Thrombus aspiration in acute coronary syndromes: prevalence, procedural success, change in serial troponin T levels and clinical outcomes in a contemporary Swiss cohort

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Abstract

Background: Randomised controlled trials have provided conflicting results regarding procedural and clinical outcomes of thrombus aspiration combined with percutaneous coronary intervention, when compared with primary percutaneous coronary intervention alone in patients with acute coronary syndromes.

Methods: Acute coronary syndrome patients referred for coronary angiography to four Swiss university hospitals between 2009 and 2012 were enrolled in the SPUM-ACS cohort. At the discretion of the interventional cardiologist, patients underwent thrombus aspiration with percutaneous coronary intervention or percutaneous coronary intervention alone. Procedural success was defined as post-procedural thrombolysis in myocardial infarction III flow in the infarct-related artery. Serial changes in high-sensitivity troponin T (Δ hsTnT) and adjudicated 30 days (1 year) clinical events defined as the composite of cardiac death, recurrent myocardial infarction or clinically indicated coronary revascularisation were assessed.

Results: Among 1641 patients, 777 (47.4%) had angiographic evidence of coronary thrombus. Patients were categorised into thrombus aspiration with percutaneous coronary intervention ($n=663$) or percutaneous coronary intervention alone ($n=144$). ST-segment elevation myocardial infarction (STEMI) patients more often received thrombus aspiration with percutaneous coronary intervention (87.8%) than non-STEMI patients (73.5%), $P<0.001$. Procedural success was not different in thrombus aspiration with percutaneous coronary intervention compared with percutaneous coronary intervention alone (93.8% vs. 90.7%, $P=0.243$). Δ hsTnT was similar in STEMI patients (3.09 ± 4.52 vs. 2.19 ± 4.92 $\mu\text{g/l}$,

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$P=0.086$) as was clinical outcome in the entire cohort at 30 days (2.9% vs. 3.6%, $P=0.76$) and 1 year (7.2% vs. 5.3%, $P=0.55$) regardless of whether thrombus aspiration was used during primary percutaneous coronary intervention or not.

Conclusions: In this real-world acute coronary syndrome cohort, patients treated by thrombus aspiration with percutaneous coronary intervention showed no difference in the restoration of coronary blood flow compared with percutaneous coronary intervention alone immediately after the procedure. Furthermore, Δ hsTnT and clinical outcomes at either 30 days or 1 year were similar between thrombus aspiration with percutaneous coronary intervention or percutaneous coronary intervention alone.

Clinical Trials Registration: SPUM–ACS cohort NCT01000701

Keywords

Acute coronary syndromes, percutaneous coronary intervention, thrombus aspiration, TIMI flow, biomarkers, clinical outcome

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Introduction

Thrombus aspiration (TA) has routinely been used in recent years as an adjunct to primary percutaneous coronary intervention (PCI) in acute coronary syndrome (ACS) patients with angiographic evidence of intracoronary thrombi, in order to restore perfusion, prevent peripheral embolisation and potentially microvascular obstruction (MVO) and no-reflow. MVO was recently identified as an independent predictor of adverse clinical events in patients with ST-segment elevation myocardial infarction (STEMI).¹ Several randomised controlled trials have been performed comparing TA combined with PCI or primary PCI alone in patients with STEMI^{2–7} and non-ST-segment elevation myocardial infarction (NSTEMI),⁸ respectively. The TAPAS trial demonstrated that manual aspiration of thrombotic material can be performed in the large majority of patients presenting with STEMI, and that reperfusion of the ischaemic myocardium as assessed by myocardial blush grade can be improved compared with primary PCI alone.² Of note, ST-segment elevation and ST-segment deviation normalised faster than with PCI alone.² Furthermore, a follow-up study of the TAPAS trial showed that the improvement in myocardial reperfusion was associated with a reduced rate of cardiac death and non-fatal myocardial infarction (MI) at 1 year.³ In contrast to the above, in the INFUSE–AMI trial infarct size as assessed by cardiac magnetic resonance imaging at 30 days was not reduced by TA with PCI compared with PCI alone in patients with anterior STEMI.⁴ However, the TASTE trial showed that in STEMI patients, TA with PCI compared with PCI alone did not reduce all-cause mortality at 30 days,⁵ nor the prespecified secondary endpoint of all-cause mortality at 1 year.⁶ Finally, the TOTAL trial also enrolling STEMI patients confirmed the results of the TASTE trial that TA with PCI compared with PCI alone did not alter the composite endpoint of cardiovascular death, recurrent MI, cardiogenic shock, or New York Heart Association (NYHA) class IV heart failure within 180 days.⁷ Interestingly, in NSTEMI patients, the

TATORT–NSTEMI trial showed that TA with PCI did not reduce MVO defined as the primary endpoint compared with PCI alone.⁸

In this context, it was the primary aim of this study to assess the prevalence of TA during primary PCI, procedural success, change in serial troponin T levels and clinical outcomes defined as the composite of cardiac death, recurrent MI and clinically indicated coronary revascularisation in a cohort of STEMI/NSTEMI patients in a real-world setting of a prospective cohort recruited at four Swiss university hospitals.

Materials and methods

Patient population

The Special Program University Medicine Acute Coronary Syndromes and Inflammation (SPUM–ACS) was established by four Swiss university hospitals (Bern, Geneva, Lausanne and Zurich) to prospectively recruit and analyse a real-world cohort of patients with ACSs.^{9–11} Consecutive patients with a diagnosis of ACS that underwent coronary angiography were enrolled between December 2009 and October 2012. Inclusion criteria comprised patients of both genders, aged 18 years and older, presenting within 5 days (preferably within 72 hours) after pain onset with a main diagnosis of STEMI, NSTEMI or unstable angina. Enrolled patients had symptoms compatible with angina pectoris (chest pain, dyspnoea) and fulfilled at least one of the following criteria: (a) ECG changes, such as persistent ST-segment elevation or depression, T-inversion or dynamic ECG changes or new left bundle branch block; (b) evidence of positive (predominantly conventional) troponin by local laboratory reference values (with a rise and/or fall in serial troponin levels); (c) known coronary artery disease specified by its status after MI, coronary artery bypass graft, or PCI or newly documented 50% or greater

stenosis of an epicardial coronary artery during the initial catheterisation. Exclusion criteria comprised severe physical disability, inability to comprehend the study, or less than 1 year of life expectancy for non-cardiac reasons. The study was approved by the local ethical committees and all patients gave written informed consent in compliance with the Declaration of Helsinki as listed under ClinicalTrials.gov number NCT01000701.

Angiographic parameters

Prespecified angiography case report forms were used for each patient at index coronary angiography to ascertain angiographic evidence of coronary thrombus adjacent to the culprit lesion, and also to establish whether TA was performed with pertinent documentation of thrombolysis in myocardial infarction (TIMI) flow¹² before and after PCI. Procedural success was defined as post-procedural TIMI-III flow in the infarct-related artery. Peri-procedural medications administered were ascertained and all data were entered into an electronic database.

Serial change in high-sensitivity troponin T

Blood was drawn at the time of index coronary angiography (high-sensitivity troponin T (hsTnT₁)) and 12–24 hours later (hsTnT₂). Serum aliquots were frozen at –80°C after centrifugation at 2700g for 10 minutes at room temperature until serial measurement (no prior freeze–thaw cycles) in the Zurich core laboratory. Aliquots were measured, blinded to patient data by means of numbered ID codes, to determine the concentration of hsTnT in one serum aliquot per patient using an electrochemiluminescence immunoassay analysed on a Cobas e 602 reader (Roche Diagnostics, Mannheim, Germany), with assay characteristics as reported by the manufacturer.

Clinical endpoints

Follow-up was performed at 30 days (phone call) and at 1 year (clinical visit) with events adjudicated by three independent experts using prespecified event adjudication forms. Major adverse cardiac events (MACE) were defined as the composite of cardiac death, non-fatal recurrent MI (according to the universal definition comprising both spontaneous and peri-procedural MI)¹³ or clinically indicated coronary revascularisation.

Clinical risk score calculation

The Global Registry of Acute Coronary Events (GRACE) risk score was used to calculate both in-hospital and long-term predictions of mortality and to assess the degree of disease severity in patients included in the current study. The GRACE risk criteria used to assess the score for in-hospital mortality comprised age, heart rate, systolic

blood pressure, initial serum creatinine, Killip class, cardiac arrest on admission, elevated cardiac markers (conventional troponins as per local laboratories) and ST-segment deviation.¹⁴ The GRACE risk criteria used to calculate the score for long-term mortality comprised age, heart rate, systolic blood pressure, initial serum creatinine, history of congestive heart failure, history of MI, elevated cardiac markers (conventional troponins as per local laboratories), ST-segment depression and no in-hospital PCI.¹⁵ The GRACE risk scores (short and long-term) were calculated using a programme written in Stata statistical software (version 13; Stata Corp, College Station, TX, USA) and we used the standard scoring of the GRACE risk score as mentioned in the reference publications.^{14,15}

Statistical analyses

The aim of the study was to assess the effect of TA with PCI in patients with angiographic documentation of coronary thrombus. Among these, we categorised patients based on whether TA with PCI or PCI alone was performed. Throughout, results are reported at the patient level (lesion-level analysis is available in the supplementary material).

The clinical characteristics of each group are presented for baseline continuous variables as means with standard deviations and *P* values from *t*-tests. Categorical variables are shown as counts with percentages and *P* values from χ^2 or Fisher's exact tests. *P* values for procedural success are derived from logistic regression. We compared medians of Δ hsTnT, with *P* values from the Wilcoxon rank-sum test.

Odds ratios (ORs) and 95% confidence intervals (CIs) were estimated using logistic regression with *P* values from the Wald test.

Clinical outcomes were analysed, censoring patients at 30 days (data for 1-year follow-up are shown in the supplementary material), at death, or at last valid contact date using Cox's regression. For procedural success and clinical outcomes we used inverse propensity score estimators to obtain covariate adjusted results. To calculate the propensity score, we included in a logistic regression (with outcome receiving TA with PCI) baseline variables which had a *P* value less than 0.2: age (≥ 75 vs. < 75 years), weight (< 60 vs. ≥ 60 kg), renal failure (< 60 vs. ≥ 60 glomerular filtration rate), STEMI (yes, no; no refers to NSTEMI), stent deployment ('stenting') and balloon angioplasty ('ballooning') classified as yes/no. The inverse probability of treatment weights (IPTW) were calculated as the inverse of the probability of receiving the treatment actually received.¹⁶ Then we conducted a logistic regression (for procedural success) and Cox's regressions (for the clinical outcomes) weighted by IPTW with robust standard errors for obtaining 95% CIs. Furthermore, we stratified these analyses according to specified subgroups of baseline variables as mentioned above and also dichotomised pre-procedural TIMI flow (0/I vs. II/III).

P values for interactions were obtained with approximate χ^2 tests for interaction across subgroups, i.e. from logistic regression (procedural success) or Cox's regression (clinical outcomes). Two sided *P* values were reported throughout and *P* values smaller than 0.05 were considered statistically significant. Statistical analyses were performed using Stata statistical software (version 14; Stata Corp, College Station, TX, USA).

Results

Prevalence of TA

Among 1641 patients with either STEMI or NSTEMI, 777 patients (47.4%) had angiographic evidence of coronary thrombus (Figure 1) and were treated by TA with PCI (*n*=663) or PCI alone (*n*=114). Overall, in both STEMI (641) and NSTEMI patients (136), TA with PCI was performed more frequently (*P*<0.001) than PCI alone 87.8% (563/641) versus 73.5% (100/136), respectively (Table 1). The baseline characteristics of the two patient groups indicate that PCI with deployment of a stent and the use of glycoprotein IIb/IIIa antagonists or bivalirudin were more frequent in patients treated by TA with PCI compared with PCI alone. In turn, PCI with balloon angioplasty alone and the use of proton pump inhibitors were found more frequently in patients treated by PCI alone compared with TA with PCI (Table 1).

Post-procedural coronary blood flow

Procedural success was defined as post-procedural TIMI-III flow in the infarct-related artery. Procedural success was similar in patients treated by TA with PCI compared with PCI alone (93.8% vs. 90.7%, *P*=0.243; Table 2). Stratified and IPTW adjusted analysis of procedural success in all lesions yielded similar results across major subgroups with an overall OR of 0.73 (95% CI 0.34–1.58, *P*=0.429; Figure 2). Restoration of coronary blood flow was obtained in both groups despite a higher rate of vessel occlusion (TIMI-0 or I flow) prior to intervention in patients treated by TA with PCI compared with PCI alone (93.8% vs. 90.7%, *P*=0.243; Table 2). These results were similar when analysed on a lesion level (Supplementary Table 1). The rate of direct stenting in the lesion was more frequent in patients treated by TA with PCI compared with patients treated by PCI alone (34.9% vs. 24.5%, *P*=0.021) (Supplementary Table 1).

Serial change in high-sensitivity troponin T

Procedural success (restoration of TIMI-III flow) and hsTnT measurements from baseline (hsTnT₁) and 12–24 hours later (hsTnT₂) were available in 541 patients. The absolute change in concentration of hsTnT (Δ hsTnT = hsTnT₂ – hsTnT₁), median $\pm$ interquartile range was

similar between patients treated by TA with PCI and patients treated by PCI alone, for STEMI patients (3.09 $\pm$ 4.52 vs. 2.19 $\pm$ 4.92 μ g/l, *P*=0.086; Table 3). In patients with STEMI stratified by time of chest pain onset to first blood draw the change in the level of hsTnT was similar in patients treated by TA with PCI compared with PCI alone, both in early and late presenters (Figure 3, Supplementary Table 2).

Clinical outcome

Follow-up data until 30 days were complete in 99.7% of patients treated by TA with PCI and in 99.1% of patients treated by PCI alone (Figure 1). Individual adjudicated events are shown in Supplementary Table 3. The composite endpoint at 30 days (MACE) occurred in 2.9% (19/663) of patients treated by TA with PCI and 3.6% (4/114) of patients treated by PCI alone (*P*=0.76). For 30 days (MACE) stratified IPTW adjusted analysis of all 777 patients with the presence of coronary thrombus and undergoing interventional treatment showed similar clinical outcomes between patients treated by TA with PCI and patients treated by PCI alone across subgroups with an overall hazard ratio (HR) of 1.03 (95% CI 0.31–.43, *P*=0.968; Figure 4). At 1 year the composite endpoint was found in 7.2% (48/663) of patients treated by TA with PCI and 5.3% (6/114) of patients treated by PCI alone (*P*=0.55) (Supplementary Table 3). Stratified IPTW adjusted analysis for 1 year MACE showed no difference in clinical outcome across major subgroups (Supplementary Figure 1).

Discussion

Our study shows that in a real-world prospective contemporary ACS cohort: (a) patients treated by TA during primary PCI showed no difference in restoration of coronary blood flow after the procedure compared with those treated with PCI alone; (b) the absolute change in hsTnT as a measure of infarct size was similar between groups, as were (c) the clinical short and long-term outcomes.

Corroborating prior data from a large Japanese registry reporting a 63.3% rate of TA during primary PCI in STEMI patients,¹⁷ we found a high rate of 87.8% (563/641) of TA in STEMI patients. Conversely, 73.5% (100/136) of the NSTEMI patients enrolled in the present cohort were treated by TA with PCI, in contrast to data from a subanalysis of the ACUITY trial,¹⁸ in which the rate of TA with PCI was 9.4% in NSTEMI patients presenting with an angiographically visible thrombus.

Of note, restoration of TIMI-III flow (as a reflection of procedural success) was achieved in 93.8% of the patients in whom TA was performed during primary PCI (and in 90.7% of the patients by the use of PCI alone), also corroborating findings from the randomised TAPAS² and INFUSE-AMI⁴

Figure 1. Study flow: flow chart shows the distribution of STEMI/NSTEMI patients with available baseline biomarker data (hsTnT₁) and comprehensive procedural data. (Data analysed $n=777$).

trials reporting success rates of 86% and 92.6% in STEMI patients, respectively. Furthermore, and in line with data from the TASTE, TOTAL and NSTEMI-TATORT^{5,7,8} trials, primary stenting was more frequently performed in patients treated by TA compared with those that were treated by PCI alone.

Despite improved restoration of coronary blood flow upon TA with PCI, serial measurement of hsTnT as an index of infarct size revealed similar changes in concentration both for patients treated by TA with PCI or PCI alone, respectively. This extends the results of the TATORT-NSTEMI trial⁸ to a real-world ACS cohort.

Table 1. Baseline characteristics.

	Thrombus present		P value
	TA + PCI	PCI alone	
Total number of patients	n=663	n=114	
Age (years)	n=663, 60.99±12.15	n=114, 61.99±13.55	0.422
≥75 years	n=663, 103 (15.5%)	n=114, 24 (21.1%)	0.169
Gender (female)	n=663, 118 (17.8%)	n=114, 23 (20.2%)	0.514
Weight (kg)	n=654, 80.69±14.94	n=111, 77.91±14.09	0.068
<60 kg (yes)	n=654, 34 (5.2%)	n=111, 11 (9.9%)	0.077
Body mass index (kg/m ²)	n=653, 26.95±4.09	n=111, 26.72±4.26	0.597
Medical history			
Diabetes mellitus	n=663, 74 (11.2%)	n=114, 17 (14.9%)	0.269
Hypertension	n=663, 323 (48.7%)	n=114, 56 (49.1%)	1.000
Hypercholesterolemia	n=663, 382 (57.6%)	n=114, 69 (60.5%)	0.608
Current smoker	n=655, 290 (44.3%)	n=112, 45 (40.2%)	0.471
Family history of CAD	n=654, 163 (24.9%)	n=114, 22 (19.3%)	0.235
Renal failure ^a	n=661, 74 (11.2%)	n=114, 6 (5.3%)	0.065
History of stroke or TIA	n=663, 18 (2.7%)	n=114, 3 (2.6%)	1.000
Previous myocardial infarction	n=662, 62 (9.4%)	n=114, 15 (13.2%)	0.234
Previous PCIs	n=663, 84 (12.7%)	n=113, 14 (12.4%)	1.000
Previous CABG	n=663, 21 (3.2%)	n=114, 4 (3.5%)	0.776
Clinically relevant valvular disease	n=663, 8 (1.2%)	n=114, 1 (0.9%)	1.000
Clinical presentation		Pearson χ^2 (1)=18.33, P<0.001	
STEMI	n=663, 563 (84.9%)	n=114, 78 (68.4%)	–
NSTEMI	n=663, 100 (15.1%)	n=114, 36 (31.6%)	–
Killip class			
I	n=663, 553 (84.2%)	n=114, 94 (82.4%)	0.679
II	n=663, 66 (10.0%)	n=114, 18 (15.8%)	0.074
III	n=663, 13 (2.0%)	n=114, 1 (0.9%)	0.706
IV	n=663, 25 (3.8%)	n=114, 1 (0.9%)	0.157
Systolic blood pressure (mmHg)	n=659, 127.2±23.7	n=114, 128.4±22.7	0.603
Heart rate (beats per minute)	n=660, 76.7±17.05	n=114, 77.54±16.87	0.632
Index procedure			
Stenting ^b	n=663, 641 (96.7%)	n=114, 102 (89.5%)	0.002
Any drug-eluting stent	n=663, 483 (72.9%)	n=114, 79 (69.3%)	0.430
Any bare-metal stent	n=663, 170 (25.6%)	n=114, 26 (22.8%)	0.561
Ballooning	n=663, 53 (8.0%)	n=114, 18 (15.8%)	0.013
Pre-procedural medication			
No P2Y12 inhibitor	18 (2.7%)	3 (2.6%)	0.960
Clopidogrel	299 (45.1%)	55 (48.3%)	0.533
Prasugrel	164 (24.7%)	21 (18.4%)	0.155
Ticagrelor	14 (2.1%)	2 (1.8%)	0.804
Mixture of (P2Y12) inhibitors	168 (25.3%)	33 (28.9%)	0.417
Glycoprotein IIb/IIIa inhibitors	283 (42.7%)	30 (26.3%)	<0.001
Unfractionated heparin	638 (96.2%)	110 (96.5%)	0.557
Bivalirudin	58 (8.7%)	2 (1.8%)	0.010
Baseline medication			
Proton-pump inhibitor	n=660, 71 (10.8%)	n=113, 20 (17.7%)	0.040
Oral anticoagulation	n=660, 18 (2.7%)	n=113, 2 (1.8%)	0.754
GRACE risk score			
In-hospital	n=663, 147.51±33.48	n=114, 144.29±33.46	0.343
Long-term	n=663, 118.11±25.48	n=114, 119.23±27.84	0.669

Depicted are counts (%) or means ± SDs.

^aBased on creatinine-estimated glomerular filtration rate clearance of <60 mL/min/1.73 m², using the modification of diet in renal disease (MDRD) formula.^bMix of drug-eluting stents and bare-metal stents possible.

CABG: coronary artery bypass graft; CAD: coronary artery disease; LMWH: low-molecular weight heparin; TA: thrombus aspiration; PCI: percutaneous coronary intervention; TIA: transient ischaemic attack; GRACE: Global Registry of Acute Coronary Events.

Table 2. Procedural success and TIMI flow.

	Thrombus present		P value
	TA + PCI	PCI alone	
Number of patients with PCI^a	n=648	n=107	
TIMI flow pre-procedure^b			
0/I	528 (81.5%)	68 (63.6%)	<0.001
II	67 (10.3%)	17 (15.9%)	0.106
III	53 (8.2%)	22 (20.6%)	<0.001
TIMI flow post-procedure^b			
0/I	12 (1.9%)	3 (2.8%)	0.534
II	28 (4.3%)	7 (6.5%)	0.334
III ^c	608 (93.8%)	97 (90.7%)	0.243

^aOnly analysed for patients with pre- and post-procedural TIMI flow data available (n=755).

^bWorst TIMI flow per patient reported.

^cProcedural success was defined as post-procedural TIMI-III flow in the infarct-related artery.

TA: thrombus aspiration; PCI: percutaneous coronary intervention.

Figure 2. Inverse probability of treatment weighting (IPTW) adjusted analysis of procedural success across major subgroups according to baseline characteristics. ^aOnly analysed for patients with pre- and post-procedural TIMI flow data available (n=755).

^bRenal failure is defined as creatinine-estimated glomerular filtration rate clearance of <60 mL/min/1.73 m², using the modification of diet in renal disease (MDRD) formula. ^cNo refers to NSTEMI patients.

Table 3. Serial change in high-sensitivity troponin T in patients with procedural success.

	Thrombus present		P value
	TA + PCI	PCI alone	
Number of patients with PCI^a	n=472	n=69	
Level of hsTnT in ΔT (μg/l) ^b			
STEMI	n=402	n=45	
Mean±SD	4.25 ± 4.36	3.07 ± 3.28	0.079
Median±IQR	3.09 ± 4.52	2.19 ± 4.92	0.086
NSTEMI	n=70	n=24	
Mean±SD	1.06 ± 1.64	0.29 ± 0.61	0.027
Median±IQR	0.45 ± 1.46	0.20 ± 0.67	0.012

^aOnly analysed for patients with pre- and post-procedural TIMI flow data and measurement of hsTnT₂ available. Patients with no improvement in TIMI flow were discarded from this analysis.

^bΔhsTnT = hsTnT₂ - hsTnT₁.

TA: thrombus aspiration; PCI: percutaneous coronary intervention; IQR: interquartile range.

Importantly, stratification by the time interval of chest pain onset to first blood draw in patients with STEMI yielded similar results, suggesting a true disparity of blood flow restoration and infarct size under these conditions.

Figure 3. Serial change in high-sensitivity troponin T (hsTnT) levels ($\Delta\text{hsTnT} = \text{hsTnT}_2 - \text{hsTnT}_1$) in STEMI patients with procedural success stratified by the onset of chest pain to first blood draw ($n=403$).

Corresponding to troponin data, rates of MACE at 30 days and 1 year were similar in patients that were treated by TA during primary PCI or by PCI alone. These outcome data generated by our sizeable real-world cohort are in line with the results obtained from large randomised trials carried out in STEMI^{5,7} and NSTEMI⁸ patients, respectively. A recent meta-analysis of randomised trials reporting mortality as a primary outcome (either all-cause or cardiovascular) similarly concluded that, contrary to the expectations of many interventionalists, TA with PCI does not reduce the rates of death, nor secondary endpoints, such as reinfarction, stent thrombosis or stroke.¹⁹ Furthermore, our data extend registry data from STEMI patients^{20,21} to patients with NSTEMI/STEMI.

Limitations

The current study reports observational data from patients in the SPUM-ACS cohort, in which ACS patients were prospectively enrolled in four Swiss university hospitals. In our study, TA was performed at the discretion of the treating interventional cardiologist. This design differs from interventional trials in which patients were randomly assigned to TA with PCI or PCI alone. Nevertheless, the SPUM-ACS cohort provides real-world data which add important information for clinical practice.

Figure 4. Inverse probability of treatment weighting (IPTW) adjusted analysis of clinical outcome (major adverse cardiac events (MACE)) at 30 days across major subgroups according to baseline characteristics ($n=777$). ^aRenal failure is defined as creatinine-estimated glomerular filtration rate clearance of $<60 \text{ mL/min/1.73 m}^2$, using the modification of diet in renal disease (MDRD) formula. ^bSTEMI (yes, no; no refers to NSTEMI). ^cPre-procedural TIMI flow: analysed only for patients with pre-procedural TIMI flow data available ($n=755$).

Among the endpoints analysed, TIMI flow was assessed by experienced interventional fellows, which may contain a certain level of variability unlike centralised analysis by a core laboratory. Furthermore, clinically indicated revascularisation was used as a component in the composite clinical endpoint rather than target lesion revascularisation based on the low event rates to allow for meaningful statistical analysis. Low patient numbers obviated a meaningful interpretation of data on biomarker concentrations in NSTEMI patients. Patients with unstable angina were not included in the study due to the low patient number (62 patients) and due to the fact that the prevalence of coronary thrombus was previously described to be very low in unstable angina patients.²²

Conclusion

Based on real-world data derived from the current analysis of the SPUM-ACS cohort, patients with STEMI and NSTEMI showed no difference in procedural success when treated by TA with PCI, compared with PCI alone, as assessed by TIMI flow. Furthermore, serial hsTnT levels, as a measure of infarct size, and short and long-term adjudicated clinical events were similar in patients treated by TA with PCI, compared with PCI alone.

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Conflict of interest

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received speaker or consultant fees from Amgen, Astra-Zeneca, MSD, and Sanofi-Aventis; TFL received research grants to the institution from AstraZeneca, Bayer Healthcare, Biosensors, Biotronik, Boston Scientific, Eli Lilly, Medtronic, MSD, Merck, Roche and Servier, including speaker fees by some of them. CMM received research grants to the institution from Eli Lilly, AstraZeneca, Roche and MSD including speaker or consultant fees. LR received speaker fees and research grants to the institution from St Jude Medical. MZ has received grant support from the European Union, Swiss National Science Foundation and from the Swiss Cancer league which paid for PhD and postdoc positions. Furthermore, he has consulted for Biotronik AG and Novigenix, Switzerland, and has or had projects which received unrestricted research support from following firms: AstraZeneca AG, Switzerland, Aptalis Pharma Inc., Dr. Falk Pharma GmbH, Germany, Glaxo Smith Kline AG, Nestlé SA, Switzerland, Receptos Inc., Regeneron Inc. All other authors have no conflict of interest to declare. CTU Bern, which is part of the University of Bern, has a staff policy of not accepting honoraria or consultancy fees. However, CTU Bern is involved in the design, conduct, or analysis of clinical studies funded by several companies and non-profit organisations (an up-to-date list can be found here: http://www.ctu.unibe.ch/research/declaration_of_interest/index_eng.html).

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