Thrombus Aspiration Reduces Contrast Volume in ST-Elevation Myocardial Infarction Patients with High Thrombus Burden

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Research Article

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Abstract

BACKGROUND Due to the emergency nature of ST-elevation myocardial infarction (STEMI), the estimation of the risk of contrast-induced nephropathy (CIN) is always limited and the hydration prior to primary percutaneous coronary interventions (PCI) is difficult. Minimizing contrast volume (CV) might be the most desirable option for preventing the incidence of CIN in these patients. The improvement of myocardial perfusion due to thrombus aspiration (TA) may be accompanied by a reduction in CV.

OBJECTIVES This study aimed to determine the effect of TA prior to angioplasty on CV in patients with STEMI.

METHODS Consecutive 380 STEMI patients undergoing primary PCI with high thrombus burden (HTB) were randomly assigned to receive either TA before primary PCI (TA group, $n = 190$) or primary PCI alone (control group, $n = 190$). Serum creatinine was detected at the time of admission, and 48 and 72 hours after primary PCI.

RESULTS Baseline characteristic were well matched. Although the CV in TA group was significantly lower than that in control group (71.5 ± 15.7 mL vs. 82.3 ± 17.5 mL, $p = 0.000$), the incidence of CIN was comparable (9.5% vs. 13.3%, $p = 0.249$). During hospitalization, no significant differences between groups was observed in major bleeding, urgent dialysis, stroke, reoperation after 72 hours, death after 72 hours and total incidence of adverse clinical events.

CONCLUSIONS The study demonstrated that TA reduces CV but not CIN or adverse clinical events during hospitalization in STEMI patients undergoing primary PCI with HTB.

Introduction

Rapid revascularization by primary percutaneous coronary intervention (PCI) is the optimal treatment for patients with ST-elevation myocardial infarction (STEMI). However, contrast-induced nephropathy (CIN) after PCI is a serious complication that negatively affects in-hospital and long-term morbidity and mortality (1). And the incidence of CIN in patients undergoing primary PCI patients is significantly higher than in those undergoing elective PCI, even in patients with normal basic renal function (2, 3). It's a common view that basic renal dysfunction is the most important risk factor for CIN. Other major risk factors include use of large contrast doses, diabetes mellitus and other comorbidities. There is no effective therapy once CIN has occurred; therefore, prevention is the cornerstone for all patients at risk for CIN. Although numerous methods have been explored to prevent renal contrast damage, hopeful strategies for reducing the incidence of CIN have not been well established. CIN continues to be a concern in high-risk patients. Hydration prior to use of contrast and preferably for a period of time is still considered as the most effective preventive measure of CIN. Due to the emergency nature of STEMI, the estimation of the risk of CIN, especially basic renal function, is always limited and the hydration prior to PCI is difficult to perform. Furthermore, available data have showed that iso-osmolar contrast media could not provide additional benefit beyond low-osmolar contrast media, especially for patients with

healthy renal function (4). Previous studies have indicated that the contrast volume (CV) correlated with CIN incidence (5, 6). Thus, minimizing CV might be the most desirable option for preventing the incidence of CIN in STEMI patients undergoing primary PCI (1).

Several procedural strategies have been established to reduce CV during PCI, such as maximizing the use of intravascular imaging (7, 8). But some of them are difficult or infeasible for STEMI patients. The rationale for thrombus aspiration (TA) during primary PCI is the removal of intracoronary thrombus, thus avoiding distal embolization leading to impaired myocardial reperfusion. Although evidence from major randomized controlled trials (notably TASTE and TOTAL) does not support the routine use of TA in STEMI patients undergoing primary PCI (9), the use of TA prior to angioplasty is associated with a significant improvement in myocardial reperfusion (10), especially in those with high thrombus burden (HTB), which shorten the time of single angiography and reduce the frequency of angiography during the procedure. This has led to speculation that the dose of contrast might be reduced by TA through the improvement of myocardial perfusion. Therefore, we performed this prospective study to evaluate the effect of TA prior to angioplasty on CV in STEMI patients undergoing primary PCI with HTB.

Methods

STUDY POPULATION

From January 2019 to July 2020, 380 consecutive STEMI patients undergoing primary PCI within 12 hours of evolution after emergency coronary angiography with HTB were enrolled at the Department of Cardiovasculology of Yue Bei People's Hospital, Shaoguan city, the northern Guangdong Province, with a population estimated at 3,366,000 inhabitants. The hospital has passed the certification of standard edition of China chest pain center on November 2, 2018. Now there are more than 1,500 PCIs annually, including more than 500 emergency primary PCIs annually. STEMI was defined by the classic symptoms of myocardial ischemia for at least 30 minutes, and serial changes on electrocardiogram: the detection of a ≥ 0.1 mV ST-segment elevation in the inferior leads or a ≥ 0.2 mV ST-segment elevation in the anterior chest leads occurring in two or more contiguous leads, or the presence of a new (or presumably new) left bundle branch block. HTB was defined as the presence of Thrombolysis in Myocardial Infarction (TIMI) thrombus grades 4 or 5.

Exclusion criteria included: previous history of myocardial infarction, PCI, coronary artery bypass grafting (CABG), stroke or serious peripheral vascular disease; serious heart, liver or kidney dysfunction or exposure to contrast media or nephrotoxic medicine within 2 weeks before procedure; hemodynamic instability, treatment with an intra-aortic balloon pump, cardiopulmonary resuscitation or thrombolysis before primary PCI; severe left main disease, target vessel diameter < 2.5 mm, or more than one intervention vessel; special lesion not suitable for PCI or TA, as judged by the treating cardiologist; TIMI thrombus grades 0 to 3, primary PCI failed to open culprit vessel or need of emergency CABG; reoperation or death within 72 hours.

All patients signed an informed consent form and the study was approved by the Human Ethics Committees of Yue Bei People's Hospital.

This study adheres to the 2017 ESC Guidelines for the management of AMI-STEMI (11).

STUDY PROTOCOL

Eligible patients were randomly assigned, in a 1:1 ratio, to receive either manual TA before primary PCI (TA group) or primary PCI alone (control group). Manual TA will be the only difference between the groups, and all other therapeutic procedures will be similar in both treatment arms. Primary PCI was performed by one of three experienced interventionists who had performed more than 300 cases of primary PCI. Standard coronary angiography was performed via radial approach using 5-Fr Judkins catheters and manual 12ml Coronary Control Syringes (Merit Medical Systems, Inc.). The second option was the brachial approach if the puncture of radial artery was failure. All angiographic views were obtained with a single plane cineangiographic system utilizing low-osmolar ioversol 350mgI/mL (Optiray®) contrast. The dosage of contrast was left to the discretion of the interventionist. And the premise is to ensure that the imaging video is clear and the operation is completed. For patients randomized to TA group, guidewire placement was followed by TA with a manual TA catheter (Export® Catheter; Medtronic, Inc, Minneapolis, MN, USA), according to previous protocol (12). TA was terminated when successful TA was followed by aspiration without any debris, or when 7 aspiration attempts did not show any visible material. Additional use of predilation or postdilation was based on the interventionist's decision. Only culprit lesions were treated with drug eluting stent (Firebird2™ Rapamycin-Eluting Coronary CoCr Stent System; Shanghai MicroPort Medical (Group) Co., Ltd, Shanghai, China) and a 6-Fr guiding catheter (Cordis®; Cordis Corporation, Miami Lakes, FL, USA). According to patient's characteristics and angiographic features, the decision to use a stent was left to the discretion of the interventionist.

After contrast exposure, hydration started with 1ml·kg⁻¹·h⁻¹ of physiologic saline intravenously for 18–24 hours. In patient with left ventricular ejection fractions of < 40% or severe heart failure, the rate of hydration was halved (0.5ml·kg⁻¹·h⁻¹), and the time of hydration was determined by the clinician according to the disease situation. All patients received a loading dose of 300 mg aspirin and 300–600 mg clopidogrel immediately after admission, a bolus of unfractionated heparin (100U/kg) during the primary PCI, and subcutaneous low molecular weight heparin therapy for 2–7 days after PCI.

Glycoprotein IIb/IIIa inhibitor (tirofiban) was used at the discretion of interventionist. A 75mg clopidogrel dose was administered for 12 months after the primary PCI and 100 mg aspirin was prescribed indefinitely. In view of the effects of statins on CIN (13), a dosage of 40 mg atorvastatin daily for 7 days followed by 20 mg daily for 12 months post PCI for all patients. Other standard therapy post primary PCI included beta-blockers and angiotensin converting enzyme inhibitors or angiotensin II receptor blockers, according to current society guidelines (11).

Angiographic analysis was performed by two experienced observers blinded to treatment allocation and clinical data. TIMI thrombus grade (14), TIMI flow grade, myocardial blush grade (MBG), and corrected TIMI frame count (cTFC) were noted as previously defined (15–17). Final TIMI flow grade < 3, and final MBG < 2 were described as angiographic no-reflow (18).

BLOOD SAMPLES AND MEASUREMENTS

Serum creatinine (SCr) was measured at the time of admission before primary PCI, 48, and 72 hours after primary PCI and discharge. Other laboratory data including the lipid profile, glycosylated hemoglobin, uric acid, and other standard clinical parameters were measured at admission before PCI. And the left ventricular ejection fraction was measured within 24 hours of symptom onset (Vivid E9; GE Vingmed Ultrasound AS, Horten, Norway)

DEFINITIONS AND FOLLOW-UP

CIN was defined as an increase in SCr by $\geq 25\%$ or ≥ 0.5 mg/dL from baseline within 48–72 hours of contrast exposure (19). Angiographic success for stent placement was defined as a residual stenosis $< 20\%$ in diameter with final TIMI flow grade 3. During hospitalization, the following adverse clinical events were considered: major bleeding (intracranial bleeding, clinically significant sign of bleeding associated with the drop in hemoglobin > 5 g/dL or fatal bleeding), urgent dialysis, reoperation for recurrent acute myocardial infarction (AMI) or stent thrombosis, stroke, and death.

STATISTICAL ANALYSIS

Continuous variables are presented as mean $\pm$ standard deviation and categorical variables as percentage (%). For comparison between two groups, the Student's t-test was used for continuous variables and the Pearson's chi-square test for categorical ones. A 2-sided $p < 0.05$ was considered significant. All statistical analyses were performed with SAS 9.1.3.

Results

CLINICAL CHARACTERISTICS

No patient was lost to follow-up during the course of this study. Two patients (1 per group) died of cardiac shock and one patient assigned to control group underwent reoperation due to stent thrombosis within 72 hours after primary PCI and therefore were taken off the sample. No patient underwent dialysis within 72 hours after admission.

The clinical characteristics are summarized in Table 1. There were no significant differences between two groups in the following: demographical, medical history, baseline laboratory parameters, indexes of cardiac function, preoperative systolic blood pressure and perioperative medications. No statistically differences were also observed between the groups in baseline SCr, baseline estimated glomerular filtration rate, and the rate of baseline SCr > 2.0 mg/dL. And the change in SCr from baseline to peak level within 48–72 hours after PCI was not significantly different between two groups. The rate of patients with CV > 200 mL was not significantly different between two groups, but the total CV used in TA group (71.5 ± 15.7 mL) was significantly lower than that in control group (82.3 ± 17.5 mL, $p = 0.000$).

TABLE 1 Clinical Characteristics

	TA (n = 189)	Control (n = 188)	p value
Age, y	65.2 ± 14.7	63.7 ± 13.4	0.317
Male	144 (76.2)	152 (80.9)	0.271
Body Surface, m ²	1.81 ± 0.24	1.79 ± 0.23	0.389
Body mass index, kg/m ²	25.6 ± 3.7	25.2 ± 3.4	0.378
Hypertension	77 (41.0)	89 (47.3)	0.197
Diabetes mellitus	41 (21.7)	37 (19.7)	0.630
Current smoker	82 (43.4)	76 (40.4)	0.560
Dyslipidemia	94 (49.7)	98 (52.1)	0.642
Heart rate, beats/min	81.2 ± 16.9	82.4 ± 16.3	0.480
Systolic blood pressure, mmHg	120.6 ± 22.0	118.2 ± 20.7	0.262
Killip class (I / II / III)	113 / 57 / 19	126 / 42 / 20	0.223
LVEF, %	54.9 ± 6.3	55.6 ± 8.2	0.330
NT-proBNP, pg/mL	1042.6 ± 774.2	947.3 ± 527.3	0.163
TC, mmol/L	4.93 ± 0.91	4.80 ± 1.02	0.192
TG, mmol/L	1.43 ± 1.01	1.51 ± 0.89	0.420
LDL-C, mmol/L	3.38 ± 0.82	3.24 ± 0.79	0.092
HDL-C, mmol/L	1.13 ± 0.31	1.09 ± 0.34	0.328
Hemoglobin, g/L	129.5 ± 22.7	133.2 ± 24.6	0.139
Platelet, ×10 ⁹ /L	226.1 ± 68.7	218.0 ± 66.3	0.241
	TA (n = 189)	Control (n = 188)	p value
HbA1c, %	6.33 ± 1.23	6.28 ± 2.22	0.787
Uric acid, μmol/L	378.3 ± 113.5	394.2 ± 110.2	0.169
Baseline SCr, mg/dL	0.99 ± 0.30	1.01 ± 0.27	0.518
Baseline SCr > 2.0 mg/dL	7(3.7)	5(2.7)	0.564
Baseline eGFR, ml·min ⁻¹ ·1.73m ⁻²	81.3 ±22.2	79.1 ±20.5	0.313
SCr at 48 hours, mg/dL	1.08 ± 0.31	1.10 ± 0.32	0.493
SCr at 72 hours, mg/dL	1.10 ± 0.35	1.13 ± 0.42	0.509
Contrast volume, mL	71.5 ±15.7	82.3±17.5	0.000
Volume of contrast >200 mL	13 (6.9)	10 (5.3)	0.447
Tirofiban administration	139 (73.5)	131 (69.7)	0.405
Aspirin	189 (100.0)	188 (100.0)	1.00
Clopidogrel	189 (100.0)	188 (100.0)	1.00
ACEI/ARB	72 (38.1)	76 (40.4)	0.643

β-blocker	36 (19.0)	42 (22.3)	0.430
CCB	28 (14.8)	24 (12.8)	0.564
Diuretics	16 (8.5)	22 (11.7)	0.297
Statins	188 (99.5)	185 (98.4)	0.312

Values are mean ± SD or n (%)

ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin II receptor blocker; CCB = calcium channel blocker; eGFR = estimated glomerular filtration rate; HbA1c = glycosylated hemoglobin; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-brain natriuretic peptide; SCr = serum creatinine; TA = thrombus aspiration; TC = total cholesterol; TG = triglyceride.

PROCEDURAL CHARACTERISTICS

Procedural characteristics of two groups are shown in Table 2, from which TA group were with significantly lower prevalence of predilatation, smaller number of stents, shorter total stent length and higher rate of TIMI flow grade 3 immediately after stent implantation(all $p < 0.05$). However, the difference in the rate of TIMI flow grade 3 between two groups disappeared at the completion of procedure ($p = 0.256$). Compared to control group, the percent of MBG ≥ 2 was higher and cTFC was lower in TA group (both $p < 0.05$). The other comparisons of procedural characteristics between two groups did not differ.

TABLE 2 Procedural Characteristics

	TA (n = 189)	Control (n = 188)	p value
Pain-wire time, min	345.4 ± 152.3	328.2 ± 139.7	0.256
IRA (LAD / LCX RCA)	110 / 18 / 61	95 / 22 / 71	0.324
TIMI flow at presentation (≤ 1 / ≥ 2)	152 / 37	159 / 29	0.298
TIMI thrombus at presentation (4 / 5)	68 / 121	61 / 127	0.470
Predilatation	28 (14.8)	79 (42.0)	0.000
Stent deployed	174 (92.1)	179 (95.2)	0.211
Number of stents	1.22 ± 0.52	1.35 ± 0.65	0.031
Stent length, mm	30.3 ± 17.6	34.5 ± 20.2	0.032
TIMI flow grade 3 after stent	146 (77.2)	125 (66.5)	0.011
Final TIMI flow grade 3	181 (95.8)	175 (93.1)	0.256
Angiographic success	181 (95.8)	175 (93.1)	0.256
MBG ≥ 2	163 (86.2)	143 (76.1)	0.012
cTFC	24.3 ± 10.5	28.2 ± 12.7	0.001

Values are mean $\pm$ SD or n (%)

cTFC = corrected TIMI frame count; MBG = myocardial blush grade; IRA = infarct related artery; LAD = left anterior descending artery; LCX = left circumflex artery; RCA = right coronary artery; TA = thrombus aspiration; TIMI = Thrombolysis in Myocardial Infarction.

INHOSPITAL ADVERSE CLINICAL EVENTS

Overall, 43 (11.4% of all patients) patients developed CIN. And, the incidence of CIN did not differ between the TA group and control group (9.5% vs. 13.3%, $p = 0.249$). During the period of hospitalization, there were no significant differences in the incidence of major bleeding, urgent dialysis, stroke, reoperation after 72 hours, death after 72 hours and total incidence of adverse clinical events between the two groups (all $p > 0.05$; Table 3).

TABLE 3 Inhospital Adverse Clinical Events

	TA ($n = 189$)	Control ($n = 188$)	p value
CIN	18	25	0.249
Major bleeding	1	2	0.559
Urgent dialysis	1	2	0.559
Stroke	2	1	0.565
Reoperation after 72 hours	2	3	0.648
Death after 72 hours	2	3	0.648
Total events	8	11	0.473

Values are mean $\pm$ SD or n (%)

CIN = contrast-induced nephropathy; TA = thrombus aspiration.

Discussion

CIN has become a serious complication with wide application of PCI technology in patients with coronary artery disease. The key step to safer CIN is to identify patients at risk and applying proven preventive measures. The incidence of CIN in the interventional therapy of AMI is much higher than that in the selective therapy. After cardiac catheterization, the incidence of CIN may be as low as 6% in patients undergoing elective catheterization (20); and reaching 23.8% in samples involving urgent catheterization (21). Our study showed the incidence of CIN was 11.4% in patients undergoing emergency primary PCI due to STEMI, lower than previous studies (21, 22). From the results of statistical analysis, we could find several characteristics concerning the low occurrence of CIN in our samples, such as lower mean age; lower infused CV (in our study the mean CV was 76.8 ml, well below the cut off points of risk listed in the literature: for some > 200 mL and others > 300 mL) (23, 24); lower baseline SCr and only 12 (3.2%) patients with SCr greater than 2 mg/dL, which prevented comparisons of statistical value among patients

with SCr equal to or lower versus higher than 2 mg/dL (SCr limit is usually used as a risk criteria for CIN); exclusion of patients with previous AMI, PCI, CABG, stroke, serious peripheral vascular disease, chronic kidney disease, or recent exposure of contrast; elimination of high risk cases and failed operation, such as severe left main disease, preoperative hemodynamic instability, and reoperation or death within 72 hours; and mean total ischemic time was less than 6 hours. And total ischemia time greater than six hours were risk factors in previously published studies (24, 25).

From previous studies, a strong correlation has been identified between CIN and adverse clinical outcome, especially in acute coronary syndrome patients (26, 27). The prevention of CIN is a key strategy for improving the clinical outcome after various vascular interventions (28). The treatment of established CIN is limited to supportive measures and dialysis, therefore, prevention is the cornerstone for all patients at risk for CIN. The most effective preventive measure, which is hydration prior to use of contrast, is difficult to perform due to the emergency nature of AMI. Choosing a safer contrast medium may be a wise choice. However, the accumulated data do not provide clear evidence that the whole iso-osmolar contrast media offers an improvement over the low-osmolar contrast media except iohexol and ioxaglate (4). Previous studies have shown that CV correlated with CIN incidence, the ratio of CV-to-SCr clearance was a predictor of CIN after PCI, and the ratio of CV to glomerular filtration rate was a strong predictor of CIN and of 1-month and long-term mortality in patients undergoing primary PCI for STEMI (5, 6, 29). Thus, minimizing contrast dose is possible and pretty much encouraging (30), and it might be the most desirable option for preventing the incidence of CIN in STEMI patients undergoing primary PCI.

In the present study, although the incidence of CIN didn't decrease, TA did reduce the dosage of contrast agent in STEMI patients with HTB. Manual TA is a simple, intuitive idea to alleviate microvascular obstruction and improve flow in STEMI during PCI. It doesn't take substantial persuasion to understand that removing part or all of the thrombus blocking the artery before implanting a stent is beneficial, both in terms of the obstruction, and at the level of the microcirculation. In the initial clinical trials, TA in addition to conventional PCI demonstrated benefits regarding coronary flow and myocardial perfusion, and was therefore recommended in practice guidelines. However, recent large scale clinical trials (including TASTE and TOTAL) did not demonstrate survival benefits. Hence the recent guidelines do not suggest routine TA, however, selected use may facilitate the improvement of TIMI 3 flow or prevent stent thrombosis (31). In other words, it is reasonable to perform selective TA for patients with large thrombus burden or as a rescue strategy for distal embolization. As well as in large-scale clinical studies recently (32, 33), our study had confirmed that TA can improve myocardial perfusion in STEMI patients with HTB during primary PCI, similar to our previous research (12). Once the slow-flow / no-reflow phenomenon occurs during PCI, several measures including intracoronary injection of tirofiban or sodium nitroprusside should be required. And repeated angiography is necessary to verify whether the phenomenon has been eliminated, that followed by the increase of the contrast dose. The improvement of myocardial perfusion in TA group is helpful to shorten the operation time and reduce the frequency of angiography, that is the main reason for the decrease of contrast dose. The reduction of CV by TA may be limited, certainly, for experienced interventionists.

The pathogenesis behind CIN has not been completely understood. Although the contrast agent with less toxicity is often used, previous studies show that the CV is still an independent risk factor for CIN (34, 35), and it is considered that the dosage of contrast less than 70 mL can effectively reduce the risk of CIN (36). There is no linear relationship between the dosage of contrast agent and the incidence rate of CIN, but a threshold relationship. In a study of patients undergoing coronary angiography, each 100 mL of contrast medium administered was associated with a significant increase of 12% in the risk of CIN (37). Although the study confirmed that TA can reduce CV, the incidence of CIN did not decrease in STEMI patients with HTB. The reasons were chiefly as follows: first, as mentioned above, our study had excluded a large proportion of high-risk STEMI patients, who were high-risk factors for CIN. Moreover, the reduction of the proportion of high-risk cases also decreased the CV. In the present study, not only the mean CV (76.8 mL), but also the proportion of cases with dose greater than 200ml (6.1%) were lower than those of previous studies (6, 21, 22, 29). Meanwhile, the CV was only reduced by 10.8 ml in TA Group. Finally, the nephrotoxicity of ioversol is relatively low (4).

The present study had confirmed that TA before primary PCI, as compared with primary PCI alone, do not demonstrate additional clinical benefits during hospitalization in STEMI patients with HTB. In addition to the small sample size and the large proportion of high-risk STEMI excluded, the fact that the incidence of CIN did not decline may be another reason for the lack of benefits during hospitalization. Our previous research work also confirmed that, in comparison with PCI alone, administration of TA before stent implantation did not reduce the major adverse cardiovascular and cerebrovascular events (MACCE) within 30 days after procedure, although improved myocardial perfusion (12). Therefore, long-term follow-up was not carried out in the present study.

LIMITATIONS

There are also several potential limitations of the study. First, neither researcher nor patients will be blinded to the study allocation. And it is impossible to standardize the dosage of contrast agent in this study. Second, the present study based on a small number of patients enrolled from a single center. Third, a large proportion of high-risk STEMI patients were excluded, including those who died or reoperation within 72 hours after primary PCI. In this case, the incidence of CIN is relatively low, and so is the incidence of MACCE in hospital. Consequently, it caused an underestimation of the true incidence of CIN in this population. Therefore, it is not allowed to extend the results of this study to other STEMI with HTB. Furthermore, only the MACCE during hospitalization was collected and the occurrence after discharge was unknown. Future multicenter studies with larger sample sizes are needed to confirm these findings, especially focus on high-risk patients with STEMI.

Conclusions

This study demonstrated in STEMI patients with HTB that the contrast volume is significantly lowered by TA before primary PCI compared to primary PCI alone. However, TA did not reduce the incidence of CIN, or bring additional clinical benefit during in-hospital period.

Abbreviations

CIN = contrast-induced nephropathy;

CV = contrast volume;

HTB = high thrombus burden;

PCI = percutaneous coronary intervention;

SCr = serum creatinine;

STEMI = ST-elevation myocardial infarction;

TA = thrombus aspiration

Declarations

Author contributions

Conceptualization, B.F.C. and Y.D.; methodology, B.F.C. and L.Q.T.; formal analysis: B.F.C.; M.L.L and Y.D.; writing—original draft preparation, B.F.C. and Y.D; writing—review and editing, All authors.

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Competing interests

The authors declare no competing interests.

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