Clinical and Experimental Dermatology and Therapies

OPEN ACCESS

Alzendani AS, et al. Clin Exp Dermatol Ther 8: 213.

www.doi.org/10.29011/2575-8268.100213

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Research Article

# Prevalence and Risk Factors of Rheumatic Heart Disease and its Relation to Skin in Dhamar City, Yemen

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**Citation:** Alzendani AS, Al-Najjar AM, Alhaj AA, Al-Hothi MA, Jayed D (2023) Prevalence and Risk Factors of Rheumatic Heart Disease and its Relation to Skin in Dhamar City, Yemen. Clin Exp Dermatol Ther 8: 213. DOI:10.29011/2575-8268.100213

**Received Date:** 23 July 2023; **Accepted Date:** 01 September 2023; **Published Date:** 05 September 2023

## Abstract

**Objective:** To determine the pattern of rheumatic heart disease in Dhamar city and its frequency distribution, severity and secondary prophylaxis. **Methods:** We conducted this research in a sole regional echocardiographic department for Dhamar city at a referral hospital located in Dhamar city, from August 2020 to January 2021. It was a retrospective study focused on echocardiographic findings in 100 patients affected by rheumatic heart disease. **Results:** Out of 100 patients, 27 (27%) were male and 73 (73%) were female (age range, 10-70 years; mean age, 30-40 years). Heart valve damage was distributed among patients as follow: mitral 72 (72%), aorta 9 (9%) and both valves 12 (12%); prosthetic valve 2(2%); tricuspid 2(2%); and mitral with tricuspid 3(3 %); mitral regurgitation 26 (26%), mitral stenosis 15 (15%), aortic regurgitation 6 (6%) and aortic stenosis 3 (3%). Adolescents, young adults and adults had regurgitation. Stenosis and multiple valve lesions predominated in adults and old. In the face of used secondary prophylaxis, the patient still suffering of rheumatic heart disease. **Conclusion:** Rheumatic heart disease takes an aggressive course in Dhamar city. Children, adolescents and young adults of both genders are the victims. Complications appear early with scanty opportunity to reach advanced age. There is a little chance for palliative treatment.

**Keywords:** Rheumatic heart disease; Prevalence; Yemen

## Introduction

Acute rheumatic fever and rheumatic heart disease thought to result from an autoimmune response, but the exact pathogenesis remains unclear. Although rheumatic heart disease was the leading cause of death 100 years ago in people aged 5-20 years in the United States, incidence of this disease has decreased in developed countries, and the mortality rate has dropped to just above 0% since the 1960s. Worldwide, rheumatic heart disease remains a major health problem. Rheumatic heart disease estimated to occur in 5-30 million children and young adults; 90,000 individuals die from this disease each year. The mortality rate from this disease remains 1-10%. A comprehensive resource provided by the World Health Organization (WHO) addresses the diagnosis and treatment [1].

Rheumatic fever and rheumatic heart disease are major causes of death and disability in the inhabitants of developing countries. Decline in its prevalence is due to the identification and eradication of the causative organism, improved socioeconomic conditions, and elimination of overcrowding. In Arab and Asian countries, rheumatic heart disease progresses rapidly, often requiring surgical intervention before patients are 20 years. Few studies of rheumatic fever and rheumatic heart disease have been conducted in Yemen, although the importance of rheumatic heart disease has been noted [2].

Acute rheumatic heart disease often produces a pancarditis characterized by endocarditis, myocarditis, and pericarditis. Endocarditis manifested as valve insufficiency. The mitral valve is most commonly and severely affected (65-70% of Patients), and the aortic valve is second in frequency (25%). The tricuspid valve is deformed in only 10% of patients and is usually associated with mitral and aortic lesions. The pulmonary valve is rarely affected. Severe valve insufficiency during the acute phase may result in congestive heart failure and even death (1% of patients). Whether myocardial dysfunction during acute rheumatic fever is primarily related to myocarditis or is secondary to congestive heart failure from severe valve insufficiency is not known. Pericarditis, when present, rarely affects cardiac function or results in constrictive pericarditis.

Rheumatic fever thought to result from an inflammatory autoimmune response. Rheumatic fever only develops in children and adolescents following group A beta- hemolytic streptococcal pharyngitis, and only streptococcal infections of the pharynx initiate or reactivate rheumatic fever. Genetic studies show strong correlation between progression to rheumatic heart disease and human leukocyte antigen (HLA)-DR class II alleles and the inflammatory protein- encoding genes MBL2 and TNFA [3].

A diagnosis of rheumatic heart disease made after confirming antecedent rheumatic fever. The modified Jones criteria (revised in 1992) provide guidelines for the diagnosis of rheumatic fever [4].

The Jones criteria require the presence of 2 major or 1 major and 2 minor criteria along with evidence for recent streptococcal infection for the diagnosis of rheumatic fever.

Regarding to the relation between rheumatic heart disease and skin, rheumatic heart disease is a major complication of streptococcal infections, and streptococcal species can cause skin infections [5]. Furthermore, recent reports have described streptococcal skin infections that resulted in an acute rheumatic fever and rheumatic heart disease [6,7]. In developing countries, skin ulcers may present as a key focus of Group A Strep in the aetiology of rheumatic heart disease [8].

Jones criteria that are used in the diagnosis of acute rheumatic fever include erythema marginatum and subcutaneous nodules. Erythema marginatum is macular rash that is generally seen on the trunk and extremities and it is commonly associated with acute rheumatic fever [9]. Subcutaneous nodules are rare granulomatous lesions that are formed as a result of the delayed hypersensitivity reaction to the Group A Strep antigen [8].

### Objectives

This study aimed to:

1. To detect the prevalence of Rheumatic Heart Disease in Dhamar city.
2. To identify the risk factor for Rheumatic Heart Disease.
3. To identify the relation between rheumatic heart disease and skin.

### Literature Review

#### Rheumatic Heart Disease

Rheumatic Heart Disease (RHD) is damage to one or more heart valves that remains after an episode of Acute Rheumatic Fever (ARF) is resolved. It is caused by an episode or recurrent episodes of ARF, where the heart has become inflamed [10].

### Acute Rheumatic Fever

Acute rheumatic fever and its sequel, rheumatic heart disease, cause significant morbidity and mortality in developing countries including Yemen [11].

Acute Rheumatic Fever (ARF) is a multisystem disease resulting from an autoimmune reaction to infection with group A streptococcus. Although many parts of the body may be affected, almost all of the manifestations resolve completely. The major exception is cardiac valvular damage Rheumatic Heart Disease

[RHD], which may persist after the other features have disappeared [12].

**Prevention of Recurrent Rheumatic Fever**

Improvement in socioeconomic conditions and public health are critical to reducing bouts of rheumatic fever [13].

The initial episode of rheumatic fever can usually be prevented by early treatment of streptococcal. The preferred method of prophylaxis is with benzathine penicillin G, 1. 2 million units intramuscularly every 4 weeks. Oral penicillin (200, 000-250, 000 units twice daily) is less reliable.

If the patient is allergic to penicillin, sulfadiazine (or sulfisoxazole), 1g daily, or erythromycin, 250 mg orally twice daily, may be substituted. The macrolide azithromycinis similarly effective against group A streptococcal infection. If the patient has not had an immediate hypersensitivity (anaphylactic-type) reaction to penicillin, then cephalosporin may also be used pharyngitis [14].

Isolated mitral stenosis accounts for about 25% of all cases, and an additional 40%have mixed mitral stenosis and regurgitation [15].

**Principal causes of valve disease**

Valve regurgitation

- Congenital
- Acute rheumatic carditis
- Chronic rheumatic carditis
- Infective endocarditis Cardiac failure
- Syphilitic aortitis
- Traumatic valve rupture
- Senile degeneration
- Damage to chordae and papillary muscles Valve stenosis
- Congenital
- Rheumatic carditis
- Senile degeneration [16]

## Mitral Stenosis

Mitral stenosis is almost always rheumatic in origin, although in older people it can be caused by heavy calcification of the mitral valve. There is also a rare form of congenital mitral stenosis [17].

**Causes**

Most patients with mitral stenosis are usually presumed to have underlying rheumatic heart disease, though a history of rheumatic fever is usually noted in only about one-third. Rheumatic mitral stenosis results in thickening of the leaflets, fusion of the mitral commissures, retraction, thickening and fusion of the chordae, and calcium deposition in the valve.

Mitral valve obstruction may also develop in patients who have had mitral valve repair with a mitral annular ring that is too small, or in patients who have had a surgical valve replacement (prosthetic valve-patient mismatch) [18].

**Clinical Picture**

Patients with mitral stenosis usually remain asymptomatic until the valve area is reduced to about one-third its normal size of 4 to 5 cm2. Then the symptoms typical of left-sided failure-dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea develop. As the disease progresses and right ventricular failure occurs, ascites and edema are common. Hemoptysis, which is common in mitral stenosis but uncommon in other causes of left atrial hypertension, develops when high LA pressure ruptures the anastomoses of small bronchial veins. In some cases, a large LA may impinge on the left recurrent laryngeal nerve and cause hoarseness (Ortner syndrome) or may impinge on the esophagus and cause dysphagia.

The physical signs of mitral stenosis are often found before symptoms develop and their recognition is of particular importance in pregnancy. The forces that open and close the mitral valve increase as left atrial pressure rises. The first heart sound (S1) is therefore loud and can be palpable (tapping apex beat). An opening snap may be audible and moves closer to the second sound (S2) as the stenosis becomes more severe and left atrial pressure rises. However, the first heart sound and opening snap may be inaudible if the valve is heavily calcified. Turbulent flow produces the characteristic low-pitched middiastolic murmur and sometimes a thrill. The murmur is accentuated by exercise and during atrial systole (pre-systolic accentuation). Early in the disease, a pre-systolic murmur may be the only auscultatory abnormality, but in patients with symptoms, the murmur extends from the opening snap to the first heart sound. Coexisting mitral regurgitation causes pansystolic murmur that radiates towards the axilla. Pulmonary hypertension may ultimately lead to right ventricular hypertrophy and dilatation with secondary tricuspid regurgitation, which causes a systolic murmur and giant ‘v waves’ in the venous pulse [19].

## Diagnosis

**Electrocardiography** (ECG), Atrial fibrillation is common, but left atrium (LA) abnormality is generally present on the ECG if the patient is in sinus rhythm. If pulmonary hypertension has developed, there is often evidence of Right Ventricular Hypertrophy (RVH).

**Chest radiograph**, LA enlargement produces straightening of the left heart border and a double density at the right heart border because of the combined silhouettes of the RA and LA. Pulmonary venous hypertension produces increased vascularity. Kerley B lines, which represent thickening of the pulmonary septa secondary to chronic venous engorgement, may also be seen. The echocardiogram produces excellent images of the mitral valve and is the most important diagnostic tool in confirming the diagnosis.

**Transthoracic echocardiography** or, if necessary, transesophageal **echocardiography** makes the diagnosis in nearly 100% of cases and accurately assesses severity. Mitral stenosis, similar to aortic stenosis, can be quantified by assessing the transvalvular gradient with the modified Bernoulli principle. The stenosis is considered severe when the area is smaller than 1. 5 cm2 and very severe when valve area is smaller than 1. 0 $cm^2$ [20].

Doppler, Pressure gradient across mitral valve, Pulmonary artery pressure, and Left ventricular function [21].

**Management**

Mitral stenosis progresses slowly and medical therapy is used before serious adverse events warrant surgical intervention. However, medical therapy has not been shown to affect outcome.

Exercise-induced tachycardia can lead to decompensation; in patients with mild symptoms, β-blockers or rate-limiting calcium antagonist scan be used.

Intermittent pulmonary oedema controlled by diuretics. AF is common, although not invariable, in mitral stenosis. This poses the dual threat of uncontrolled rate and emboli. The former can be controlled with β-blockade or calcium antagonists. Digoxin may also be used but is not as helpful for controlling exercise induced tachycardia.

Anticoagulation is initially with heparin; warfarin is then required, whether AF is paroxysmal or permanent. Anti-thrombin agents are now being used. When symptoms worsen (NYHA class III–IV) and the echo appearance is consistent with severe mitral stenosis (valve area < 1 cm2), mechanical intervention should be considered. Patients may be suitable for percutaneous mitral valvuloplasty and this should always be discussed with a cardiologist. Alternatively, surgery with mitral valvuloplasty or mitral valve replacement can be performed [22].

**Mitral Regurgitation**

The mitral valve is composed of the mitral annulus, the leaflets, the chordate tendineae, and the papillary muscles. Abnormalities in any of these structures may lead to mitral regurgitation [23].

Causes of mitral regurgitation

- Mitral valve prolapses
- Dilatation of the left ventricle and mitral valve ring (e. g. coronary artery disease, cardiomyopathy)
- Damage to valve cusps and chordae (e. g. rheumatic heart disease. endocarditis)
- Ischemia or infarction of the papillary muscle
- Myocardial infarction [24].

**Clinical Features**

Symptoms of mitral regurgitation occur late in the disease and comprise fatigue and breathlessness. Examination findings include a forceful, displaced apex beat with apan-systolic murmur that does not vary in intensity throughout its duration. Radiation is typically to the axilla but it may be to the base. The presence of a third heart sound indicates volume overload [25].

**Investigation**

Echocardiographic information demonstrating the underlying pathologic process (rheumatic, prolapse, flail leaflet, cardiomyopathy), LV size and function, LA size, PA pressure, and Right Ventriclei (RV) function can be invaluable in planning treatment as well as in recognizing associated lesions.

An ECG should be performed and commonly shows AF, as a consequence of atrial dilatation. Doppler techniques provide qualitative and semi quantitative estimates of the severity of mitral regurgitation. Cardiac catheterization is commonly used for assessment of right and left heart pressures. However, it can also be used as an additional tool to assess the degree of regurgitation.

Chest X-ray appear Enlarged left atrium, Enlarged left ventricle, Pulmonary venous congestion and Pulmonary oedema (if acute) [26].

**Treatment**

Acute mitral regurgitation. In acute severe mitral regurgitation, surgical intervention is urgent. Other measures should be aimed at optimizing the patient for anesthesia and avoiding organ hypoperfusion. If BP is normal, vasodilatation can aid forward cardiac output and reduce pulmonary congestion via the regurgitating valve. This can be achieved by the use of IV sodium nitroprusside. If BP falls, an alternative to nitroprusside alone is a combination with dobutamine or intra-aortic balloon counter pulsation.

Chronic mitral regurgitation. If mitral regurgitation has resulted from left ventricle (LV) dilatation (functional mitral

regurgitation), treatment with angiotensine converting inhibitors (ACE inhibitors) and/or β-blockers is recommended. There is no indication for these drugs where LV function is normal [27].

**Aortic Stenosis**

There are two common clinical scenarios in which aortic stenosis is prevalent. The first is due to a congenitally abnormal unicuspid or bicuspid valve, rather than tricuspid.

Symptoms occur in young or adolescent individuals if the stenosis is severe, but more often emerge at age 50–65years when calcification and degeneration of the valve becomes manifest. A second group develops what has traditionally been called degenerative or calcific aortic stenosis, which is thought to be related to calcium deposition due to processes similar to what occurs in atherosclerotic vascular disease.

Approximately 25% of patients over age 65 years. Aortic stenosis has become the most common surgical valve lesion in developed countries, and many patients are elderly. The risk factors include hypertension, hyper cholesterolemia, and smoking. Hypertrophic obstructive cardiomyopathy (HOCM) may also coexist with valvular aortic stenosis [28].

**Causes**

Infants, children, adolescents

- Congenital aortic stenosis
- Congenital subvalvular aortic stenosis
- Congenital supravalvular aortic stenosis Young adults to middle-aged
- Calcification and fibrosis of congenitally bicuspid aortic valve
- Rheumatic aortic stenosis Middle-aged to elderly
- Senile degenerative aortic stenosis
- Calcification of bicuspid valve
- Rheumatic aortic stenosis [29].

**Clinical Features**

Exertional chest pain can result from a mismatch between myocardial perfusion and hypertrophy or from coexisting CAD. Syncope is generally exertional and can be heralded by episodes of exertional pre-syncope. LV dysfunction, which tends to be predominantly diastolic in nature, can lead to breathlessness. The onset of systolic dysfunction and LV dilatation carries a particularly poor prognosis. In such circumstances, the pressure generated within the ventricle may be reduced, leading to an under estimate of the valve stenosis if measured by gradient across the valve.

Examination reveals a slow-rising pulse, a narrow pulse pressure (generally a used by low systolic pressure and maintained diastolic pressure) and a pressure-overloaded apex beat. There is typically a crescendo–decrescendo ejection systolic murmur, which radiates to the carotid arteries [30].

**Investigation**

The electrocardiogram (ECG) in patients with aortic stenosis usually shows Left ventricular hypertrophy (LVH). In some cases of even severe aortic stenosis, however, LVH is absent on the ECG, possibly because of the lack of LV dilation. Left atrial (LA) abnormality is common because the stiff LV increases LA after load and causes the LA to dilate. The chest radiograph in aortic stenosis is generally nondiagnostic. The cardiac silhouette is not usually enlarged but may assume a boot-shaped configuration. In advanced cases, there may be signs of cardiomegaly and pulmonary congestion; aortic valve calcification may be seen in the lateral view [31].

**Treatment**

Medical management. The aim is to control cardiovascular risk factors. Treatment of hypertension can be difficult, as vasodilators can worsen the gradient across the valve. Surgical management. Surgery is the definitive treatment. Patients should be operated on before symptoms and with good LV systolic function.

Percutaneous Aortic Valve Replacement (PAVR). This treatment is generally reserved for patients with severe co-morbidities that make surgical aortic valve replacement too high-risk. The short-term results are excellent but the long-term results are as yet unclear. These patients are assessed and treated as appropriate by a multi-disciplinary team, including an interventional cardiologist, cardiac surgeon and cardiac anaesthetist at a tertiary centre [32].

**Aortic Regurgitation**

Regurgitation of blood through the aortic value causes the LV to dilate as cardiac output increases to maintain the demands of the circulation.

**Congenital**

- Bicuspid valve or disproportionate cusps

**Acquired**

- Rheumatic disease
- Infective endocarditis
- Trauma
- Causes of aortic dilatation:
- Marfans syndrome

- Aneurysm
- Aortic dissection
- Syphilis
- Ankylosing spondylitis [33].

**Clinical Picture**

Acute aortic regurgitation. Patients may present with sudden breathlessness, chest pain and cardiogenic shock. Tachycardia is common. The murmur can be shorter in acute severe aortic regurgitation, as there is rapid pressure equalization between ventricle and aorta in diastole. The diagnosis is a priority, as is exclusion of aortic dissection, even if the patient does not complain of chest pain.

l Chronic aortic regurgitation. There is a wide difference between systolic and diastolic BP (pulse pressure). The arterial pulse is bounding and collapsing. The apex beat is typically displaced owing to LV dilatation and it may feel hyperdynamic. The murmur of aortic regurgitation is diastolic and decrescendo in nature, occurring immediately after the second heart sound. A systolic flow murmur is commonly heard due to the need for increased forward flow through the aortic valve to compensate for the regurgitation [34].

**Investigation**

Electrocardiography In patients with chronic severe AR, the ECG signs of left ventricular hypertrophy become manifest in addition, these patients frequently exhibit ST-segment depression and T-wave inversion leads 1aVL and V6 ("LV strain"). Left-axis deviation and/or QRS prolongation denote diffuse myocardial disease generally associated with patchy fibrosis, and usually a poor prognosis. Echocardiogram L V size is increased in chronic AR and systolic function is normal or even supernormal until myocardial contractility declines, as signalled by a decrease in EF or increase in the end-systolic Dimension. Chest X-Ray In chronic severe AR, the apex is displaced downward and to the left in the frontal projection. In the left anterior oblique and lateral projections, the L V is displaced posteriorly and encroaches on the spine [35].

**Treatment**

Due to increased afterload, therapy with vasodilators such as nitroprussideand hydralazine may improve haemodynamic function. ACE inhibitors are less effective in this context. Surgery with mechanical prosthesesor tissue valve replacement is performed before symptoms occur when themyocardium is failing (i. e. LV ejection fraction < 50%). A repair of the aortic valve may occasionally be surgically possible [36].

**Tricuspid Stenosis**

Tricuspid stenosis is usually rheumatic in origin. Tricuspid disease occurs in fewerthan5% of patients with rheumatic heart disease and then nearly always occurs in association with mitral and aortic valve disease. Tricuspid stenosis and regurgitation may also occur in the carcinoid syndrome [37].

**Clinical Features and Investigations:**

Although the symptoms of mitral and aortic valve disease predominate, tricuspid stenosis may cause symptoms of right heart failure, including hepatic discomfort and peripheral oedema.

The main clinical feature is a raised JVP with a prominent a wave, and a slow y descent due to the loss of normal rapid right ventricular filling. There is also a mid-diastolic murmur, best heard at the lower left or right sterna edge. This is generally higher- pitched than the murmur of mitral stenosis and is increased by inspiration. Right heart failure causes hepatomegaly with presystolic pulsation (large a wave), ascites and peripheral oedema. The diagnosis can be confirmed by Doppler echocardiography, which shows similar appearances to those of rheumatic mitral stenosis [38].

**Management**

In patients who require surgery to other valves, either the tricuspid valve can also be replaced or treated with valvotomy. Balloon valvuloplasty can be used to treat rare cases of isolated tricuspid stenosis [39].

**Tricuspid Regurgitation**

There is almost always a minor degree e of tricuspid regurgitation on echocardiography. l Significant tricuspid regurgitation is usually secondary to right ventricular dilation of any cause, e. g. cor pulmonale Rarely, it can be organic, e. g. rheumatic heart disease. It is usually detected clinically by giant 'v' waves in the Jugular Venous Pressure (JVP), a pulsatile, palpable liver and a blowing pan-systolic murmur best heard on inspiration. Management is that of right heart failure. The occasional organic disease might require surgical replacement [40].

**Pulmonary Stenosis**

Pulmonary stenosis is a congenital disease resulting from fusion of the pulmonary valve cusps. It is usually detected and corrected during childhood, but occasionally cases are diagnosed for the first time in adulthood. Symptoms of pulmonary stenosis include angina and syncope. Occasionally, symptoms of right sided heart failure develop. During physical examination, the uncalcified valve in pulmonary stenosis produces an early systolic ejection click on opening. During inspiration, the click diminishes or disappears because increased flow into the right side of the heart during inspiration partially opens the pulmonary valve in diastole

so that systole causes less of an opening sound. The click is followed by a systolic ejection murmur that radiates to the base of the heart. If the transvalvular gradient is severe, RVH develops and produces a parasternal lift. The diagnosis of pulmonary stenosis is confirmed by echocardiography, which quantifies the transvalvular gradient and the degree of right ventricular hypertrophy and dysfunction [41].

### Pulmonary Regurgitation

Pulmonary Regurgitation (PR) may develop as a consequence of primary valve pathology annular enlargement, or their combination; after surgical treatment of RVOT obstruction in children with such disorders as Tetralogy of Fallot; or after pulmonary balloon valvotomy Carcinoid usually causes mixed pulmonary valve disease with PR and PS. Long-standing severe PA hypertension from any cause can result in dilation of the pulmonary valve ring and PR [42].

### 2 Chronic rheumatic heart disease

Chronic valvular heart disease develops in at least half of those affected by rheumatic fever with carditis. Two-thirds of cases occur in women. Some episodes of rheumatic fever pass unrecognized and it is possible to elicit a history of rheumatic fever or chorea in only about half of all patients with chronic rheumatic heart disease. The mitral valve is affected in more than 90% of cases; the aortic valve is the next most frequently involved, followed by the tricuspid and then the pulmonary valve [43].

## Subjective and Methods

### Subjective

Scanning of both gender and all age group patients referred for echocardiography whatever the cause, in three hospitals (Taiba hospital, Al-musalli hospital and cardiologist private clinic in Dhamar city.

### Study Design

This study was a cross sectional conducted between October 2020, to February 2021 to evaluate patients coming into (Taiba hospital, Al-musalli hospital, and cardiologist private clinic).

### Study Population

This study included 1325 patients coming for echocardiography evaluations, at Dhamar hospitals. The inclusions patients that had one or more heart valves lesion (anatomical damage) with certain dysfunction (mitral regurgitation, mitral stenosis, mitral prolapse, aortic regurgitation, aortic stenosis, tricuspid regurgitation) and those with trivial regurgitation, simple thickening of the heart valve without dysfunction were included in this study exclusion lesion attributed to degenerative valve disease were excluded.

### Data Collection

The data were collected by transthoracic echocardiography; m-mode and two- dimensional echocardiography were used to display the anatomical, and pathology feature of the lesion and heart dimensions; color flow doppler imaging.

## Results

Out of 100 patients, (27%) were male and (73%) were female (age range, 10-70 years; mean age, 30-40 years). Heart valves damage were distributed among patients as following: mitral (72%), aorta (9%) and both valves (12%); prosthetic valve (2%); tricuspid (2%); and mitral with tricuspid (3 %); mitral regurgitation (26%), mitral (15%), aortic regurgitation (6%) and aortic stenosis (3%) Regurgitation was more prevalence among adolescents and young adults. multiple valve lesions predominant in adults and old (Table 4.1-Table 4.5) and (Figure 4.1-Figure 4.4).

| | Frequency and percentage |
|---|---|
| **Age category/year** | |
| 10 - 25 | 34 |
| 26- 40 | 43 |
| 41-60 | 13 |
| > 60 | 10 |
| **Gender** | |
| Male | 27 |
| Female | 73 |
| Total | 100% |

**Table 4.1:** Frequency and percentage that including Age category/ year and Gender.

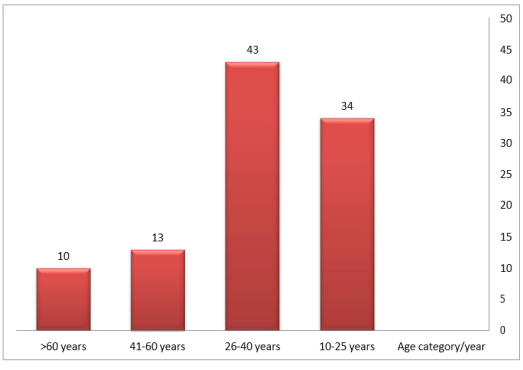


**Figure 4.1:** Relation of rheumatic heart disease with age categories/ year in this study.

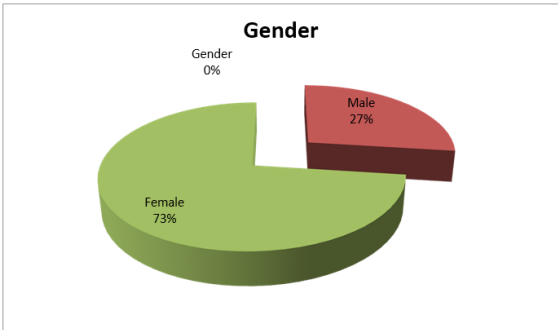


**Figure 4.2:** Relation of rheumatic heart disease with gender in this study.

| Age category | Total | Male | | Female | |
|---|---|---|---|---|---|
| | | No | % | No | % |
| 10 - 25 | 34 | 9 | 26.47 | 25 | 73.53 |
| 26- 40 | 43 | 9 | 21 | 34 | 79 |
| 41-60 | 13 | 3 | 23 | 10 | 77 |
| > 60 | 10 | 6 | 60 | 4 | 40 |
| **Total** | 100 | 27 | 27 | 73 | 73 |

**Table 4.2:** Relation of age category with gender of the rheumatic heart disease.

| Age category | Total | planes for secondary prophylaxis | |
|---|---|---|---|
| | | | |
| | | Yes | No |
| Oct-25 | 34 | 30 | 4 |
| 26- 40 | 43 | 32 | 11 |
| 41-60 | 13 | 7 | 6 |
| >60 | 10 | 2 | 8 |
| Total | 100 | 71 | 29 |

**Table 4.3:** Relation of age category with secondary prophylaxis of the rheumatic fever.

| Valve dysfunction | | Gender | | | Total |
|---|---|---|---|---|---|
| | Male | | Female | | |
| | no | % | no | % | |
| Mitral stenosis | 2 | 13.3 | 13 | 86.7 | 15 |
| Mitral regurge | 8 | 30.8 | 18 | 69.2 | 26 |
| Mitral valve prolapse | 1 | 9 | 10 | 91 | 11 |
| Aortic stenosis | 1 | 33.3 | 2 | 66.7 | 3 |
| Aortic regurge | 4 | 66.7 | 2 | 33.3 | 6 |
| MR & AR | 4 | 57.2 | 3 | 42.8 | 7 |
| Tricuspid regurge | 0 | 0 | 2 | 100 | 2 |
| MS with MR & AR | 0 | 0 | 5 | 100 | 5 |
| MR & TR | 1 | 33.3 | 2 | 66.7 | 3 |
| MVP & MR | 5 | 25 | 15 | 75 | 20 |
| Prosthetic valve | 1 | 50 | 1 | 50 | 2 |
| **Total** | 27 | | 73 | | 100 |

**Table 4.4:** The frequency distribution of valve dysfunction with gender.

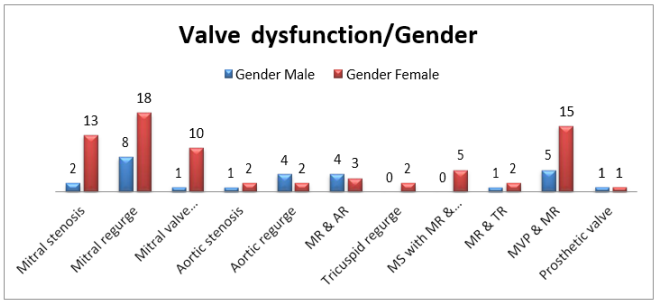


**Figure 4.3:** Relation of valve dysfunction with gender in the rheumatic heart disease.

| Valve Dysfunction / Age Categories Cross tabulation | | | | | |
|---|---|---|---|---|---|
| Valve Dysfunction | Age Categories | | | | Total |
| | 10 - 25years | 26- 40 | 41-60 | > 60 | |
| Mitral Stenosis | 3 | 5 | 5 | 2 | 15 |
| Mitral Regurge | 10 | 12 | 2 | 2 | 26 |
| Mitral Valve Prolapse | 4 | 6 | 1 | 0 | 11 |
| Aortic Stenosis | 2 | 0 | 1 | 0 | 3 |
| Aortic Regurge | 0 | 3 | 0 | 3 | 6 |
| MR + AR | 3 | 2 | 2 | 0 | 7 |
| TR | 1 | 1 | 0 | 0 | 2 |
| MS + MR + AR | 1 | 3 | 0 | 1 | 5 |
| MR + TR | 0 | 2 | 1 | 0 | 3 |
| MVP + MR | 10 | 9 | 0 | 1 | 20 |
| Prosthetic Valve | 0 | 0 | 1 | 1 | 2 |

| | | | | | |
|---|---|---|---|---|---|
| Total | 34 | 43 | 13 | 10 | 100 |

**Table 4.5:** Relation of age category with valve dysfunction of the rheumatic heart disease.

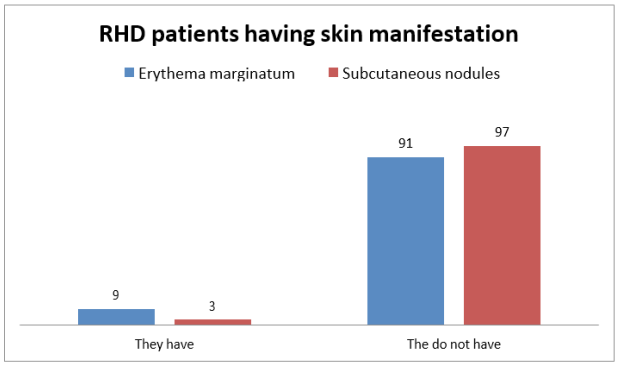


**Figure 4.4:** RHD patients having skin manifestation.

## Discussion

Using clinical and echocardiography criteria our study showed that the prevalence of RHD among cardiac patients through august 2018 to January 2019 was 7.5%. This finding was slightly lower than that reported in similar studies which done in Yemen for example in Dhamar city from January 2017 to march 2018 where the result was 11.9% out of 3612 cases referred for echocardiography. Also, in other study donein ADEN CITY from January 1999to December 2003 the result was 805 (8.5%) out of 9452 cases.

On the other hand, study was done for adult patients over 3 months form 1/5/2009 to 1/8/2009 in cardiac centre AL-Thawra general hospital, Sana'a governorate and the result was higher than our study 172 (34.4%) cases were having RHD out of 500cases referred for echocardiography

In our study done in Dhamar city showed high prevalence of about (7.5%) cases with high prevalence between 26-40 years (77%) with female predominant (73%), majority of those patient has mitral regurgitation (26%) which was more predominant in female (18%) then mixed MR+MVP was (20%) which was in female (15%),then MS (15%) which was in female (13%), MVP (11%) in female (10%) and mixed MR+AR (7%) with male predominant(4%) then AR(6%) with male predominant (4%) then MS+MR+AR(5%) which was all in female ,lastly AS(3%) with female predominant(2%).

Tow definite pattern of rheumatic heart disease are established:- One observed in developed countries The other in under developed countries the pattern in developed countries which have high socioeconomic and health care status has significantly changes in the last decade were the severe form of RHD is generally present in people in their late thirties or above manifesting itself as mitral stenosis with or without concurrent regurgitation In those countries, acute rheumatic fever diagnosed early in children and young patients and disappear by using antibiotics.

It is obvious that due to adequate preventive measures including the usage of prophylactic penicillin therapy and avoiding recurrence only few cases develop Chronic valve lesion.

On the other hand, the pattern of RHD in developing countries characterized by high incidence mitral regurgitation and mitral stenosis among people.

The virulent nature of the RHD is strongly related to number of risk factor confirmed in our study including low socioeconomic status, high prevalence of group A streptococcus, overcrowding, inadequate medical services and noncompliance to chemoprophylaxis. This suggest that in Yemen like developing countries the rheumatic fever is left untreated and lesion may progress rapidly to severe form of pure mitral regurgitation. The prevalence was also significantly higher among people living in rural area of 20hamar government than those living in city.

Repeated episode sore throat was identified in our study as predisposing factor.

In this study sore throat was not treated in most of patient. Also, in our study we found correlation between occurrence of RHD and history of arthritis.

A monthly injection Benzathine Penicillin G is the most effective and cheaper method of preventing of RHD. The definitive form of primary prevention of RF is would be streptococcal vaccine but unfortunately it is not available yet, so RF continue to have high prevalence, recurrence rate and aggressive pattern. Unfortunately, only few of them will have the chance of surgical intervention (Table 5.1 and Table 5.2).

| City | Prevalence Of RHD | Duration Of study |
|---|---|---|
| Aden | 8.5% | From/January/1999 To/ December/2003 |
| Sana'a | 34.9% | 1/5/2009 To 1/8/2009 |
| Thamar | 11.9% | From 1/1/2017 to1/3/2018 |

**Table 5.1:** prevalence of RHD in Aden – Sana'a and Thamar cities.

| Country | Prevalence of RHD | Duration |
|---|---|---|
| Oman | 8/10000 | From 1992 to 1993 |
| India | 6-46% | 2007-2008 |
| Pakistan | 22% | |
| Sudan | 3% | 2016-2017 |
| South Africa | 16/100 | |

**Table 5.2:** prevalence of RHD in neighboring countries.

Concerning the relation between RHD and skin, in this study 9% of the patients had erythema marginatum, and 3% of them had subcutaneous nodules. In a study that was done in Italy, they found out that erythema marginatum was in 11.4% of the participants, and 4.6% of them had subcutaneous nodules [44].

## Conclusions and Recommendations

### Conclusion

Rheumatic heart disease more prevalence rate in Dhamar governorate rural areas which is similar to that of developing countries suffering from deterioration of socioeconomic and hygienic condition, children, adolescents and young adults are the principle victims with little chance of reaching an advanced age.

Sadly, RHD Diagnosis in Dhamar government usually established at an advanced stage of the disease when severe valve lesions become symptomatic and significant intervention is indicated reducing the incidence of RHD should be through the implementation appropriate policies targeting at risk population and focus on awareness and early detection.

### Recommendation

According to our study the main reasons of high incidence of rheumatic heart disease in Dhamar city are the absence of specific program for prevention and control of RHD the lack of government decisions to adopt such a program and the inadequate use of penicillin,the monthly use of Benzathine penicillin G is the most effective and cheaper method of preventing of rheumatic heart disease; a specific program for the prevention and control of rheumatic fever and rheumatic heart disease is strongly recommended Consisting of The intra muscular administration of weight adjusted penzathine penicillin or daily Aral penicillin V for entire duration and follow up. Also, all patients with episode of sore throat should receive primary prophylaxis and refer to echocardiography.

The proper evaluation is by Echocardiography screening allow diagnosis of RHD at an earlier stage across continuous spectrum compered to cardiac auscultation. Also, the effective measures to reduce the global burden of RHD represent ongoing challenge involving reduction in overcrowding and improving hygiene increasing public awareness and facilitating access to health caring on awareness early detection.

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