Original Article

Association between delayed transthoracic echocardiography and in-hospital mortality in type A acute aortic dissection-associated ST-segment elevated myocardial infarction

Bei Liu[#], Li-Dong Cai[#], Yi Wang

Department of Cardiology, Shanghai General Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, China

Contributions: (I) Conception and design: B Liu, Y Wang; (II) Administrative support: Y Wang; (III) Provision of study materials or patients: Y Wang; (IV) Collection and assembly of data: B Liu, LD Cai; (V) Data analysis and interpretation: B Liu, Y Wang; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

[#]These authors contributed equally to this work.

Correspondence to: Yi Wang. Department of Cardiology, Shanghai General Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, China. Email: wangyi2016@sjtu.edu.cn.

Background: This study evaluates the association between transthoracic echocardiography (TTE) timing and in-hospital mortality among individuals presenting with ST-segment elevated myocardial infarction (STEMI) complicating type A acute aortic dissection (TAAAD).

Methods: This cohort study obtained the data of previously published case reports from searches of PubMed (1990–2020), and adults with STEMI secondary to TAAAD were finally included. Delayed TTE (dTTE) exposure was defined as when the TTE test was made available after antithrombotic management for STEMI due to an initially missed diagnosis of TAAAD. The primary outcome of interest was in-hospital mortality, comparing individuals with dTTE and those with emergency TTE (eTTE). The odds ratio (OR) with 95% confidence interval (CI) were calculated to provide an estimate of association.

Results: A total of 109 individuals with a mean age of 56.7 [standard deviation (SD) 12.9] years, and of whom 75 were men (68.8%) presenting with STEMI complicating TAAAD were included. Of all patients, 68 (62.4%) had a dTTE test, which tended to be associated with increased in-hospital mortality after adjustment (OR, 2.320; 95% CI, 0.743–7.248). The association between dTTE and in-hospital death was significant only among patients presenting with a high-risk examination (HRE) (OR, 11.196; 95% CI, 1.322–94.803) and with surgical therapy (OR, 5.375; 95% CI, 1.080–26.700), and not among those presenting with negative HRE (OR, 0.150; 95% CI, 0.016–1.397) and no surgical therapy (OR, 0.177; 95% CI, 0.008–4.018).

Conclusions: This study found an association between dTTE and increased in-hospital mortality in TAAAD-associated STEMI patients with surgical management. This association warrants further investigation.

Keywords: ST-segment elevated myocardial infarction (STEMI), type A acute aortic dissection (TAAAD), transthoracic echocardiography (TTE)

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Introduction

As two common causes of acute fatal chest pain, ST-segment elevated myocardial infarction (STEMI) and type A acute aortic dissection (TAAAD) are poles apart regarding their therapeutic strategies. The antithrombotic therapy required in cases of the former is an absolute contraindication for the latter, and timely diagnosis and emergency therapy are vital for the successful management of both. However, the

rare incidence of coronary malperfusion complicates 10–15% of TAAAD cases, making both diagnosis and therapy intractable (1-6). The high mortality rate of 1–2% per hour after symptom onset renders immediate surgical treatment vital for TAAAD. However, its relative infrequency, coupled with its similar clinical presentation to acute coronary syndromes (ACS), can significantly delay hospital arrival and definitive diagnosis and treatment, potentially worsening the prognosis. Thus, distinguishing TAAAD from STEMI at the initial medical contact is of great clinical significance.

Transthoracic echocardiography (TTE) is safe, can be easily applied at the bedside, and is recommended as an appropriate first-line imaging technique for suspected acute aortic dissection (AAD) (7-9). Direct TTE signs can rapidly identify patients requiring advanced imaging despite low clinical probability, and in integrated bundles with D-dimer, negative TTE is useful for ruling out AAD (10). While TTE can also benefit early diagnosis, medical management, and even prognosis of patients with STEMI (11), its use is limited before emergent coronary reperfusion therapy to avoid an increase in total ischemic time. Previous data reveals the proportion of patients eventually diagnosed with AAD who receive an initial emergency diagnosis and management plan for ACS can be as high as 80%. For such a population, TTE timing might be instructive to inform clinical decisions and guide the use of antithrombotic medications and procedures. Thus, in the setting of STEMI complicating TAAAD, the association between TTE timing and prognosis is of considerable interest and is the subject of the present study.

We sought to understand the association between TTE timing and in-hospital death and hypothesized that delayed TTE (dTTE), defined as its availability after antithrombotic therapy as contraindicated management for TAAAD, increases the in-hospital mortality in this patient population.

We present the following article in accordance with the STROBE reporting checklist (available at http://dx.doi.org/10.21037/jtd-20-3470).

Methods

Study design, population, and setting

This retrospective cohort study included patients (aged ≥18 years) with STEMI complicating TAAAD from previously published case reports. All observations recorded in reports ceased when patients were discharged from the hospital or died. Case reports were identified from the PubMed (1990–2020) database using the search terms "aortic dissection and acute myocardial infarction" and "acute aortic syndromes and acute myocardial infarction". Abstracts of the identified papers were reviewed for appropriateness, and the reference lists of articles were manually checked for additional papers. Cases were included if they appeared in published case reports in which patients had been clinically diagnosed with STEMI caused by TAAAD. Cases in which an electrocardiograph (ECG) showed the presence of ST depression ≥1 mm in six or more surface leads (inferolateral ST depression), coupled with ST-segment elevation in aVR and V1, were also included due to the possibility of left main coronary artery obstruction (12,13).

The exclusion criteria were as follows: patients diagnosed with Stanford type B aortic syndrome, STEMI patients with a non-TAAAD cause, patients with non-ST segment elevation, percutaneous coronary intervention (PCI) induced iatrogenic aortic dissection (AD), patients with spontaneous coronary dissection, case reports with no clear ECG description, and in-hospital STEMI as a complication of surgery for AAD.The study was conducted following the Declaration of Helsinki (as revised in 2013) and approved by the Shanghai General Hospital ethics committee affiliated with Shanghai Jiaotong University (NO.: [2018] 24). Informed consent was not required as the data relating to included patients were from previously published case reports.

Clinical data collection

Two investigators independently screened all titles and abstracts to identify cases that met the inclusion criteria. When discordance occurred, cases were adjudicated after discussion, and disagreements were resolved by joint review. Inpatient data regarding baseline demographics, including age, sex, and medical history, pain features, physical examination findings, and imaging modalities parameters were extracted (as available) from individual papers.

dTTE exposure and outcomes

Patients who underwent TTE after antithrombotic therapy (antiplatelet aggregation and anticoagulation treatment) were initiated as part of a therapeutic plan for ACS, with a missed TAAAD diagnosis were defined as dTTE. If TTE was not performed during the management process, patients

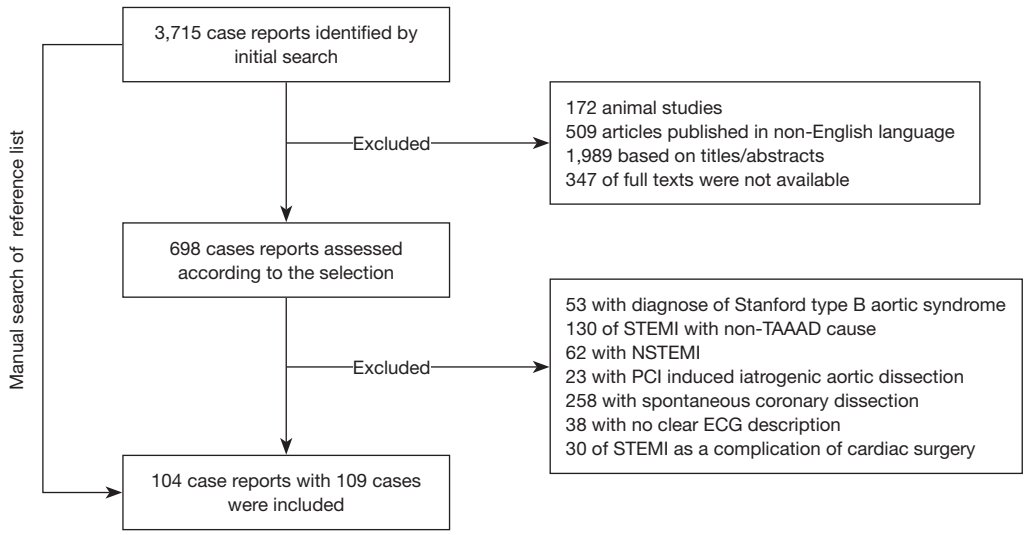

Figure 1 Flow diagram of case reports included in the final analysis. STEMI, ST-segment elevated myocardial infarction; TAAAD, type A acute aortic dissection; PCI, percutaneous coronary intervention; ECG, electrocardiograph.

were also classified as dTTE, and if emergency TTE (eTTE) was performed on admission before antithrombotic therapy for any reason, it was defined as no delay. The primary outcome of interest was in-hospital mortality, and cases with no reported in-hospital outcome were excluded.

Statistical analysis

IBM SPSS Statistic 25 was used to perform all statistical analyses. All normally distributed continuous variables were expressed as mean [standard deviation (SD)], and categorical variables were expressed as frequencies (%). Univariate comparisons between groups were performed using the χ^2 or Fisher's exact test for categorical data and the *t*-test for normally distributed continuous variables. In all cases, missing data were not defaulted to negative, and denominators reflect only cases reported. Univariate logistic regression and multivariate logistic regression models were used to examine whether dTTE and surgery had an independent effect on in-hospital death. In addition to demographic variables such as age, covariates associated with either dTTE or in-hospital mortality ($P<0.05$) were introduced into multivariate logistic regression. As dTTE was defined based on antithrombotic therapy, relevant variables such as CAG and aortography were rejected for multivariate analysis. Odds ratios (ORs) and hazard ratios with their corresponding 95% confidence intervals (CIs) were generated to provide an estimate of these associations, and a pre-specified subgroup analysis of dTTE and in-hospital death in patients with high-risk examinations (HREs) and surgery was respectively performed. All P values were calculated using two-tailed statistical significance tests with a type I error rate of 5%. Finally, as this was a retrospective analysis of the data from previously published cases, it does not apply to the sample size calculation.

Results

Demographics

The initial search identified 3,715 case reports (*Figure 1*). After excluding articles that were animal studies, not published in English, were not full texts, or were judged as irrelevant, the remaining 698 case reports were examined according to our defined selection criteria. Among these, 595 were excluded based on the exclusion criteria, and one case published in 1989 was manually found from the references list, leaving 104 reports with 109 cases eligible for final inclusion (see online Table S1). As shown in *Table 1*, 68.8% of patients were male, and all patients' mean age was 56.7±12.9 years. On admission, the mean systolic blood pressure (BP) of patients was 100.4±34.2 mmHg, the mean diastolic BP was 59.9±21.4 mmHg, and the mean heart rate was 74.5±25.5 bpm. Patients with dTTE had lower levels of BP compared with patients with eTTE. The reported aortic dissection risk scores (ADRS) included ADRS =0 in 1.1% of the patients, =1 in 33.0%, =2 in 61.7%, and =3 in 4.3% each.

Table 1 Baseline clinical characteristics and management

Variable	Rate of missing data	Total (n=109)	eTTE (n=41)	dTTE (n=68)	P value
Age, mean ± SD, year	0	56.7±12.9	55.4±13.4	57.6±12.6	0.394
Male, n (%)	0	75 (68.8)	26 (63.4)	49 (72.1)	0.345
SBP, mean ± SD, mmHg	40/109	100.4±34.2	110.2±40.7	93.7±27.5	0.048
DBP, mean ± SD, mmHg	50/109	59.9±21.4	66.7±24.6	55.9±18.4	0.059
HR, mean ± SD, bpm	40/109	74.5±25.5	73.3±23.5	75.3±27.0	0.748
Published date	0				0.467
Before 2008	–	31 (28.4)	10 (24.4)	21 (30.9)	
After 2008	–	78 (71.6)	31 (75.6)	47 (69.1)	
Hypotension/shock, n (%)	0	50 (45.9)	17 (41.5)	33 (48.5)	0.473
Chest pain, n (%)	0	105 (96.3)	39 (95.1)	66 (97.1)	0.602
Back pain, n (%)	0	16 (14.7)	7 (17.1)	9 (13.2)	0.583
Abdominal pain, n (%)	0	4 (3.7)	1 (2.4)	3 (4.5)	0.586
HRC, n (%)	0	12 (11.0)	7 (17.1)	5 (7.4)	0.116
HRP, n (%)	15/109	85 (90.4)	28 (84.9)	57 (93.4)	0.176
HRE, n (%)	6/109	66 (64.1)	26 (70.3)	40 (60.6)	0.327
ADRS	15/109				0.327
ADRS =0, n (%)	–	1 (1.1)	0 (0.0)	1 (1.6)	
ADRS =1, n (%)	–	31 (33.0)	10 (30.3)	21 (34.4)	
ADRS =2, n (%)	–	58 (61.7)	20 (60.6)	38 (62.3)	
ADRS =3, n (%)	–	4 (4.3)	3 (9.1)	1 (1.6)	
ADRS Grouping	15/109				0.574
ADRS 0–1, n (%)	–	32 (34.0)	10 (30.3)	22 (36.1)	
ADRS 2–3, n (%)	–	62 (66.0)	23 (69.7)	39 (63.9)	
CR, n (%)	0	35 (32.1)	17 (41.5)	18 (26.5)	0.104
CAG, n (%)	0	81 (74.3)	25 (61.0)	56 (82.4)	0.013
Type of STEMI, n (%)	0				
Anterior STEMI	–	49 (46.7)	17 (43.6)	32 (48.5)	0.627
Inferior STEMI	–	56 (53.3)	22 (56.4)	34 (51.5)	0.100
Aortogram, n (%)	0	51 (46.8)	12 (29.3)	39 (57.4)	0.004
IVUS/OCT, n (%)	0	15 (13.8)	8 (19.5)	7 (10.3)	0.176
CTA, n (%)	0	71 (65.1)	30 (73.2)	41 (60.3)	0.172
MRI, n (%)	0	1 (0.9)	1 (2.4)	0 (0.0)	0.196
TEE, n (%)	1/109	25 (23.2)	3 (7.5)	22 (32.4)	0.003
Anti-thrombotic therapy, n (%)	0	86 (78.9)	28 (68.3)	58 (85.3)	0.035
Thrombolysis, n (%)	0	9 (8.3)	3 (7.3)	6 (8.8)	0.782
PCI, n (%)	0	25 (22.9)	10 (24.4)	15 (22.1)	0.779
Surgery, n (%)	0	87 (79.8)	32 (78.0)	55 (80.9)	0.721
Death, n (%)	0	34 (31.2)	10 (24.4)	24 (35.3)	0.234

SD, standard deviation; TTE, transthoracic echocardiography; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; ADRS, aortic dissection risk score; HRC, high-risk condition; HRP, high-risk pain; HRE, high-risk examination; CR, chest radiograph; CAG, coronary angiography; STEMI, ST-segment elevated myocardial infarction; IVUS, intravascular ultrasound; OCT, optical coherence tomography; CTA, computed tomography angiography; MRI, magnetic resonance imaging; TEE, transesophageal echocardiography; PCI, percutaneous coronary intervention.

Data on the type of acute myocardial infarction (AMI) based on ECG were available in 105 patients, of which 49 (46.7%) presented with anterior STEMI and 56 (53.3%) with inferior STEMI. No differences in ADRS and myocardial infarction (MI) type between groups were observed. Finally, more patients in dTTE received antithrombotic therapy than those in the eTTE group (85.3% *vs.* 68.3%, P=0.035).

Imaging tests and TTE timing exposure

Coronary angiography (CAG) was performed in 81 (74.3%) patients, and of these, 15 (13.8%) received intravascular imaging tests. In all patients, 71 (65.1%) had CTA, and 25 (23.2%) had transesophageal echocardiography (TEE). While most patients (90.8%) received TTE at some point during their management, in more than half (62.4%), this was delayed. In patients with dTTE, more had CAG, aortogram, and TEE compared with patients with eTTE. Interestingly, more direct TTE signs of TAAAD, such as an intimal flap, were reported in patients with the dTTE test than those with eTTE (*Figure 2*).

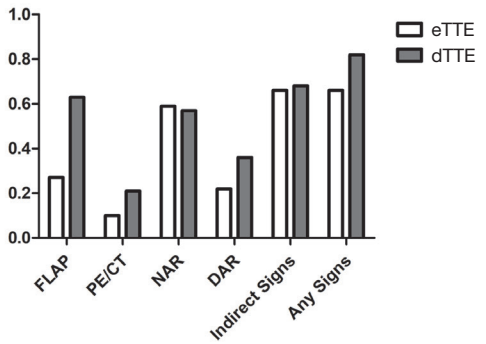

Figure 2 Differences of TTE signs between groups. The results were compared between groups based on TTE timing (before and after antithrombotic therapy). Direct signs include flap, penetrating aortic ulcer, and intramural hematoma. Indirect signs include new aortic regurgitation (NAR), dilated aortic root (DAR), and pericardial effusion/cardiac tamponade (PE/CT). eTTE, emergency transthoracic echocardiography; dTTE, delayed TTE.

Association between dTTE and outcomes

Among all included cases, 34 (31.2%) were in-hospital deaths. As shown in Table S2, patients with a positive HRE had a higher risk of in-hospital death than those with a negative HRE (78.12% *vs.* 57.75%, P=0.046), and patients with dTTE had a higher hazard for in-hospital mortality (OR, 1.691; 95% CI, 0.709–4.033) at univariate analysis. At the multivariable regression analysis, dTTE tended to increase in-hospital death (OR, 2.320; 95% CI, 0.743–7.248) after being adjusted for age, surgery, TEE, and HRE (*Table 2*). In a further subgroup analysis, dTTE in patients with positive HRE (OR, 11.196; 95% CI, 1.322–94.803) and patients receiving surgery (OR,5.375; 95% CI, 1.080–26.700) were significantly associated with higher in-hospital mortality respectively (*Table 3*).

Remedial clues for suspected TAAAD during CAG

A delay in diagnosing TAAAD may be unavoidable in patients presenting as STEMI with non-specific conditions/clinical manifestations or low ADRS (0–1). In these cases, TAAAD should be considered when catheter operation is difficult, characteristic angiographic abnormalities, and an inconsistency between angiographic imaging and clinical manifestations during the CAG procedure, and further imaging tests should be planned

Table 2 Association between delayed transthoracic echocardiography and in-hospital mortality

Variable	Univariable analysis				Multivariable analysis*			
	OR	95% CI		P value	OR	95% CI		P value
dTTE	1.691	0.709	4.033	0.236	2.320	0.743	7.248	0.148
Age	1.016	0.984	1.049	0.342	0.995	0.952	1.039	0.805
Surgery	0.071	0.023	0.221	0.000	0.044	0.010	0.189	0.000
TEE	1.032	0.395	2.694	0.949	0.824	0.210	3.223	0.780
HRE	2.613	0.999	6.834	0.050	4.357	1.196	15.875	0.026

*, adjusted for age, surgery, TEE, HRE. dTTE, delayed transthoracic echocardiography after electrocardiograph; TEE, transesophageal echocardiography; HRE, high-risk examination.

Table 3 Subgroup analysis of delayed transthoracic echocardiography on in-hospital mortality

Variables	OR	95% CI
Shock		
Without shock	1.398	0.269–7.273
With shock	4.327	0.440–42.593
HRE		
HRE =0	0.150	0.016–1.397
HRE =1	11.196	1.322–94.803
ADRS		
ADRS =0–1	0.298	0.014–6.537
ADRS =2–3	4.741	0.863–26.030
Surgery		
No surgery	0.177	0.008–4.018
Surgery	5.375	1.080–26.700

HRE, high-risk examination. ADRS, aortic dissection risk score.

(*Figure 3*).

Discussion

In this retrospective cohort study, dTTE tended to be associated with a higher risk-adjusted in-hospital mortality in STEMI, complicating TAAAD patients receiving surgical therapy. While establishing a causal link between TTE timing and patient outcomes was not the aim of this study, the observed association may assist physicians in management decisions (14).

In-hospital mortality and delay in TAAAD diagnosis and therapy

With the significant increase in surgical management employed to treat TAAAD over the past 20 years, the overall in-hospital mortality has decreased significantly from 31% to 22% (15), while in those managed medically, the rate has remained unchanged at 57% (16). The in-hospital mortality in the present study was 31.2%, which is slightly higher than that in the International Registry of Acute Aortic Dissection (IRAD). In those patients who managed surgically, the rate was similar to that seen in IRAD (20.0% *vs.* 18%), but those receiving medical therapy without surgery had higher mortality than IRAD (77.3% *vs.* 57.0%). As an independent predictor of in-hospital mortality, shock in the present cases was almost twice as high as in IRAD (44.4% *vs.* 15.0%) (16,17). This may reflect the complication of coronary malperfusion. Also, previous studies have demonstrated patient outcomes can be compromised if therapeutic measures are delayed or misappropriated. In cases where symptoms are not specific, ECG with new Q wave or ST-segment changes has been documented to cause delays in diagnosis (18,19) and contraindicated management, including antithrombotic therapy and even thrombolysis, in TAAAD. Other variables associated with a delayed diagnosis include female sex, the absence of typical historical features or HRE findings, including hypotension or pulse deficits, and initial visitation to nontertiary hospitals. These findings suggest the need for earlier detection and aggressive surgical treatment to provide a reasonable chance of survival to these patients with an otherwise dismal prognosis.

Underused TTE in TAAAD screening

TTE is a tool providing relevant bedside data and has a sensitivity of 78% to 100% with a specificity of 93–96% for TAAAD (20-22). The advantages of TTE, including its ready availability and ability to be performed quickly and at the bedside, make it the test of choice to be used in early screening for TAAAD. Combined with ADRS ≤1, a negative TTE had a sensitivity of 93.8% and a failure rate of 1.9% for ruling out AAD. However, data from the IRAD showed TTE was used in only 25% of cases as an initial diagnostic study (16), indicating underuse of TTE in AD pre-inspection. In the present study, although 90.8% of cases underwent TTE during in-hospital management, only 37.6% did so immediately on admission before antithrombotic therapy. Of these, at least one suspicious sign of TAAAD was found in 65.9%, including 11 (26.8%) with flap, 4 (9.8%) with pericardial effusion, 24 (58.5%) with new aortic regurgitation, and 9 (22.0%) with a dilated aortic root. In STEMI patients, routine TTE before reperfusion therapy is unnecessary in order to reduce the total ischemic time (12). However, eTTE is reasonable for a particular STEMI population with cardiac arrest, cardiogenic shock, hemodynamic instability or suspected mechanical complications, and an uncertain diagnosis. In this study, in patients with a high probability of AAD, only one-third (34.0% in shock and 37.0% in ADRS of 2–3) received eTTE, which was not in compliance with the recommendation of STEMI guidelines and indicated underuse of eTTE for appropriate indications. Equally

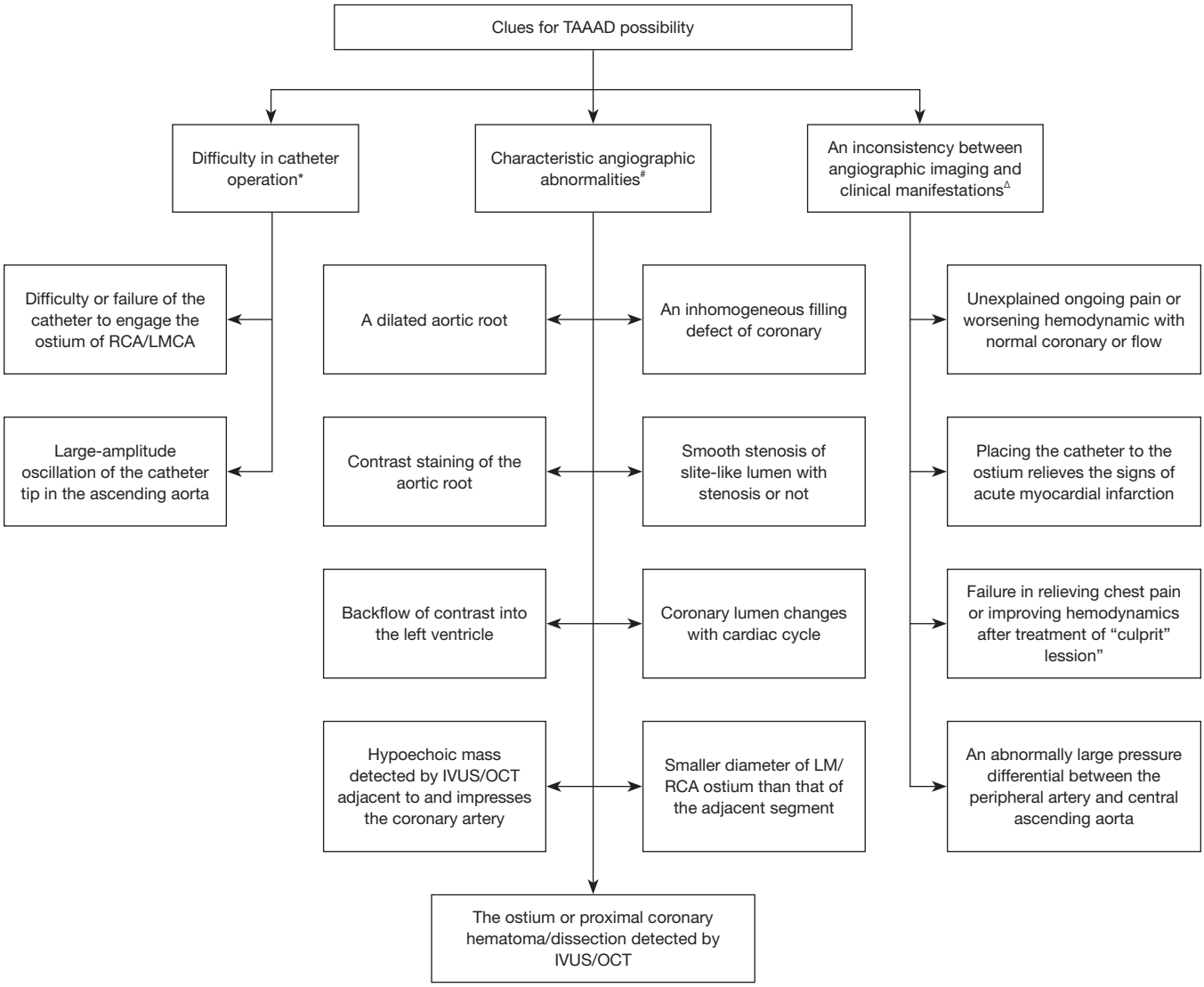

Figure 3 Remedial clues for suspected TAAAD during coronary angiography. *, case reports No. of 10, 12, 15, and 17; #, case reports No. of 6, 8, 10, 12, 14, 16–25, 44, 51, and 101; △, case reports No. of 22, 48, 56, 64, 65, 67, 73, 77, 86, 87, 99, and 104. Case reports No. can be available in Table S1. TAAAD, type A acute aortic dissection; CAG, coronary angiography; PCI, percutaneous coronary intervention; RCA, right coronary artery; LMCA, left main coronary artery; IVUS, intravascular ultrasound; OCT, optical coherence tomography.

concerning is the fact that more TTE signs of TAAAD, including intimal flap, were detected in patients with dTTE than those immediately on admission. A possible explanation for this may be that clues for TAAAD available during CAG (summarized in *Figure 3*) could remind sonographers to look more closely for signs of TAAAD. Therefore, it may be advisable to introduce eTTE into the STEMI process, especially in cases with a high risk of TAAAD, including those with shock or ADRS of 2–3. While this may result in concerns over the delay in coronary perfusion, further shortening door-to-balloon times within 90 minutes bring no significant overall change in mortality (23), and as bedside eTTE can provide red flags warranting emergency aortic imaging just within minutes (10), this might not necessarily delay reperfusion time. Further prospective studies are required to explore whether the total ischemic time is prolonged and the STEMI prognosis is affected by eTTE or whether more TAAAD is identified

and the prognosis improves.

TTE timing and in-hospital mortality of TAAAD

Previous studies have established a link between TTE and mortality among patients hospitalized for five specific cardiovascular diseases (24), but the timing has not been considered. In the present study, patients undergoing dTTE had a higher risk of developing in-hospital mortality than eTTE patients in a subgroup of both (I) patients with positive HRE and (II) patients receiving surgery. Several hypotheses have been forwarded to account for this increase in mortality. The first hypothesis suggests dTTE would increase delays to diagnosis, which would worsen outcomes (25), and many patients who experienced delays to diagnosis also experienced delays to surgical treatment of AD (26). As the preferred initial imaging modality in IRAD, CT is associated with longer surgery delays regardless of the transfer. An aortogram may be performed as part of an evaluation for ACS or in patients undergoing CAG before surgery, which also slows the surgery path. Dissection was more rapidly diagnosed when TTE formed part of the diagnostic workup (17). The imaging study's results showed that very critical patients who have a bedside TTE confirming a diagnosis of proximal AD are frequently sent directly to the operating room. Consistent with the above background, the association between dTTE and in-hospital death was significant only among patients presenting with a positive HRE and surgical therapy, not among those presenting with negative HRE and no surgical treatment. This suggests the benefits of eTTE in improving the prognosis might come from guiding early diagnosis and surgical initiation in high-risk patients.

A second hypothesis suggests the increased use of antithrombotic therapy observed in dTTE cases may increase the risk of complications such as shock and tamponade, as well as perioperative bleeding. The use of preoperative antithrombotic therapy could increase the risk of intraoperative bleeding, thus delay surgery. Moreover, aiming to decrease wall stress and limit the extension of dissection using initial medical therapy is also vital to reduce the risk of developing end-organ damage and rupture (27). Thus, information gained by eTTE to avoid contraindications to management can also optimize the medical therapy related to AD. It is vital to reduce early risk and delay surgery to allow patients to transfer to expert aortic surgery centers. More cases in this study who underwent dTTE received antithrombotic therapy, which may indicate a lack of timely optimal medical management and might be one of the causes for higher in-hospital mortality. However, it is impossible to assert this, given the small sample size and incomplete relevant data.

Thirdly, a focused TTE on the aortic valve complex and distinct aorta segments along with a glimpse into left heart structure and function may provide essential information needed in both anesthetic and surgical protocols for clinical decision-making (28). TTE is an excellent tool to quickly detect the potentially lethal complications of TAAAD, such as aortic regurgitation, pericardial effusion, cardiac tamponade, and wall motion abnormalities. Preoperative TTE findings provided crucial diagnostic information and prognostic value and were useful for improving the management strategy. Pericardial effusion, tamponade, periaortic hematoma, and false patent lumen were more frequent in non-survivors (29). As the least expensive and lowest-risk cardiac imaging modality available, eTTE is a first-tier diagnostic tool that routinely leads to the earlier initiation of various therapeutic interventions such as heart failure regimens, use of implantable devices, surgery, and revascularization, all of which have been shown to improve mortality rates in the appropriate clinical setting.

Limitations

Our study has some limitations. First, the sample size was small, although the association between dTTE and in-hospital mortality in patients receiving surgery was highly significant. Secondly, the data could be influenced by selection and ascertainment bias. However, as a rare condition of STEMI complicating TAAAD, the most complicated cases were likely reported, but the patients that have been successfully managed will also be useful experiences to be shared, which reduces selection bias to some extent. Thirdly, detailed data on the use of different surgical techniques and medical management, which may have influenced in-hospital mortality, were not included in this study as our study focused on TTE timing. Finally, our study results should be considered hypothesis-generating because of the study's observational nature.

Conclusions

The delayed performance of TTE was associated with an increase in the in-hospital mortality in STEMI, complicating TAAAD patients with surgery management based on previously published case reports. This detriment's mechanism remains to be explored but may be related

to the delayed diagnosis and surgery, increased use of antithrombotic therapy, and inadequate medical management as indicated and guided by TTE results. As the incidence of STEMI complicating TAAAD is a rare, randomized controlled trial (RCT)-based data are lacking, and no RCT will likely be performed to provide evidence in the future. The real-world data application provides a way to assess management's clinical effectiveness, such as TTE timing. Our findings should not be taken as the final and definitive word concerning the value of TTE timing in the management of TAAAD associated STEMI. As a reviewed analysis of data from previously published case reports, the potential issues of residual confounding by variables not captured require additional investigation.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at http://dx.doi.org/10.21037/jtd-20-3470

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at http://dx.doi.org/10.21037/jtd-20-3470). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the ethics committee of Shanghai General Hospital affiliated to Shanghai Jiaotong University. (NO.: [2018] 24). Informed consent was not required as the data relating to included patients were from previously published case reports.

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Table S1 The list of case reports

Number	Author	Published year	Title
1	Blankenship JC, *et al.* (30)	1989	Cardiovascular complications of thrombolytic therapy in patients with a mistaken diagnosis of acute myocardial infarction
2	Marian AJ, *et al.* (31)	1993	inadvertent administration of rtPA to a patient with type 1 aortic dissection and subsequent cardiac tamponade
3	Luciani GB, *et al.* (32)	1993	Mechanical assist device for right ventricular failure after acute aortic dissection
4	Garvey P, *et al.* (33)	1998	Aortic dissection and myocardial infarction in a pregnant patient with Turner syndrome
5	Shapira OM, *et al.* (34)	1998	Functional Left Main Coronary Artery Obstruction Due to Aortic Dissections
6	Tominaga R, *et al.* (35)	1999	Acute Type A Aortic Dissection Involving the Left Main Trunk of the Coronary Artery A Report of Two Successful Cases
7	Chang HC, *et al.* (36)	2004	Aortic intramural hematoma presenting as acute inferior wall MI with cardiogenic shock
8	Ashida K, *et al.* (37)	2000	A case of aortic dissection with transient ST-segment elevation due to functional left main coronary artery obstruction
9	Hu SY, *et al.* (38)	2001	Acute Myocardial Infarction Caused by Aortic Dissection
10	Pinney SP, *et al.* (39)	2002	Anterior myocardial infarction, acute aortic dissection, and anomalous coronary artery
11	Scarabeo V, *et al.* (40)	2002	dissection of the ascending aorta mimicking an acute coronary syndrome: usefulness of transthoracic echocardiography for the differential diagnosis
12	Chan KC, *et al.* (41)	2003	Acute aortic dissection developing acute myocardial infarction diagnosed by accidentally inserting the catheters into the false lumen during catheterization: a pitfall and rare image
13	Kamada M, *et al.* (42)	2003	Acute Type A aortic dissection complicated with acute inferior myocardial infarction following aortic valve replacement
14	Hibi K, *et al.* (43)	2003	Images in cardiovascular medicine. Intracoronary ultrasound diagnosis of an aortic dissection causing anterior acute myocardial infarction
15	Almassi GH, *et al.* (44)	2003	Proximal prolapse of aortic intimal flap: a rare complication of acute Type A aortic dissection
16	Kwon KS, *et al.* (45)	2004	A Case of Left Main Coronary Stenting for Acute Myocardial Infarction Complicated by Ascending Aortic Dissection
17	Sung L, *et al.* (46)	2005	Acute Myocardial Infarction Caused by Acute Ascending Aortic Dissection
18	Imoto K, *et al.* (47)	2005	Stenting of a left main coronary artery dissection and stent-graft implantation for acute Type A aortic dissection
19	Kawano H, *et al.* (48)	2006	Aortic dissection associated with acute myocardial infarction and stroke found at autopsy
20	Na SH, *et al.* (49)	2006	Images in cardiovascular medicine. Acute myocardial infarction caused by extension of a proximal aortic dissection flap into the right coronary artery: an intracoronary ultrasound image
21	Gu YL, *et al.* (50)	2006	Aortic dissection presenting as acute myocardial infarction: potential harm of antithrombotics and antiplatelet therapy
22	Horszczaruk GJ, *et al.* (51)	2006	Aortic dissection involving ostium of right coronary artery as the reason of myocardial infarction
23	Lee C H, *et al.* (52)	2007	Type A aortic dissection: a hidden and lethal cause for failed thrombolytic treatment in acute myocardial infarction
24	Tarver K, *et al.* (53)	2007	Extensive aortic dissection presenting as acute inferior myocardial infarction
25	Omar AR, *et al.* (54)	2007	Peripartum acute anterior ST-segment elevation myocardial infarction: an uncommon presentation of acute aortic dissection
26	Cook J, *et al.* (55)	2007	Aortic dissection presenting as concomitant stroke and STEMI
27	Zegers ES, *et al.* (56)	2007	Acute myocardial infarction due to an acute type A aortic dissection involving the left main coronary artery
28	Radwan K, *et al.* (57)	2007	Acute aortic dissection imitating ST-elevation myocardial infarction with accompanying pulmonary embolism
29	Jo SH, *et al.* (58)	2008	Coronary artery dissection associated with ascending aortic dissection
30	Forbes RA, *et al.* (59)	2008	Aortic dissection mimicking acute myocardial infarction: the perils of prehospital care
31	Marek D, *et al.* (60)	2008	Mistakes in dealing with aortic dissection. Lessons from three warning cases
32	Camaro C, *et al.* (61)	2009	Acute myocardial infarction with cardiogenic shock in a patient with acute aortic dissection
33	Bilge M, *et al.* (62)	2009	Penetrating atherosclerotic aortic ulcer mimicking acute inferior myocardial infarction
34	Patanè S, *et al.* (63)	2009	Obstruction of the right coronary artery ostium due to acute aortic dissection
35	Ohki S, *et al.* (64)	2009	Pulsatile coronary artery luminal compression caused by aortic dissection
36	Sir JJ, *et al.* (65)	2009	Acute Myocardial Infarction Due to Aortic Dissection
37	Luo JL, *et al.* (66)	2009	Type A aortic dissection manifesting as acute myocardial infarction: still a lesson to learn
38	Dermengiu S, *et al.* (67)	2009	Spontaneous aortic dissection due to cystic medial degeneration. Report of a sudden death case and literature review
39	Dorman SH, *et al.* (68)	2008	Acute aortic dissection mimicking an acute coronary syndrome through occlusion of the right coronary artery
40	Yunoki K, *et al.* (69)	2010	Stenting of right coronary ostial occlusion due to thrombosed Type A aortic dissection: One-year follow-up results
41	Chamnarnphol N, *et al.* (70)	2010	Coronary spasm due to Type A aortic dissection complicated by hemopericardium: a case report of another possible cause of coronary malperfusion
42	Arrivi A, *et al.* (71)	2010	Undetected acute aortic dissection in a patient referred for primary coronary angioplasty: a successful treatment of perioperative bleeding after abciximab administration
43	Kaya MG, *et al.* (72)	2010	Acute Type A aortic dissection in a patient with ventricular septal defect
44	Ravandi A, *et al.* (73)	2011	Percutaneous intervention of an acute left main coronary occlusion due to dissection of the aortic root
45	Tsigkas G, *et al.* (74)	2011	A successfully thrombolysed acute inferior myocardial infarction due to Type A aortic dissection with lethal consequences: the importance of early cardiac echocardiography
46	Saxena P, *et al.* (75)	2011	Left main coronary artery stenting prior to surgical repair of a type A aortic dissection
47	Kaul P, *et al.* (76)	2011	Innominate truncal dissection and rupture into right pleural cavity following acute Type A dissection of the aorta with right coronary ostial avulsion and inferior STEMI
48	Gong HR, *et al.* (77)	2012	A case report of misdiagnosis of acute myocardial infarction complicated with aortic dissection
49	Cockburn JA, *et al.* (78)	2012	Novel use of a guiding catheter to relieve left main stem occlusion complicating Stamford type A aortic dissection
50	Arrivi A, *et al.* (79)	2012	Acute Aortic Dissection Mimicking STEMI in the Catheterization Laboratory: Early Recognition Is Mandatory
51	Cai J, *et al.* (80)	2012	Inferior myocardial infarction secondary to aortic dissection associated with bicuspid aortic valve
52	Ali OM, *et al.* (81)	2012	Trapdoor ischaemia: coronary ostial occlusion by an aortic dissection
53	D'Aloia A, *et al.* (82)	2012	A Type A Aortic Dissection Mimicking an Acute Myocardial Infarction
54	Almansori M, *et al.* (83)	2012	Aortic dissection compressing the left circumflex coronary artery
55	Leonida C, *et al.* (84)	2013	An uncommon presentation of acute Type A aortic dissection: left main coronary artery obstruction
56	Okamoto M, *et al.* (85)	2012	A Case of Acute Myocardial Infarction due to Left Main Trunk Occlusion Complicated with Aortic Dissection as Diagnosed by Intravascular Ultrasound
57	Januszewski M, *et al.* (86)	2012	Haemodynamic tracing pattern reveals acute Type A aortic dissection
58	Fernández-Jiménez R, *et al.* (87)	2012	Acute aortic dissection with ongoing right coronary artery and aortic valve involvement
59	Güvenç TS, *et al.* (88)	2013	Acute aortic regurgitation with myocardial infarction: an important clue for aortic dissection
60	Dalén M, *et al.* (89)	2013	Ticagrelor-associated bleeding in a patient undergoing surgery for acute Type A aortic dissection
61	Lentini S, *et al.* (90)	2013	Hybrid management of acute Type A aortic dissection presenting as acute coronary syndrome
62	Yang EH, *et al.* (91)	2013	Circumferential type A aortic dissection and intimal intussusception of the aorta causing severe aortic regurgitation and obstruction of the left main coronary artery
63	Katayama A, *et al.* (92)	2013	Partial root remodeling on the two sinuses for acute Type A aortic dissection with right coronary arterial dissection
64	Kodera S, *et al.* (93)	2015	Percutaneous coronary intervention is a useful bridge treatment for acute myocardial infarction due to acute Type A aortic dissection
65	Wu BT, *et al.* (94)	2014	Type A Aortic Dissection Presenting with Inferior ST-Elevation Myocardial Infarction
66	Tang L, *et al.* (95)	2014	Acute Stanford Type A Aortic Dissection Mimicking Acute Myocardial Infarction: A Hidden Catastrophe Which Should Prompt Greater Vigilance
67	Kim SH, *et al.* (96)	2014	IVUS images of the left main intramural hematoma from aortic dissection
68	Kızıltan HT, *et al.* (97)	2014	Intimal intussusception in aortic dissection and coexisting coronary artery disease
69	Li CH, *et al.* (98)	2014	Myocardial hypoperfusion in acute aortic dissection
70	Hanaki Y, *et al.* (99)	2015	Coronary stenting with cardiogenic shock due to acute ascending aortic dissection
71	Ichihashi T, *et al.* (100)	2016	Acute myocardial infarction due to spontaneous, localized, acute dissection of the sinus of Valsalva detected by intravascular ultrasound and electrocardiogram-gated computed tomography
72	Wang ZG, *et al.* (101)	2015	Successful treatment of a case of acute myocardial infarction due to Type A aortic dissection by coronary artery stenting: A case report.
73	Ruisi M, *et al.* (102)	2015	Aortic dissection presenting as acute subtotal left main coronary artery occlusion: a case approach and review of the literature
74	Hawatmeh A, *et al.* (103)	2016	A case of ascending aortic dissection mimicking acute myocardial infarction and complicated with pericardial tamponade
75	Chen A, *et al.* (104)	2015	Aortic Dissection Manifesting as ST-Segment-Elevation Myocardial Infarction
76	Erkan H, *et al.* (105)	2015	Aortic dissection flap imitating intracoronary thrombus: an extremely rare angiographic presentation of acute myocardial infarction
77	Ahn JH, *et al.* (106)	2015	Paraparesis after Primary Percutaneous Coronary Intervention for ST-segment Elevation Myocardial Infarction: Combined Uncommon Complications of Acute Aortic Syndrome in a Patient
78	Magno Palmeira M, *et al.* (107)	2016	Aortic aneurysm with complete atrioventricular block and acute coronary syndrome
79	Treptau J, *et al.* (108)	2016	Angiographic detection of fatal acute aortic dissection Stanford type A under resuscitation
80	Takahashi K, *et al.* (109)	2016	Intravascular Ultrasound-Diagnosed Acute Aortic Dissection Involving Left Main Closure
81	Doksöz A, *et al.* (110)	2017	A case of aortic dissection complicating right subclavian artery occlusion and mimicking inferior myocardial infarction
82	She J, *et al.* (111)	2016	Chest Pain after Aortic Valve Replacement: Rupture of Right Sinus of Valsalva Presenting as Myocardial Infarction
83	Prakash B, *et al.* (112)	2017	A Case of Acute Paraplegia Due to Aortic Dissection in Marfan Syndrome
84	Wang D, *et al.* (113)	2017	The missed diagnosis of aortic dissection in patients with acute myocardial infarction: a disastrous event
85	Sadaba M, *et al.* (114)	2017	STEMI with fluctuating closing of LAD and Cx: Do not concentrate on the finger
86	Attia T, *et al.* (115)	2017	Successful treatment of aortic root dissection complicated with extensive myocardial infarction using the total artificial heart
87	Zhu Q, *et al.* (116)	2017	ST-segment elevation myocardial infarction could be the primary presentation of acute aortic dissection
88	Gohbara M, *et al.* (117)	2017	Left main trunk stenting in a case of acute aortic dissection: a case report.
89	Rodrigues RC, *et al.* (118)	2017	Aortic Dissection Mimicking ST-Elevation Myocardial Infarction
90	Kunwor R, *et al.* (119)	2017	Rare Case of Cocaine-Induced Aortic Aneurysm: A Near Dissection Event
91	Chenkin J, *et al.* (120)	2017	Diagnosis of Aortic Dissection Presenting as ST-Elevation Myocardial Infarction using Point-Of-Care Ultrasound
92	Yukawa T, *et al.* (121)	2017	treatment of a patient with acute aortic dissection using extracorporeal cardiopulmonary resuscitation after an out-of-hospital cardiac arrest: a case report
93	Massetti M, *et al.* (122)	2003	Flap suffocation: an uncommon mechanism of coronary malperfusion in acute Type A dissection
94	Vianna CB, *et al.* (123)	2007	Spontaneous aortic dissection limited to sinus of Valsalva and involving the left main coronary artery
95	Choi CH, *et al.* (124)	2012	Acute type A aortic dissection initially diagnosed with myocardial infarction
96	Barabas M, *et al.* (125)	2000	Left main stenting-as a bridge to surgery-for acute Type A aortic dissection and anterior myocardial infarction
97	Cardozo C, *et al.* (126)	2004	Acute myocardial infarction due to left main compression aortic dissection treated by direct stenting
98	Nordt TK, *et al.* (127)	1991	Acute myocardial infarction due to proximal aortic dissection in giant cell aortitis
99	Chen HY, *et al.* (128)	2019	Acute myocardial infarction and coronary intramural haematoma: a clue to aortic dissection
100	Domingues K, *et al.* (129)	2019	Left Main Occlusion - A True or False (Lumen) STEMI Diagnosis?
101	Yennu Nandan A, *et al.* (130)	2019	Suspected ST-segment elevation myocardial infarction referred for primary angioplasty: a masquerader
102	Abrams E, *et al.* (131)	2019	Aortic Dissection with Subsequent Hemorrhagic Tamponade Diagnosed with Point-of-care Ultrasound in a Patient Presenting with STEMI
103	Wang W, *et al.* (132)	2019	Type-A aortic dissection manifesting as acute inferior myocardial infarction: 2 case reports
104	Huang CY, *et al.* (133)	2020	Catheter directed diagnosis of ST-segment elevation myocardial infarction induced by Type A aortic dissection: A case report

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Table S2 Baseline variables associated with in-hospital mortality

Variables	Survive (n=75)	Death (n=34)	P value
Age, mean ± SD, years	56.11±12.28	58.65±14.32	0.345
Male, n (%)	53 (70.67)	22 (64.71)	0.534
SBP, mean ± SD, mmHg	98.15±27.29	105.57±46.71	0.410
DBP, mean ± SD, mmHg	57.88±17.10	64.61±28.93	0.269
HR, mean ± SD, bpm	74.75±23.76	73.90±29.66	0.900
Hypotension/shock, n (%)	33 (44.00)	17 (50.00)	0.560
Chest pain, n (%)	74 (98.67)	31 (91.18)	0.054
Back pain, n (%)	10 (13.33)	6 (17.65)	0.555
Abdominal pain, n (%)	1 (1.35)	3 (8.82)	0.056
HRC, n (%)	9 (12.00)	3 (8.82)	0.624
HRP, n (%)	59 (90.77)	26 (89.66)	0.865
HRE, n (%)	41 (57.75)	25 (78.12)	0.046
CR, n (%)	21 (28.00)	14 (41.18)	0.172
CAG, n (%)	59 (78.67)	22 (64.71)	0.122
Type of STEMI, n (%)			
Anterior STEMI	33 (45.83)	16 (48.48)	0.800
Inferior STEMI	39 (54.17)	17 (51.52)	0.800
Aortogram, n (%)	37 (49.33)	14 (41.18)	0.429
IVUS/OCT, n (%)	13 (17.33)	2 (5.88)	0.108
CTA, n (%)	51 (68.00)	20 (58.82)	0.352
MRI, n (%)	1 (1.33)	0 (0.00)	0.499
TEE, n (%)	17 (22.67)	8 (24.24)	0.858
Anti-thrombotic therapy, n (%)	61 (81.33)	25 (73.53)	0.355
Thrombolysis, n (%)	5 (6.67)	4 (11.76)	0.370
PCI, n (%)	19 (25.33)	6 (17.65)	0.377
Surgery, n (%)	70 (93.33)	17 (50.00)	<0.001

SD, standard deviation; TTE, transthoracic echocardiography; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; HRC, high-risk condition; HRP, high-risk pain; HRE, high-risk examination; CR, chest radiograph; CAG, coronary angiography; STEMI, ST-segment elevated myocardial infarction; IVUS, intravascular ultrasound; OCT, optical coherence tomography; CTA, computed tomography angiography; MRI, magnetic resonance imaging; TEE, transesophageal echocardiography; PCI, percutaneous coronary intervention.

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