Atrial Fibrillation in Rheumatic Heart Disease - Clinical and Echocardiographic study

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A thesis Submitted in partial fulfillment for the requirements of the Degree of Clinical MD in Medicine, April, 2004

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Dedication

To

My parents
ACKNOWLEDGMENT

I am mostly appreciate the continuous encouragement of my supervisor Prof. Siddig Ahmed Ismail, Professor emeritus of Internal Medicine, Faculty of Medicine, University of Khartoum, I am indebted to his close supervision and meticulous revision of my thesis through all the steps of this study. His advice and continuous guidance are highly appreciated.

I would like to express thank to Dr. Mohamed Saeed Elkhalifa, for his help.

My appreciation goes to my family for their continuous support and encouragement.

My thanks extend to my colleagues, for their encouragement.

My thanks to Mr. Hassan Ali who analyzed the data and to Widad for typing.
## ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Meaning</th>
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<tr>
<td>RF</td>
<td>Rheumatic fever</td>
</tr>
<tr>
<td>RHD</td>
<td>Rheumatic heart disease</td>
</tr>
<tr>
<td>Echo</td>
<td>Echocardiography</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>MS</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>MVA</td>
<td>Mitral valve area</td>
</tr>
<tr>
<td>AR</td>
<td>Aortic regurgitation</td>
</tr>
<tr>
<td>AF</td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>TTE</td>
<td>Transthoracic echocardiography</td>
</tr>
<tr>
<td>TEE</td>
<td>Trans oesophageal echocardiography</td>
</tr>
<tr>
<td>LA thrombus</td>
<td>Left atrial thrombus</td>
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ABSTRACT

Background: Rheumatic heart disease still is a major health problem in developing countries. One of its serious complication is atrial fibrillation, it has high incidence of morbidity and mortality. Its treatment is another hurdle specially in countries such as Sudan. It has been noticed by some workers that AF occurs in relatively young Sudanese patients with RHD and that many young patients and children develop congestive cardiac failure in the absence of AF hence the objective is to find out how left atrial size plays a role in the development of rheumatic atrial fibrillation and to report the echocardiographic findings in these patients.

Methods: This was descriptive prospective study conducted in 200 Sudanese patients with rheumatic heart disease and atrial fibrillation in Elshaab and Ahmed Gasim Teaching Hospitals in the period from June 2003 to February 2004. Cases were seen in the referred clinic as well as admitted cases history, physical examination, ECG, and transthoracic echocardiographic were used in the study.

Result: In our study we found that, the ratio of female to male was 1.7:1, the frequency of occurrence of atrial fibrillation due
to rheumatic heart disease was high in older patients. Those patient who were above 30 years or more had greater chance to develop AF.

There is correlation between the development of atrial fibrillation and left atrial size the greater the left atrial size the greater the chance to develop AF in patients with R.H.D. Patient with atrial fibrillation and had normal left atrial size were found to be 2.5% in this study. All patients had mitral valve involvement, 183(91.5%) had mitral valve stenosis, 115 patients had severe mitral stenosis, 71 patients had aortic valve lesion, left atrial thrombus was found in 18 patients (9%).

**Conclusion:** Rheumatic heart disease has high predilection to occur in patients, whose ages were above 30 years. There is correlation between its occurrence and left atrial size, the degree of mitral valve stenosis had another role for AF development.
연구 주제: QDZ

연구 대상: {사용자 지정}

연구 목표: {사용자 지정}

연구 방법: {사용자 지정}

연구 결과: {사용자 지정}

주요 발견: {사용자 지정}

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INTRODUCTION AND LITERATURE REVIEW

1.1. Prevalence of rheumatic fever (RF) and rheumatic heart disease (RHD) in developing countries:

The prevalence of rheumatic heart disease among school children aged 5-14 years varies from 1.36 to 6.4/1000 in developing countries.(1)

In Yemen(2) Rheumatic fever was estimated to be 3.6 per 1000. Which is higher than that reported from neighbouring countries.

In Kenya prevalence in sub-urban area is 22/1000 and in urban area is 4/1000.

The prevalence of rheumatic heart disease is now recognized to be very high, particularly among children and young adults.

Between 1984 and 1995 the rate varied from 1 to 5.4 per 1000. By comparison, in western countries the prevalence of RHD in children aged between 5-15 years is below 0.5 per 1000, and for rheumatic fever it is below 1 per 1000(3).

Resurgence of R.F occurred in the U.S.A in 1980s (1).
1.2. Rheumatic fever (RF) and rheumatic heart disease (RHD) in Sudan:

Few studies were done and published in this issue.

• In 1970 Ismail S A and Gabir$^4$ studied rheumatic heart disease in Sudan. They studied the pattern of rheumatic heart disease in 200 patients with RHD in Khartoum hospital, and they observed that atrial fibrillation developed while some of these patients were below the age of twenty and that more children developed congestive heart failure, while still in sinus rhythm$^4$.

• Atiyat$^5$ studied 100 patients attending the out patient clinic and the cardiac clinic at paediatric hospital at Khartoum during the period, April 1982 to September 1983, she studied patients below 18 years. History, physical examination and laboratory investigation were done but echo was done for selected cases. The majority of patients came from Khartoum (78%) and central part of Sudan. Atiyat reported that the incidence of RHD was 25% of all cardiac cases as compared to 14% in 1945.$^5$

• Khalil SI, et al, studied the prevalence of rheumatic fever and rheumatic heart disease in Sahafa town. The study involved school children (5-15 years) of age in the period 1986-1989. The prevalence for all ages were 10 /1000 for boys and 14/1000 for
girls. The prevalence of RF was 8/1000 and prevalence of RHD was 3/1000. The prevalence rate was increased among the centre of the city inhabitant (15/1000) compared to the outskirts inhabitant (4/1000).\(^{(6)}\)

- In the Sudan, RHD is still the most frequent cause of heart disease in the 5 to 30 years age group and accounts for 36% of the total hospital admission for cardiovascular disease.\(^{(6)}\)

- In 1937, RHD formed only 5% of the total hospital admissions for cardiovascular disease in Khartoum, by, 1945, the figure had risen to 14%, in 1960 to 26% and in 1984 to 36%.\(^{(6)}\)

The lack of bio-statistical study about RF and RHD makes the estimation of its incidence in Sudan so difficult and non accurate. There is no published data about morbidity and mortality of RF and RHD.

Most of the data was obtained basically from the record of the admitted cases.

- In 2003 data obtained from Ahmed Gasim Cardiac centre were reported as follow:
  - RHD constitute 50% of the total admission, where congenital heart disease 22%, ischaemic heart disease 21% and cardiomyopathy 7%.
1.3. Rheumatic Fever (RF):

Acute rheumatic fever, caused by a systemic immunologic reaction to group A *Streptococcus pyogenes* pharyngitis, is a clinical syndrome of fever, arthritis, rash, chorea and carditis\(^{(7)}\).

Its prevalence in Western Europe and North America has progressively declined to very low level, but it remains common in parts of Asia, Africa and South America, where it is still the most common cause of acquired heart disease in childhood and adolescence\(^{(8)}\).

Acutely there can be aortic and mitral regurgitation, while aortic and mitral stenosis are characteristic of long term sequelae\(^{(7)}\).

The initial valvular pathological lesion in acute rheumatic fever consists of valvulitis manifesting itself as a series of translucent nodules along the line of closure of the mitral valve. With chronic mitral stenosis; the mitral valve is thickened fibrotic and often calcified. Subvalvular apparatus are the hallmark of RF.

Aschoff bodies in the myocardium demonstrate that an attack of rheumatic fever had occurred in the past. They are located within the myocardium or the endocardium or both\(^{(9)}\).
1.4. Rheumatic Heart Disease (RHD):

The main pathological process in chronic rheumatic heart disease is a progressive fibrosis particularly affecting the heart valves.

1.4.1. Rheumatic mitral stenosis:

Rheumatic fever results in fusion of the mitral valve apparatus leading to stenosis, commissural, cuspal, chordal, and combined. Progressive fibrosis, thickening and calcification of the valve apparatus occurs.

Enlargement of the left atrium and result in elevation of the left main bronchus, calcification of the left atrial wall, development of mural thrombi, and obliteratorive changes in the pulmonary vascular bed may all result from chronic rheumatic mitral stenosis\(^{(10)}\)

Pathophysiology:

In mitral stenosis, there is elevated left atrial pressure which, in turn raises pulmonary venous and capillary pressure, resulting in exertional dyspnoea. The first bout of dyspnoea in patient with mitral stenosis is usually precipitated by exercise,
emotional stress, infections or atrial fibrillation.

**Pulmonary hypertension**: Result from:

1. Passive backward transmission of the elevated left atrial pressure.

2. Pulmonary arteriolar constriction, which is triggered by left atrial and pulmonary venous hypertension.

In some patients with moderately severe mitral stenosis (MS) mitral valve area (MVA) 1 to 1.5 cm², the high transvalvular pressure gradient causes a marked elevation of left atrial and pulmonary capillary pressure. This leads to severe pulmonary congestion during exertion\(^{(10)}\).

In patients with severe mitral stenosis (MVA <1.0 cm²) particularly when pulmonary vascular resistance is elevated, cardiac output is usually depressed at rest. These patients frequently have severe weakness and fatigue secondary to low cardiac output.

**Left atrial changes:**

The combination of mitral valve disease and atrial inflammation secondary to rheumatic carditis causes left atrial dilatation, fibrosis of the left atrial wall, and disorganization of the atrial muscle bundle\(^{(10)}\). The last leads to disparate conduction velocities and inhomogeneous refractory periods.
Premature atrial activation, due either to an automatic focus or to reentry, may stimulate the left atrium during the vulnerable period and thereby precipitate atrial fibrillation. The development of this arrhythmia correlates independently with severity of the mitral stenosis and the height of left atrial pressure.

**Clinical manifestations of mitral stenosis:**

Asymptomatic.

Recurrent chest pain.

- Exertional dyspnoea, nocturnal dyspnoea and cough.
- Symptoms of right heart failure.
- Symptoms of acute pulmonary oedema.
- Haemoptysis: either due to rupture of thin walled, dilated bronchial veins.
- Chest pain.
- Palpitations.
- Recent chest infection

Systemic embolism: when embolization occurs in patients with sinus rhythm, the possibility of transient atrial fibrillation (AF) or underlying infective endocarditis should be considered.

**Infective endocarditis:**
This complication tends to occur less frequently in rigid MS.

Compression of left recurrent laryngeal nerve by a greatly dilated left atrium, enlarged tracheobronchial lymph nodes and a dilated pulmonary artery causes hoarseness of voice (Ortner's Syndrome).

A history of repeated haemoptysis, systemic venous hypertension, hepatomegaly, oedema, ascites, and hydrothorax are all signs of severe MS with pulmonary vascular resistance and right sided heart failure\(^{(10)}\).

**Signs of mitral stenosis:**

Severe mitral stenosis with pulmonary hypertension is associated with the so-called mitral facieses or malar flush\(^{(11)}\).

The pulse in mitral stenosis is small volume. The development of AF in patients with mitral stenosis often causes a dramatic clinical deterioration.

The turbulent flow, which is heralded by the opening snap, causes the characteristic low-pitched diastolic murmur and often a thrill\(^{(8,11)}\). Pulmonary hypertension is recognized by a right ventricular heave, a loud pulmonary component of the second heart sound and signs of right heart failure\(^{(11)}\).

Pulmonary hypertension results in pulmonary valvular regurgitation, which causes an early diastolic murmur in the
pulmonary area known as a Graham-steel murmur.

Investigations:

- Chest X ray usually shows an enlarged left atrium (picture 1).
- E.C.G: Shows a bifid p wave due to delayed left atrial activation. AF is frequently present.
- Echo: M. mode echocardiography is sufficient to judge the severity of mitral stenosis\(^{(11)}\).
- Cardiac catheterization:

  The typical findings in mitral stenosis are a diastolic pressure that is higher in the left atrium than in the left ventricle. This gradient of pressure is usually proportional to the degree of the stenosis.

1.4.2. Mitral regurgitation:

In mitral regurgitation part of the left ventricular stroke output is ejected backwards into the left atrium\(^{(12)}\).

Clinical findings:

In acute, severe mitral regurgitation, left atrial pressure rises abruptly, leading to pulmonary oedema. When it is chronic, the left atrium enlarges progressively, but the pressure in pulmonary veins and capillaries rises transiently during exertion. Mitral regurgitation,
like mitral stenosis, predisposes to atrial fibrillation; but this arrhythmia is less likely to provoke acute pulmonary congestion, and fewer than 5% of patients have peripheral arterial emboli. Mitral regurgitation more often predisposes to infective endocarditis\(^{(13)}\) (picture 2).
1.4.3. Aortic stenosis:
The major physiological effects of aortic stenosis are rising in left ventricular systolic pressure, so that a pressure drop or gradient occurs across the aortic valve. A gradient more than 50mmhg indicate severe aortic stenosis\(^{(12)}\).

Symptoms of failure may be sudden in onset or may progress gradually. Common Symptoms Angina pectoris frequently occurs in aortic stenosis. Syncope is typically exertional and may be due to arrhythmia, hypotension, or decreased cerebral perfusion.

In mild or moderate cases, the characteristic signs are systolic thrill, systolic ejection murmur at the aortic area transmitted to the neck and apex; in severe cases, a palpable left ventricular heave or thrill, a soft to absent aortic second sound, or reversed splitting of the second heart sound are present. When the valve area is less than 0.8-1 cm\(^2\), normal (3-4 cm\(^2\)) ventricular systole becomes prolonged and the typical carotid pulse pattern of delayed upstroke and low amplitude is present.\(^{(13)}\)

1.4.4. Aortic regurgitation:

In aortic regurgitation or incompetence, there is a free back flow of blood out of the left ventricle and into it the left ventricle\(^{(12)}\).

1.4.5. Tricuspid and pulmonary valve:

The tricuspid and pulmonary valves are rarely involved in
chronic rheumatic heart disease although the tricuspid valve may become incompetent as a result of right ventricular dilation due to PH in MVD.\textsuperscript{(12)}

Tricuspid and pulmonary stenoses are uncommon.

**Percentages of valvular involvement in rheumatic heart disease as follow\textsuperscript{(14)}:**

<table>
<thead>
<tr>
<th>Valve involved</th>
<th>Percentage of cases</th>
</tr>
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<tbody>
<tr>
<td>Mitral valve alone</td>
<td>50</td>
</tr>
<tr>
<td>Mitral and Aortic</td>
<td>40</td>
</tr>
<tr>
<td>Mitral, Aortic and tricuspid</td>
<td>5</td>
</tr>
<tr>
<td>Aortic valve alone</td>
<td>2</td>
</tr>
<tr>
<td>All other combination</td>
<td>3</td>
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### 1.5. The role of Echo in rheumatic heart disease:

#### 1.5.1. Normal echocardiography Study:

Echocardiography (ultrasonic imaging of the heart) is now an established, invaluable diagnostic tool, often permitting direct visualization of cardiac pathology. Echo is extremely useful in measuring dimension of cardiac chambers as pattern of motion of walls and valves (picture 3).

Despite the advances in M-mode echo that have been
achieved by combining scanning from multiple transducer locations with a deductive approach, interest has increased over the past several years in developing an echocardiographic system that can provide specially correct dynamic image of cardiac structure.(15)

**Contrast echocardiography:**

The technique of contrast echo involves the intravenous injection of a liquid that contains micro bubbles that create a cloud of echoes on the echocardiogram.

The visualization of these micro bubbles is useful in identifying intra cardiac shunts and right sided valvular insufficiency.

**Doppler echocardiography:**

Early exploration of the Doppler technique in cardiac examination made use of continuous wave technique and concerned with movement of the heart walls and valves and blood flow velocity in the aorta and major thoracic vessels.
Picture 3

Apical four-chamber view: Seen is the apical four-chamber view from a 2-D echocardiogram. The display shows the apex at the top of the screen with the left ventricle (LV) to the viewer’s right. aML, anterior mitral leaflet; LA, left atrium; pML, posterior mitral valve leaflet; RA, right atrium; RV, right ventricle.

Transoesophageal echo (TEE):
TEE has been shown to be a reliable method for measuring the aortic valve orifice in aortic stenosis. TEE also has higher sensitivity for detecting left atrial thrombus. The M-mode echo pattern of the mitral valve is one of the most distinctive of any of the anatomic elements of the heart and also one of the easiest to record.

**Aortic valve:**

Satisfactory echoes are more difficult to obtain from the aortic valve than the mitral valve.

**Tricuspid valve:**

Recording the echoes from the tricuspid valve is more difficult than in the case of either the mitral or the aortic valve.

**Pulmonary valve:**

This valve technically is most difficult to record echocardiographically.\(^{(15)}\)

**Left atrium:**
The standard left atrial dimension is the distance at end-systole between the atrial side of the posterior aortic wall and the anteroposterior left atrial wall at the level of the aortic valve. This dimension in normal adult is measures 19 to 40 mm\(^{(15)}\). In a normal person, the diameter of the left atrium at the end of systole is the same as the diameter of the aortic root\(^{(16)}\). A large left atrium occurs in mitral regurgitation or stenosis and in patient with chronic left sided heart failure\(^{(15)}\).

**Mitral valve area:**

The normal orifice of the mitral value is about 4.5 cm\(^2\)\(^{(17)}\).

**1.6. Echocardiographic study in rheumatic heart disease:**

The echocardiographic findings reflect the varied anatomic and path physiologic derangements of different valves.

**Mitral stenosis:**

The images reflect an obliteration of the response to the atrial systole, movement of the posterior leaflet in the same direction as the anterior cusp in diastole, and thickening of the leaflet. The mobility of the mitral valve can also be measured and evaluated accurately. The extent of fibrosis and calcification and
the mobility of the mitral valve are accurately reflected in the echocardiogram by an amorphous, thick multi layered echo. In the absence of calcification which favors the feasibility of commissurotomy, where as heavy calcification and poor mobility usually point to the need for valve replacement.\(^{(18)}\) It is now evident that the assessment of mitral stenosis by echoes best achieved by cross-sectional echo, which permits measuring the true orifice of the mitral valve. Many studies show excellent correlation with mitral valve area calculated at cardiac catheterization or at the time of surgery\(^{(18)}\). Echo is now the corner stone of the diagnostic assessment of patient with mitral stenosis (picture 4). Two-dimensional transthoracic or transoesophageal echo demonstrates a thickened, calcified, stenotic rheumatic valve\(^{(10)}\). The principle diagnostic feature of mitral stenosis is doming of the anterior mitral leaflet in diastole. Doming indicates that the valve can not accommodate all the blood available for delivery into the left ventricle. Thus, the body of the leaflets separates more widely than do the edges. Doming is one of the main two - dimensional features of any stenotic valve. The aortic valve may also opens poorly in patients with a low cardiac output. Doppler echo offers an opportunity to assess the haemodynamic consequences of mitral stenosis\(^{(19)}\).
Calcified mitral annulus:

The principle observation of the M-mode echo is a band of dense high intensity echoes between the anterior mitral valve leaflet and the posterior left ventricle wall. This band of echoes is immediately posterior to the posterior mitral valve leaflets (picture...
Calcification may not be limited to only the annular or sub mitral area, but frequently extends throughout the base of the heart. It may extend into both mitral and aortic valves.

The calcium also spreads into the posterior mitral leaflets. The calcific process may extend deeply into the left ventricle and involve the aorta and aortic valve\(^{(19)}\).

Two-dimensional echo may be helpful in recognizing, left atrial thrombus preoperatively and assessing mitral valve calcification and left ventricular contractility\(^{(20)}\) (picture 6).

Doppler echocardiography is the most accurate noninvasive technique available for quantifying the severity of MS and for estimating pulmonary arterial pressure.

Color flow Doppler imaging can enhance the accuracy of the Doppler data detected by determining whether MR, aortic regurgitation, and other valvular abnormalities coexist.
Mitral stenosis Apical long axis view from a 2-D echocardiogram shows thickened mitral valve leaflets which have marked limitation of mobility and show doming, with failure to open normally during diastole.
Mitral regurgitation:

Doppler flow imaging, or color flow Doppler, is the initial approach currently used for diagnosis of mitral regurgitation. The multicolored turbulent mitral regurgitating jet is seen easily within
left atrium. This examination is a two-chamber view. The Doppler flow study displayed in the variance mode and the large multicolored, somewhat greenish jet is visualized within the left atrium (19).

A major feature of color flow Doppler in assessing mitral regurgitation is the display of the regurgitant jet within the left atrium. The location and direction of jet is readily apparent with this technique (picture 6). There also is an intuitive desire to quantitate mitral regurgitation based on the size of the regurgitant jet. The pulsed Doppler techniques also gives information with regard to the timing of regurgitation. For examples one can detect late systolic regurgitation in a patient with mitral valve prolapse. Pulsed Doppler techniques can also be used to quantitative the severity of mitral regurgitation by calculating regurgitate volumes or regurgitant fractions (19).

The intensity of the regurgitant jet is greater with more severe regurgitation. Several secondary signs of mitral regurgitation can be detected on M-mode and two-dimensional. These signs include the size of the left atrium, pulsations of the left atrial wall, the size of the left ventricle, aortic valve motion and the pattern of the interventricular septum (19).

The Doppler method is most frequently used in clinical
practice to evaluate the severity of mitral regurgitation and to map the extent of flow disturbances in the left atrium. M-mode and two dimensional echo are of limited use in the diagnosis of mitral regurgitation because in contrast to mitral stenosis, there are no characteristic signs\(^{(15)}\).

**Aortic valve disease:**

The pathologic changes in the aortic valve of the adult with aortic stenosis may range from mild thickening of the cusps that are functionally normal to extreme calcification with marked obstruction\(^{(15)}\). Recently, investigators have utilized Doppler echo as a means of identifying the severity of aortic stenosis. Doppler echo is the most sensitive ultrasonic technique for detecting aortic regurgitation\(^{(15)}\)(picture 7).

The valve becomes thickened and is frequently seen in diastole (picture 8). A more important sign of valvular stenosis is systolic doming. The principle means of quantitating aortic stenosis is with Doppler echo. Valvular aortic stenosis is an important valvular problem, especially in the older population. The need to quantitate the degree of aortic stenosis is great, and Doppler techniques are playing a major role in this effort. It must also be recognized that the pressure gradient does not always reflect the severity of the aortic stenosis. In a setting of low cardiac output, the
amount of the flow through the stenotic valve is reduced. Thus, the ultimate answer is not just pressure gradient but aortic valve area\(^{(19)}\).

**Aortic regurgitation (AR):**

All of the Doppler techniques are sensitive and reliable for qualitative diagnosis of AR. The degree of AR is to measure the size of regurgitant jet with color flow imaging. It was realized that, the jet is only used to distinguish mild from severe regurgitation. The AR velocity is indicative of the pressure gradient between the aorta and the left ventricle during diastole. Pulsed Doppler recording of aortic flow can also be helpful in judging the presence and the severity of aortic regurgitation\(^{(19)}\).

An indirect, two-dimensional echo cardiographyic sign of AR is reverse doming of the mitral leaflet with moderate to sever AR, the regurgitant jet produces diastolic indentation of the anterior mitral leaflet. There are several useful echocardiographic signs of severe and acute AR an important sign is premature closure of the mitral valve. The mitral valve is almost completely closed before ventricular systole. This sign is indicative of a high left ventricular diastolic pressure, which produces virtual closure of the mitral valve early in diastole\(^{(19)}\).

**Tricuspid valve:**
Echo diagnosis of tricuspid valve disease has not been extensively studied for the diagnosis of the mitral valve, because visualization of the tricuspid valve is more difficult and the two leaflets can usually be seen in patients with a dilated right ventricle\textsuperscript{(15)}.

The hallmark of the diagnosis is doming of the tricuspid valve. Doppler echo can also assist with the diagnosis of tricuspid stenosis and it offers better opportunity for judging the severity of stenosis\textsuperscript{(19)}.

In tricuspid stenosis, the M-mode echo of the anterior leaflet resembles that of the anterior leaflet of the mitral valve.

The pulse Doppler can readily detect the turbulent systolic jet of moderate to severe tricuspid regurgitation behind the valve in the right atrium. This was also used in pulmonary valve insufficiency\textsuperscript{(15)}. The Doppler technique can also be used to examine the inferior vena cava and or the hepatic vein for the presence of tricuspid regurgitation. This will document the reversal of flow during systole\textsuperscript{(19)}(picture 9).

Dense mitral valve echoes suggest rheumatic valvular disease but don’t provide reliable information about the degree of mitral regurgitation. The three alteration in the leaflet structure and function (thickening, calcification and decreased mobility)
characteristic of all types of acquired stenosis, may be reflected on the echocardiogram\textsuperscript{(21)}.

The pulmonary arterial pressure also can be estimated from the tricuspid regurgitation velocity signal \textsuperscript{(16)}. Most important complication of rheumatic mitral valve disease is cardiac failure, atrial fibrillation, systemic embolization and infective endocarditis \textsuperscript{(11)}. 
**Picture 7**

Continuous wave Doppler of mitral regurgitation. The transmural continuous wave Doppler in severe mitral regurgitation shows an E wave dominant, restrictive inflow pattern; there is a high velocity E wave, which in this example is 1.7 m/sec. The A wave is small and the E/A ratio is therefore increased.
Aortic regurgitation Long-axis view of 2-D echocardiogram of showing dilated left ventricle and thickening of aortic valve.
1.7. Rhythm disturbances due to rheumatic heart disease:

Supraventricular tachycardia, atrial flutter and AF are the most common rhythm disturbances in rheumatic heart disease.

Atrial flutter:
This is a rhythm disturbance that is usually associated with organic heart disease. The atrial rate varies between 280-350 per minute the ECG shows regular saw tooth-like atrial flutter waves (F waves) between QRST complexes\(^8\).

Atrial flutter is usually less long-lived, than is atrial fibrillation, although on occasion it may persist for month to years. Most commonly, if it lasts for more than one week, atrial flutter, will convert to AF. Systemic embolization is less common in atrial flutter than atrial fibrillation\(^22\).

Treatment of a symptomatic acute of a paroxysm is electrical cardioversion (50 J, 100 J,) and class III drugs are also effective\(^14\).

1.8. Atrial Fibrillation:

1.8.1 Definition:

In atrial fibrillation, the normal contractions are replaced by a continuous series of rapid, irregular, fibrillatory waves at a range of 380 to 600 or more a minute, which are ineffective in emptying the atria\(^23\).

The occurrence of A.F seems to be related to the extent of left atrial dilatation and its incidence also increases in older patient
so that it is usually present by the age 35 or 40\(^{(11)}\).

Less than 3% of patients with rheumatic mitral disease and echocardiographic left atrial dimension of $<4$ cm are in AF compared to more than 80% of patients over the age of 40 with a left atrial dimension over 4.5 cm\(^{(11)}\).

Rheumatic involvement of the left atrial wall may also play a part in the development of AF.

**Incidence of atrial fibrillation in rheumatic heart disease:**

Atrial fibrillation is a chronic disorder of rhythm in approximately 40% of patient with rheumatic mitral valve disease. It is uncommon in young patients but is present in most who are 45 years of age or older\(^{(18)}\).

The incidence of recognized paroxysmal AF is much less than that of chronic AF but is the precursor of the chronic arrhythmia.

**1.8.2. Common causes of atrial fibrillation:**\(^{(14)}\)

- Coronary artery disease (including acute myocardial infarction).
- Valvular heart disease (especially rheumatic mitral valve).
- Idiopathic (lone AF disease).
  - Hypertension.
• Thyrotoxicosis.

• Alcohol cardiomyopathy, congenital heart disease, pulmonary embolism, pericarditis and pneumonia.

1.8.3. Mechanism of arrhythmogenesis:

The principal mechanisms responsible for arrhythmia are those of abnormal automaticity, triggered activity and re-entry\(^{(24)}\).

1.8.4. Mechanism of Atrial Fibrillation:

It is now known that foci of rapid ectopic activity, often located in muscular sleeves that extend from the left atrium into the proximal parts of pulmonary veins, play a pivotal role in the initiation of AF in human\(^{(25)}\).

Proposed mechanisms for generation of abnormal. Focus activity include increased autonomic, triggered activity, and micro re-entry- changes in autonomic tone around the time of initiation of AF paroxysms, with an increased in sympathetic activity followed by an abrupt change to mal-sympathetic predominance, have also recently been demonstrated\(^{(26)}\).

1.8.5. Electrophysiological Features:

The resultant atrial abnormality (frequently, inflammation or fibrosis) acts as substrate for the development of the arrhythmia \(^{(27)}\).

In addition, the onset of atrial fibrillation usually requires a
trigger that may initiate the arrhythmia including alterations in autonomic tone, acute or chronic changes in atrial wall tension atrial ectopic foci, and local factors\(^{(28)}\).

1.8.6. **Classifications of atrial fibrillation:**

Classifications system based on the temporal pattern of arrhythmia has been recently recommended. Patients presenting for medical attention may have a first detected episode of AF or, if previous episodes have been documented, recurrent arrhythmia.

Episodes themselves may be paroxysmal, if they terminate spontaneously, usually within seven days, or persistent if the arrhythmia continues requiring electrical or pharmacological cardioversion for termination. AF that can not be successfully terminated or has not been attempted is termed permanent \(^{(29)}\).

The term “Lone AF” describes atrial fibrillation in the absence of demonstrable underlying cardiac disease or a history of hypertension or systemic disease.

1.8.7. **Hemodynamic Effects:**

Atrial fibrillation is associated with loss of the atrial contribution to ventricular filling. This may result in a decrease in ventricular stroke volume of up to 20 percent\(^{(30)}\).

The irregularity of the ventricular response may also contribute to haemodynamic impairment\(^{(29,30)}\).
In some patients with poorly controlled ventricular rate (generally, a mean of more than 100 beats per minute), persistent tachycardia results in ultra structural changes that cause ventricular dysfunction. This tachy-mediated cardiomyopathy is often reversible after sinus rhythm has been restored or when the heart rate during atrial fibrillation is controlled\(^{29,31}\).

1.8.8. Clinical Manifestations:

The onset of AF can cause palpitations and precipitate or aggravate cardiac failure in patient with an abnormal heart, especially those with mitral stenosis or poor left ventricular function. Nevertheless, atrial fibrillation is often asymptomatic particularly in the elderly\(^8\).

Symptoms may range from palpitations to acute pulmonary oedema, but fatigue and other nonspecific symptoms are probably the most common\(^{32}\).

Not all episodes of arrhythmia are symptomatic, and monitoring studies in patients with paroxysmal atrial fibrillation demonstrate that asymptomatic episodes occur more frequently than thought before, 21% of the patients were asymptomatic on presentation\(^{33}\). Among the 79% of patients with symptoms, palpitation was the presenting symptom occurred in 50%, chest pain and fatigue in more than 25% and dizziness presyncope or
syncope in about 25%.

Women had a significantly more impaired quality of life than men. When at cause by rheumatic stenosis, the onset of A.F results in considerable worsening of cardiac failure\(^{(33)}\).

Slight variation in intensity of the first heart sound occurs often with fast ventricular rates. A significant pulse deficit appears, during which the auscultated or palpated apical rates is faster than the rate palpated at the wrist (pulse deficit) because each contraction is not sufficiently strong to open the aortic value or transmit an arterial pressure wave through the peripheral artery\(^{(34)}\).

1.8.9. Clinical Evaluation:

The initial evaluation of patient with AF begins with a thorough history focused on identifying precipitants, defining associated cardiac or extra cardiac factors and characterizing the pattern of arrhythmia (e.g. symptoms, duration, proximal versus persistent, first episode versus recurrent)\(^{(35)}\).

Physical examination typically reveals an irregularly irregular pulse, irregular jugular venous pulsations with absent a wave and variation in the intensity of the first heart sound.

Associated valvular disease, primary or secondary (i.e. tachycardia-induced) cardiomyopathies, or heart failure may also be identified.
1.8.10. Diagnosis:

Definitive diagnosis of AF requires at least one (electrocardiogram) ECG lead documenting the arrhythmia from a rhythm strip, standard 12 leads EEG, holter monitoring.

**ECG:**

Electrical activity of the atrium can be detected on ECG as small irregular baseline undulations of variable amplitude and morphology called (F) waves at a rate of 350 to 600 beats /min. The verticular response is grossly irregular (irregular, irregular)(34) (pictures 10 & 11).
PICTURE 9-ECG RHYTHM STRIP SHOWING FAST ATRIAL FIBRILLATION
PICTURE 10 - ECG RHYTHM STRIP SHOWS CONTROLLED ATRIAL FIBRILLATION
1.8.11. Transthoracic echocardiographic study in atrial fibrillation:

- Transthoracic echo is really necessary for the initial evaluation and management in patients who have AF.
- The transthoracic echo is very useful to evaluate the dimensional areas and volumes of the left atrium, sharing their changes in the evolution of AF, to such point that the left atrial enlargement in the chronic AF is constant\(^{(36)}\).
- Less than 3% of patients with rheumatic mitral disease and echocardiographic left atrial dimension of < 4 cm are in AF compared to more than 80% of patients over the age of 40 with a left atrial dimension over 4.5 cm. Rheumatic involvement of the left atrial wall may also play a part in the development of AF\(^{(11)}\).
- Echocardiography study data has demonstrated that, chronic AF is associated with enlargement of the left atrium and that the conversion to a sinus rhythm is rare and not persistent.\(^{(37)}\)
1.9.12 Complications of rheumatic AF:

**Heart failure:**

When atrial fibrillation is caused by rheumatic mitral stenosis, the onset of AF results in considerable worsening of cardiac failure\(^{(14)}\).

**Thromboembtism in atrial fibrillation:**

The incidence of thromboembolic complications is higher in patients with rheumatic mitral stenosis and is a major cause of morbidity and mortality\(^{(38)}\).

The most feared complications of AF is stroke, which is often caused by thromboembolism from clotting in the relatively static blood pool of the fibrillating atrium, particularly in the left atrial appendage\(^{(39)}\).

Patient with paroxysmal AF had the same risk of stroke as subjects with AF.

Various factors determine the individual risk for the development of left atrial clot and thromboembolization in patient with rheumatic mitral valve disease which include atrial fibrillation, left atrial size, duration of symptoms and older age and severity of mitral stenosis\(^{(39)}\).
Sustained AF causes important reduction in cellular contractility, resulting in a tachycardia-induced atrial cardiomyopathy that may be responsible for late occurrence of thromboembolic events as contractility recovers later after cardioversion\(^{(27)}\).

The exact incidence of systemic emboli in rheumatic mitral valve disease is obviously not known. Most series have reported from 9 to 20 percent\(^{(21)}\).

**Left atrial thrombus (LA thrombus):**

The presence of left atrial spontaneous echo contrast which observed by TTE or TEE in patients with mitral valve disease indicates the presence of a thrombus, has been associated with increased risk of clot formation in the left atrium and systemic thromboembolization\(^{(40)}\).

**1.9.13  Treatment of atrial fibrillation:**

**Aim of treatment:**

1. Restoration to sinus rhythm.
2. Control ventricular rate.

**Restoration to sinus Rhythm:**

Spontaneous conversion to sinus rhythm within 24 hours after the onset of (AF); is common occurring in up two third of
Once the duration of (AF) exceeds 24 hours, the likelihood of conversion decreases. After one week of persistent arrhythmia, spontaneous conversion is rare.\(^{(41)}\).

**Ventricular rate control:**

In the absence of an urgent need for cardioversion, consideration should be given to pharmacologic rate control. Although the atrial rate usually exceeds 350 beats per minute, the mean resting ventricular rate in a patient with AF of new onset is between 110 and 130 beats per minute\(^{(42)}\).

Digoxin is somewhat effective for slowing the ventricular rate in a patient at rest, but its maximal action is achieved only after several hours. and it is of little value in patients who are in an adrenergic state\(^{(43,45)}\).

Intravenous beta-blocking or calcium channel-blocking drugs produce more rapid rate control, regardless of the level of sympathetic tone. However, in a recent small trial control of the heart rate with the use of diltiazem during atrial fibrillation produced as much relief of symptoms as did with amiodarone\(^{(43,44)}\).

**Antiarrhythmic-drug therapy:**

Early drug therapy to restore sinus rhythm is considered in patients in whom the arrhythmia has lasted less than 48 hours or
who are receiving long-term warfarin therapy. Digoxin is not effective in converting atrial fibrillation to sinus rhythm. But antiarrhythmic therapy increases the likelihood of conversion to as much as 90 percent, if the drug is administered early and in adequate doses\(^{(43,44)}\).

**Recurrent paroxysmal atrial fibrillation:**

Several drugs have been shown to be effective in the treatment of paroxysmal atrial fibrillation. These include propafenone, flecainide and Sotalol\(^{(45)}\).

Often they do not totally abolish the arrhythmias, but increase the length of the interval between the paroxysms. Although this decrease in frequency of paroxysm is often satisfactory for a reduction of symptoms, there are no data supporting the possibility that having fewer episodes of atrial fibrillation decreases the risk of thromboembolism.

Furthermore, patients who have symptomatic episodes of paroxysmal arrhythmia may also have multiple episodes of asymptomatic atrial fibrillation. Although symptomatic episodes tend to be shorter than the asymptomatic episodes, they may still have a possible a risk of thromboembolism\(^{(46)}\).

**Persistent atrial fibrillation:**

Once an episode of atrial fibrillation has lasted more than
seven days, spontaneous conversion is rare and the conditions can be defined as persistent.

The decision to attempt to restore a sinus rhythm in a patient with persistent atrial fibrillation is not always clear-cut.

Restoration of sinus rhythm will generally improve the patient’s symptoms, but not all patients have symptoms\(^{(46,47)}\).

**Prevention of thromboembolism:**

**Anticoagulation therapy:**

In the absence of contraindication, patients should be considered for anticoagulation if they have one or more risk factors for thromboembolism\(^{(44,48)}\).

Patients at low or intermediate risk in whom warfarin is contraindicated may benefit from antiplatelet treatment.

The risk of bleeding substantially increases at INRs greater than 3.0 and thus the optimal INR range for patients with AF is 2.0-3.0.

**Cardio-version:**

Restoration of sinus rhythm in patient with atrial fibrillation frequently requires at least 300J of energy with most defibrillators currently in use. However, the recent introduction of defibrillators with a biphasic wave form, rather than the traditional monophonic damped-sine wave form, is associated with a marked decrease in
the energy repulsed for atrial defibrillation and with fewer failure
(49).

If a patient fails to return to sinus rhythm even for one beat
despite these measures, transvenous internal cardioversion may
be successful. The decision regarding, which antiarrhythmic agent
to use for the maintenance of sinus rhythm should be based on
properties of the drugs, their side effects, their safety in the
presence of structural heart disease. There are few studies of
comparative efficacy, but amiodarone has been shown to be
superior to both sotalol and propafenone for the maintenance of
sinus rhythm\(^{(50)}\).

Most recurrence of atrial fibrillation occur within three
months after cardioversion of the first episode of atrial fibrillation
regardless antiarrhythmic agent used\(^{(51)}\).

**Drug-Refractory atrial fibrillation:**

There are different modalities of treatments:

1. Ablation of the atrioventricular node and implantation of a
pace-maker. The ablation of the atrioventricular node along with
the implantation of a pacemaker significantly improves quality-of
life.\(^{(52)}\)
2. Focal ablation: the fact that at least some subgroups of AF may be amenable to cure by radio frequency catheter ablation has been increasingly evident in recent years.

OBJECTIVES

1. To study the correlation between the left atrial size and atrial fibrillation due to rheumatic heart disease in Sudanese patients at different age groups.

2. To determine the relation between the mitral valve area in
rheumatic mitral stenosis and the incidence of atrial fibrillation.

3. To study the occurrence of atrial fibrillation in different age group in patients with RHD.
RATIONAL

Rheumatic heart disease is still a major health problem in our country as well as in developing countries e.g. (India).

It was noticed that many young Sudanese patients with rheumatic heart disease develop severe congestive heart failure while in sinus rhythm, other patients develop atrial fibrillation while they are quite young and with relatively recent attack of rheumatic fever.

Atrial fibrillation is one of the commonest complications of rheumatic heart disease. It has a high incidence with increased morbidity and mortality among our patients. There was no previous study addressing this issue.

The use of anticoagulation is a big hurdle, in patient with these deficient lab facilities and where the prothrombin time and INR estimation is not done in most of towns.
PATIENTS AND METHODS

Study design:

This is a prospective cross-sectional study.

Study population:

We studied patient presenting for the first time or in the referred clinic for follow up.

The studied population consisted of a 200 random cases with rheumatic heart disease and atrial fibrillation. All male and female at any age on presentation were included.

Incidence of atrial fibrillation in Sudanese patient with RHD:

500 cases were studied from the records to estimate the incidence of AF among Sudanese patients with rheumatic heart disease. It was found to occur in 107 patients i.e. comprising an incidence of 21.4%.

Place and time of the study:

The study was conducted in Elshaab and Ahmed Gasim cardiac centre.

The period was from June 2003 to February 2004.

Data sources:

Information was obtained through examining patients in the referral clinic and in patients in both hospitals mentioned.
Data collection and methods of the study:

Data was collected by self-administered questionnaire. This was constructed in section to address the different aspects of the study as follows:

- Personal data.
- Presentations.
- Physical examinations.
- Investigations.

Different age groups and of both sexes were included in this study.

Patient assessment:

This was done in the normal manner by history, examination and investigations.

Investigations:

Electrocardiogram (ECG).

Chest X ray.

Echocardiography (ECHO).

Transthoracic Echo was used as the procedure of choice in the study, but in some cases TEE was used because, it was difficult to obtain satisfactory echo by TTE.

Data analysis:

All the collected data was entered in a master sheet and then
analyzed by SPSS (Statistical packages for social sciences).

RESULTS

This is cross-sectional prospective study in Sudanese
patient with rheumatic heart disease and atrial fibrillation. The number of studied cases was 200 cases.

The age of these patients was ranging between 10-49 years. Most of the patients were in the age group of 30-39 years (Fig. 1).

Female to male ratio was 63%: 37% (1.7:1) (Fig. 2).

Most of our patients studied resided in Khartoum (48%), Central (Gezera) (22%), Western (12.5%), Eastern (11%), Northern (6%) and Southern Sudan (0.5%). (Fig.3)

History suggestive of rheumatic fever was found in 73 cases (36.5%). 127 (63.5%) did not give history suggestive of rheumatic fever. (Fig.4).

The main presenting symptoms to the Emergency and Referred Clinic were shortness of breath (85%), palpitations (74%) cough (4%), chest pain (28%), dizziness (16%), embolic phenomenon (8%) and syncopal attack (2.5%) (Table 1).

Dyspnoea was classified according to New York Heart Association as class I, II, III and IV.

Cases studied were as follows:

class I 19.5%

class II 37%
The rhythm pattern was reported by ECG, showed 41% to have fast AF and 59% were controlled AF, because they were on digoxin therapy (Fig. 6).

Regarding the chest X-ray findings, were cardiomegaly in 85%, and pulmonary oedema in 45%.

The echocardiographic findings concentrated on the following:
- Determination of left atrial size.
- Presence of left atrial thrombus.
- Nature of valvular lesion involved in these patients.

Mitral valve was involved in all patients. 89 (44.5%) patients had mitral stenosis and 94 (47%) patients had mitral stenosis and regurgitation and 17 (8.5%) patients had pure mitral regurgitation (Fig. 7).

The degree of stenosis studied by echocardiography according to the mitral valve area was classified as follow:

Severe  <1 cm²
Moderate  1-1.5 cm²
Mild      1.5-2.5 cm²

The following result was obtained: 20 patients (11%) had
mild mitral stenosis, 48 patients (26%) had moderate mitral stenosis and 115 patients (63%) had severe mitral stenosis (Table 2).

The nature of the mitral valve whether calcified or not was determined through the echocardiographic study which showed 80 patients (40%) to have calcified mitral valve and 120 patients (60%) had non calcified mitral valve (Fig. 8).

Seventy-one patients had aortic valve lesion. 2 patients had aortic stenosis, 54 had aortic regurgitation and 15 had mixed aortic valve disease (Table 3).

Two patients (1%) had tricuspid stenosis, while 58 patients (29%) had functional tricuspid regurgitation (Fig. 9).

Left atrial size was found to be normal in only 5 patients (2.5%).

And dilated in 195 patients (97.5%) (Table 4).

(Table 5) shows the left atrial diameter in mm in the studied group.

Left atrial thrombus was found in 18 patients (9%) only (Fig. 10 & picture 12).
Picture 12

Left atrial appendage thrombus
Fig. 1: Distribution of age among the study group
Fig. 2: Gender distribution among the study group

- Female: 126 (63%)
- Male: 74 (37%)
Fig. 3: Geographical Distribution Among the study Group

- Khartoum: 48%
- Central (Aljazera): 22%
- Western: 12.5%
- Eastern: 11%
- Northern: 6%
- Southern: 0.5%
Fig. 4: History suggestive of rheumatic fever among the study group

Yes
73 (36.5%)

No
127 (53.5%)
Fig. 5: Dyspnoea according to NYHA classification

- Class I: 39 (19.50%)
- Class II: 74 (37%)
- Class III: 62 (31%)
- Class IV: 25 (12.5%)
Fig. 6: Rhythm Pattern on ECG among the study group

- Fast atrial fibrillation: 41%
- Controlled atrial fibrillation: 59%
Fig. 7: Pattern of Mitral Valve involvement among the study group

Mixed Mitral Lesion 94(47%)
Mitral Stenosis 89(44.5%)
Mitral Regurgitation 17(8.5%)
Fig. 8: Nature of mitral valve lesion among the study group

- Non-calcific mitral valve: 60%
- Calcific mitral valve: 40%
Fig. 9: Tricuspid valve lesion among the study group

58 (29%)

2 (1%)

Percentage

Tricuspid stenosis

Tricuspid regurgitation
Fig. 10: Presence of left atrial thrombus among the study group
The studied patients were classified as below 20 and above 20 years of age named group 1 and group II respectively. Then the incidence of AF was analyzed according to age as follow:

The incidence of AF was found in group I as (7.5%), and in...
group II (92.5%) (**Fig. 11**).

The above two groups were also studied with relation to mitral valve lesion, that in group I, two patients had pure mitral stenosis, 2 patients had pure mitral regurgitation and 11 patients had mixed mitral valve lesion. In group II, the lesion of the mitral valve was as follows: 87 patients had mitral stenosis, 15 had regurgitation and 83 had mixed mitral valve disease (**Table 6**).
<table>
<thead>
<tr>
<th>Percentage</th>
<th>No. of patients</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>85%</td>
<td>170</td>
<td>Shortness of breath</td>
</tr>
<tr>
<td>74%</td>
<td>148</td>
<td>Palpitations</td>
</tr>
<tr>
<td>46%</td>
<td>92</td>
<td>Cough</td>
</tr>
<tr>
<td>28%</td>
<td>52</td>
<td>Chest pain</td>
</tr>
<tr>
<td>16%</td>
<td>32</td>
<td>Dizziness</td>
</tr>
<tr>
<td>8%</td>
<td>16</td>
<td>Embolic phenomenon</td>
</tr>
</tbody>
</table>

**Table 2: Degree of mitral valve stenosis among the study group (n= 183)**
<table>
<thead>
<tr>
<th>Percentage</th>
<th>No. of patients</th>
<th>Degree of mitral stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>11%</td>
<td>20</td>
<td>Mild</td>
</tr>
<tr>
<td>26%</td>
<td>48</td>
<td>Moderate</td>
</tr>
<tr>
<td>63%</td>
<td>115</td>
<td>Severe</td>
</tr>
<tr>
<td>100%</td>
<td>183</td>
<td>Total</td>
</tr>
</tbody>
</table>

Table 3: Number of patients with aortic valve disease in the study group (n= 71)

<table>
<thead>
<tr>
<th>Percentage</th>
<th>No. of patients</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.8%</td>
<td>2</td>
<td>Aortic stenosis</td>
</tr>
<tr>
<td>Percentage</td>
<td>No. of patients</td>
<td>Size</td>
</tr>
<tr>
<td>------------</td>
<td>----------------</td>
<td>------------</td>
</tr>
<tr>
<td>76.1%</td>
<td>54</td>
<td>Aortic regurgitation</td>
</tr>
<tr>
<td>21.12%</td>
<td>15</td>
<td>Mixed aortic lesion</td>
</tr>
<tr>
<td>100%</td>
<td>71</td>
<td>Total</td>
</tr>
</tbody>
</table>

Table 4: Left atrial size (dimension) among the study group

<table>
<thead>
<tr>
<th>Percentage</th>
<th>No. of patients</th>
<th>Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5%</td>
<td>5</td>
<td>Normal</td>
</tr>
<tr>
<td>97.5%</td>
<td>195</td>
<td>Dilated</td>
</tr>
<tr>
<td>100%</td>
<td>200</td>
<td>Total</td>
</tr>
</tbody>
</table>
Table 5: Left atrial size diameter among the study group by echocardiography.

<table>
<thead>
<tr>
<th>Percentage</th>
<th>No. of patients</th>
<th>Left atrial diameter (millimeter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5%</td>
<td>5</td>
<td>( \leq 40 )</td>
</tr>
<tr>
<td>8%</td>
<td>16</td>
<td>41 – 45</td>
</tr>
<tr>
<td>12%</td>
<td>24</td>
<td>46 – 50</td>
</tr>
<tr>
<td>25%</td>
<td>50</td>
<td>51 – 55</td>
</tr>
<tr>
<td>52%</td>
<td>104</td>
<td>&gt; 55</td>
</tr>
<tr>
<td>100%</td>
<td>200</td>
<td>Total</td>
</tr>
</tbody>
</table>
Table 6: Mitral valve lesion in relation to age in the study groups

<table>
<thead>
<tr>
<th>Group I (&gt; 20 years)</th>
<th>Group I (&lt; 20 years)</th>
<th>Mitral valve</th>
</tr>
</thead>
<tbody>
<tr>
<td>87 (47.02%)</td>
<td>2 (13.33%)</td>
<td>Stenosis</td>
</tr>
<tr>
<td>15 (8.10%)</td>
<td>2 (13.33%)</td>
<td>Regurgitation</td>
</tr>
<tr>
<td>83 (44.0%)</td>
<td>11 (73.33%)</td>
<td>Mixed lesion</td>
</tr>
<tr>
<td>185 (100%)</td>
<td>15 (100%)</td>
<td>Total</td>
</tr>
</tbody>
</table>
DISCUSSION

This prospective study on 200 patients with AF and RHD was carried in El Shaab Teaching Hospital and Ahmed Gasim Cardiac Centre. It was conducted during the period from June 2003 to February 2004. ECG and echocardiographic were performed for all patients.

Shimada S. reported a 13 years follow-up study in 301 patients of rheumatic valvular disease\textsuperscript{(55)}. He stated that male to female ratio was 78: 145. 51\% of his patient had mitral valve disease, 40\% combined valvular disease and 29\% of aortic valve disease. In his study atrial fibrillation was observed in 50\% of the patients. Cerebral emboli occurred frequently in patients with mitral
stenosis (MS), mitral stenoregurgitation and combined valvular diseases, which were associated with atrial fibrillation. However cerebral emboli were rarely found in cases in sinus rhythm.

Melka\(^{(56)}\) studied one hundred and fourteen patients with rheumatic heart disease between January 1994 and January 1995 in Gender and he found, the mean and median age of the patients were 23±8 years and 22 years, respectively (range 5 - 50 years). 66% of the patients were females with female to male ratio of 1.9:1. He reported history suggestive of rheumatic fever (RF) in 26% of his patients. Frequently encountered valve lesions were combined mitral regurgitation and stenosis in 29 (25-5%) followed by pure mitral stenosis in 25(21.9%) mitral regurgitation in 21 (18.4%). The commonest arrhythmia was atrial fibrillation observed in 22.8% of the cases. Regarding dyspnoea, he classified studied patients according to the New York Heart Association’s classification as 17%, 25%, 26%, and 32%, for classes I, II, III, IV respectively. The common precipitating factor was drug discontinuation followed by infection and arrhythmia.

Study done in Sudan\(^{(5)}\) by Atiyat et al, in 1983 among pediatric, (age < 18 years) reported that, the ratio of female to male was 1 : 1, mitral incompetence was the dominant lesion. 33% of the studied patients were in heart failure. The mitral valve affected
was three times as common as the aortic valve. The mitral valve was involved in 82%, mitral and aortic valve in 5%, aortic valve only in 3%. The incidence of AF was 5% and the thromboembolic incidence was 1%.

Shyama S, Budhani, V.A Kothiware\(^{(57)}\). Studied patient with respects to aetiology and complications in KLES Hospital “Belgaum”. They reported that in patient with atrial fibrillation due to rheumatic heart disease female to male ratio was 65.7% : 34.28%. Mitral valve stenosis was commonest valvular lesion (88.75%), 71.42% of cases had mitral valve orifice less than 1cm\(^2\) and 95.3% of patients of mitral valve disease had left atrial size above 40 mm. They concluded that mitral stenosis was the commonest valvular lesion. Development of atrial fibrillation correlates fairly well with mitral valve orifice area and left atrial size.

Henry, Morganorth, Pearlman, Clark \textit{et al}\(^{(58)}\) studied the relation between echocardiographically determined left atrial size and atrial fibrillation in attempts to define quantitatively the relation between left atrial size, and atrial fibrillation. 85 patients were studied with isolated mitral valve disease. Atrial fibrillation was rare when the left atrial dimension was less than 40 mm (3%), but was common when this dimension exceeded 45 mm (54%). These data suggest that left atrial size is an important factor in the
Cheng-Wen Chiang, Sing-Kai Lo et al.\textsuperscript{(59)} studied 534 patients with a mitral valve area of 2 cm or less. 132 patients were in sinus rhythm and 402 were in atrial fibrillation. 60 patients developed systemic embolism and were in atrial fibrillation (11.3%). They concluded that patients age, the presence of left atrial thrombus, mitral valve area and the presence of significant aortic regurgitation were positively associated with embolism. Patients with atrial fibrillation, previous embolism was positively associated with embolism. Percutaneous balloon mitral commissurotomy was a negative predictor.

Shrestha, Horeno, Narciso, \textit{et al.}\textsuperscript{(60)} studied 293 patients with rheumatic heart disease with two-dimensional echo. LA thrombus was found in 33 (8.8%). Twenty-one patients had left atrial thrombus but were not detected by two-dimensional echocardiography (sensitivity 58%). Ten of these had thrombi in the left atrial cavity. Eleven were located in the left atrial appendage, all of which were missed by two-dimensional echo. Excluding these left atrial appendage thrombi, the sensitivity of two-dimensional echo for detecting left atrial cavity thrombi was 75%.

Srimannaryana, Varma \textit{et al.}\textsuperscript{(61)} studied patients with
severe mitral stenosis and AF, who underwent a transoesophageal echo (TEE) evaluation. A total of 490 patients (343 female "70%" and 147 males "30%") were included in the study, the mean age of these patients was $27.4 \pm 8.41$ years (range 21-65 years). LA clot were present in 163 (33.3%). Isolated LA appendage clots were found in 88 patients (18%). Isolated LA body clot or LA appendage clots extending into the LA body were found in 75 patients (15.3%). They reported that one out of every three patients with mitral stenosis and atrial fibrillation will have left atrial thrombus. It has been shown that transoesophageal echocardiography is highly sensitive for the detection of left atrial clot, especially, in the left atrial appendage\(^{(61)}\).

In our study male to female ratio was (37%: 63%) 1:1.7 which is similar to Shyma\(^{(57)}\) who reported the female to male ratio is 1.9:1 (65.7%:34%).

It was also similar to Shiamada\(^{(55)}\) who reported that male to female ratio was (78%:145%) 1:1.8, whereas Melka\(^{(56)}\), found the ratio between female to male as 1.9:1. It was shown that rheumatic heart disease is more common in females as well as atrial fibrillation. History suggestive of rheumatic fever was found in 73 (36.5%) patients. Melka\(^{(56)}\) reported it as (26%).

Regarding geographical distribution most of our patients
came from Khartoum 96 patients (48%), Central 44 (22%), Western 25(12.5%), and Eastern 22(11%), Northern12 (6%) and one patient referred from Southern 0.5%. This was related to presence of advanced technology and facilities and specialized centre, in Khartoum for those patient whom were referred from different states of Sudan, the other factor, is that Khartoum constitutes this high number of patients ,most of them resides in the peripheries of the capital.

In our study it was found that atrial fibrillation due to rheumatic heart disease occurs more commonly at the age between  30-35 years. In the literature\textsuperscript{(11)} the incidence of atrial fibrillation increases in older patient , usually it presents by the age of 35 to 40.

Atiyat\textsuperscript{(5)} found the incidence of atrial fibrillation to be (5%) among patients with rheumatic heart disease in the paediatric age.

In our study groups, the percentage of atrial fibrillation was found to be (7.5%) in patients below the age of 20 years. This is comparable with Dr. Atiyat s` patients.

The functional classes of patients according to the NYHA association's classification were 19.5%, 37%, 31% and 12.5%.to class I, II, III and IV respectively .In our study we found that mitral valve disease was involved in all patients. Mitral stenosis was
found in 183 patients (91.5%).

The severity of mitral stenosis was assessed by two-dimensional transthoracic echocardiography. 115 (63%) patients had severe mitral stenosis in which the mitral valve area was less than 1 cm². Our study was similar and comparable to Shyma (57) who stated that mitral valve stenosis was involved in 88.57%, However 71.4% of his patients with rheumatic heart disease and atrial fibrillation had mitral valve area less than 1 cm². The study concluded that the severity of stenosis was a strong predictor for the development of atrial fibrillation. The transthoracic two-dimensional echocardiographic study of the left atrial size showed normal left atrium in 5 patients (2.5%) and abnormal LA diameter more than 40 mm in 195 patients (97.5%). This was similar to the findings of Hall R.J.C. (10) who stated that the occurrence of atrial fibrillation seems to be related to the extent of left atrial dilatation. It was reported that less than 3% of patients with rheumatic heart disease and echocardiographic left atrial dimension of < 4 cm are in atrial fibrillation compared to more than 80% of patients over the age of 40 with left atrial dimension over 4.5 cm.

Our study results were also similar to those of Shyma (57) study who stated that the left atrial size was abnormal in 95.3% of patients with mitral valve disease. They had left atrial size above
40mm. So this supports the previous study and indicates that an abnormal left atrial dimension is a strong factor in the development of rheumatic atrial fibrillation.

Most studies concluded that there is a significant relation between the development of atrial fibrillation due to rheumatic heart disease and increased left atrial dimension exceeding the normal size.

In our study the occurrence of embolic phenomenon was detected in 16 (8%) patients who had cerebrovascular accident. Cheng (59) who reported embolic phenomenon was found in (11.9%) in patient with rheumatic heart disease who had atrial fibrillation, but he studied 402 patients, and we studied 200 cases.

The occurrence of left atrial thrombus was found in 18 patients (9%). This was different from Srimmangyaan (61) who studied the prevalence of left atrial thrombus in rheumatic mitral stenosis by transoesophageal echocardiography and reported that left atrial clot was found in one third of patients. He studied 490 patients with rheumatic mitral stenosis where left atrial clot were present in 163 (33.3%).

Another study done by NK Shrestha (60) who studied 293 patients with rheumatic heart disease, left atrial thrombus was found by transthoracic two-dimensional echocardiography in 8.8%
of patients.

It was observed in NK Shrestha\textsuperscript{(60)} study as well as in our study, the low sensitivity of transthoracic echo in the detection of left atrial thrombus. Transoesophageal echo is the best method with higher sensitivity in detecting the presence of left atrial thrombus.
In summary in our study we found that:

• The incidence of AF below 20 years was 7.5%, this was similar to Dr. Atiyat\(^{(5)}\) study who stated that the incidence among paediatrics was found to be 5%.

• Severe mitral stenosis was present in 115(63%) while in Shyma\(^{(57)}\) study it was 71%.

• The left atrial dimension exceeded the normal size in 97.5%, Shyma stated it as 95.3%.

• Left atrial thrombus was detected in 9% only, in Shrestha\(^{(60)}\) study it was found in 8.8%. Both studies differ from Srimannaryanno\(^{(61)}\) who detected it in 33%, but he used TEE.
CONCLUSION

- Rheumatic heart disease is still a major health problem in Sudan.
- Atrial fibrillation is noticed with high predilection in patients with rheumatic mitral valve disease.
- There is strong correlation between age and the occurrence of atrial fibrillation. Patients with rheumatic heart disease their age range between 30 - 49 years has greater chance to develop atrial fibrillation.
- There is correlation between atrial fibrillation due to rheumatic heart disease and left atrial diameter.
- A reduced mitral valve area less than 1 cm$^2$ is also another factor for the development of atrial fibrillation in rheumatic heart disease the greater the severity of mitral valve stenosis the greater the chance of development of atrial fibrillation.
- Atrial fibrillation has lots of complications such as left atrial thrombus and thromboembolic phenomenon.
- Transthoracic echocardiographic has low sensitivity in detecting the presence of left atrial thrombus.
RECOMMENDATIONS

• Early diagnosis of rheumatic fever is needed as well as prescription of secondary prophylaxes is of a vital role to prevent or reduce the incidence of long term sequelae of chronic rheumatic valvular disease.

• Early detection of rheumatic valvular lesions and referral to special centre for earlier and easier surgical intervention whenever indicated is recommended.

• Regular follow up clinical and echocardiographic is useful especially in patients with previous systemic embolism and also to those patients showing left atrial thrombus on echo cardiography.

• The hazard of anticoagulation should not be underestimated as patients might develop bleeding at a risk area (haemorrhagic stroke) if the INR is not adjusted properly.

• A well-established centre for INR adjustment in different states of our country is needed.

• Transoesophageal echo is useful for detecting left atrial thrombi, though it is of great help if instituted to patient with abnormal left atrial size.

• Early percutaneous balloon mitral commissurotomy may also
help in preventing systemic embolism in patients with pliable mitral valve stenosis.

- Biostatistical studies to estimate the incidence of rheumatic heart disease is of great help, to recognize the real size of this problem in different states of our country.


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