Original Article

Diagnostic performance of transthoracic echocardiography in screening acute type A aortic dissection from ST-segment elevated myocardial infarction

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Background: When patients with type A acute aortic dissection (TAAAD) present with changes to their ST-segment, diagnostic and treatment delays increase significantly. The performance of transthoracic echocardiography (TTE) screening of TAAAD in patients with ST-segment elevated myocardial infarction (STEMI) is yet to be validated.

Methods: The diagnostic performance of TTE alone and combined with the aortic dissection risk score (ADRS) in TAAAD was evaluated. In this retrospective study (ChiCTR, No. 2000031291), TTE was reviewed to detect direct/indirect signs of TAAAD. The ADRS of each patient was calculated according to guidelines. Case adjudication was based on advanced imaging and surgery.

Results: Among a total of 442 patients, TAAAD was diagnosed in 146 (33.0%). The presence of direct TTE signs had a sensitivity of 43.0% [95% confidence interval (CI): 35.0% to 52.0%] and specificity of 97.0% (95% CI: 95.0% to 99.0%), and the presence of any TTE sign had a sensitivity of 97.0% (95% CI: 93.0% to 99.0%) and specificity of 78.0% (95% CI: 73.0% to 82.0%) for TAAAD. The additive value of TTE was most evident in patients with low clinical probability for TAAAD (ADRS ≤1). The presence of ADRS ≤1 plus an absence of direct TTE signs for TAAAD rule-out had a sensitivity of 98.4% (95% CI: 96.1% to 99.6%).

Conclusions: The use of TTE adds value in the screening of TAAAD in STEMI patients. In patients with low clinical probability for TAAAD, direct TTE signs can be used to rapidly identify those who require advanced imaging.

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

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Introduction

Coronary malperfusion complicates 10–15% of type A acute aortic dissection (TAAAD) cases (1,2). Myocardial infarction (new Q waves or ST segments) is observed in 7.1% TAAAD cases and leads to delays in diagnosis and surgical treatment of TAAAD (3). Moreover, antithrombotic or thrombolytic therapy are contraindicated in TAAAD and may be inappropriately used by clinicians after the detection of electrocardiogram (ECG) ischemic abnormalities (4). In this context, an aortic dissection risk score (ADRS) was developed to identify acute aortic dissection (AAD) at initial presentation (5,6). Furthermore, D-dimer (D-D) with a cutoff value of 0.5 μg/mL can stratify patients suspected of AAD within the first 24 hours after symptom onset (7). Further study has demonstrated that negative D-D combined with the use of ADRS could enable AAD to be ruled out without performing conclusive imaging (8). Nevertheless, ST-segment elevated myocardial infarction (STEMI) guidelines recommend that patients undergo reperfusion therapy without needing to wait for myocardial injury markers to be available (9). However, the D-D test is not applied to screen TAAAD in the STEMI patients.

For patients with suspected AAD, transthoracic echocardiography (TTE) is proposed as the first-line imaging test by the European Society of Cardiology (ESC) and the European Association of Echocardiography, especially when patients have a low probability for AAD according to ADRS (10,11). Notably, in the STEMI patients, TTE is only suggested when the diagnosis is unclear or when complications occur. However, routine TTE tests before coronary angiography (CAG) are contraindicated because they may delay perfusion (9). Therefore, we found it necessary to explore the necessity and accuracy of TTE for TAAAD screening in STEMI patients.

According to the above background, the following hypotheses were made in the present study: (I) TTE can help to rapidly screen TAAAD patients for early computed tomography angiography (CTA) in STEMI; and (II) negative TTE combined with low clinical probability for AAD can be a safe strategy to rule out TAAAD in STEMI patients. We present the following article in accordance with the STARD reporting checklist (available at https://cdt.amegroups.com/article/view/10.21037/cdt-22-59/rc).

Methods

Study design

This was a retrospective study (ChiCTR, No. 2000031291) of data from two centers (Shanghai General Hospital and Nanchong Central Hospital). 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 Jiao Tong University (No. [2018] 23). Informed consent was not required due to the retrospective study design.

Study population, clinical data collection, and definitions

Data of TAAAD patients were retrospective obtained from Nanchong Central Hospital between January 2017 and December 2019, and data of STEMI patients were obtained from Shanghai General Hospital between January 2017 and December 2017. Patients who met all the following criteria were included: aged >18 years, presenting with chest pain within 14 days of onset, and a final diagnosis of either TAAAD or STEMI. Those ultimately diagnosed with any of the following causes were excluded: type B aortic dissection, non-ST segment elevated acute coronary syndrome, pulmonary embolism, acute pericarditis, non-cardiogenic or unexplained chest pain, and those without TTE available during hospitalization. We defined STEMI as patients with an ECG that showed ST-segment elevation and positive myocardial injury biomarkers, which was confirmed by angiography (9). We defined TAAAD as any dissection of the ascending aorta detected within 14 days of symptom onset (10). Patients were identified by searching the discharge diagnosis records and the TTE laboratory and surgical databases. Data including demographics, medical history, clinical presentation, physical findings, and imaging studies were collected by a retrospective physician review of hospital records.

Aortic imaging and final diagnosis

Two independent senior physicians established the final diagnosis by reviewing all available clinical data, such as aortic imaging studies and medical and surgical records. The following diagnoses were considered TAAAD: any dissection, intramural hematoma (IMH), or penetrating aortic ulcer (PAU) developed in the ascending part of the aorta. Cases with discordant diagnoses were discussed by specialized cardiologists who were not involved in the present study to resolve disagreements.

Aortic dissection detection risk score classification

Based on a review of medical charts for each patient, the

ADRS was calculated by an independent physician on 12 high-risk markers in the clinical categories of predisposing conditions, pain features, and physical findings (5). Patients with ADRS of 0 (without all risk markers) were classified as a low risk for AAD, those with ADRS of 1 (with any risk markers in any single category) were classified as an intermediate risk of AAD, while patients with ADRS >1 (with any risk markers in two or three categories) were classified as a high risk of AAD.

TTE

The results of TTE were in the form of reports as a post hoc analysis, therefore, the data about TTE in this study were recorded in the original form according to the report results. Direct signs of TAAAD were defined as the presence of either an intimal flap or aortic wall thickening (≥5 mm) in the ascending aorta. Indirect signs of TAAAD were defined as the following TTE findings: pericardial effusion (PE)/cardiac tamponade (CT), enlarged ascending aorta with diameter ≥4 cm, and aortic valve regurgitation (AVR).

Sample size

The aim of this study was to evaluate the performance of TTE to differentiate the signs for TAAAD from those for STEMI. The study assumed that the area under the curve (AUC) of the receiver operating characteristic (ROC) curve for TTE any signs plus ADRS predicting TAAAD was greater than 0.75. The AUC of the ROC curve for TTE any signs plus ADRS was determined, with reference to related studies, to be 0.88 (12). By setting the ratio of N– to N+ to 2, PASS 2021 (NCSS, Kaysville, UT, USA) determined the total sample of 81 to detect a difference of 0.13 between the AUC under the null hypothesis of 0.75 and an AUC under the alternative hypothesis of 0.88 using a one-sided z-test at a significance level of 0.15. Considering a possible 40% exclusion rate, we estimated that at least 135 patients needed to be included.

Statistical analysis

Categorical data were shown as frequencies and percentages, continuous variables with normal data distributions were shown as mean ± standard deviation (SD), and continuous variables with skewed distributions were shown as medians with first and third quartiles. to perform univariate comparisons between groups, a Student's *t*-test was used for continuous variables with normal data distributions, the χ^2 or Fisher's exact test were used for categorical data, and a nonparametric test of medians was used for continuous variables with skewed distributions. We used ROC analysis was used to determine the accuracy of TTE in predicting TAAAD. The number of cases with true positive (TP), true negative (TN), false positive (FP), and false negative (FN) were assessed to evaluate diagnostic performance of TTE in predicting TAAAD. The specificity, sensitivity, accuracy, positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio, and negative likelihood ratio of each significant feature and combinations of these features were calculated. to determine the model's clinical usefulness, decision curve analysis (DCA) was applied. At increasing decision thresholds, the TP and FP classifications were considered. The net benefit (NB) of using a model at different thresholds was demonstrated by the decision curve. A nomogram was developed by using counts of TTE signs for TAAAD and ADRS. The P values were two-sided, and $P<0.05$ was considered statistically significant. Patients with missing data on the TTE test were excluded. Empower (R) (www.empowerstats.com, X&Y solutions, Inc., Boston, MA, USA) and R (http://www.R-project.org) were applied to perform all statistical analyses.

Results

Baseline demographics and characteristics

Data of 3,177 potentially eligible patients were retrospectively reviewed, and data of 442 patients were finally analyzed (*Figure 1*). Among all patients included in the present study, 146 were TAAAD patients. Overall, TAAAD patients were younger than the other patients included in the study. The level of myocardial injury markers, including cardiac troponin I (cTnI), myohemoglobin, and creatine kinase (CK)-MB on admission was significantly lower in the TAAAD group than in the STEMI group. Both D-D and C-reactive protein (CRP) on admission in the TAAAD group were markedly higher than those in the STEMI group. No Marfan syndrome was reported in the STEMI group patients; 6 (4.1%) were recorded in the TAAAD cohort. History of aortic valve disease was 7.6× more common in patients with TAAAD, and previous aorta aneurysm was ≈5.1-fold higher in the TAAAD cohort compared with STEMI patients. More patients experienced syncope and back or abdominal pain in the TAAAD group than in the STEMI

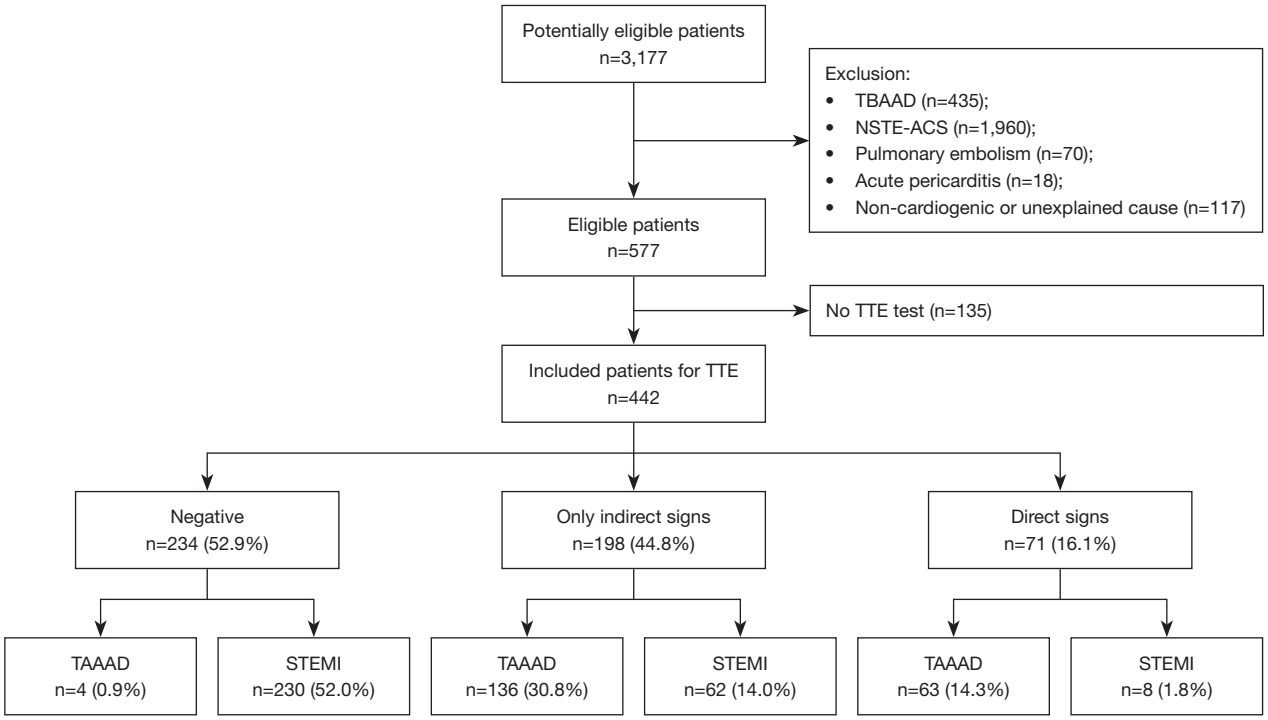

Figure 1 A flow diagram of the study. TTE negative: no direct or indirect signs of acute aortic syndrome; indirect signs: ascending aorta dilatation, pericardial effusion/tamponade, or aortic valve regurgitation; direct signs: intimal flap, intramural aortic hematoma, or penetrating aortic ulcer. % refers to 442 study patients. TAAAD, type A acute aortic dissection; TBAAD, type B acute aortic dissection; NSTE-ACS, non-ST-segment elevated acute coronary syndrome; TTE, transthoracic echocardiography; STEMI, ST-segment elevated myocardial infarction.

group, and presentation with tearing pain was 12.5-fold higher among TAAAD patients than in the STEMI group. The combination of shock and hypotension was 3-times higher among patients with TAAAD. Totals of 25 (17.12%), 22 (15.07%), and 13 (8.90%) patients in the TAAAD cohort reported pulse deficit, new aortic murmur, and focal neurological, respectively, and no STEMI patients reported those high-risk signs. Accordingly, there were more patients with ADRS ≥2 in the TAAAD group than in the STEMI group. Other demographics, presenting symptoms, and clinical features were similar between TAAAD and STEMI patients (*Table 1*).

Thoracic echocardiography

Direct TTE signs of TAAAD were detected in 8 (2.7%) STEMI patients, of which 4 (1.4%) intimal flap and 4 (1.4%) IMH were found. In 63 (43.2%) TAAAD patients, 39 (26.7%) had intimal flap, 18 (12.3%) IMH, and 6 (4.1%) PAU were recorded. There were 8 FP cases and 288 FN cases. Indirect TTE signs were reported in 62 (20.9%) STEMI patients, of which 49 (16.6%) had dilated ascending aorta, 13 (4.4%) had new detected aortic regurgitation, and 12 (4.05%) had PE upon computed tomography. In 136 (93.2%) TAAAD patients, 107 (73.3%) had dilated ascending aorta, 40 (27.4%) had new detected aortic regurgitation, and 71 (48.6%) had PE/CT. The diagnostic performance of TTE for TAAAD is presented in *Table 2*. The ROC analysis further demonstrated that the integration of TTE with ADRS remarkably increased the accuracy for TAAAD diagnosis (*Figure 2*). The performance of a diagnostic rule-out strategy integrating ADRS and TTE is detailed in *Table 3*.

Development of a nomogram for TAAAD diagnosis and internal validation

The nomogram for predicting TAAAD, based on the

Table 1 Demographics, history, clinical symptoms and signs between groups

Variables	STEMI (n=296)	TAAAD (n=146)	P value
Age (years), median [Q1–Q3]	65 [61–69]	53 [44–60]	<0.0001
Male, n (%)	175 (59.12)	97 (66.44)	0.1370
Diabetes, n (%)	97 (32.77)	47 (32.19)	0.9029
Current smoking, n (%)	122 (41.22)	57 (39.04)	0.6613
Hypertension, n (%)	205 (69.26)	101 (69.18)	0.9866
Old myocardial infarction, n (%)	36 (12.16)	10 (6.85)	0.0854
Systolic blood pressure (mmHg), median [Q1–Q3]	132 [119–148]	135 [118–160]	0.4260
Ascending aorta (mm), median [Q1–Q3]	38.18 [36.74–39.39]	47.35 [41.64–55.69]	<0.0001
cTnI (ng/mL), median [Q1–Q3]	2.40 [0.38–13.62]	0.10 [0.04–0.89]	<0.0001
Myoglobin >85 μg/L, n (%)	227 (76.7)	50 (34.2)	<0.0001
D-dimer (ng/mL), median [Q1–Q3]	662.5 [374.0–971.0]	3,856.5 [2,247.8–4,481.8]	<0.0001
D-dimer >500 (ng/mL), n (%)	196 (66.2)	143 (97.9)	<0.0001
C-reactive protein (mg/L), median [Q1–Q3]	6.20 [4.78–8.95]	15.10 [7.75–25.85]	<0.0001
CKMB (U/L), median [Q1–Q3]	95.50 [12.01–178.25]	4.55 [1.90–10.00]	<0.0001
Serum creatinine (μM), median [Q1–Q3]	115.0 [104.0–128.2]	97.2 [68.0–125.7]	0.190
Direct TTE signs, n (%)	8 (2.7)	63 (43.2)	<0.0001
Flap	4 (1.4)	39 (26.7)	<0.0001
Intramural hematoma	4 (1.4)	18 (12.3)	<0.0001
Aortic ulcer	0 (0.0)	6 (4.1)	<0.0001
Indirect TTE signs, n (%)	62 (20.9)	136 (93.2)	<0.0001
Ascending aorta enlargement (diameter ≥4 cm)	49 (16.6)	107 (73.3)	<0.001
Pericardial effusion	11 (3.7)	66 (45.2)	<0.0001
Cardiac tamponade	1 (0.34)	5 (3.42)	0.0083
Aortic regurgitation	13 (4.4)	40 (27.4)	<0.0001
High-risk conditions, n (%)	24 (8.11)	32 (21.92)	<0.0001
Marfan syndrome	0 (0.00)	6 (4.11)	0.0004
Family history of aortic dissection	7 (2.36)	5 (3.42)	0.5191
Aortic valve disease	4 (1.35)	15 (10.27)	<0.0001
Recent aortic manipulation	1 (0.34)	1 (0.68)	0.6091
Previous aorta aneurysm	4 (1.35)	10 (6.85)	0.0019
High-risk pains, n (%)	282 (95.27)	134 (91.78)	0.1425
Abrupt pain	272 (91.89)	134 (91.78)	0.9680
Severe pain	229 (77.36)	98 (67.12)	0.02
Tearing pain	6 (2.03)	37 (25.34)	<0.0001

Table 1 (*continued*)

Table 1 (*continued*)

Variables	STEMI (n=296)	TAAAD (n=146)	P value
High-risk examination, n (%)	19 (6.42)	56 (38.36)	<0.0001
Pulse deficit	0 (0.00)	25 (17.12)	<0.0001
New aortic murmur	0 (0.00)	22 (15.07)	<0.0001
Focal neurological deficit	0 (0.00)	13 (8.90)	<0.0001
Shock	11 (3.72)	10 (6.85)	0.1453
Hypotension	9 (3.04)	20 (13.70)	<0.0001
Syncope	7 (2.36)	14 (9.59)	0.0008
Chest pain, n (%)	260 (87.84)	119 (81.51)	0.0733
Back pain, n (%)	27 (9.12)	69 (47.26)	<0.0001
Abdominal pain, n (%)	24 (8.11)	31 (21.23)	<0.0001
ADRS, n (%)			<0.0001
0	12 (4.05)	3 (2.05)	
1	244 (82.43)	77 (52.74)	
2	39 (13.18)	53 (36.30)	
3	1 (0.34)	13 (8.90)	

ADRS, aortic dissection risk score; CKMB, MB isoenzyme of creatine kinase; cTnI, cardiac troponin I; Q1, the first quartile; Q3, the third quartile; STEMI, ST-segment elevated myocardial infarction; TAAAD, type A acute aortic dissection; TTE, transthoracic echocardiography.

Table 2 Diagnostic variables of TTE for diagnosis of TAAAD

TTE signs	TP	FP	TN	FN	Sensitivity (95% CI)	Specificity (95% CI)	PPV (95% CI)	NPV (95% CI)	+LR (95% CI)	–LR (95% CI)
Any TTE signs	142	66	4	230	0.97 (0.93, 0.99)	0.78 (0.73, 0.82)	0.68 (0.61, 0.75)	0.98 (0.96, 0.99)	4.36 (3.52, 5.41)	0.04 (0.01, 0.09)
Direct signs	63	8	83	288	0.43 (0.35, 0.52)	0.97 (0.95, 0.99)	0.89 (0.79, 0.95)	0.78 (0.73, 0.82)	15.97 (7.86, 32.42)	0.58 (0.51, 0.67)
Indirect signs	136	62	10	234	0.93 (0.88, 0.97)	0.79 (0.74, 0.84)	0.68 (0.62, 0.75)	0.96 (0.93, 0.98)	4.45 (3.55, 5.57)	0.08 (0.05, 0.16)
Thoracic aorta dilatation	107	49	39	247	0.73 (0.65, 0.80)	0.84 (0.79, 0.88)	0.69 (0.61, 0.76)	0.86 (0.82, 0.90)	4.43 (0.37, 5.82)	0.32 (0.24, 0.42)
AVR	40	13	106	283	0.27 (0.20, 0.35)	0.96 (0.93, 0.98)	0.75 (0.62, 0.86)	0.73 (0.68, 0.77)	6.24 (3.45, 11.29)	0.76 (0.69, 0.84)
PE/CT	66	11	80	285	0.45 (0.37, 0.54)	0.96 (0.93, 0.98)	0.86 (0.76, 0.93)	0.78 (0.73, 0.82)	12.16 (6.63, 22.31)	0.56 (0.49, 0.66)
ADRS grouping	66	40	80	256	0.45 (0.37, 0.54)	0.86 (0.82, 0.90)	0.62 (0.52, 0.72)	0.76 (0.71, 0.81)	3.35 (2.38, 4.64)	0.63 (0.54, 0.74)

ADRS, aortic dissection risk score; AVR, aortic valve regurgitation; 95% CI, 95% confidence interval; FN, false negative; FP, false positive; –LR, negative likelihood ratio; +LR, positive likelihood ratio; NPV, negative predictive value; PE/CT, pericardial effusion or cardiac tamponade.; PPV, positive predictive value; TAAAD, type A acute aortic dissection; TN, true negative; TP, true positive; TTE, transthoracic echocardiography.

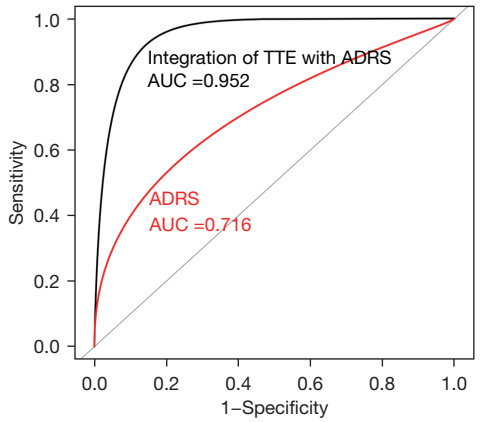

Figure 2 Receiver operating characteristic curves for TAAAD screening of integration of TTE with ADRS (black line) and ADRS (red line). ADRS, aortic dissection risk score; AUC, area under the curve; TAAAD, type A acute aortic dissection; TTE, transthoracic echocardiography.

combination with any TTE signs and ADRS, is provided in *Figure 3*. The AUC of the model was 0.963 (95% CI: 0.912 to 0.969). For nomogram interpretation, vertical lines should be drawn from each prognostic factor's correct status to the top axis (points). to convert this into a TAAAD probability, a vertical line should be drawn from the "total points" axis to the bottom axes after adding all the points. Decision curves for the TTE signs and ADRS are demonstrated in *Figure 4*. All models were useful between threshold probabilities of 30–80%. The clinical impact of ADRS to identify individuals with TAAAD was observed at a threshold of ≥30%, and of TTE signs combining with ADRS was at a threshold ≥5%; maximal utility occurred at 33.0%. By applying the model that integrated TTE signs with ADRS, higher NBs than the ADRS model for every patient could be achieved for a risk threshold above 5%.

Table 3 Diagnostic performance of strategies integrating ADRS, and TTE for rule-out of TAAAD

Variables	ADRS ≤1 and TTE negative	ADRS ≤1 and absence of direct TTE signs
Sensitivity (95% CI)	0.8203 (0.7677–0.8653)	0.9844 (0.9605–0.9957)
Specificity (95% CI)	0.9750 (0.9126–0.9970)	0.3750 (0.2692–0.4904)
Diagnose accuracy (95% CI)	0.8571 (0.8151–0.8928))	0.8393 (0.7956–0.8769)
PPV (95% CI)	0.9906 (0.9663–0.9989)	0.8344 (0.7876–0.8745)
NPV (95% CI)	0.6290 (0.5377–0.7140)	0.8824 (0.7255–0.9670)
+LR (95% CI)	32.8125 (8.3406–129.0862)	1.5750 (1.3282–1.8677)
–LR (95% CI)	0.1843 (0.1415–0.2400)	0.0417 (0.0151–0.1147)

+LR, positive likelihood ratio; –LR, negative likelihood ratio; NPV, negative predictive value; PPV, positive predictive value; 95% CI, 95% confidence interval; TTE, transthoracic echocardiography; ADRS, aortic dissection risk score.

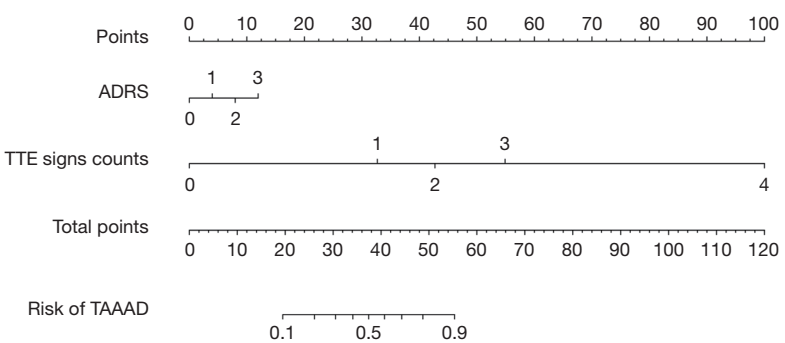

Figure 3 Nomogram of screening TAAAD in the STEMI group patients. ADRS, aortic dissection risk score; TAAAD, type A acute aortic dissection; STEMI, ST-segment elevated myocardial infarction.

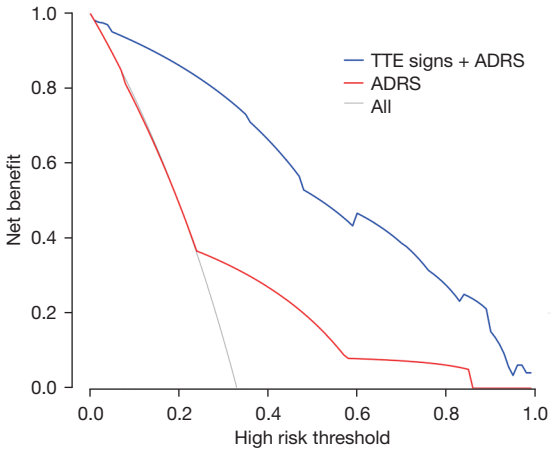

Figure 4 Decision curve analysis of TTE and ADRS for TAAAD probability. The net benefit curve is shown. The horizontal x-axis line indicates net benefit when all patients are considered as not having the TAAAD diagnosis; the light gray dashed line indicates net benefits when all patients are considered having the TAAAD diagnosis. The preferred model is the model with the highest net benefit at any given threshold. Model 1, integration of TTE with ADRS (blue line); Model 2, ADRS (red line). TTE, transthoracic echocardiography; ADRS, aortic dissection risk score; TAAAD, type A acute aortic dissection.

Discussion

As a rare complication of TAAAD, the occurrence of STEMI is of particular importance since the rapid reperfusion process, including either thrombolysis or primary percutaneous coronary intervention (PPCI), risks causing further extension or rupture of the aortic dissection. Moreover, relevant data has shown that ECG with ST-T changes on admission is associated with diagnosis and surgery delay (13-15). However, no existing studies had evaluated the methods and feasibility of screening TAAAD in STEMI patients. The results here demonstrated that TTE has additive value to differentiate between TAAAD and STEMI. Direct signs of TTE can quickly screen patients with low clinical probability of TAAAD for advanced imaging.

For life-threatening conditions including TAAAD and STEMI, it is critical to select rapid and safe imaging for etiologic screening or differentiation. Although CTA is the gold standard of imaging for the diagnosis of AAD and has accounted for 69% of initial diagnostic studies (2), timing is also a critical consideration in conjunction with the ability to screen for TAAAD in a specific STEMI population. Existing data from the International Registry of Acute Aortic Dissection (IRAD) demonstrate that CTA is associated with longer surgical delays (16), which may also apply to delayed reperfusion in the STEMI group. The utility of TTE for emergency physicians to diagnose acute cardiovascular diseases is well established. As a readily available imaging modality, bedside TTE can potentially identify direct and/or indirect signs that are valuable to diagnose TAAAD. Relevant guidelines indicate that TTE is a first choice of imaging modality for patients with suspected aortic dissection (10). Nevertheless, for patients with acute chest pain with ST-segment elevation on admission, emergency TTE at presentation is suggested only in patients with uncertain diagnoses, cardiogenic shock, cardiac arrest, or suspected mechanical complications. Routine TTE before CAG is not suggested by current guidelines to avoid reperfusion therapy delay (9). However, some TAAAD patients do not have specific clinical manifestations or hemodynamic instability. In such cases, especially with ECG displaying ST-segment elevation, the TAAAD diagnosis is delayed. Moreover, the urgency of reducing total ischemic time does not allow for time-consuming tests such as CTA, D-D, or myocardial injury biomarkers. Therefore, routine use of TTE to screen for TAAAD in the STEMI group should be further explored.

So far, varied performance of TTE with sensitivity (from 57% to 88%) and specificity (from 65% to 96%) for the TAAAD detection have been reported, indicating that specialized training largely affects the sensitivity of TTE (17). A recent predefined secondary analysis of the ADVISED study showed that the presence of direct TTE signs had a low sensitivity of 45.2% (95% CI: 37.0% to 53.6%) but high specificity of 97.4% (95% CI: 95.9% to 98.4%), while the presence of any signs had an increased sensitivity (89%, 95% CI: 82.8% to 93.6%) and decreased specificity (74.5%, 95% CI: 71.0% to 77.7%) for TAAAD (12). Similarly, the results of the present study demonstrated the presence of direct TTE signs with a sensitivity of 43.0% (95% CI: 35.0% to 52.0%) and specificity of 97.0% (95% CI: 95.0% to 99.0%), while any TTE signs had a sensitivity of 97.0% (95% CI: 93.0% to 99.0%) and specificity of 78.0% (95% CI: 73.0% to 82.0%) for TAAAD.

The present study was impacted by the limitations of TTE for thoracic aorta evaluation. Therefore, the diagnostic sensitivity of TTE cannot be a stand-alone test to conclusively differentiate TAAAD from STEMI. Nonetheless, the present study's key finding is that the

integration of TTE with ADRS demonstrated a reasonably efficient and exceptionally safe rule-out strategy for TAAAD. Based on the results, patients with ADRS ≤1 but without direct TTE signs had an extremely low probability of TAAAD. Among patients with ADRS ≤1, it is not advised for those with only indirect TTE signs to have further aorta imaging.

For STEMI patients, routine TTE before reperfusion therapy is not recommended because it prolongs total ischemic time (9). Nevertheless, endless shortening of door-to-balloon times does not result in the expected mortality reduction. Data of 96,738 STEMI patients in the CathPCI Registry demonstrated improvements in door-to-balloon times within 90 minutes. However, there was no significant overall change in either unadjusted or risk-adjusted in-hospital mortality, nor was there a significant difference observed in unadjusted 30-day mortality, which suggests that additional strategies are needed to reduce in-hospital mortality (18). Notably, the total ischemic time for STEMI consists of patient delay, emergency medical services (EMS) delay, and in-hospital delay. Therefore, we believe that it is a matter of system optimization as to whether routine TTE examination should be performed in STEMI patients before reperfusion therapy rather than only thinking that routine TTE will cause reperfusion delay. After all, within minutes, bedside TTE can indicate red flags that warrant urgent aortic imaging. Therefore, it is necessary to consider introducing TTE as a routine part of the STEMI process. Before that, the following issues, among others, should be considered: (I) strengthen TTE training, and require clinicians involved in whole management to be proficient in performing TTE, ensuring accurate ultrasound information available within minutes on patient's presentation; (II) the pre-hospital transport system should be routinely equipped with TTE, and TTE should be tested on the way to guide the patient's transfer; and (III) optimization of related departments layouts is required in the percutaneous coronary intervention (PCI) center. The catheter center should be close to the imaging department. Once TAAAD is ruled out by CTA/transesophageal echocardiography (TEE), patients with direct or indirect TTE signs initially could be immediately transferred to the receiving catheter procedure.

This was a retrospective study performed at two single centers, which may limit its generalizability. Further well-designed studies on new cohorts are essential for external validation. In the present study, TTE was routinely performed after a definite diagnosis. Therefore, physicians were not blinded to existing imaging results of coronary/aortic artery, which could have influenced the final TTE results. Well-designed prospective studies are needed to overcome such information bias.

Conclusions

This is the first study designed to investigate TTE screening of TAAAD in patients with STEMI. Our results demonstrate that TTE can be used as a rapid bedside test to screen for TAAAD in the STEMI group. However, the strategy of ruling out TAAAD by TTE alone, even in combination with ADRS, needs to be interpreted and applied with caution.

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Footnote

Reporting Checklist: The authors have completed the STARD reporting checklist. Available at https://cdt.amegroups.com/article/view/10.21037/cdt-22-59/rc

Data Sharing Statement: Available at https://cdt.amegroups.com/article/view/10.21037/cdt-22-59/dss

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://cdt.amegroups.com/article/view/10.21037/cdt-22-59/coif). 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 Jiao Tong University (No. [2018] 23). Informed consent was not required because this is a retrospective design study.

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