Original Article

Relationship between valvular structure and biochemical indices of non-valvular atrial fibrillation and senile degenerative valvular heart disease

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Background: Valvular heart disease (VHD) is a common clinical condition in geriatric-related cardiovascular diseases that is connected to heart dysfunction. Atrial fibrillation (AF) is the most frequent arrhythmia. Considering these two common clinical conditions, so far no sufficient data on the relationship between degenerative VHD and non-valvular atrial fibrillation (NVAF). We aimed to explore the relationship between valvular structure and biochemistry of nonvalvular AF and degenerative valvular heart disease in the elderly.

Methods: In our study, 234 VHD patients who were diagnosis evaluated by transthoracic echocardiography were enrolled in this retrospective study from January 2015 and December 2018. Significant valvular diseases were defined according to ACC/AHA Classification as any moderate or severe mitral regurgitation (MR), aortic regurgitation (AR), tricuspid stenosis, regurgitation, or aortic stenosis (AS). Data on relevant laboratory indicators were also collected.

Results: A total of 234 patients with degenerative VHD were enrolled, of whom 81 had NVAF and 153 had sinus rhythm. Gender, smoking history, and some comorbidities, such as coronary artery disease, diabetes, and renal dysfunction, did not differ significantly between the two groups, but there were significant differences in age and hypertension {79 [74–83] *vs.* 70 [65–79] years} After propensity-score matching (PSM), we identified 68 VHD patients with NVAF and 68 VHD patients without NVAF. The NVAF + VHD had higher low-density lipoprotein (LDL) cholesterol (2.94±0.84 *vs.* 2.26±1.33 mmol/L, P=0.001), lower high-density lipoprotein (HDL) cholesterol [1.03 (0.89–1.34) *vs.* 1.56 (0.99–2.71) mmol/L, P<0.001], and higher uric acid (UA) (438.18±145.83 *vs.* 376.67±148.03 μmol/L, P=0.02) than the VHD group. The ejection fraction (EF) of the NVAF + VHD group was lower than that of the VHD group {63 [51–68] *vs.* 66 [62–69], P=0.013}. In addition, the left atrial size, MR, and calcification of the NVAF + VHD group were higher than those of the VHD group.

Conclusions: Pronounced MR, valve calcification and hyperlipidemia were more likely in VHD patients with NVAF. These structures and biomarkers changes maybe important clinical parameters for disease prevention and management, which indicate early drug intervention to AF and hyperlipidemia is necessary.

Keywords: Valvular heart disease (VHD); non-valvular atrial fibrillation (NVAF); mitral regurgitation (MR)

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Introduction

Valvular heart disease (VHD) is a significant cause of mortality and morbidity in elderly patients and is a common heart disorder. The decrease in rheumatic heart disease has been accompanied by an increase in degenerative valve disease (1). Due to these epidemiological changes, the clinical presentations and management of VHD have also changed. The latest study estimated that the prevalence of VHD was around 2.5% in the United States, and noted that this figure increases greatly after the age of 65 years because of degenerative etiologies (2). The Euro Heart Survey findings also revealed the etiologies of various types of VHD. Notably, the degenerative disease represents 63% of all cases of native heart valve disease, patients with aortic and mitral valve diseases have a mean age of >50 years, and rheumatic heart disease represents 22% of all cases (3,4). The prevalence of VHD increases substantially with age (1), and the term 'degenerative' is commonly used to define the most frequent etiology of senile valvular disease.

Unlike hypertension or coronary artery disease, some difficulties exist in the management of VHD, as these kinds of conditions cannot be diagnosed reliably based only on clinical information, and the symptoms are usually only evident at an advanced stage (5). Degenerative valvular diseases are often related to aging, and as such, patients often meet with indifference in the decision making process for intervention management. Following adequate clinical evaluation, echocardiography can be used to diagnose VHD and is a key technique in assessing the severity and prognosis of VHD (6). A hospital-based survey of 139,496 patients with severe VHD in China revealed that mitral regurgitation (MR) was the most frequent in patients (n=946, 0.68%) followed by mitral stenosis (MS) (n=524, 0.38%), aortic stenosis (AS) (n=392, 0.28%), and aortic regurgitation (AR) (n=371, 0.27%) (6). Notably, degenerative valvular disease remains the primary cause of severe MR. Degenerative valvular heart disease mainly focuses on the mitral valve and aortic valve in patients aged older ones, and the prevalence rates for severe MR and AS increase sharply with age (7).

Li *et al.* (8) enrolled and surveyed 134,874 Chinese subjects on the prevalence and causes of MR, and found that 42.44%, 1.63%, and 1.44% of the patients had mild MR (+), moderate MR (2+), and severe MR (3+/4+), respectively. The detection rate of patients with mitral regurgitation (MR) was much higher, as the enrolled patients were from a heart center (rather than communities); however, the results still show that severe MR is common and a great number of individuals may require transcatheter mitral valve treatment in China, Thus, prevention and early intervention measures are very important for the management of VHD.

Atrial fibrillation (AF) is the most frequent arrhythmia. The incidence of AF with its associated morbidities continues to rise worldwide, creating a public health burden. Under the 2014 AHA/ACC/HRS guidelines for the management of patients with AF, non-valvular atrial fibrillation (NVAF) is defined as AF in the absence of rheumatic MS, or a mechanical valvular AF heart valve, and explicitly added no bioprosthetic heart valves or no mitral valve repair (9). In patients with severe MR, degenerative causes or organic valvular abnormalities are associated with the incidence of AF, and even asymptomatic organic MR has been shown to increase the risk of AF and adverse cardiovascular outcomes (10,11). Theoretically, it seems plausible that moderate MR may be a risk factor for the development of lone AF, as it may lead to the mechanical stretching of the left atrium, or the AF itself may exert some anatomical effect on the mitral annulus that worsens the progression and severity of MR over time (12).

In patients with valvular heart disease, hemodynamic changes due to valvular disease lead to atrial and ventricular remodeling. At present, it is considered that all kinds of heart valves can be changed to cause AF. For rheumatic heart valve disease, the main mechanism may be that it not only causes atrial enlargement and annulus dilatation after hemodynamic changes, but also causes atrial myocardial fibrosis, ultrastructural abnormalities of atrial myocytes,

Highlight box

Key findings

- VAD patients with NVAF had more pronounced MR and valvular calcification and were more likely to have hyperlipidemia than those without NVAF.

What is known and what is new?

- VHD patients with NVAF who were referred for echocardiography had a higher prevalence of echo evident moderate MR than VHD patients in sinus rhythm.
- Our study sought to evaluate the effect of NVAF on the structure and biochemical parameters of patients with degenerative VHD.

What is the implication, and what should change now?

- This study provides information on the association between the prevalence of severe VHD and NVAF. In the future, further prospective research should be conducted with larger sample sizes and at multi-centers.

significantly increased gap junction of myocardial cells, atrial electrical remodeling, etc., thus leading to the occurrence of AF (13). The pathophysiological process of AF involves atrial remodeling, the role of renin-angiotensin-aldosterone system, inflammatory factors and oxidative stress, and the role of the autonomic nervous system (14). Among them, the role of renin-angiotensin-aldosterone system, inflammatory factors and oxidative stress, the role of the autonomic nervous system and renal insufficiency are also involved in the pathophysiological mechanism of degenerative heart valves in the elderly (15).

There is no sufficient data on the relationship between degenerative VHD and NVAF. We found that VHD patients with NVAF who underwent echocardiography had a higher prevalence of echo evident moderate MR than VHD patients in sinus rhythm. Thus, our study sought to understand the relationship between valvular structure and biochemical indices of non-valvular AF and senile degenerative valvular heart disease. Specifically, it sought to determine the exact clinical manifestations of cardiac structure changes by echocardiology in NVAF patients with degenerative VHD compared to patients in normal sinus rhythm. To understand the relationship between AF and degenerative valvular heart disease, so as to find possible structures or biomarkers, and provide reference data for further exploration of the effect of AF on degenerative valvular heart disease in the elderly. In addition, it also provides a research basis for the prevention and early intervention of VHD. We present the following article in accordance with the STROBE reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-61/rc).

Methods

Patients

This retrospective analysis used data from the database of The First Affiliated Hospital of Shantou University. It was designed to include all degenerative VHD patients between January 2015 and December 2018. Patients who had undergone transthoracic echocardiography for diagnosis confirmation were included in the study, but those with poor echocardiograms were excluded. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by Ethics Committee of The First Affiliated Hospital of Shantou University (No. 2020-138) and informed consent was taken from all the patients.

Echocardiography

All the patients underwent echocardiography in the ultrasound laboratory. After the patient had rested for at least 10 min, the echo measurements were taken in supine position. Transthoracic echocardiography was performed with the SIEMENS SC 2000 and GE VIVID7. The parasternal long-axis, apical 4 chamber, 2 chamber, and long-axis projections were obtained. Left atrial diameter (LAD) and left ventricular end-diastolic and end-systolic diameters were measured. Hemodynamic parameters were obtained by pulsed-wave and continuous-wave Doppler ultrasound.

According to the ACC/AHA Classification (16), the severity of the valve disease was classified as mid, moderate, or severe. Significant valvular diseases were defined as follows: any mitral or AS severity, moderate or severe valve regurgitation, including MR and AR, and moderate or severe tricuspid stenosis or regurgitation.

Risk factors and laboratory data

Blood was collected in the morning at fasting hours to determine white blood cell (WBC), hemoglobin, total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, total albumin, albumin, and fasting blood glucose levels. The plasma levels of indicators such as total cholesterol, triglycerides, HDL cholesterol, creatinine, and glucose were measured in clinical laboratories using a standard automated analyzer. Risk factors such as coronary artery diseases, diabetes, and renal dysfunction were studied.

Statistical analysis

The normally distributed continuous variables are presented as the mean ± standard deviation, and the non-normally distributed continuous variables are presented as the median and interquartile range. The baseline characteristics were compared among the groups using an analysis of variance or Wilcoxon rank-sum test as appropriate.

A logistic regression model was used for the different covariate adjusted models. To generate propensity scores, we used a logistic regression model using a 1:1 nearest-neighbor matching algorithm with no replacement and a caliper width of 0.2 of the standard deviation of the logit of

Table 1 Baseline characteristics of VHD patients with and without NVAF

Variables	NVAF + VHD (n=81)	VHD (n=153)	P value
Age, years	79 [74–83]	70 [65–79]	<0.001*
Sex: male	46 (56.8)	94 (61.4)	0.317
History of smoking	32 (39.5)	73 (47.7)	0.0.63
History of hypertension	57 (70.4)	65 (42.4)	<0.001*
History of diabetes mellitus	21 (25.6)	28 (18.3)	0.173
History of CAD	22 (27.2)	33 (21.6)	0.337
History of renal dysfunction	14 (17.3)	15 (9.8)	0.099

Data are presented as median [range] or n (%). *, there were significant differences between the VHD patients with NVAF and patients with VHD. VHD, valvular heart disease; NVAF, non-valvular atrial fibrillation; CAD, coronary atherosclerotic heart disease.

the propensity score. The covariates were age, sex, history of smoking, history of hypertension, history of diabetes mellitus, history of coronary heart disease (CAD), and history of renal dysfunction.

All the tests were 2-sided, and a P value <0.05 was considered significant. SPSS for Windows version 20.0 (SPSS Inc., Chicago, IL, USA) was used for the statistical analyses.

Results

Patient characteristics

A total of 234 patients with degenerative VHD were enrolled in this, of whom 81 had NVAF and 153 had normal sinus rhythm. The patients had a median age of 74 [67–80] years. *Table 1* shows the clinical features of the VHD patients with and without NVAF, including the comorbidities and risk factors, such as smoking history, coronary artery disease, hypertension, diabetes mellitus, and renal insufficiency. The Kolmogorov-Smirnov test was used to examine any imbalances in the variables of age. As the VHD patients with NVAF were significantly older than patients with VHD in age, Mann-Whitney U was used to test the statistical difference between the two groups.

As *Table 1* shows, gender, smoking history, and some comorbidities, such as coronary artery disease, diabetes, and renal dysfunction, did not differ significantly between the 2 groups, and the VHD patients with NVAF were more likely to be older in age and have hypertensive conditions than patients with VHD. The median age was 79 [74–83] years and 70.4% hypertensive patients with NVAF in the VHD NVAF patient group. In addition, *Table 2* shows the differences between the two groups in valve structure and biochemical parameters.

Risk factors and laboratory measurements

Propensity-score matching (PSM) 1:1 was performed using a calipers value M=0.02 based on the above 7 risk factors in *Table 1*. After PSM, there were 68 patients in each group. *Table 3* shows the WBC count measurement results, the renal, liver and thyroid function evaluation results, and the serum electrolytes and blood lipids measurement results. Notably, there were significant differences in the lipid levels and uric acid (UA) between the groups, and VHD patients with NVAF had higher LDL cholesterol (2.86±0.12 *vs.* 2.36±0.17 mmol/L, P=0.014), lower HDL cholesterol [1.10 (0.90–1.32) *vs.*1.33 (1.08–2.67) mmol/L, P<0.001], and higher UA [447 (336–534) *vs.* 372 (309–452) μmol/L, P=0.009] than the VHD patients without NVAD.

Echocardiography manifestations of VHD

Table 4 shows the comparison results of the echocardiogram structure and function between the 2 groups after the 1:1 PSM analysis based on the above 7 factors. There was no significant difference in the left ventricular diastolic diameter (LVEDD) between the 2 groups after PSM (P>0.05), but there were significant differences in the LAD, left ventricular ejection fraction (LVEF%), MR classification, and calcification classification between the 2 groups. The ejection fraction (EF) of the NVAF + VHD group was lower than that of the VHD group [63 (51–68) *vs.* 66 (62–69), P=0.013]. In addition, the left atrial size, MR, and calcification of the NVAF + VHD group were

Table 2 Baseline characteristics of VHD patients with and without NVAF about valve structure and biochemical parameters

Variables	NVAF + VHD (n=81)	VHD (n=153)	P value
WBC, 10E+9/L	7.60 [5.76–9.95]	7.15 [5.86–8.70]	0.195
Hgb, g/L	126 [114–138]	126 [113–138]	0.920
TP, g/L	67.20±6.10	68.39±7.17	0.132
ALB, g/L	34.63±3.91	36.14±4.93	0.003*
UA, μmol/L	435 [334–534]	338 [267–426]	<0.001*
Blood calcium, mmol/L	2.18 [2.10–2.27]	2.23 [2.13–2.29]	0.063
Glucose, mmol/L	5.75 [4.91–6.9]	5.33 [4.68–6.19]	0.073
TC, mmol/L	4.45±1.23	4.84±1.30	0.067
TG, mmol/L	0.98 [0.76–1.28]	1.02 [0.77–1.37]	0.547
HDL, mmol/L	1.10 [0.90–1.32]	1.24 [0.99–1.67]	0.001*
LDL, mmol/L	2.83±0.94	2.76±1.23	0.694
LAD (mm)	41 [37–45]	30 [27–35]	<0.001*
LVEDD (mm)	45 [40–52]	44 [41–48]	0.183
LVEF (%)	63 [53–68]	67 [63–70]	<0.001*

Data are presented as median [range], n (%) or mean ± SD. *, there were significant differences between the VHD patients with NVAF and patients with VHD. VHD, valvular heart disease; NVAF, non-valvular atrial fibrillation; WBC, white blood cell; Hgb, hemoglobin; TP, total protein; ALB, albumin; UA, uric acid; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LAD, left atrial diameter; LVEDD, left ventricular diastolic diameter; LVEF, left ventricular ejection fraction.

higher than those of the VHD group.

Discussion

Along with the aging Chinese population, the etiology of degenerative, age-related, VHD is an important health issue. However, epidemiology data on the prevalence of VHD in older Chinese patients are lacking. Clinically, senile degenerative VHD and AF often occur at the same time, which are mutually causal. Additionally, due to the aging of the population in China, there are more and more elderly patients with AF and degenerative VHD, and the number of their combined occurrence is also increasing (17). A study has shown that about 80% of senile degenerative heart valve disease (SDHVD) patients also suffer from arrhythmia, and AF occurs easily in the early stage of SDHVD (18). In elderly patients with degenerative heart valve disease, MR causes atrial enlargement and annulus dilatation and may also lead to atrial myocardial fibrosis electrical remodeling, which leads to the occurrence of AF (19). AF itself can lead to left atrial dilatation and mitral valve dilatation, leading to functional Type I MR, which is known as atrial functional MR.

MR is a common condition of VHDs, and there has been particular interest in the occurrence of AF, especially in the elderly (20). Sustained MR tends to cause left atrial enlargement, which is a possible precursor of AF, and research has demonstrated that MR patients with preoperative AF had a worse postoperative outcome than those in sinus rhythm (21,22). As noted in our study, VHD patients with NVAF were older in age and had more severe echo findings. Grigioni *et al.* (10) found that left atrium (LA) enlargement is not a benign compensatory phenomenon in MR but leads to AF and its complications. In view of the present clinical practice, transient or unrecorded AF episodes may be ignored, and the link between degenerative MR and NVAF needs to be questioned. LA enlargement corresponds to an abnormal LA size and could be a marker of AF in VHD patients. Thus, echocardiographic diagnosis could be an important marker for those at high risk of both VHD and chronic AF.

Hypertension is an independent risk factor for both VHD and AF. Hypertension is very common in AF patients, both conditions often coexist and are responsible for considerable morbidity and mortality. There is extensive evidence that high blood pressure is a significant contributor to the incidence of AF (23). In our study population, hypertensive

Table 3 Comparison of laboratory test results between the 2 groups

Variables	NVAF + VHD (n=68)	VHD (n=68)	P value
Age, years	78 [73–80]	79 [73–83]	0.014*
Sex: male	41 (60.29)	44 (64.70)	0.595
History of smoking	27 (39.71)	32 (47.06)	0.387
History of hypertension	46 (67.65)	41 (60.29)	0.372
History of diabetes mellitus	18 (26.47)	14 (20.59)	0.420
History of CAD	19 (27.94)	18 (26.47)	0.847
History of renal dysfunction	13 (19.12)	9 (13.24)	0.354
WBC, 10E+9/L	7.18 [5.68–9.42]	7.69 [5.69–8.79]	0.624
Hgb, g/L	126 [115–138]	125 [106–138]	0.282
TP, g/L	67.23±0.77	67.99±1.00	0.096
ALB, g/L	34.63±0.50	35.88±0.60	0.096
UA, μmol/L	447 [336–534]	372 [309–452]	0.009*
Blood calcium, mmol/L	2.21 [2.11–2.27]	2.22 [2.12–2.32]	0.189
Glucose, mmol/L	5.79 [4.86–6.8]	5.49 [4.60–6.64]	0.244
TC, mmol/L	4.52±1.10	4.64±1.32	0.603
TG, mmol/L	0.98 [0.76–1.34]	1.06 [0.77–1.42]	0.802
HDL, mmol/L	1.10 [0.90–1.32]	1.33 [1.08–2.67]	<0.001*
LDL, mmol/L	2.86±0.12	2.36±0.17	0.014*

Data are presented as median [range], n (%) or mean ± SD. *, there were significant differences between the VHD patients with NVAF and patients with VHD. NVAF, non-valvular atrial fibrillation; VHD, valvular heart disease; CAD, coronary atherosclerotic heart disease; WBC, white blood cell; Hgb, hemoglobin; TP, total protein; ALB, albumin; UA, uric acid; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 4 Comparison of echocardiogram structure and function between the 2 groups

Echocardiogram data	NVAF + VHD (n=68)	VHD (n=68)	P value
LAD (mm)	41 [38–45]	31 [28–38]	<0.001
LVEDD (mm)	46 [42–52]	45 [41–48]	0.234
LVEF (%)	63 [51–68]	66 [62–69]	0.013
MR grading			
Slight	6 (8.82)	8 (11.76)	0.003
Mild	25 (37.76)	42 (61.76)	
Moderate	19 (27.94)	10 (14.71)	
Serious	18 (26.47)	8 (11.76)	
MAC grading			
Slight	21 (30.9)	30 (44.1)	0.04
Mild	30 (44.1)	30 (44.1)	
Moderate	13 (19.1)	7 (10.3)	
Serious	4 (5.9)	1 (1.5)	

Data are presented as median [range] or n (%). NVAF, non-valvular atrial fibrillation; VHD, valvular heart disease; LAD, left atrial diameter; LVEDD, left ventricular diastolic diameter; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; MAC, mitral annulus calcification.

patients also accounted for a higher proportion in the NVAF group (70.4%) than the sinus rhythm group (42.4%). Through augmented mechanical stress and increased regurgitation volume, it has been proven that hypertension may also worsen the progression and prognosis of MR (24). As hypertension and AF are common in the general population and the prevalence of both conditions increases with age, MR also could be the high co-prevalence of hypertension and AF, especially in older populations, which indicates that there may be an association between these disorders. Awareness of the association risk between AF and hypertension in the development of MR in VHD patients may be of great importance and a focus on the prevention of the development of risk factors and optimal treatment could reduce morbidity, mortality, and health care expenditures.

The levels of HDL of patients with NVAF combined with SDHDVD were lower than those of the SDHDVD group, while the levels of UA and LDL were significantly higher than those of the SDHDVD group (both $P<0.05$). A 2009 long-term follow-up study of the American Heart Association Foundation on lipid-lowering therapy found that patients with low HDL cholesterol had a higher incidence of AF (25). A follow-up study in Japan in 2011 also found that for every 10% reduction in HDL cholesterol in women, the risk of AF in their patients was 28% (26). However, the specific related mechanism of AF and HDL is not clear. This study showed that the elderly patients with degenerative VHD had a lower HDL level and a higher possibility of AF. A low HDL level might participate in the pathophysiological mechanism of SDVHD and thus affect the occurrence and development of AF, but the specific mechanism is still unclear.

In addition, UA levels were higher in patients with concomitant AF, which suggests that UA may be involved in the mechanism by which AF aggravates the development of degenerative heart valve disease in the elderly. Studies have shown that hyperuricemia is involved in the occurrence of a variety of cardiovascular diseases and causes endothelial dysfunction (27). It may be that hyperuricemia participates in the mechanism of endocardial injury, resulting in damaged heart valves, the deposition of inflammatory infiltration calcium salt, etc.

In this study, the LDL level of the patients with NVAF combined with SDHDVD was higher than that of the patients with simple SDHDVD, which suggests that the combination of AF causes higher LDL and aggravates the valvular lesion of senile degenerative VHD. Inconsistent with foreign studies, a study in the United States showed that LDL cholesterol and total cholesterol were equally and negatively correlated with AF risk, and it might be that hyperthyroidism reduces the total cholesterol and LDL cholesterol levels and is secondary to AF (28). AF is negatively correlated with LDL cholesterol; however, studies outside China have also shown that elevated blood cholesterol aggravates valve calcification (29), but the specific mechanism requires further exploration.

In this study, the ultrasonic examination indexes were compared, and it was found that the left atrial size, reflux degree, and calcification degree of the NVAF group and the aged degenerative VHD group were higher than those of the aged degenerative VHD without AF group, and the EF value was significantly lower than that of the aged degenerative VHD simple group ($P=0.013$). Thus, AF may be a risk factor for the development of degenerative VHD in the elderly.

The 2015 guidelines for the understanding and treatment of AF in China state that the pathophysiological mechanisms of AF can lead to atrial mechanical remodeling, including atrial myofibers, AF, and atrial enlargement (30). As the LA expands, it aggravates the pull on the posterior leaflet of the mitral valve, which may lead to lobular malposition and insufficiency, causing regurgitation. Silbiger used the term "atriogenic leaflet tethering" to describe the possible mechanisms by which anatomical changes in LA due to long-term changes in left atrial pressure and volume may lead to functional MR (19). The present study showed that the left atrial size of SDVHD with AF was larger than that of SDVHD without AF, and this confirms that atrial enlargement can be caused by AF. This study also found that patients with AF had more severe MR, which confirms that AF can lead to functional MR, and thus also aggravate the degree of regurgitation of degenerative VHD in the elderly.

This study revealed that valvular calcification is more obvious in patients who also have AF, which indicates that the combination of NVAF can aggravate the calcification of degenerative VHD in the elderly. It may be that AF not only aggravates the activation of the renin-angiotensin system and the microinflammatory state of the body, but also causes renal insufficiency, which is a risk factor for degenerative VHD in the elderly. It may also be that the insufficient vitamin D production and insufficient activation of parathyroid hormone is due to renal insufficiency, which results in bone salt dissolution and deposition on the heart valve. However, the specific mechanism needs further study (31). A large number of studies have shown that

AF can aggravate heart failure, and AF and heart failure interact. In this study, we found that the LVEF of patients with NVAF combined with SDHDVD was lower than that of patients with SDVHD without AF, indicating that the cardiac function of patients with NVAF combined with SDHDVD should be significantly decreased.

Study limitations

Our study provided information on NVAF differences in the prevalence of severe VHD. We also presented unique echocardiology data on severe VHD manifestations with and without AF. Due to the relatively small sample size and as this was only a single-center study, selection bias exists. As a retrospective analysis was conducted in this study, a prospective, multi-center study needs to be conducted to further determine the exact prevalence and risk factors for VHD in patients with NAVF.

Conclusions

The prevalence of moderate to severe MR was significantly higher in patients with NVAF, who had more pronounced MR and valve calcification and were more likely to have hyperlipidemia than those with VHD in sinus rhythm.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-61/rc

Data Sharing Statement: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-61/dss

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jtd.amegroups.com/article/view/10.21037/jtd-23-61/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 Ethics Committee of The First Affiliated Hospital of Shantou University (No. 2020-138) and informed consent was taken from all the patients.

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