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Evaluation of Mitral and Aortic Valvular Disease and Left Ventricular Dysfunction in a Lebanese Population: Retrospective Single-Center Experience

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Background: Recently, new therapeutic approaches have revolutionized the management of left ventricular dysfunction (LVD) and valvular heart disease (VHD), which are a growing public health problem. In parallel, there are no available epidemiological data about LVD and VHD in developing countries, especially in the Mediterranean area. This retrospective study was conducted at a single center and aimed to evaluate the associations between mitral and aortic valvular disease and left ventricle systolic and diastolic dysfunction in the Lebanese population.


Material/Methods: A retrospective study was conducted of 4520 consecutive patients aged >18 years who were referred to the Cardiovascular Department of Notre Dame de Secours-University Hospital in Jbeil-Lebanon for transthoracic echocardiography between December 2016 and December 2019. The study population was divided into different groups based on types of LVD and VHD. Left ventricle systolic dysfunction was defined as a left ventricle ejection fraction (EF) ≤40%. Statistical analysis was carried out using SPSS software version 20.

Results: VHD and systolic dysfunction were more common in men, whereas diastolic dysfunction was more common in women. Being older than age 65 years and smoking were significantly associated with heart failure with preserved EF, whereas female sex was a significant preventive factor against heart failure with reduced EF. Systemic hypertension was correlated with mitral stenosis and tricuspid regurgitation, whereas diabetes mellitus was associated with tricuspid regurgitation (TR). Smoking and older age also appeared to be associated with aortic stenosis.

Conclusions: Mitral valve disease (regurgitation and stenosis) was significantly correlated with systolic dysfunction, whereas aortic and mitral regurgitation were associated with diastolic dysfunction. Better monitoring of cardiovascular disease risk factors may lead to a reduced burden of LVD and VHD.

Keywords: Heart Failure • Heart Valve Diseases • Risk Factors • Ventricular Dysfunction, Left

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Background

Cardiovascular disease is a major cause of mortality. The global increase in life expectancy has resulted in a rising incidence of valvular heart disease (VHD) and left ventricular dysfunction (LVD) among populations, and their impacts on health systems constitute a growing public health problem [1].

VHD is damage to any cardiac valve that alters its capacity to fully open or close. LVD is mainly divided into 2 groups: systolic LVD, characterized by a depressed ejection fraction (EF), and diastolic LVD (LVDD), characterized by stiff heart muscles that cause inadequate relaxation. Statistics from published epidemiological data have shown an increasing prevalence of VHD and heart failure in European and American populations, and the trend is expected to continue [2,3]. Moreover, the widespread use of echocardiography – a simple, noninvasive diagnostic tool – has led to discovery of silent cases of VHD and LVD, which suggests that these 2 independent clinical entities progress slowly over time, from an asymptomatic stage to physical disability or sudden death [4-6].

Transthoracic echocardiography is the diagnostic tool recommended for first-line assessment of left ventricle function, valvular stenosis, and regurgitation [7,8]. Quantitative evaluation of left ventricle EF is usually calculated by dividing the stroke volume by the end-diastolic volume. A value $\leq 40\%$ is defined as left ventricular systolic dysfunction (LVSD) [8,9]. Transesophageal echocardiography is usually performed to grade the severity of valvular stenosis or regurgitation, particularly in patients with heart failure who have regurgitation [7].

Different imaging modalities are integrated into the management of VHD. For example, exercise testing unmasks symptoms, particularly in asymptomatic patients with severe aortic stenosis (AS), and exercise echocardiography identifies the cardiac origin of dyspnea in addition to assessing the prognosis for mitral regurgitation (MR) and AS [8]. Cardiac magnetic resonance imaging is useful in patients with when echocardiogram quality is poor or results are conflicting, and it is the criterion standard imaging modality for evaluating right ventricle function, which is important in cases of tricuspid regurgitation (TR) [8].

In parallel, novel medical and interventional therapeutic approaches recently have revolutionized the management of VHD and LVD, improving the quality of life and survival of patients. However, critically ill patients are not candidates for surgery or standard treatments and they may be unable to afford the new transcatheter therapies [10].

Historically, VHD has been a main cause of and poor prognostic predictor for LVD and it is associated with high rates of

morbidity and mortality [11,12]. Given the paucity of epidemiological data in the Mediterranean area and the absence of surveys in Lebanon, cardiology data for that population have been extrapolated from experience in the United States or Europe. Therefore, the aim of the present study, which was retrospective and single-center, was to evaluate associations between mitral valve disease (MVD) and aortic valve disease (AVD) and LVSD and LVDD in the Lebanese population.

Material and Methods

Study Design and Population

A retrospective, observational, single-center study was conducted of patients referred for transthoracic echocardiography to the Cardiovascular Department of Notre Dame de Secours-University Hospital (NDS-UH), Jbeil-Lebanon between December 2016 and December 2019. All patients aged >18 years who were not known to have or not being followed for LVD and VHD and who underwent echocardiography during the study period were consecutively enrolled. Patients previously diagnosed with rheumatic heart disease, LVD, and/or VHD were excluded. Patients with physiological or insignificant valvular regurgitation and LVSD with mid-range EF (40% to $<50\%$) were excluded so that LVSD and LVDD could be compared. The study population was divided into groups, based on the different types of VHD: 1) AVD (AS or aortic regurgitation [AR]); 2) Mitral valve disease [MVD] (mitral stenosis [MS] or MR); 3) Combined aorto-mitral valve disease and TR; and LVD (LVSD or LVDD).

Data Collection and Endpoints

Echocardiographic studies were performed by the same reference physicians using a General Electric machine. Standard gray-scale and color Doppler images were acquired at a depth of 16 cm in the parasternal (standard long and short axis) and apical views (2- and 4-chamber and apical long axis). The semi-quantitative and quantitative methods recommended by the American Society of Echocardiography were used to assess VHD (regurgitation or stenosis). LVSD was defined as an EF $\leq 40\%$ and diastolic LVD as an early mitral inflow velocity (E)-to-tissue Doppler mitral annular early diastolic velocity (E') ratio (E/E') <8 and EF $\geq 50\%$. EF was calculated in the apical view by using Simpson's biplane method [13]. In the present study, significant VHD was defined as MS or AS of any severity and mild, moderate, or severe MR, AR, or TR. The primary aim was to evaluate the associations between LVD and VHD in a Lebanese population based on echocardiographic criteria. Our study was approved by the Ethics Committees at NDS-UH and written consent for data collection was obtained from the hospital.

Statistical Analysis

Quantitative data were summarized with means and standard deviations, whereas qualitative data were summarized with counts and percentages. A chi-square test was used during the first step in statistical analysis to assess the differences between the groups. Multivariate logistic regression was performed to investigate the association between each type of VHD and LVD as a dependent variable. $P < 0.05$ was considered statistically significant. Incidence of LVD was calculated by dividing the number of new cases during the specified time interval by the total person-years of observation.

Results

Baseline characteristics of the 4520 patients included in the study are shown in **Table 1**. Of the patients, 2575 (57%) were men with a mean age of 65.70 ± 38.60 and 1945 (43%) were women with a mean age of 64.87 ± 40.61 , 1355 (30%) had arterial hypertension, 1040 (23%) had diabetes mellitus (DM), 1045 (23.1%) had dyslipidemia, and 1900 (42%) were smokers. Nearly half of the studied population (49.2%; 2225/4520) were age >65 years and 50.8% (2295/4520) were age <65 years, with an overall mean age of 63.7 years. Regarding the different types of VHD, AVD was present in 1440 patients (31.9%), of whom 1040 (23%) had AR and 400 (8.9%) had AS. MVD was present in 2110 (46.7%), of whom 2100 (46.5%) had MR and 50 (1.1%) had MS. Aorto-MVD was present in 970 (21.5%) and 1870 (41.4%) had TR. Of the patients, 2705 (59.8%) were diagnosed with diastolic dysfunction and 810 (17.9%) with systolic dysfunction (**Table 1**). The incidences of LVSD and LVDD in the Lebanese population we studied were 11.9 per 100 person per year and 39 per 100 person per year, respectively.

Considering sex, MR was the most common type of VHD and was equally distributed among men (46.4%) and women (46.5%). However, diastolic dysfunction was more common in women (62% vs 58%) while systolic dysfunction was more common in men (22% vs 12%). No statistically significant association was found between sex and VHD, but being female was positively correlated with diastolic dysfunction (odds ratio [OR]=1.17; 95% confidence interval [CI] 1.04-1.33) and negatively correlated with systolic dysfunction (OR=0.48; CI 0.41-0.57)]. The incidence of systolic dysfunction was 8% in women and 14.9% and for diastolic dysfunction, it was 41.4% in women and 38.7% in men.

Regarding age, VHD (52.6% vs 47.4%), diastolic (65% vs 55%), and systolic dysfunction (20% vs 16%) were significantly more common in patients age >65 years compared with those age ≤ 65 years. MR was the most common VHD in both age groups. Age >65 years was positively correlated with incidence of AS

Table 1. Baseline characteristics of the studied population.

Variable	N (%)	Total
Age		4520
≥65 years	2225 (49)	
<65 years	2295 (51)	
Sex		4520
Male	2575 (57)	
Female	1945 (43)	
Smoking	1900 (42)	1900
Arterial hypertension	1355 (30)	1355
Diabetes mellitus	1040 (23)	1040
Dyslipidemia	1045 (23.1)	1040
Aortic valve disease		1440
Aortic regurgitation	1040 (23)	
Aortic stenosis	400 (8.9)	
Mitral valve disease		2150
Mitral regurgitation	2100 (46.5)	
Mitral stenosis	50 (1.1)	
Aorto-mitral valve disease	970 (21.5)	970
Tricuspid regurgitation	1870 (41.4)	1870
Diastolic dysfunction	2705 (59.8)	2120
Systolic dysfunction	810 (17.92)	810

(OR=2.65; CI 2.09-3.37), TR (OR=1.1; 95% CI 1.05-1.3), MS (OR=3.83; 95% CI 1.9-7.7), MR (OR=1.16; 95% CI 1.03-1.3), systolic dysfunction (OR=1.3; 95% CI 1.1-1.5), and diastolic dysfunction (OR=1.4; 95% CI 1.2-1.6). The incidence of systolic dysfunction was 13.5% in patients age >65 years and 10.4% in those ≤ 65 years, whereas the incidence of diastolic dysfunction was 43% in those age >65 years and 37% in those age ≤ 65 years.

LVD and VHD were more likely in patients with multiple risk factors. Indeed, multivariate logistic regression showed a positive correlation between smoking and all types of VHD and between systemic arterial hypertension and MS (OR=10.8; 95% CI 3.9 to 29.3) and TR (OR=1.5; 95% CI 1.2 to 1.8). It also showed a negative association between DM and TR (OR=0.55; 95% CI 0.4 to 0.7). Moreover, smoking and arterial hypertension were the 2 CVD risk factors studied that were positively correlated with diastolic dysfunction (OR=1.4; 95% CI 1.2 to 1.7; $P < 0.001$), while no correlation was found between CVD risk factors and systolic dysfunction (**Tables 2, 3**). Both diastolic and systolic dysfunction were more common in patients with VHD, particularly in those with MVD (**Table 4**). Multivariate

Table 2. Prevalence of valvular heart disease and left ventricular dysfunction according to several cardiovascular risk factors.

		Sex		Age		Smoker		AH		DLP		DM	
		Male	Female	[18-65]	>65	No	Yes	No	Yes	No	Yes	No	Yes
AVD	No	1740 68%	1340 69%	1660 72%	1420 64%	2300 88%	780 41%	2515 80%	565 42%	2510 72%	570 55%	2520 72%	560 54%
	Yes	835 32%	605 31%	635 28%	805 36%	320 12%	1120 59%	650 21%	790 58%	965 28%	475 46%	960 28%	480 46%
	<i>P</i>	0.36		<0.001		<0.001		<0.001		<0.001		<0.001	
AR	No	1970 77%	1510 78%	1765 77%	1715 77%	2340 89%	1140 60%	2640 83%	840 62%	2745 79%	735 70%	2760 79%	720 69%
	Yes	605 23%	435 22%	530 23%	510 23%	280 11%	760 40%	525 17%	515 38%	730 21%	310 30%	720 21%	320 31%
	<i>P</i>	0.39		0.96		<0.001		<0.001		<0.001		<0.001	
AS	No	2365 92%	1755 90%	2180 95%	1940 87%	2595 99%	1525 80%	3050 96%	1070 79%	3250 94%	870 83%	3245 93%	875 84%
	Yes	210 8%	190 10%	115 5%	285 13%	25 1%	375 20%	115 4%	285 21%	225 7%	175 17%	235 7%	165 16%
	<i>P</i>	0.06		<0.001		<0.001		<0.001		<0.001		<0.001	
MVD	No	1345 52%	1025 53%	1260 55%	1110 50%	1550 59%	820 43%	1810 57%	560 41%	1890 55%	480 46%	1920 55%	450 43%
	Yes	1230 48%	920 47%	1035 45%	1115 50%	1070 41%	1080 57%	1355 43%	795 59%	1585 46%	565 54%	1560 45%	590 57%
	<i>P</i>	0.76		<0.001		<0.001		<0.001		<0.001		<0.001	
MR	No	1380 53.6%	1040 53.5%	1280 56%	1140 52%	1575 60%	845 45%	1830 58%	590 44%	1935 56%	485 46%	1965 57%	455 44%
	Yes	1195 46.4%	905 46.5%	1015 44%	1085 49%	1045 40%	1055 56%	1335 42%	765 57%	1540 44%	560 54%	1515 44%	585 56%
	<i>P</i>	0.91		0.002		<0.001		<0.001		<0.001		<0.001	
MS	No	2550 99%	1920 99%	2285 99%	2185 98%	2605 99%	1865 98%	3150 99%	1320 97%	3435 99%	1035 99%	3440 99%	1030 99%
	Yes	25 1%	25 1%	10 1%	40 2%	15 1%	35 2%	15 1%	35 3%	40 1%	10 1%	40 1%	10 1%
	<i>P</i>	0.32		<0.001		<0.001		<0.001		0.6		0.61	
AMVD	No	2020 78%	1530 79%	1860 81%	1690 76%	2385 91%	1165 61%	2720 86%	830 61%	2815 81%	735 70%	2820 81%	730 70%
	Yes	555 22%	415 21%	435 19%	535 24%	235 9%	735 39%	445 14%	525 39%	660 19%	310 30%	660 19%	310 30%
	<i>P</i>	0.86		<0.001		<0.001		<0.001		<0.001		<0.001	
TR	No	1520 59%	1130 58%	1400 61%	1250 56%	1640 63%	1010 53%	1950 62%	700 52%	2045 59%	605 58%	2035 59%	615 59%
	Yes	1055 41%	815 42%	895 39%	975 44%	980 37%	890 47%	1215 38%	655 48%	1430 41%	440 42%	1445 42%	425 41%
	<i>P</i>	0.53		<0.001		<0.001		<0.001		0.58		0.7	

Table 2 continued. Prevalence of valvular heart disease and left ventricular dysfunction according to several cardiovascular risk factors.

		Sex		Age		Smoker		AH		DLP		DM	
		Male	Female	[18-65]	>65	No	Yes	No	Yes	No	Yes	No	Yes
DD	No	1080 42%	735 38%	1025 45%	790 36%	1175 45%	640 34%	1370 43%	445 33%	1400 40%	415 40%	1405 40%	410 39%
	Yes	1495 58%	1210 62%	1270 55%	1435 65%	1445 55%	1260 66%	1795 57%	910 67%	2075 60%	630 60%	2075 60%	630 61%
	<i>P</i>	0.005		<0.001		<0.001		<0.001		0.74		0.58	
SD	No	2000 78%	1710 88%	1935 84%	1775 80%	2150 82%	1560 82%	2615 83%	1095 81%	2875 83%	835 80%	2865 82%	845 81%
	Yes	575 22%	235 12%	360 16%	450 20%	470 18%	340 18%	550 17%	260 19%	600 17%	210 20%	615 18%	195 19%
	<i>P</i>	<0.001		<0.001		0.97		0.14		0.03		0.42	

AH – arterial hypertension; AMVD – aorto-mitral valve disease; AVR – aortic valve regurgitation; AVS – aortic valve stenosis; DD – diastolic dysfunction; DLP – dyslipidemia; DM – diabetes mellitus; MVD – mitral valve disease; MVR – mitral valve regurgitation; MVS – mitral valve stenosis; R – tricuspid regurgitation; SD – systolic dysfunction; TAVD – aortic valve disease.

logistic regression showed an association between diastolic and systolic LVD and several types of VHD and AR (OR=0.44; 95% CI 0.2-0.9) and MVD (OR=0.24; 95% CI 0.09-0.65) and a positive correlation with MR (OR=6.7; 95% CI 2.5-18.2) and MS (OR=4.7; 95% CI 1.45-15.4). Systolic dysfunction was positively correlated with MVD (OR=7.2; 95% CI 4.4-11.8) and TR (OR=1.2; 95% CI 1.05-1.44) (Table 5).

Discussion

Although there are a number of therapies for LVD, epidemiologic data about this clinical entity are lacking [14]. The present study was the first epidemiological survey of LVD in a Mediterranean Lebanese population. It showed a 17.9% prevalence of LVSD and a 59.8% prevalence of LVDD. Our study also revealed a significant association between VHD and LVD, which have several CVD risk factors in common. In parallel, we showed a higher incidence of VHD and LVSD and LVDD in individuals older than age 65 years, whereas male sex was an independent predictor for LVSD. These results are consistent with previous findings in European and American populations [2,15-17]. The international incidence of LVD varies between 1% and 20.5% for LVSD [2,15] and 15.8% and 52.8% for LVDD [18]. The higher incidence of diastolic dysfunction in our study can be explained by the older age of the population studied and their ethnicity. For example, a 25.1% prevalence rate for LVDD has been reported in a study of Europeans with a mean age of 48.5±15.7 [19], while the median prevalences of LVSD and LVDD in the European population are estimated at 36% and 5.5%, respectively [18]. In Asian and African-Caribbean populations, an LVD prevalence higher than 70% has been reported [20].

The present study also showed no significant association between female sex and LVSD. This finding may be related to the fact that in women, LVD tends to be related to hypertension and DM and associated with preserved EF, whereas in men, it is more likely to be provoked by myocardial infarction [21]. Our research also showed that age >65 years is a risk factor for developing AS and diastolic dysfunction. This is due to a degenerative process that leads to active leaflet calcification and subsequent narrowing of the effective valve opening area [22]. Variant modifications in cardiac structure also occur with aging, such as replacement of myocytes by fibroblasts that produce collagen, which increases heart stiffness and affects ventricle compliance, contributing to diastolic dysfunction [23]. As shown by the present study, smoking is significantly correlated with VHD and diastolic dysfunction. Smoking cigarettes accelerates the atherosclerotic process and acutely impairs diastolic function by decreasing E wave and increasing A wave velocity, which results in a reduced E/A ratio that reflects the altered diastolic function [24].

In the present study, arterial hypertension was found to be significantly correlated with MS and TR. A literature review revealed a higher incidence of systemic hypertension in patients with MS [25] but provided no explanation for it; the etiology could be the subject of a future prospective study aimed at investigating a possible causal relationship. Indeed, blood pressure elevation recently has been investigated as a risk factor for development of AS or AR [26]. We also found a significant relationship between TR and DM, which has not been described before. Variant valvular changes caused by DM have been described but without statistical significance [27], except for the documented significant correlation between MR and Type 2 DM [28].

Table 3. Multivariate logistic regression for each type of valvular heart disease and left ventricular dysfunction, with cardiovascular risk factors as independent variables.

		Sex	Age	Smoker	AH	DLP	DM
AVR	OR		0.89	5.7	1.1	0.64	1.26
	95% CI		[0.77-1.03]	[4.7-6.9]	[0.89-1.37]	[0.28-1.4]	[0.86-1.85]
	P		0.137	<0.001	0.33	0.29	0.21
AVS	OR		2.66	18	1.7	1.95	0.49
	95% CI		[2.01-3.3]	[11.5-28.2]	[1.3-2.3]	[1.2-3.2]	[0.3-0.8]
	P		<0.001	<0.001	<0.001	0.007	0.006
MVR	OR		1.16	1.7	1.07	0.58	2.1
	95% CI		[1.03-1.3]	[1.4-1.9]	[0.88-1.3]	[0.42-0.81]	[1.5-2.9]
	P		0.01	<0.001	0.48	0.001	0.001
MVS	OR		3.8	0.75	10.8	0.67	0.35
	95% CI		[1.9-7.7]	[0.29-2.02]	[3.9-29.3]	[0.18-2.42]	[0.09-1.27]
	P		0.001	0.59	<0.001	0.54	0.11
TR	OR		1.18	1.25	1.52	1.27	0.55
	95% CI		[1.05-1.34]	[1.07-1.47]	[1.25-1.85]	[0.92-1.74]	[0.39-0.76]
	P		0.005	<0.001	<0.001	0.14	0.044
AMVD	OR		1.25	5.4	1.5	0.94	0.8
	95% CI		[1.07-1.46]	[4.4-6.5]	[1.2-1.8]	[0.65-1.37]	[0.55-1.18]
	P		0.004	<0.001	<0.001	0.76	0.26
DD	OR	1.17	1.4	1.4	1.4	0.86	0.82
	95% CI	[1.04-1.33]	[1.26-1.6]	[1.2-1.6]	[1.1-1.7]	[0.63-2]	[0.59-1.15]
	P	0.009	<0.001	<0.001	0.001	0.38	0.25
SD	OR	0.48	1.35	0.9	1.01	1.2	0.573
	95% CI	[0.4-0.56]	[1.1-1.5]	[0.5-1.5]	[0.8-1.2]	[0.9-1.5]	[0.2-1.4]
	P	<0.001	<0.001	0.72	0.89	0.054	0.2

AH – arterial hypertension; AMVD – aorto-mitral valve disease; AVR – aortic valve regurgitation; AVS – aortic valve stenosis; CI – confidence interval; DD – diastolic dysfunction; DLP – dyslipidemia; DM – diabetes mellitus; MVR – mitral valve regurgitation; MVS – mitral valve stenosis; OR – odds ratio; SD – systolic dysfunction; TR – tricuspid regurgitation.

In our study, MVD was significantly correlated with systolic dysfunction. MR enhances modification of left ventricle structure by inducing left ventricle remodeling and altering intrinsic contractility, leading to systolic dysfunction [29], whereas MS directly decreases left ventricle strain or contractility [30]. Finally, our study found a significant relationship between AR and MR and diastolic dysfunction.

Limitations

The present study has some limitations, in that it was not population-based and it was retrospective, observational, and performed at a single center. Only written medical records and

not echocardiograms were reviewed. Data from results of testing for N-terminal pro b-type natriuretic peptide, on use of medication, and for scales reflecting clinical manifestations were not collected for 2 reasons: 1) A large proportion of the patients studied were outpatients, so that information was lacking; and 2) No computerized medical files were available.

Conclusions

The incidence and epidemiological characteristics of LVD in a Mediterranean Lebanese population are similar to those found in patients in Europe. Modifiable CVD risk factors play

Table 4. Distribution of valvular heart disease according to types of left ventricular dysfunction.

		Diastolic dysfunction			Systolic dysfunction		
		No	Yes	P value	No	Yes	P value
AVD	No	1410, 78%	1670, 62%	<0.001	2580, 70%	500, 62%	<0.001
	Yes	405, 22%	1035, 38%		1130, 30%	310, 38%	
AVR	No	1485, 82%	1995, 74%	<0.001	2900, 78%	580, 72%	<0.001
	Yes	330, 18%	710, 26%		810, 22%	230, 18%	
AVS	No	1730, 95%	2390, 88%	<0.001	3395, 92%	725, 90%	0.69
	Yes	85, 5%	315, 12%		315, 8%	85, 10%	
MVD	No	1090, 60%	1280, 47%	<0.001	2125, 57%	245, 30%	<0.001
	Yes	725, 40%	1425, 53%		1585, 43%	565, 70%	
MVR	No	1115, 61%	1305, 48%	<0.001	2145, 58%	275, 34%	<0.001
	Yes	700, 39%	1400, 52%		1565, 42%	535, 66%	
MVS	No	1800, 99%	2670, 99%	0.14	3685, 99%	785, 97%	<0.001
	Yes	15, 1%	35, 1%		25, 1%	25, 3%	
AMVD	No	1540, 85%	2010, 74%	<0.001	2990, 81%	560, 69%	<0.001
	Yes	275, 15%	695, 26%		720, 19%	250, 31%	
TR	No	1115, 61%	1535, 57%	0.002	2255, 61%	395, 49%	<0.001
	Yes	700, 39%	1170, 43%		1455, 39%	415, 51%	

AMVD – aorto-mitral valve disease; AVD – aortic valve disease; AVR – aortic valve regurgitation; AVS – aortic valve stenosis; MVD – mitral valve disease; MVR – mitral valve regurgitation; MVS – mitral valve stenosis; TR – tricuspid regurgitation.

Table 5. Multivariate logistic regression taking using each type of left ventricular dysfunction as a dependent variable and different kinds of valvular heart disease as independent variables.

	Diastolic dysfunction			Systolic dysfunction		
	Adjusted OR	95% CI	P value	Adjusted OR	95% CI	P value
AVD	4.64	[2.2-9.7]	<0.001	1.119	[0.522-2.397]	0.772
AVR	0.44	[0.2-0.9]	0.031	1.1	[0.93-1.33]	0.23
AVS	0.75	[0.34-1.7]	0.48			
MVD	0.24	[0.09-0.65]	0.006	7.2	[4.4-11.8]	<0.001
MVR	6.7	[2.5-18.2]	0.001	0.34	[0.2-0.5]	0.001
MVS	4.73	[1.4-15.4]	0.01	1.8	[0.16-22]	0.6
AMVD	0.72	[0.54-0.95]	0.02	0.8	[0.6-1.05]	0.11
TR	1.00	[0.88-1.14]	0.98	1.23	[1.04-1.44]	0.01

AMVD – aorto-mitral valve disease; AVD – aortic valve disease; AVR – aortic valve regurgitation; AVS – aortic valve stenosis; CI – confidence interval; MVD – mitral valve disease; MVR – mitral valve regurgitation; MVS – mitral valve stenosis; OR – odds ratio; TR – tricuspid regurgitation.

a significant role in development of LVD and VHD. Our results underscore the need for better management of CVD risk factors to reduce the impact of VHD and LVD on the public health system.

Conflicts of Interest

None.

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