**Table 1**).

No significant differences were observed in the distribution of culprit vessels between the TA and PCI-alone groups (LAD: 54.8% vs. 50.0%; p=0.749). MVD was present in 18 patients (21.4%) in the TA group (all with 2-vessel disease) and 12 patients (26.1%) in the PCI-alone group (ten with 2-vessel and two with 3-vessel disease; the latter were referred for staged CABG). ST-segment resolution at 90 minutes post-PCI was categorized into <30%, 30-70%, and >70%; the distribution was comparable between groups (p=0.154). At discharge, patients received guideline-directed

medical therapy, including maximally tolerated heart failure-specific treatments where indicated, although no patient received Sodium-Glucose Cotransporter-2 (SGLT2) inhibitors, as the enrollment period predated the approval of this drug class for this indication (**Table 1**).

Baseline CMR Assessment

CMR assessments confirmed significant differences in risk profile characterization between the two groups. Prognostically unfavorable CMR markers were more prevalent in the TA group compared to the non-TA group.

Specifically, functional assessment confirmed a more severe impairment in the TA group, which displayed significantly lower LVEF [46% (41; 54) vs. 54% (42; 60), p=0.0285] and worse global longitudinal strain (GLS) [-8.27% (-10.56; -5.71) vs. -10.12% (-12.47; -7.19), p=0.0279] compared to the PCI-alone group.

Furthermore, index CMR scans revealed a significantly higher prevalence and greater extent (in grams) of MVO, and a higher prevalence of IMH in patients who underwent TA.

At baseline CMR, patients in the TA group had a more extensive myocardial edema [38% (25.5; 52.3) vs. 27% (16.75; 43.83), p = 0.04] and greater extent of LGE [24.2% (15.1; 39.1) vs. 17.53% (7.8; 25.8), p = 0.001) and compared to the non-TA group (**Table 2**).

Spearman's correlation analysis revealed a positive correlation between MVO and myocardial edema extent (R = 0.39, p < 0.0001) and between MVO and LGE extent (R = 0.526, p < 0.0001). MVO negatively correlated with LVEF (R = -0.44, p < 0.0001).

In the non-TA group, baseline MVO presence correlated positively with the Thrombus Grade score (R = +0.3, p = 0.01), while this correlation was not significant in the TA group (R = -0.03, p = 0.7) (**Table 3**).

Follow-up CMR Assessment

At follow-up CMR scans, differences between the two groups persisted for both LVEF and GLS. We observed a positive correlation between MVO and LGE extent ($R = +0.442$, $p < 0.0001$).

At follow-up CMR, as compared to baseline scan, patients in the TA group showed a significantly greater reduction in LGE mass than patients in the PCI-only group [-5.0% (-21.8; 1.4) vs. -3.28% (-17.9; 4.6), $p<0.05$]. Whereas no significant differences were observed in indexed left ventricular volumes, there was a trend towards ventricular dilatation in the TA group. Moreover, we observed complete resolution of MVO in both groups at 12 months (**Table 3**).

Additionally, the LGE reduction was significantly more pronounced in patients with high Thrombus Grade scores (>3) and baseline MVO [-10.3% (-19.8; -2.5) vs. -3.7% (-9.5; 1.2), $p<0.05$]. **Figure 1** shows an explicative clinical case.

DISCUSSION

The main findings of our study are that a favorable relative reduction in infarct size in STEMI patients treated with TA was observed, particularly among those with high thrombus burden.

STEMI remains one of the leading causes of cardiovascular morbidity and mortality worldwide. Despite significant therapeutic advances with primary PCI, a substantial proportion of patients develop reperfusion-related complications, including distal embolization and the no-reflow phenomenon, leading to the development of MVO.

Among the proposed strategies to mitigate microvascular injury, TA has received particular attention over the years. Although not routinely recommended by ESC guidelines, TA may still be considered in patients with a high thrombus burden, as it aims to remove thrombotic material before revascularization, thereby reducing distal embolization and improving microcirculatory perfusion.[5]

The clinical efficacy of TA has been extensively investigated. While initial data suggested benefits, large-scale randomized clinical trials—including TAPAS, TASTE, and TOTAL—ultimately demonstrated no significant reduction in hard clinical endpoints with routine TA use, although a potential signal for reduced cardiovascular mortality was noted in patients with high thrombus burden.[3,6–10] Alongside these major trials, several smaller mechanistic studies utilizing imaging modalities like CMR have explored the impact of TA on tissue-level healing (**Supplementary Table 1**). These studies have yielded conflicting results regarding MVO mitigation and infarct size reduction.[11–19]

To the best of our knowledge, this is the first study evaluating the myocardial damage by using sequential CMR in a long-term follow-up.

Our study highlighted significant differences between patients treated with and without TA during primary PCI. Patients undergoing TA had a higher risk profile, as they presented worse baseline clinical conditions, a higher thrombus burden, more severe pre-procedural TIMI flow impairment,

significantly higher peak high-sensitivity troponin levels and lower baseline LVEF on admission echocardiography. Consistent with the clinical presentation, index CMR scans of the TA group showed a higher prevalence of MVO and IMH, as well as a larger myocardial scar extent.

At follow-up CMR, patients in the TA group had more pronounced improvements in myocardial tissue characterization, including a greater reduction in myocardial scar (-5.0% vs -3.3%). Although the numerical difference may appear modest, its clinical relevance is substantial. According to Stone et al., each 5% increase in myocardial infarct size is associated with a 19% relative increase in 1-year all-cause mortality and a 20% rise in heart failure hospitalization.[20] A 5.0% reduction in scar size thus directly targets a prognostically meaningful threshold. Notably, achieving this reduction in a high-risk cohort, characterized by extensive baseline MVO, reduced LVEF, and greater thrombus burden, represents a clinically significant outcome compared to a 3.3% reduction observed in the PCI-only group.

When interpreting these results, it is crucial to consider the natural temporal evolution of infarcted tissue. As elegantly demonstrated by the TIME trial and by Bodí et al., larger baseline infarcts inherently undergo a greater absolute and relative mass reduction over time, driven by the physiological resolution of acute edema and necrotic tissue clearance.[21,22] Given the significantly larger baseline infarcts in our TA group, the observed scar shrinkage is undoubtedly influenced by natural history of infarct healing and the statistical phenomenon of regression to the mean.

However, a distinctive aspect of our TA cohort is the high baseline prevalence of MVO. Typically, extensive MVO is a strong predictor of adverse remodeling and blunted scar regression. Consistent with the TIME trial, the higher baseline MVO mass in the TA group was associated with a trend towards adverse remodeling with left ventricular dilation. The observation that patients in the TA group achieved significant scar shrinkage despite a disproportionately severe initial MVO burden and a ‘hostile’ mechanical environment, suggests that alleviating the macroscopic thrombotic burden

might optimize the microenvironment for tissue repair, counteracting the expected adverse natural history of extensive 'no-reflow' lesions.

Moreover, myocardial scar reduction was more pronounced in patients with a high Thrombus Grade (>3) and baseline MVO, underscoring the specific efficacy of TA in mitigating myocardial damage within this high-risk phenotype where distal embolization is most detrimental.

At follow-up, we did not observe significant improvements in left ventricular remodeling in the TA group, as reported in other studies. This could be attributed to the significantly worse initial clinical conditions of the TA group and to the higher prevalence of MVO, which itself is a well-established negative prognostic factor in STEMI patients, as previously demonstrated in the PROSPECT study.[23]

Our findings are consistent with those of a retrospective study by Meier et al., which, similarly to our study, reported statistically significant differences in pre-procedural thrombus burden (TIMI Thrombus Grade) and TIMI flow impairment, with worse values in the TA group. However, unlike our study, Meier et al. did not perform follow-up CMR, thus lacking data on scar evolution and myocardial edema.[24]

Prospective randomized trials, such as the MUSTELA and EXPIRA trials, directly evaluated MVO reduction with TA. While the MUSTELA trial did not include follow-up CMR, and the EXPIRA trial had a short follow-up (CMR at 3 months), both studies demonstrated a reduction in MVO incidence among patients treated with TA. However, neither trial found a significant impact on infarct size or left ventricular remodeling, likely due to the absence or limited duration of follow-up CMR.[12,13]

A crucial aspect of our findings lies in the interplay between the timing of recovery, ventricular remodeling, and potential clinical outcomes. While previous studies with shorter follow-up often failed to capture the dynamic evolution of myocardial damage, our 12-month assessment highlights a significant biological recovery in the TA group. Although this tissue-level improvement, manifested

as a greater reduction in scar size, did not translate into a superior functional remodeling compared to the lower-risk control group, it indicates that TA acts by optimizing the conditions for myocardial repair. Specifically, by alleviating the microvascular burden, TA appears to foster a more efficient scar consolidation, thereby limiting the final infarct extension even in patients with an adverse hemodynamic baseline. Given that final infarct size is a strong independent predictor of long-term mortality, the tissue recovery facilitated by TA might represent a mechanistic bridge to improved clinical outcomes, particularly in high-risk patients where the burden of necrotic tissue is the primary driver of prognosis.

Our result provides evidence supporting the efficacy of TA in modifying the underlying mechanisms of microvascular injury, particularly in patients with high thrombus burden, and warrants further investigation in future studies.

Study Limitations

The single-center nature of this study represents an intrinsic limitation, associated with a potential selection bias due to the absence of true randomization and clinical indication for TA: it was preferentially used in patients with a worse clinical and angiographic profile. Additionally, the relatively small sample size limits the statistical power of subgroup analyses. Finally, the lack of prospective data prevents establishing a direct causal relationship between TA and long-term clinical outcome improvements. Thus, our findings should be interpreted as exploratory and hypothesis-generating rather than confirmatory of a causal treatment effect.

CONCLUSIONS

Our study explored the role of TA in improving microvascular perfusion and modulating left ventricular remodeling in STEMI patients, specifically analyzing changes observed on CMR performed during the index hospitalization and at long-term follow-up.

Although patients undergoing TA had worse initial clinical conditions, with a significantly higher thrombus burden and a greater prevalence of MVO, the follow-up CMR results suggested a potential benefit of TA in promoting myocardial scar reduction.

The findings of this study suggest that, in high-risk STEMI patients, TA could be considered as an adjunctive strategy to enhance myocardial recovery, particularly in cases with a high thrombus burden, emphasizing the need for careful patient selection.

Further prospective, randomized trials with larger cohorts and long-term follow-up are necessary to confirm the role of TA in improving clinical outcomes in STEMI patients and to identify subpopulations that may derive the greatest benefit from this technique.

Conflict of interests
the authors have no conflicts of interests to declare.

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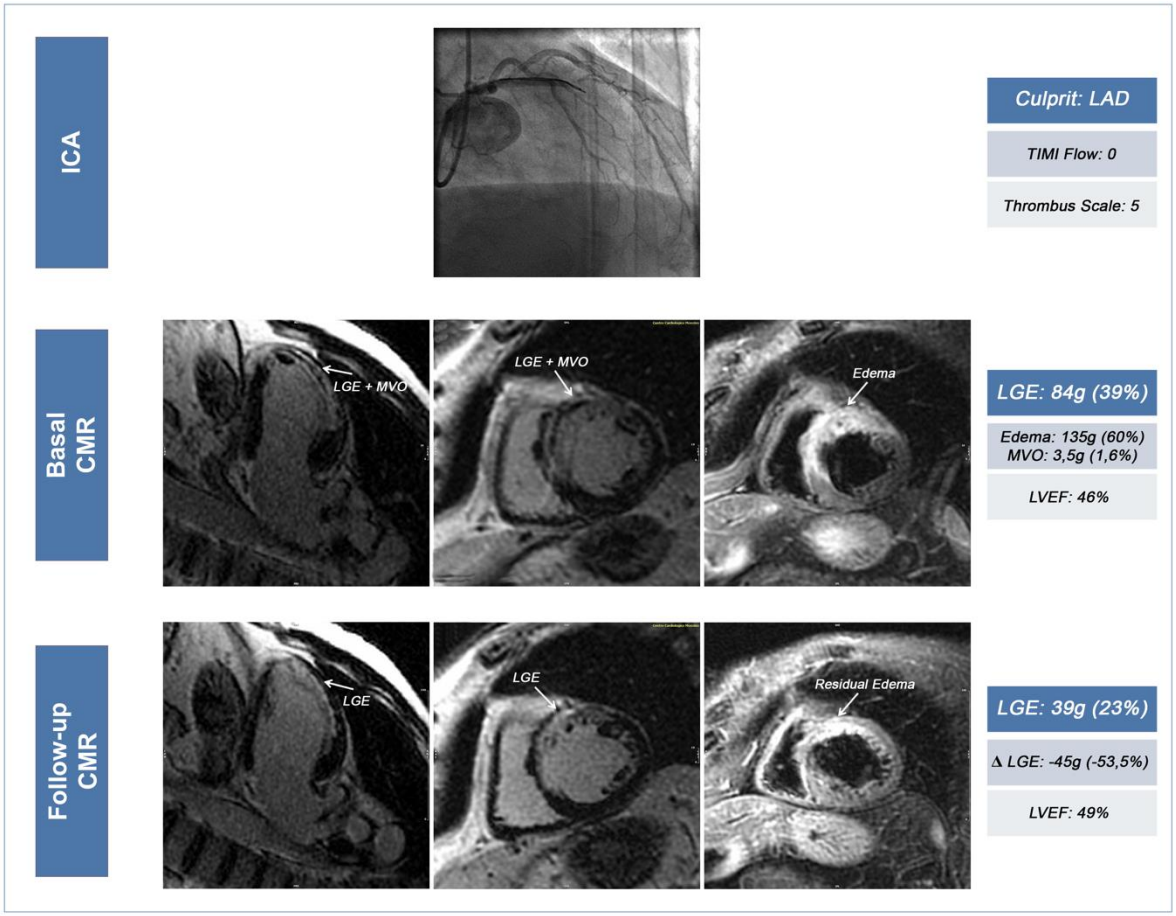
Figure caption

Figure 1. Representative Case of a Patient Undergoing Thrombus Aspiration.



ICA: invasive coronary angiography; CMR: cardiac magnetic resonance; LGE: late gadolinium enhancement; MVO: microvascular obstruction; LVEF: left ventricle ejection fraction.

Table 1. Clinical and angiographic baseline characteristics

Variables	TA+PCI (n=84)	PCI-alone (n=46)	*P* Value
Age, years	59.61±8.93	58.72±10.21	0.60
Male sex, n (%)	56	29	0.81
Body Mass Index, kg/m²	26.83±3.69	27.11±4.51	0.70
Cardiovascular Risk Factors			
Family history of CVD, n (%)	19(22.6%)	19(41.3%)	0.02
Smoking, n (%)	33(39.3%)	19(41.3%)	0.51
Diabetes, n (%)	13(15.5%)	7(15.2%)	0.96
Hypertension, n (%)	38(45.2%)	21(45.7%)	0.96
Dyslipidemia, n (%)	30(35.7%)	20(43.5%)	0.38
Cardiovascular condition			
Ejection Fraction (echo), %	44.96±12.39	49.98±11.55	0.02
ST Resolution at 90', n (%)			0.15
< 30%	23 (27.4%)	10 (22.2%)	
30% - 70%	27 (32.1%)	9 (20.0%)	
> 70%	34 (40.5%)	26 (57.8%)	
Cardiogenic Shock, n (%)	13(15.5%)	3(6.7%)	0.17
MACE, n (%)	18(21.4%)	5(11.1%)	0.14
MI Location, n (%)			
Anterior	46(54.8%)	23(50%)	0.75
Infero-Posterior	33(39.3%)	19(41.3%)	
Lateral	4(4.8%)	4(8.7%)	
Blood Tests			
Hemoglobin, g/dL	14.31±2.23	14.45±1.72	0.71
Platelet Count, UI×10³/mm³	222(196;268)	231(202;271)	0.50
Creatinine, mg/dL	0.94±0.2	0.94±0.32	0.97
GFR, mL/min	83.99±18.44	86.82±23.56	0.45
hsTnI Admission, pg/mL	175(30;4065)	389.55(68;2010)	0.48
hsTnI Peak, pg/mL	75846.3(22285;160180.7)	24030.7(6107;46730)	<0.001
Home Therapy n (%)			
Aspirin	10(11.9%)	10(21.7%)	0.13
Anticoagulant	24(28.6%)	12(26.7%)	0.81
Angiographic characteristics			
Time from Diagnosis to PCI, hours	3(2;6)	3(2;6)	0.53
TIMI Flow 0 pre-PCI, n (%)	67(79.8%)	19(41.3%)	<0.001
TIMI Flow 3 post-PCI, n (%)	74(88.1%)	39(84.8%)	0.13
Stent Diameter, mm	3(3;3.5)	3(2.5;3.5)	0.01
Stent Length, mm	23.5(15.5;37.5)	22(15;30)	0.15
Adenosine, n (%)	24(28.6%)	4(8.7%)	0.008
No-Reflow, n (%)	24(28.6%)	4(8.7%)	0.007
Thrombus Grade	5(5;5)	3(2;5)	<0.001
0, n (%)	1(1.2%)	5(10.9%)	
1, n (%)	1(1.2%)	2(4.3%)	
2, n (%)	2(2.4%)	10(21.7%)	
3, n (%)	5(6%)	8(17.4%)	

4, n (%)	9(10.7%)	5(10.9%)	
5, n (%)	66(78.6%)	16(34.8%)	
Extent of CAD, n (%)			0.92
1-vessel disease	66 (78.6%)	34 (73.9%)	
2-vessel disease	18 (21.4%)	10 (21.7%)	
3-vessel disease	0 (0%)	2 (4.3%)	
Antithrombotic treatments at discharge, n (%)			
Aspirin	81(96.4%)	45(100%)	0.55
GPIIb/IIIa inhibitors	22(26.2%)	6(13.3%)	0.09
Clopidogrel	17(20.2%)	8(17.8%)	0.51
Prasugrel	15(17.9%)	5(11.1%)	0.51
Ticagrelor	52(61.9%)	32(71.1%)	0.51
Other treatments at discharge, n (%)			
Beta-blockers	80 (95.2%)	44 (95.6%)	0.99
ACE-i or ARB	46 (54.8%)	32 (69.6%)	0.15
ARNI	32 (38.1%)	10 (21.7%)	0.18
MRA	30 (35.7%)	10 (21.7%)	0.21

TA: Thrombus Aspiration; PCI: percutaneous coronary intervention MACE: Major Adverse Cardiac Event; GFR: Glomerular Filtration Rate; TIMI: Thrombolysis in Myocardial Infarction; ACE-i: Angiotensin-Converting Enzyme inhibitor; ARB: Angiotensin Receptor Blocker; ARNI: Angiotensin Receptor-Neprilysin Inhibitor; MRA: Mineralocorticoid Receptor Antagonist.

Table 2. Differences between Major Baseline and Follow-up CMR Parameters

	Baseline CMR			**Follow-up CMR**		
CMR Variables	TA+PCI (n=84)	PCI-alone (n=46)	*P* Value	TA+PCI (n=84)	PCI-alone (n=46)	*P* Value
MVO, n (%)	37 (44.6)	10 (25)	0.03	5 (6.7%)	1 (2.7%)	0.66
MVO mass, g	0 (0; 1.82)	0 (0; 0.03)	0.08	0 (0; 0)	0 (0; 0)	0.38
MVO, % of LV mass	0 (0; 0.5)	0 (0; 0.1)	0.17	0 (0; 0)	0 (0; 0)	0.39
Myocardial Scar, g	34.5 (20.75;58.95)	27.3 (9.3;35.67)	0.005	22.47 (14.59;41.8)	18.12 (10.15;30.22)	0.04
Myocardial Scar, % of LV mass	24.2 (15.1;39.1)	17.53 (7.8;25.8)	0.001	19.2 (13.16;28.8)	14.25 (8.25;23.77)	0.03
Myocardial Edema, g	61.1 (41,37;87)	49.09 (20.48;78.45)	0.07	0(0;0)	0(0;0)	0.54
Myocardial Edema, % of LV mass	38 (25.5;52.3)	27 (16.75;43.83)	0.04	0(0;0)	0(0;0)	0.52
Intramyocardial Hemorrhage, n (%)	16(27.6)	2(8)	0.04	1(7.7%)	0(0%)	1
MSI	0.37 (0.22;0.56)	0.53 (0.45;0.66)	<0.001			

LVEDV index, ml/m²	86.8 (76; 102)	84.7 (76; 91)	0.20	91.5 (73; 108)	80.5 (70; 93)	0.09
LVESV index, ml/m²	46.3 (34; 57)	37.0 (29; 51)	0.03	47.0 (31; 63)	34.9 (27; 48)	0.01
GLS, %	-8.27 (-10.56;-5.71)	-10.12 (-12.47;-7.19)	0.02	-9.54 (-11.51;-7.24)	-11.47 (-13.2;-9.48)	0.001
LVEF, %	46 (41;54)	54 (42;60)	0.02	50 (40;57)	54.5 (48;62)	0.005

CMR: cardiac magnetic resonance; TA: Thrombus Aspiration; PCI: percutaneous coronary intervention; MVO: microvascular obstruction; MSI: myocardial salvage index; LVEDV: left ventricle end diastolic volume; LVESV: left ventricle end sistolic volume; GLS: global longitudinal strain, LVEF: left ventricular ejection fraction.

Table 3. Spearman Correlation Analysis of Thrombus Grade with the Absolute and Relative Mass of MVO Detected on Baseline CMR

Variables	Absolute Mass of MVO	P Value	Relative Mass of MVO	P Value
PCI-alone				
Thrombus Grade	0.372	0.01	0.347	0.02
TA+PCI				
Thrombus Grade	-0.031	0.78	-0.053	0.63

MVO: microvascular obstruction.

Highlights

- In a real-word STEMI population, thrombus aspiration was used in patients with higher thrombus burden.
- Thrombus aspiration promotes superior 12-month myocardial scar reduction.
- Benefits are most pronounced in patients with baseline microvascular obstruction.