Int J Clin Exp Med 2017;10(10):14911-14918
www.ijcem.com /ISSN:1940-5901/IJCEM0048319

*Original Article*

# Risk factors of moderate-to-severe aortic regurgitation in patients with aortic dissection

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Received January 7, 2017; Accepted May 4, 2017; Epub October 15, 2017; Published October 30, 2017

**Abstract:** *Background:* Aortic regurgitation is an important clue for diagnosing aortic dissection (AD). This study is to investigate the risk factors for moderate-to-severe aortic regurgitation in patients with AD. *Methods:* We retrospectively reviewed and compared clinical data of 281 AD patients and 838 healthy subjects, and further compared the clinical manifestations of AD and transthoracic echocardiography findings between patients having acute AD and those having chronic AD. Univariate and multivariate binary logistic regression analyses were performed to identify the risk factors in all AD patients, in either acute AD or chronic AD patients, and related to moderate-to-severe aortic regurgitation. *Results:* A multivariate analysis showed that Stanford type A aortic dissection was a positive predictor of moderate-to-severe aortic regurgitation, while hypertension was a negative predictor. Compared with normal subjects, AD patients showed significant differences ($P < 0.05$) in the mean aortic root diameter, left ventricular end-diastolic diameter, left ventricular end-systolic diameter, interventricular septal end-diastolic dimension, posterior wall thickness, and ejection fraction. Additionally, patients with acute AD showed significantly lower values of interventricular septal end-diastolic dimension and posterior wall thickness when compared to patients with chronic AD ($P < 0.05$). *Conclusions:* Hypertension is a negative predictor, and Stanford type A AD is a positive predictor of moderate-to-severe aortic regurgitation in AD patients. Transthoracic echocardiography is valuable in discriminating moderate from severe aortic regurgitation and differentiating acute AD from chronic AD.

**Keywords:** Aortic regurgitation, aortic dissection, transthoracic echocardiography

## Introduction

Aortic dissection (AD), which is caused by tearing in the intima of aorta, is divided into type A (involving ascending aorta) and type B (not involving ascending aorta) based on the Stanford classification [1]. Based on the time of diagnosis, AD is also classified into acute (i.e. diagnosed within two weeks following symptom onset), and chronic (i.e. diagnosed longer than 2 weeks after symptom onset) [2]. Acute AD is a catastrophic acute disorder and is associated with high mortality, as evidenced by the findings that the one-week mortality rates for patients with acute AD ranged from 60% to 70% [3]. Hence, an earlier diagnosis of AD at emergency room is important to choose correct effective treatment strategy and thus reduce mortality. However, the clinical presentations of AD are highly diversified, and no single set of symptoms can be regarded as typical. For instance, the majority of patients reported a painful tearing sensation on the back, while others, and especially elderly patients, may be free of this symptom [4]. Therefore, the highly variable clinical symptoms make the early accurate diagnosis of acute AD challenging. Although the recognition of some specific clinical signs of this fatal disorder by clinical techniques such as chest X-ray, computed tomography (CT), and magnetic resonance imaging (MRI) may be helpful for decreasing the rate of misdiagnosis, these imaging techniques require an excessive amount of time to provide definitive results.

One of the most important physical examinations for possible AD is heart auscultation, as it

**Table 1.** Demographic data for participants enrolled in the present study

| Gender | Control subjects (n = 838) | Acute aortic dissection (n = 222) | Chronic aortic dissection (n = 59) | *P*-value |
|---|---|---|---|---|
| Male (n) | 576 (68.7%) | 170 (75.6%) | 45 (76.3%) | 0.757 |
| Female (n) | 262 (31.3%) | 52 (23.4%) | 14 (23.7%) | |
| Age (years) | 49.28 ± 10.77 | 49.98 ± 11.98 | 50.00 ± 12.83 | 0.845 |

**Table 2.** Demographics and medical histories of 281 patients with aortic dissection

| | Number | Stanford type A | Stanford type B |
|---|---|---|---|
| Male | 215 | 159 | 56 |
| Female | 66 | 53 | 13 |
| Age (years) | 281 | 50.1 ± 12.3 | 49.6 ± 11.5 |
| History of hypertension | 182 | 136 | 46 |
| Acute course of disease | 222 | 175 | 47 |
| Chronic course of disease | 59 | 37 | 22 |
| Important medical history | | | |
| Prior cardiac surgery | 10 | | |
| Aortic valve replacement | 5 | | |
| Marfan syndrome | 12 | | |
| Bicuspid aortic valve | 4 | | |
| Giant cell arteritis | 1 | | |
| Surgical trauma | 2 | | |
| Late pregnancy | 1 | | |
| Infective endocarditis | 1 | | |
| Renal transplantation | 1 | | |

allows a physician to detect a murmur (i.e. aortic regurgitation) resulting from valvular dysfunction caused by AD. Aortic regurgitation has been shown to be existent in 44% of patients with type A AD [5], and AD-related mortality increased as the severity of aortic regurgitation increased (i.e. from mild, moderate, moderate-to-severe) [6, 7], suggesting that aortic regurgitation as an important indicator of AD severity. Aortic regurgitation is usually first detected during auscultation, and then confirmed by transthoracic echocardiography (TTE). However, the risk factors for moderate-to-severe aortic regurgitation in AD patients remain unclear.

In this study, we retrospectively reviewed clinical data obtained from AD patients and healthy subjects, and compared the clinical manifestations of AD and the TTE findings of acute AD patients with those of chronic AD patients. We aimed to provide clinically valuable predictor(s) for moderate-to-severe aortic regurgitation in AD patients.

## Materials and methods

### *Participants*

The protocol of our study was approved by the Ethics Committee of local Hospital. Clinical data obtained from 361 AD patients between January 2003 and June 2008 were retrospectively reviewed. AD was diagnosed by CT or MRI, or confirmed during surgery. Eight of the 361 patients were excluded from our study due to the lack of TTE results. A group of 838 healthy individuals who had undergone TTE as part of a routine medical checkup between January and May 2008 served as a control cohort. Individuals with liver/kidney dysfunction, chronic pulmonary disease, valvular heart disease, congenital heart disease, or a history of heart or large artery surgery were excluded.

### *Study protocol*

We reviewed the baseline data, demographic characteristics (age and sex), medical history, and clinical courses of all participants, and also recorded the type of AD (acute or chronic dissection; Stanford type A or B dissection). Acute and chronic AD were defined as AD with symptoms lasting ≤ 14 days and > 14 days, respectively, as previously defined [8]. Based on the Stanford classification, Stanford type A dissection involves the ascending aorta with or without extension to the descending aorta, and Stanford type B dissection does not involve the ascending aorta [9]. In addition, we also reviewed findings obtained by TTE and heart auscultation (i.e. the presence or absence of regurgitation murmurs). Hypertension was defined as the blood pressure ≥ 140/90 mmHg or the use of any antihypertensive agents. Marfan syndrome was diagnosed using the Ghent criteria.

### *Transthoracic echocardiography*

A total of 281 AD patients and 838 healthy participants had undergone TTE (Philips IE33 Ultrasound System; Philips Healthcare, Netherlands). The vast majority of parameters had been collected from the parasternal left ventricular long axis view of a 2D examination, and

**Table 3.** Distribution of moderate-to-severe aortic regurgitation among aortic dissection patients

| | MAR | N MAR | Total | Percentage | *P*-value |
|---|---|---|---|---|---|
| Sex | | | | | NS |
| Male | 33 | 182 | 215 | 15.3% | |
| Female | 16 | 50 | 66 | 24.2% | |
| Age (yrs) | | | | | NS |
| < 30 | 4 | 14 | 16 | 20.0% | |
| 30-40 | 9 | 21 | 30 | 35.5% | |
| 40-50 | 19 | 68 | 87 | 31.0% | |
| 50-60 | 13 | 77 | 90 | 32.0% | |
| 60-70 | 4 | 38 | 42 | 14.9% | |
| > 70 | 2 | 14 | 16 | 5.7% | |
| Hypertension | | | | | S |
| Present | 22 | 160 | 182 | 12.1% | |
| Absent | 27 | 72 | 99 | 27.3% | |
| Stanford type | | | | | S |
| A | 48 | 164 | 212 | 22.6% | |
| B | 1 | 68 | 69 | 1.5% | |
| Course | | | | | NS |
| Acute | 43 | 179 | 222 | 19.4% | |
| Chronic | 6 | 53 | 59 | 10.2% | |
| Pain | | | | | NS |
| Present | 43 | 204 | 247 | 17.4% | |
| Absent | 6 | 28 | 34 | 17.6% | |

MAR: moderate-to-severe aortic regurgitation; NS: no significant difference ($P > 0.05$); NMAR: the patients who suffered aortic dissection has no moderate-to-severe aortic regurgitation; S: statistically significant difference ($P < 0.05$).

data from other views including parasternal left ventricular short axis views, apical 2 and 4 chamber views, and suprasternal notch aortic long and short axis views (AD was mainly found in this view) were also available. All TTE procedures were performed by a specialist with at least 3-year-experience in using the echocardiography system.

*Aortic regurgitation*

The severity of aortic regurgitation was classified according to 2014 AHA/ACC guidelines established for evaluating valvular heart disease [10]. The following parameters were evaluated for diagnosis of moderate-to-severe aortic regurgitation by TTE: (1) Structural parameters: left atrium size, (normal or dilated); aortic leaflets, (normal or abnormal). (2) Doppler parameters: jet width of the left ventricular outflow tract [(LVOT)-color flow, intermediate]; jet density on continuous wave (CW) Doppler, dense; jet deceleration rate on CW Doppler [(pressure half-time), medium, 500-200 ms]; diastolic flow reversal on descending aorta pulsed wave (PW) Doppler, intermediate. (3) Quantitative parameters: VC [(vena contracta)-width (cm), 0.3-0.60]; jet width/LVOT width (cm), 25-45/46-64; jet cross-sectional area (CSA)/LVOT CSA (mL/beat), 5-20/21-59; regurgitant volume (%), 30-44/45-59; regurgitant fraction (%), 30-39/40-49; effective regurgitant orifice area ($cm^2$), 0.1-0.19/0.20-0.29.

*Data collection*

All demographic and clinical data of individual participant, including age, sex, medical history of hypertension, Marfan syndrome status, history of cardiac surgery and/or aortic valve replacement, clinical presentations, and TTE findings, were recorded on a single form.

*Statistical analysis*

All data were analyzed using IBM SPSS Statistics for Windows (Version 21.0. Armonk, NY: IBM Corp). Values for quantitative variables and categorical variables are presented as the mean ± standard deviation (SD) and percentages, respectively. Comparisons between results in the patient and control groups were performed using the $\chi^2$ test or Student's t-test. Univariate and multivariate binary logistic regression analyses were performed to identify the risk factors for moderate-to-severe aortic regurgitation. *P*-values < 0.05 were considered statistically significant.

## Results

*Demographic and basal clinic characteristics of participants*

This study included 222 acute AD patients (170 men, 52 women; mean age 49.98 ± 11.89 years), 59 chronic AD patients (45 men, 14 women; mean age 50.00 ± 12.83 years), and 838 healthy control subjects (576 men, 262 women; mean age 49.28 ± 10.77 years). The demographic characteristics of control and AD patients (acute and chronic AD) are presented in **Table 1**, and the baseline clinical characteristics of the 281 AD patients are summarized in **Table 2**. 75.4% patients (n = 212) had Stanford type A, and 64.8% (n = 82) had a medical history of hypertension. Among the AD patients with a history of hypertension, 136 had Stanford type A and 46 had Stanford type B. A

**Table 4.** Univariate analysis of risk factors related to aortic regurgitation with aortic dissection

| | BiLA | B | S.E. | Wald | OR | 95% CI | *P*-value |
|---|---|---|---|---|---|---|---|
| Sex | U | -0.568 | 0.344 | 2.728 | 0.567 | 0.289-1.112 | 0.099 |
| Age | U | -0.029 | 0.013 | 4.952 | 0.971 | 0.947-0.997 | 0.026* |
| Hypertension | U | -1.003 | 0.32 | 9.808 | 0.367 | 0.196-0.687 | 0.002* |
| Stanford type | U | 2.991 | 1.021 | 8.588 | 19.902 | 2.693-147.112 | 0.003* |
| Course | U | 0.752 | 0.463 | 2.64 | 2.122 | 0.856-5.258 | 0.104 |
| Pain | U | -0.016 | 0.480 | 0.001 | 0.984 | 0.384-2.521 | 0.973 |

Sex: male or female; Hypertension: present or absent; Stanford type: A or B; Course: acute or chronic; Pain: present or absent; BiLA: Binary Logistic Analysis; U: Univariate analysis; M: Multivariate analysis. *: $P < 0.05$.

**Table 5.** Multivariate analysis of risk factors related to aortic regurgitation with aortic dissection

| | BiLA | B | S.E. | Wald | OR | 95% CI | *P*-value |
|---|---|---|---|---|---|---|---|
| Sex | M | -0.656 | 0.375 | 3.058 | 0.519 | 0.249-1.082 | 0.080 |
| Age | M | -0.022 | 0.015 | 2.075 | 0.979 | 0.95-1.008 | 0.150 |
| Hypertension | M | -0.885 | 0.365 | 5.867 | 0.413 | 0.202-0.835 | 0.015* |
| Stanford type | M | 2.955 | 1.028 | 8.267 | 19.195 | 2.562-143.836 | 0.004* |
| Course | M | 0.71 | 0.500 | 2.017 | 2.033 | 0.764-5.415 | 0.156 |
| Pain | M | -0.163 | 0.527 | 0.095 | 0.85 | 0.302-2.389 | 0.758 |

Sex: male or female; Hypertension: present or absent; Stanford type: A or B; Course: acute or chronic; Pain: present or absent; BiLA: Binary Logistic Analysis; U: Univariate analysis; M: Multivariate analysis. *: $P < 0.05$.

small number of patients with AD had undergone the following surgeries: prior cardiac surgery (n = 10), aortic valve replacement (n = 5), bicuspid aortic valve replacement (n = 4), and renal transplantation (n = 1). In addition, 12 patients had Marfan syndrome, 1 had giant cell arteritis, and 1 had infective endocarditis.

*Risk factors of moderate to severe aortic regurgitation in patients with AD*

The distribution of moderate-to-severe aortic regurgitation among the 281 AD patients is shown in **Table 3**. A total of 49 patients (17.4%; 33 males and 16 females) were found to have moderate-to-severe aortic regurgitation as confirmed by TTE. Among these 49 patients, 22 had hypertension, 48 were diagnosed as Stanford type A dissection, and only 1 patient was Stanford type B dissection. As shown in **Tables 4**, **5**, a univariate analysis revealed that both hypertension and a patient's Stanford AD classification were significant risk factors for moderate-to-severe aortic regurgitation in AD patients ($P < 0.05$). Moreover, Stanford type A classification (OR, 19.195; 95% confidence interval (CI), 2.562 to 143.836; $P = 0.004$) was a significant positive predictor, while a diagnosis of hypertension (OR, 0.413; 95% CI, 0.202 to 0.835; $P = 0.015$) was a significant negative predictor. We also found that AD patients had significantly higher mean values of aortic root diameter, left ventricular end-diastolic diameter, left ventricular end-systolic diameter, interventricular septal end-diastolic dimension, posterior wall thickness, and ejection fraction (all *P*-values < 0.05) compared with controls (**Table 6**). Additionally, compared with patients with chronic AD, patients with acute AD had lower interventricular septal end-diastolic dimension (10.6 ± 1.9 vs. 11.4 ± 2.1, respectively; $P = 0.011$) and posterior wall thickness (10.5 ± 1.7 vs. 11.0 ± 1.9, respectively; $P = 0.047$).

## Discussion

The major findings of our study included that 1) AD patients had significantly higher values of main cardiac structural and functional parameters as measured by TTE than control participants, 2) Stanford type A dissection served as a positive predictor for moderate-to-severe aortic regurgitation, 3) Hypertension served as a negative predictor for moderate-to-severe aortic regurgitation, and 4) Acute AD patients exhibited significantly lower interventricular septal end-diastolic dimension and posterior wall thickness than chronic AD patients.

In the present study, our univariate and multivariate analyses identified Stanford type A dissection as an independent positive predictor for the onset of moderate-to-severe aortic regurgitation in patients with AD, which was consistent with previous findings [11-14]. As patients with type A AD usually present with more severe, sudden, and extensive complications, their onset of valvular dysfunction occurs earlier, and is often accompanied by moderate-to-severe aortic regurgitation. For instance,

**Table 6.** Comparison of echocardiographic parameters among control subjects, acute and chronic AD patients

| | | N | x ± SD | 95% CI | *P* |
|---|---|---|---|---|---|
| AoRD | Control | 838 | 32.2 ± 3.1 | 8.31~10.40 | [A]0.000* |
| | Acute | 59 | 41.5 ± 10.2 | -2.15~2.56 | [B]0.862 |
| | Chronic | 222 | 41.3 ± 7.6 | 8.49~9.80 | [C]0.000* |
| LAD | Control | 838 | 34.3 ± 3.5 | -0.7~1.26 | [A]0.570 |
| | Acute | 59 | 34.6 ± 5.9 | -1.55~2.41 | [B]0.672 |
| | Chronic | 222 | 34.2 ± 7.1 | -0.81~0.52 | [C]0.672 |
| LVDd | Control | 838 | 45.7 ± 4.15 | 5.34~7.83 | [A]0.000* |
| | Acute | 59 | 52.3 ± 9.6 | -0.26~4.24 | [B]0.082 |
| | Chronic | 222 | 50.3 ± 7.2 | 3.86~5.33 | [C]0.000* |
| LVDs | Control | 838 | 28.2 ± 3.2 | 4.11~6.22 | [A]0.000* |
| | Acute | 59 | 33.3 ± 9.8 | -0.06~3.89 | [B]0.058 |
| | Chronic | 222 | 31.4 ± 5.8 | 2.68~3.83 | [C]0.000* |
| IVSd | Control | 838 | 9.1 ± 1.2 | 1.19~1.84 | [A]0.000* |
| | Acute | 59 | 10.6 ± 1.9 | -1.37~1.18 | [B]0.011* |
| | Chronic | 222 | 11.4 ± 2.1 | 2.09~2.50 | [C]0.000* |
| PWT | Control | 838 | 8.9 ± 1.0 | 1.28~1.84 | [A]0.000* |
| | Acute | 59 | 10.5 ± 1.7 | -1.09~-0.01 | [B]0.047* |
| | Chronic | 222 | 11.0 ± 1.9 | 1.92~2.29 | [C]0.000* |
| EF | Control | 838 | 68.2 ± 4.6 | -3.97~-1.26 | [A]0.001* |
| | Acute | 59 | 65.63 ± 9.931 | -3.55~0.96 | [B]0.259 |
| | Chronic | 222 | 66.92 ± 7.169 | -2.09~0.54 | [C]0.001* |

A: control vs. acute; B: acute vs. chronic; C: control vs. chronic. *: $P < 0.05$. AoRD: aortic root diameter; LAD: left atrium diameter; LVDd: left ventricular end-diastolic diameter; LVDs: left ventricular end-systolic diameter; IVSd: interventricular septal end-diastolic dimension; PWT: posterior wall thickness; EF: left ventricular ejection fraction.

Uçar et al. [15] reported a case of a 45-year-old man who was diagnosed as having acute AD (Debakey type I, Stanford type A dissection). In this case, a TEE examination confirmed prolapse of an aortic intimal flap, and revealed severe aortic regurgitation. Armstrong et al. [16] also reported a study of 45 patients with type A dissection, and found that all of those patients exhibited an intimal flap revealed by TEE. Similarly, Wernly et al. [17] reported a 60% incidence of aortic regurgitation in patients with a type A AD. These findings support the premise that moderate-to-severe aortic regurgitation is a common clinical symptom in patients with type A AD. Mechanistically, the occurrence of aortic regurgitation associated with acute AD may be attributed to: (1) the progressive aortic root dilatation leading to eventual leaflet mal-coaptation; (2) the leaflet prolapse resulting from distorted aortic root geometry; (3) the leaflet detachment by extension of a dissection through the aortic root; and (4) a prolapsing flap which impedes aortic valve closure and thus severs as a conduit for regurgitant flow [13, 18].

Our present study also identified hypertension as a negative predictor for the occurrence of moderate-to-severe aortic regurgitation in AD patients. Previously, hypertension was shown as one of the most important risk factors for AD [6]. More recently, Landenhed et al. [19] performed a prospective cohort study with 30,412 individuals to identify risk factors for AD and severe aneurysmal disease in the thoracic and abdominal aorta. They showed that hypertension was present in 86% of individuals who subsequently developed AD, and that hypertension conferred a 54% population-attributable risk for AD [19], indicating that hypertension was strongly associated with incidence of AD. Also, a previous study of 1812 acute AD patients conducted in China revealed that most acute AD patients had hypertension, and presented with abrupt onset chest and/or back pain [20]. That study also identified hypertension as a predictive factor for increased in-hospital mortality among acute AD patients [20]. Tsai et al. [21] published a review of the International Registry of Acute Aortic Dissection (IRAD) study which enrolled > 2000 patients during a 12-year period, and found that systemic hypertension was the most common risk factor for AD. In our study, although we also uncovered hypertension as a positive predictor of AD in the present study, we noticed that moderate-to-severe aortic regurgitation occurred almost exclusively in patients without a medical history of hypertension, which is inconsistent with the above-mentioned findings. One potential reason accounting for this discrepancy is that the incidence of hypertension increased with advancing of age, while moderate-to-severe aortic regurgitation was predominantly found in patients aged 40-60 years in our study. For instance, Jeong et al. [22] reported that the overall prevalence of hypertension in China is 39%, and this prevalence increased to 59.4% among individuals aged ≥ 60 years, and to 72.8% among those aged ≥ 75 years. These data suggested a lower incidence of hypertension among patients aged

< 60 years compared to those aged ≥ 60 years, which may explained why in our study, moderate-to-severe aortic regurgitation was predominantly found in patients without hypertension. Additional studies are needed to further corroborate this observation.

Improved imaging techniques such as CT and MRI are now widely used in the emergency room for diagnosing AD. Additionally, TTE, which is non-invasive, requires minimal time to perform, does not expose a patient to X-ray, is also readily available. Baliga et al. [9] reported the usefulness of TTE for identifying aortic valve dysfunction, pericardial tamponade, and abnormalities of cardiac wall motion. In patients with good echocardiographic windows, TTE can also be used to screen the proximal 4 to 8 mm of ascending aorta to just above the sinotubular junction, and a short segment of the descending aorta in patients with shock. In addition, TTE can also be used to visualize an intimal flap or thickened aortic wall, and the proximal ascending aorta in long- and short-axis parasternal views. In the current study, we chose TTE to detect aortic regurgitation and assess its severity. Our results showed that the aortic root diameter, left ventricular end-diastolic diameter, left ventricular end-systolic diameter, interventricular septal end-diastolic dimension, posterior wall thickness, and ejection fraction were significantly higher/larger in AD patients than in healthy participants, which is consistent with the results of previous studies. For instance, Polain et al. [23] reviewed the TTE data of 186 consecutive patients who had undergone valve repair surgery during a 10-year period to correct damage caused by aortic regurgitation, and identified the diameter of the aortic annulus (similar to AoRD) as a significant positive predictor of 3+ recurrent aortic regurgitation. However, another study conducted by Nienaber et al. [24] found that MRI and TEE had higher sensitivities for diagnosing type A AD than those of CT and TTE. Moreover, MRI and CT were the most specific methods for excluding a dissection involving the ascending aorta, with higher specificities compared with TEE and TTE. Therefore, that study concluded that TTE was useful for detecting aortic regurgitation and pericardial effusion but was not useful for assessing the descending aorta. Indeed, data obtained from conventional TTE alone were not always sufficiently precise to permit a diagnosis of AD, however, they were proved to be highly valuable when used as an adjunct to analyses of spin-echo MRI sequences [7]. In the present study, we found significantly smaller interventricular septal end-diastolic dimension and posterior wall thickness in the acute AD group than in the chronic AD group. Previously, positron emission tomography/computed tomography (PET/CT) and levels of several biomarkers in peripheral blood including P-selectin, thrombin activation, and fibrinolysis were used to diagnose and assess the prognosis of chronic AD patients. Based on our findings, we believe that two parameters revealed by TTE, interventricular septal end-diastolic dimension and posterior wall thickness can, may have a potential value in distinguishing acute AD patients from chronic AD patients.

This retrospective study had several limitations. First, it was performed at a single medical center, and thus its results may have been affected by the patient selection bias. Second, the study enrolled a small number of patients, and may have been underpowered for identifying additional predictive factors. Finally, the TTE examinations were performed by at least three different specialists; thus creating the possibility of differences in the examination procedures.

## Conclusion

In the present investigation, we demonstrate that hypertension is a negative predictor and Stanford type A aortic dissection is a positive predictor for the onset of moderate-to-severe aortic regurgitation in AD patients. Moreover, patients with acute AD have lower interventricular septal end-diastolic dimension and posterior wall thickness compared with patients with chronic AD, suggesting that TTE is valuable in differentiating between acute AD and chronic AD. Given the limitations of our study, our findings need to be further corroborated in a large cohort study in the future.

## Acknowledgements

This work was supported by the Key Program of Health and Family Planning Commission of Shanghai, China (201440024).

The content of this report does not necessarily reflect the position or policy of the Chinese

Government. There is no any ethical or legal conflict involved in the article.

### Disclosure of conflict of interest

None.

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