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## CLINICAL SPECTRUM AND ECHO-CARDIOGRAPHIC FEATURES IN VHD PATIENTS OF TERTIARY CARE HOSPITAL IN TAMIL NADU

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### Keywords:

Valvular heart disease,  
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**ABSTRACT: Objectives:** Valvular Heart Disease (VHD) is an important cause of cardiovascular morbidity and mortality worldwide. The aim of this study was to establish the frequency of VHD on the basis of echocardiographic parameters, assess etiological factors, quantify the clinical spectrum and evaluate the medical management of VHD. **Methods:** This observational cross-sectional study was conducted for a period of 5 months from May to September 2021 at a tertiary care hospital in Tamil Nadu. A total of 101 patients, both in-patients and out-patients visiting the cardiology department were included for the study. **Results:** Among the 101 participants, the mean age was  $55.43 \pm 14.91$  with 55.4% of them being males. The most common etiology was found to be degenerative (75.2%). The most commonly encountered valve lesions included mitral regurgitation (67.3%) followed by aortic regurgitation (36.6%), tricuspid regurgitation (33.7%) and mitral stenosis (21.8%). The prevalence of VHD and VHD of degenerative etiology increased with age  $>40$ . The occurrence of VHD symptoms was also higher with age  $>40$  ( $P < 0.001$ ). **Conclusion:** The major causes for the occurrence of VHD among the study population were degenerative followed by rheumatic heart disease. Surgical intervention was not preferred by the study participants due to lack of financial resources. Greater efforts are required to ensure the affordability of surgical procedures by the medical insurance provided by the state/country, considering the better prognosis seen with surgical interventions.

**INTRODUCTION:** The spectrum of valvular Heart Disease (VHD) spans across congenital, rheumatic, degenerative and calcified etiologies. The prevalence and incidence of VHD has increased with better prognosis seen with advances in imaging and treatment offered by cardiologists and cardiothoracic surgeons.

Effective invasive and non-invasive monitoring of ventricular function, valve reconstruction techniques and invention of prosthetic valves have improved prognosis. VHD is a major cause of cardiovascular morbidity and mortality worldwide posing a huge burden on the health care resources <sup>1</sup>.

Individual life expectancy and atherosclerotic risk factors pose increased incidence of age related degenerative valvular heart disease. In developing countries, the prevalence of VHD is found to be higher with age – 0.7% in 18-44 years and 13.3% in 75 years and older. The prevalence of VHD in regard to age was demonstrated to be higher among

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60-74 years of age (13.2%)<sup>2</sup>. Atrioventricular valves and semilunar valves are the natural heart valves which consist of an outer layer of valve endothelial cells (VECs) further surrounded by three more layers of extracellular matrix and valve interstitial cells (VICs)<sup>3</sup>. The potential variations in functionality and localization of matrix components surrounding valves leads to VHD with both genetic and acquired causes disrupting the normal organization and composition of ECM within VECs and VICs valve mechanism. Inflammation plays a vital role in macrovascular calcification of valves including CAVD involving inflammation-associated factors, including tumor necrosis factors, interleukin 1-beta, oxidized lipoprotein, and vascular signaling processes.

The treatment choices are mostly restricted to surgical valve replacement procedures with mechanical or biological prostheses. Other therapeutic options include percutaneous valve replacement, balloon aortic valvuloplasty with multiple limitations such as non-trivial complication rates, high rates of aortic complication rates, and recurrence<sup>4</sup>. Various biomarkers are employed for understanding the pathogenesis of valvular heart disease such as asymmetric dimethylarginine, calcium phosphate, fetuin-A, osteopontin, and natriuretic peptides associated with endothelial cell dysfunction, and calcification.

The diagnostic evaluation of VHD depends on echocardiography and auscultation findings. Echocardiography is predominantly used, and helps in determining the severity and prognosis of VHD. The evaluation of stenosis and regurgitation is carried out by M-Mode. Doppler measurement for all four valves and effective regurgitant orifice area, colour flow imaging and other significant diagnostic tools in VHD assessment is also done<sup>5</sup>.

In more severe cases, VHD can be accurately assessed by some techniques such as Trans-Esophageal Echocardiography (TEE) and Trans-Thoracic Echocardiography (TTE). The pharmacological treatment of VHD is not supported by evidence. Pharmacological therapies can be used pre-surgery to delay surgery and post-surgery to promote cardiac function and prevent valve inflammation. A prospective study to

evaluate efficacy of statins in aortic stenosis (Rosuvastatin Affecting Aortic Valve Endothelium to Slow the Progression of Aortic Stenosis – RAAVE) observed beneficial effects in asymptomatic patients with moderate-severe stenosis with ECG findings<sup>6</sup>. In contrast, the Scottish Aortic Stenosis and Lipid Lowering Trial Impact of Regression (SALTIRE) study found no significant results following 25 months of statin therapy<sup>7</sup>. To make patients respond to statins, multiple medication administration at the same time with specific mode of action of statins must be employed. The other miscellaneous pharmacological treatments for valvular heart disease include proprotein convertase kexin 9 (PCSK9) inhibitors to treat hypercholesterolemia.

This is found to be more effective than statin therapy in decreasing lipoprotein and low-density lipoprotein cholesterol which is linked to the development of valve disease and calcification or stenosis<sup>8</sup>. Braunwald's paper on Further Cardiovascular Outcomes Research with PCSK9 Inhibition in Subjects with Elevated Risk (FOURIER) trial has revealed PCSK9i to decrease calcified aortic valve disease incidence. On the other side Aikawa lab experimented in demonstrating the role of PCSK9 in calcification in murine models highlighting the need for translational research for therapeutic interventions.

No drug has been developed yet for VHD and future trials must be focused on negating the pharmacological therapies for VHD<sup>9</sup>. VHD resulting in abnormal functionality of valves have severe impact in impairment of valve motion, and are associated with risk factors such as age, biological sex, tobacco use, hypercholesterolemia, type 2 diabetes mellitus, and hypertension.

The huge burden of VHD upon the children and adults has resulted in premature deprivation of productivity in their lives. The inadequate research reports on the VHD spectrum and its complications in Tamil Nadu had led to the conceptualization of this study. The study outcomes may help the initiation and implementation of campaigns that assist the reduction of VHD in Tamil Nadu. The current study attempted to understand the frequency, clinical spectrum, echocardiography parameters and etiological parameters in VHD.

**METHODOLOGY:**

**Study Design/Criteria:** A descriptive cross-sectional study was conducted for a period of 5 months from May 2021 to September 2021 at a tertiary care hospital in Tamil Nadu. The study participants were recruited from the Department of Cardiology, SRM MCH &RC after getting their written informed consent. Patients of all genders and ages from both inpatient and outpatient departments of cardiology were included in the study. The past medical history or clinical diagnosis of VHD with significant echocardiographic findings of stenosis and regurgitation were included.

**Data Collection Process:** Patients were recruited as per the criteria mentioned after explaining in detail the objective of the study. The research data were collected with the help of a questionnaire which was validated and approved by the researchers and the concerned clinicians of the department. Demographic data and other clinical features and complications of the patients like the valve involved, etiology, clinical history, ECG and echocardiography findings, Doppler measurements, Color Flow Imaging, surgical procedures undergone and medication chart were collected through the questionnaire. All procedures performed in this study involving human participants were in adherence to the 1964 Helsinki declaration and its later amendments. Institutional ethics committee of SRM Medical College Hospital and Research Centre, Kattankulathur, Chengalpattu approved the study (2460/IEC/2021).

**Statistical Analysis:** Continuous variables were summarized as mean  $\pm$  standard deviation or median (IQR) and categorical data were expressed as frequency with percentages. The differences in the categorical variables between groups were analyzed using the Chi-square test. The differences in continuous variables between groups were analyzed using the Independent Samples t-test. All statistical data in this study were analyzed using SPSS software version 16.0. All values were two-sided and a P value less than 0.05 was considered statistically significant.

**RESULT:**

**Demographics:** From May 2021 to September 2021, a total number of 101 VHD patients were

included in the study. The patient characteristics such as demographic information, lab investigations, ECG findings, echocardiographic parameters, and patient medication profile were collected and documented. Out of 101 patients, the mean age was  $55.43 \pm 14.91$  and males constituted 55.4%. The mean BMI was found to be  $24.09 \pm 2.92$ . The baseline characteristics of the study population are tabulated under **Table 1**.

**Clinical Variables:** There was no significant difference between gender groups with regard to abnormal, thickened, stenosed valves and S/P AVR. The prevalence of mitral stenosis and tricuspid regurgitation was higher in females when compared to males ( $P < 0.001$ ). There was no difference in mitral regurgitation, aortic stenosis, aortic regurgitation and pulmonary regurgitation between genders. The prevalence of single valve disease was higher in males. There was no significant difference in double valve disease between gender groups. The prevalence of VHD was higher in patients above the age of 40, in particular, VHD of degenerative etiology ( $P < 0.0001$ ).

Most of the VHD symptoms and changes in valve morphology were found in patients above 40 years of age ( $P < 0.001$ ). There was no difference in mitral regurgitation and aortic stenosis with regard to age. Single valve disease was more common in the older age group ( $> 40$  years) and multiple valve disease was more common in the younger age group ( $< 40$  years). Single valve disease is increased by age and in contrast multi valve disease is decreased by age **Table 2**.

**Echocardiography:** The most common complications among the study participants were CAD (52.5%), MI (17.8%) and hypertension (11.9%). The mean aortic root was  $2.50 \pm 0.398$  (cm). The mean LV end systole diameter and LV end diastole diameter were  $3.52 \pm 0.983$  (cm) and  $4.79 \pm 0.83$  (cm), mean end systole volume and end diastole volume were  $58.02 \pm 34.56$  (ml) and  $113.25 \pm 48.75$  (ml), and mean ejection fraction was  $51.38 \pm 13.458$  % respectively. In terms of morphology, the mitral and aortic valves were most affected. The percentages of dilation of left atrium and left ventricle were 34.4% and 26.6%, while those of the right atrium and right ventricle were

20.0% and 11.4%, respectively. The characteristics of Doppler measurement such as mean mitral, aortic and pulmonary valve were  $2.94 \pm 5.11$ (m/sec),  $4.61 \pm 13.56$  (m/sec) and  $1.03 \pm 0.253$ (m/sec).46.6% of patients had sclerosed and 15.9% had thickened aortic valves. The percentage of mitral valve abnormality was 13.6 and that of PMAC was 9.1 **Table 4.**

**Type of Lesions:** The most common diseased valve was the mitral followed by the aortic.

Multiple valve disease was seen in nearly one third of the study population. The order of involvement of the valves in descending are  $MR+TR \geq MS+MR+AR+TR \geq AR+MR \geq MR+TR+MS \geq MS+MR \geq MS+TR \geq AS+AR \geq TR+AR \geq MR+AS \geq MS+AR+TR \geq AR+TR+PR \geq MS+MR+AS+AR+TR$ . The most common valve lesions were mitral stenosis (40.9%) followed by aortic stenosis (33.3%), and tricuspid regurgitation (5.8%) **Fig. 1.**

**TABLE 1: SOCIODEMOGRAPHIC CHARACTERISTICS AND LAB INVESTIGATIONS OF STUDY PARTICIPANTS (N=101)**

Characteristics (N=101)	Frequency (%) Mean ± SD
Age (Years)	55.43 ± 14.91
Gender (Male)	56 (55.4)
<b>Education</b>	
Primary Education	22 (21.8)
Secondary Education	41 (40.6)
Higher Secondary Education	12 (11.9)
Graduate	23 (22.8)
Illiterate	3 (3)
<b>Socio-Economic Status</b>	
Low	11 (10.9)
Middle	85 (84.2)
Upper	5 (5)
Height (Cms)	157.92 ± 6.69
Weight (Kgs)	60.10 ± 8.60
Body Mass Index (kg/m <sup>2</sup> )	24.09 ± 2.92
Temperature (F)	98.39 ± 0.67
Systolic Blood Pressure (mmHg)	120.03 ± 22.79
Diastolic Blood Pressure (mmHg)	73.37 ± 12.76
Pulse rate (beats/min)	84.75 ± 17.51
Respiratory rate (breaths/min)	20.68 ± 9.72
Hemoglobin (g/dl)	11.94 ± 2.16
Red Blood Cells (Million cells/cu.mm)	4.90 ± 1.45
Packed Cell Volume (%)	36.70 ± 6.05
Serum Creatinine (mg/dl)	0.99 ± 0.76
Serum Urea (mg/dl)	34.57 ± 21.77
Blood Urea Nitrogen (mg/dl)	16.28 ± 10.69
Sodium (mmol/L)	135.44 ± 3.38
Potassium (mmol/L)	3.92 ± 0.47
Chloride (mmol/L)	101.62 ± 4.55
Bicarbonate (mmol/L)	23.88 ± 2.90
Random Blood Sugar (mg/dl)	145.38 ± 60.045
Fasting Blood Sugar (mg/dl)	153.50 ± 23.33
Total Cholesterol (mg/dl)	155.50 ± 57.011
High Density Lipoproteins (mg/dl)	39.78 ± 13.98
Low Density Lipoproteins (mg/dl)	107.56 ± 45.13
Very Low-Density Lipoproteins (mg/dl)	22.44 ± 14.46
Triglycerides (mg/dl)	112.11 ± 71.39
<b>ECG findings</b>	
Heart rate (beats/min)	82 (81.21 ± 24.66)
P wave (ms)	76 (95.09 ± 30.539)
PR wave (ms)	80 (153.61 ± 42.47)
QRS wave (ms)	87 (97.48 ± 26.81)
<b>Co-morbid conditions</b>	
Coronary Artery Disease	53 (52.5)

Myocardial Infarction	18 (17.8)
Hypertension	12 (11.9)
Acute Coronary Syndrome	7 (6.9)
Diabetes Mellitus	5 (5)
Pulmonary Hypertension	2 (2)
Atrial Fibrillation	1 (1)

cms-Centimeters, kgs-Kilograms, ms-Meter per second, mg/dl-Milligram per decilitre, mmol/L-Micromoleculesper litre, g/dl-Grams per decilitre, million cells/cu.mm-million cells per cubic millimeter, F-Fahrenheit, kg/m2-Kilograms per meter square.

**TABLE 2: CLINICAL VARIABLES AND ASSOCIATION WITH GENDER AND AGE GROUPS (N=101)**

Characteristics	Age groups		P value	Gender		P value
	Age <40 (N=17)	Age >40 (N=84)		Male (N=56)	Female (N=45)	
Etiology	6 (35.3%)	70 (83.3%)	0.0001	50 (89.3%)	26 (57.8%)	0.001
Degenerative	11 (64.7%)	14 (16.6%)		6 (10.7%)	19 (42.2%)	
Rheumatic						
Symptoms		68 (80.9%)	0.139	47 (83.9%)	32 (71.1%)	0.12
Chest pain	11 (64.7%)	23 (27.4%)		13 (23.2%)	16 (35.5%)	
Palpitation	6 (35.3%)	18 (21.4%)		17 (30.3%)	6 (13.3%)	
Shortness of breath	5 (29.4%)					
Mitral	5 (29.4%)	56 (66.7%)	0.001	42 (75%)	19 (42.2%)	0.006
Normal	-	8 (9.5%)		5 (8.9%)	3 (6.7%)	
PMAC	-	2 (2.4%)		1 (1.8%)	1 (2.2%)	
S/P MVR	1 (5.9%)	2 (2.4%)		2 (3.6%)	-	
S/P CMC	5 (29.4%)	7 (8.3%)		1 (1.8%)	3 (6.7%)	
Abnormal	1 (5.9%)	1 (1.2%)		-	1 (2.2%)	
BMV						
Aortic	5 (29.4%)	23 (27.4%)	0.0001		13 (28.9%)	0.016
Normal	-	41 (48.9%)		15 (26.8%)	11 (24.4%)	
Sclerosed	1 (5.9%)	-		30 (53.5%)-	1 (2.2%)	
Abnormal	6 (35.3%)	8 (9.5%)		3 (5.3%)	11 (24.4%)	
Thickened	1 (5.9%)	2 (2.4%)		2 (3.6%)	1 (2.2%)	
Stenosed	-	1 (1.2%)		1 (1.8%)	-	
S/P AVR						
Tricuspid		75 (89.3%)	0.016	50 (89.2)	37 (82.2%)	0.39
Normal	12 (70.5%)	-		1 (1.8%)	-	
Thickened	1 (5.9%)					
Clinical spectrum	10 (58.8%)	12 (14.3%)	0.0001	3 (5.3%)	19 (42.2%)	0.001
Mitral stenosis	11 (64.7%)	57 (67.8%)	0.801	39 (69.6%)	29(64.4%)	0.580
Mitral regurgitation	1 (5.9%)	5 (5.9%)	0.474	2 (3.6%)	4 (8.9%)	0.261
Aortic stenosis	11 (64.7%)	26 (30.9%)	0.008	18 (32.1%)	19 (42.2%)	0.296
Aortic regurgitation	11 (64.7%)	23 (27.4%)	0.003	13 (23.2%)	21 (46.7%)	0.013
Tricuspid regurgitation	-	1 (1.2%)	0.651	1 (1.8%)	-	0.368
Pulmonary regurgitation						
Valve affected	6 (35.3%)	58 (69%)	0.0002	42 (75%)	22 (48.9%)	0.011
SVD	5 (29.4%)	22 (26.2%)		12 (21.4%)	15 (33.3%)	
DVD	6 (35.3%)	4 (4.7%)		2 (3.6%)	8 (17.7%)	
MVD						

PMAC-Post Mitral Anulus Calcification, MVR-Mitral Valve Replacement, CMC-Closed Mitral Commissurotomy, AVR-Aortic Valve Replacement, BMV-Balloon Mitral Valvuloplasty, SVD-Single Valve Disease, DVD-Double Valve Disease, MVD-Multi Valve Disease, S/P-Post Surgery.

**TABLE 3: CLINICAL VARIABLES AND DRUGS PRESCRIBED IN REGARD TO DIFFERENT LESIONS ENCOUNTERED (N=101)**

Variables/ Characteristics	Mitral stenosis (N=22)	Mitral regurgitation (N=68)	Aortic stenosis (N=6)	Aortic regurgitation (N=37)	Tricuspid regurgitation (N=34)
Etiology					

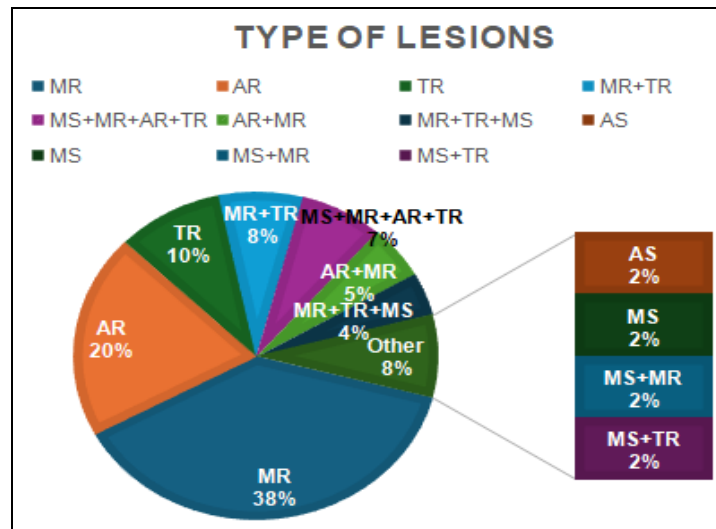
Degenerative Rheumatic	4.5	70.5	100	67.5	41.1
<b>Complaints</b>	95.4	29.4	-	32.4	58.8
Chest Pain	50	79.4	66.6	81.1	67.6
Palpitations	45.4	27.9	33.3	27	41.1
Shortness of Breath	18.1	17.6	16.6	29.7	29.4
Others	6.5	11.2	4.5	11.5	7.5
<b>Co morbidities</b>					
Coronary artery disease	-	52.9	50	43.2	29.4
Myocardial infarction	-	17.6	33.3	13.5	2.9
Acute coronary syndrome	-	4.4	16.6	13.5	20.5
Diabetes mellitus	18.1	13.2	16.6	18.9	11.7
Hypertension	9.1	1.4	-	5.4	5.9
Pulmonary hypertension	-	1.47	-	2.7	2.9
Atrial fibrillation					
<b>Drug prescribed</b>					
Penicillin	55	-	-	-	-
Aspirin + Clopidogrel	35	-	60	54.2	42.4
Furosemide+Spironolactone	35	66.6	-	-	-
Furosemide	25	-	-	-	45.4
Acenocomarol	-	53.3	66.6	40	33.3
Atorvastatin	-	53.3	53.3	34.2	30.3
Trimetazidine	-	33.3	-	42.8	36.3
Pantoprazole	-	30	-	-	-
Heparin	-	25	-	-	-
Metoprolol succinate	-	23.3	-	-	-
Furosemide					

**TABLE 4: ECHOCARDIOGRAPHY PARAMETERS OF STUDY PARTICIPANTS (N=101)**

Echocardiography Characteristics	Frequency (N=101)	Percentage/Mean ± SD
<b>Echocardiography M mode measurements</b>		
Aortic root (cm)	87	2.50 ± 0.398
Inter ventricular septum in diastole (cm)	87	0.9966 ± 0.140
Left ventricular end diastolic internal diameter (cm)	87	4.79 ± 0.83
Left ventricular posterior wall in diastole (cm)	87	1.02 ± 0.32
End diastole volume (ml)	87	113.25 ± 48.75
Ejection fraction (%)	88	51.38 ± 13.458
Left atrium (cm)	86	3.82 ± 0.777
Inter ventricular septum in systole (cm)	87	1.31 ± 0.389
Left ventricular end systolic internal diameter (cm)	87	3.52 ± 0.983
Left ventricular posterior wall in systole (cm)	87	1.28 ± 0.182
End systole volume (ml)	87	58.02 ± 34.56
Fractional shortening (%)	87	26.05 ± 6.76
<b>Echocardiography morphology</b>		
<b>Mitral</b>		
Normal	61	69.3
Post mitral annulus calcification	8	9.1
S/P mitral valve replacement	2	2.3
S/P closed mitral commissurotomy	3	3.4
Abnormal	12	13.6
BMV	1	1.1
<b>Aortic</b>		
Normal	28	31.8
Sclerosed	41	46.6
Abnormal	1	1.1
Thickened	14	15.9
Stenosed	3	3.4
S/P aortic valve replacement	1	1.1

<b>Pulmonary</b>		
Normal	87	98.9
<b>Tricuspid</b>		
Normal	87	98.9
Thickened	1	1.1
<b>Inter atrial septum</b>		
Intact	86	97.7
Atrial septal defect	2	2.3
<b>Inter ventricular septum</b>		
Intact	88	100
<b>Left atrium</b>		
Normal	59	65.6
Dilated	31	34.4
<b>Left ventricle</b>		
Normal	68	76.4
Dilated	21	23.6
<b>Right atrium</b>		
Normal	72	80.0
Dilated	18	20.0
<b>Right ventricle</b>		
Normal	78	88.6
Dilated	10	11.4
<b>Pulmonary artery</b>		
Normal	88	100
<b>Aorta</b>		
Normal	88	100
<b>Echocardiography doppler measurements</b>		
Mitral valve (m/sec)	88	2.94 ± 5.11
Aortic value (m/sec)	88	4.61 ± 13.56
Pulmonary valve (m/sec)	88	1.03 ± 0.253
Tricuspid valve (m/sec)	36	11.87 ± 49.571

Echocardiography-Echocardiography cardiogram, m/sec-Meter per second, S/P-Post Surgery, cm-Centimeter, BMV-Balloon Mitral Valvuloplasty.



**FIG. 1: VARIOUS TYPES OF LESIONS REPORTED.** MS-Mitral Stenosis, MR- Mitral Regurgitation, AS-Aortic Stenosis, AR-Aortic Regurgitation, TS-Tricuspid Stenosis, TR- Tricuspid Regurgitation, PR-Pulmonary Regurgitation.

**DISCUSSION:** This study portrays the frequency and clinical spectrum of valvular heart disease in patients visiting a tertiary care hospital in Tamil Nadu. Inadequate research reports on the VHD spectrum and its associated comorbidities in the

South Indian population led to the conceptualization of this study. Most of the studies on VHD in India are restricted to patients with RHD. To our knowledge, this is the first study to

assess the clinical spectrum of VHD in a tertiary care hospital in Tamil Nadu.

**Age and Gender:** The overall analysis of the study showed that there is no significant difference among genders. The VHD patients had a mean age of 55.4 years, suggesting an increased prevalence with age. A retrospective study by Clovis Nkoke *et al* that was conducted VHD patients between July 2016 and November 2018 reported a mean age of 54.7 years<sup>10</sup>. Shu C *et. al* 2015 conducted an epidemiological survey on VHD patients in a Chinese population and reported a mean age of 64.2 years<sup>11</sup>. These literatures project similar results with regard to the mean age range of VHD patients worldwide.

**Clinical Variables:** Etiological analysis showed that more than 75% of our study population was categorized under degenerative origin. Ana Fatima Esteves *et al* conducted a population-based study on VHD patients in Portugal between January 2014 and October 2015, which showed that 67.9% had a degenerative etiology, which was higher compared to rheumatic etiology (8.7%)<sup>12</sup>. In contrast, Karen Sliwa *et al* conducted the Heart of Soweto Study among VHD patients in South Africa and reported that rheumatic etiology was 70% more common than degenerative etiology<sup>13</sup>. This contradiction may be due to racial and geographical differences. The Heart of Soweto Study population had a mean age of 43 years compared to 55.4 years in our study. This could be the other main reason for increased frequency of rheumatic etiology as scientific literature states that degenerative VHD is more prevalent among the elderly people whereas RHD affects mostly younger population. Further analysis of the clinical variables in our study showed that the etiology of degenerative and rheumatic causes showed a significance in both age (<40 and >40) and gender (P<0.001) groups implying the crucial role of age and gender in the etiology of VHD.

Among clinical symptoms, it was seen that more than 75% of our patients experienced chest pain and more than 25% experienced palpitations in at the time of admission. Similar results have been noted in an Indian study by Prakash R Ghogale *et al* where chest pain (65.2%), palpitation (38.4%) and shortness of breath (32.1%) were reported as

the most common symptoms<sup>14</sup>. Coronary Artery Disease accounted for more than 50% of comorbidities among our study participants. Emren *et al* conducted a retrospective study to analyze the prevalence of CAD among VHD population and showed that majority of patients with VHD (57.7%) also had CAD<sup>15</sup>. Therefore, CAD is a predominant comorbidity in VHD patients.

The pattern of valve dysfunction in our VHD population was mitral and aortic followed by tricuspid. Similar patterns have been identified from literature. Ana Fatima Esteves *et al* and Prakash R Ghogale *et al* also reported valve dysfunction in mitral, aortic and tricuspid valves<sup>12, 14</sup>. With regard to valve lesions in our study, the most common valvular abnormality was MR and AR affecting more than 65% of study population. Abago balaka *et al* conducted a retrospective cross sectional, multicentric study among VHD patients that showed that more than 55% of patients had MR and AR<sup>16</sup>. Other similar studies in the Western and Central African population and community-based studies from Netherlands reported that more than 50% of the patients had either MR or AR<sup>17, 18</sup>.

Ana Fatima Esteves *et al* and Sean Coffey *et al* reported 35% of TR in their study populations<sup>19</sup>. These studies indicate that MR and AR followed by TR are the most common valve abnormalities worldwide. Certain studies have reported MS and AS in very small proportion of VHD patients. Emren *et al* (2014) and Abago balaka *et al* (2015) reported differences in MS and AS prevalence. The probable reasons are discrepancies in etiology and sampling errors. Other lesions such as pulmonary lesions and TS were found to be very rare among VHD patients. Single valve disease was seen in 63.4% of our VHD population. Abago balaka *et al* and Nkoke *et al* conducted multicentric studies in VHD populations and reported single valve disease in 77.1% and 75%, respectively. The association of age groups of <40 and >40 years and gender were found to be statistically significant with respect to single, double, and multiple valvular disease.

The most common combination of valve lesion involvement is MR + AR (4.9%). Prakash R Ghogale *et al* and Melvin DB *et al* conducted a computerized analysis in VHD population proving that MR + AR is the most common combination in



valve lesion involvement<sup>20</sup>. According to Essop *et al* 2005, MS + MR was the most common combination of valve lesions with a frequency of 40%. This variation can be explained by sampling errors and etiological differences among study participants<sup>21</sup>. Age groups and gender were found to be statistically significant in patients with mitral stenosis, aortic regurgitation, and tricuspid regurgitation. The most commonly prescribed drugs include aspirin, clopidogrel, furosemide, atorvastatin, metoprolol succinate, and trimetazidine. These drugs fall under the guidelines of the American Heart Association/American College of Cardiology. The frequency of participants undergoing surgical intervention was found to be very low. The common surgical interventions reported were mitral valve replacement and balloon valvuloplasty. Scientific studies estimate balloon valvuloplasty to be a minor intervention with very little risks involved. Most of the study participants did not prefer surgical intervention owing to their poor socio-economic status.

VHD has persisted as a silent but prevalent disease in recent decades. The older age group and those with comorbidities are at a higher risk of degenerative heart disease. The current study was carried out to understand the frequency and clinical spectrum of VHD, to improve therapeutic options and to aid in early diagnosis and management for better prognosis and quality of life.

**Limitations:** The study is limited by its shorter duration and relatively smaller sample size. This study was carried out in a single tertiary care hospital and does not represent VHD cases in the entire community. Outcome measures such as hospital readmission and mortality rates were not evaluated. Future research should be aimed at understanding the pharmacological effectiveness of therapeutic regimens and surgical interventions on VHD patients.

**CONCLUSION:** The major etiology for the occurrence of VHD in the study population was found to be degenerative heart disease. Severity of disease was higher in patients with rheumatic heart disease. The most commonly reported lesions were of the mitral and aortic valves followed by tricuspid within the regurgitation category and mitral

followed by aortic in the stenosis category. Surgical interventions such as mitral valve replacement and balloon aortic valvuloplasty were carried out in only 1.8% of study participants. Although studies have shown that surgical interventions have beneficial outcomes, a considerable proportion of our study population did not prefer surgical or interventional procedures due to limited financial resources. Thus, greater efforts are required to ensure availability and affordability of these procedures by the country for improving prognosis in VHD patients.

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