ANALYSIS OF THE AETIOLOGICAL FACTORS CLINICAL FINDINGS AND COMPLICATIONS OF ATRIAL FIBRILLATION

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CERTIFICATE

This is certify that the Dissertation entitled ***" Analysis of the aetiological factors clinical findings and complications of Atrial Fibrillation",*** *herewith submitted by* ***Dr.J.Priya*** *Post Graduate in M.D. General Medicine, Coimbatore Medical College to the Tamilnadu Dr. M.G.R. Medical University is a record of a bonafide research work carried out by her under my guidance and supervision from Jan 2006 to Jun 2007.*

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DEAN

DECLARATION

I solemnly declare that the Dissertation titled ***"Analysis of the aetiological factors and clinical findings and complications of Atrial Fibrillation ",*** *was done by me at Coimbatore Medical College & Hospital during the period from Jan 2006 to Jun 2007 under the guidance and supervision of Prof. Dr. K. Umakanthan M.D, and Prof. Dr.S .Veerakesari M.D,*

This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch I) in General Medicine.

Place: Coimbatore **Dr. J. Priya**
Date :

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INTRODUCTION

INTRODUCTION:

Atrial fibrillation is the commonest sustained disorder of cardiac rhythm. When it is present many prognostic and therapeutic implications exist as overall morbidity and mortality increase appreciably. Despite this, atrial fibrillation is sometimes regarded as a fairly trivial and unimportant disorder and is often neglected, probably because many patients have few symptoms. In fact, some patients with chronic atrial fibrillation may require long term treatment with potent antiarrhythmic and anticoagulant drugs, which may have important pharmacological interactions and adverse effects. In addition, treatment differs importantly for chronic and paroxysmal atrial fibrillation and for atrial fibrillation, atrial flutter, and the other supraventricular arrhythmias.

Atrial fibrillation is encountered in many clinical settings. It may, for example, be discovered incidentally in an asymptomatic patient, develop in a patient who merely has a chest infection, or be found in a patient with a ventricular rate of 200 beats/min who is too light-headed to stand up. Patients admitted with atrial fibrillation may have many cardio respiratory symptoms and clinical features, including syncope and stroke.

Atrial fibrillation is common in the community, affecting up to 5% of people aged 75 or over. It is a major reason for emergency admissions

and cause of cardiovascular deaths. Thus most clinicians in hospital and general practice will participate in managing such patients. As the prevalence of the condition increases with age, atrial fibrillation will become increasingly common in the increasingly aging population.

Atrial fibrillation appears to be more in **WHITES** than in **BLACKS.** Blacks have less than half the age adjusted risk of developing AF than is seen in whites.

Epidemiological studies have shown that atrial fibrillation is fairly uncommon in people aged under 50 years but is found in 0.5% of people aged 50-59, increasing to 8.8% at age 80-89. Furthermore, the arrhythmia may be either chronic or paroxysmal. In the Framingham study, hypertension, cardiac failure, and rheumatic heart disease were the commonest precursors of atrial fibrillation. Up to a third of patients with atrial fibrillation, however, may have idiopathic or "lone" atrial fibrillation, where no precipitating cause can be identified and no evidence of structural heart disease exists.

AIM OF THE STUDY

1) Analysis of etiological features of atrial fibrillation.
2) Analysis of clinical features of atrial fibrillation.
3) Analysis of Complications of atrial fibrillation.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY OF ATRIAL FIBRILLATION: [1, 2, 3, 4, 5]

For two centuries after Harv the atrial pulse was often regarded as independent of the heart beat. This misunderstanding was mainly because of the frequent failure of the irregular heart to elicit a radial pulse: the "pulse deficit" of later times. Harvey observed ineffective palpitation of the atrium just before death. This was probably atrial fibrillation. He established the origin of the heart beat in the right atrium. Harvey's observations were confirmed and extended by de Senac in the mid-eighteenth century. He correlated gross irregularities (palpitation) with necropsy observation of mitralvalve disease and dilatation of the left ventricle. He emphasised the origin of the heart's irregularity from the distended atrium consequent on distension or reflux of blood irritating the atrial wall. He also commented on disconcerted action and rippling of the ventricular wall before final cessation of movement in a dying heart (ventricular fibrillation). De Senac's ideas were a century and a half ahead of his time.

In clinical practice and with the aid of Laennec's recently invented stethoscope, Robert Adams reported in 1827 the association of irregular pulses with mitral stenosis; in 1863, Etienne Marey published a

pulse tracing from such a patient. Other early descriptions of atrial fibrillation and its importance were published early this century by Sir James Mackenzie and Heinrich Haring.

The discovery of the therapeutic properties of digitalis leaf (Digitalis purpurea) in 1785 by William Withering brought some relief to patients with severe heart failure. It is interesting that Withering recorded a patient who had a weak, irregular pulse that became "more full and more regular" after five draughts containing Fol Digital Purp oz iv. In 1935 Jean Baptiste Bouilland said that he considered digitalis to be "a sort of opium for the heart."

The main diagnostic breakthrough was the invention of the electrocardiograph by William Einthoven in 1900. A close friend of Einthoven, Sir Thomas Lewis at University College Hospital, London, was the first to record an electrocardiogram in a patient with atrial fibrillation. (1995)

DEFINITION: [6]

Atrial fibrillation is an arrhythmia that is characterised by seemingly disorganised and depolarization without effective atrial contraction.

During atrial fibrillation electrical activity of atrium can be detected on ECG as small irregular baseline undulation of variable amplitude and morphology called ‘f’ waves, at a rate of 350 to 600 beats/min

PREVALENCE: [7]

Overall prevalence of 0.5% in adult population of UK. The prevalence raises with age affecting 2-5% of 70 yrs olds and 9% of those aged over 80 yrs.

FRAMINGHAM DATA:

Atrial fibrillation found in 1% of persons older than 60 yrs to more than 5% of patients older than 69 yrs .The over all chance of atrial fibrillation developing over a period of 2 decades in patients older than 30 yrs . Estimates are that 2.5 million Americans have atrial fibrillation. [6]

CLASSIFICATION OF ATRIALL FIBRILLATION:

1. PAROXYSMAL—Intermittent self terminating episodes [7]

Duration less than 7 days with spontaneous termination [8]

2.PERSISTENT—

Prolonged episodes terminated by electrical or chemical Cardio version.

Duration greater than 7days [8]

3.PERMANENT:

Present all the time [9] Restoring sinus rhythm is either not possible or is not deemed appropriate.

4.LONE ATRIALL FIBRILLATION

AF in the absence of clinical or echocardiography findings of cardiopulmonary disease patients with LAF who are under 65 have best prognosis.

According to, American heart association, American college of cardiology and European society of cardiology have proposed the following classification system based on simplicity and clinical relevance:

1 FIRST DETECTED AYTRIAL FIBRILLATION:

Any patient newly diagnoses with atrial fibrillation fits in this category as the exact onset and chronicity of disease is often undertaken.

2. RECURRENT ATRIAL FIBRILLATION:

Any patient with 2 or more identified episodes of AF is called recurrent AF. It is classified into paroxysmal and persistent.

- **Paroxysmal—**terminates spontaneously within 7 days most commonly within 24 hrs.

- **Persistent or chronic AF—AF** established for more than 7 days.

3. LONE AF (LAF) [6,7]

GENETICS OF FAMILIAL ATRIAL FIBRILLATION: [10]

Mutations in the KCNQ1 , KCNE2 and KCNJ2 gene cause familial atrial fibrillation. In heart (cardiac) muscle, the ion channels produced from the KCNE2, KCNJ2, and KCNQ1 genes play critical roles in maintaining the heart's normal rhythm. Mutations in these genes have been identified in only a few families worldwide. These mutations increase the activity of the channels, which changes the flow of potassium ions between cells.

Familial atrial fibrillation inherited in autosomal dominant pattern.

PATHOPHYSIOLOGY: [11]

3 mechanism

1. Enhanced automaticity in the LA extending to proximal 5-6cm portion of pulmonary veins.
2. Electrical remodelling of the atria with resultant shortening of atrial refractory period increases the duration and stability of AF (Atrial fibrillation begets atrial fibrillation)
3. In chronic AF, areas of functional conduction block further divide and maintain persistently chronic electrical state.

I. ATRIAL FACTORS:

1. Atrial fibrosis:

The most frequent pathoanatomic changes in AF are atrial fibrosis and loss of atrial muscle mass.

Histologically shows – patchy fibrosis, juxtaposed with normal atrial fibrosis which may account for non homogeneity of conduction.

The SA and AV nodes may also be involved accounting for SICK SINUS SYNDROME and AV block.

Biopsy from LA post well revealed

Mild to moderate fibrosis in AF of short duration, severe fibrosis and loss of muscle mass in long standing AF.

Genetic diseases like lamin AC gene mutation play a role in atrial fibrosis.

Other trigger fibrosis includes inflammation as seen in sarcoidosis and auto immune disorders.

2. Amyloidosis

3. Hemochromatosis

4. Endomyocardial fibrosis

5. Atrial dilatation

This triggers the atrial fibrosis.

Atrial dilatation in any type of heart disease includes systemic Hypertension, Heart failure, valvular heart disease, CAHD. In this conditions stretch activates several molecular pathways, including renin angiotensin aldosterone system, Angiotensin II and transforming growth factor –B1 are unregulated in response to stretch and these molecules induce production of connective tissue growth factors.

ACE and angiotensin II receptor blockade had potential to prevent AF by reducing fibrosis.

ATRIAL MECHANISM:

1. Automatic focus theory:

In which the arrhythmia persist only in isolated regions of atrial myocardium.

This theory received minimal attention until the important observation that a focal source for AF could be identified in humans and ablation of this source could extinguish AF. While pulmonary veins are the most frequent source of these rapidly atrial impulses, foci have also been found in the **superior venacava, ligament of Marshall, left posterior free wall, christa terminalis, coronary sinus.**

2. Multiple –wavelet hypothesis:

The multiple wavelet hypothesis as the mechanism of re-entrant AF was advanced by more colleagues who proposed that fractionation of wave fronts propagating through the atria results in self perpetuating "**daughter wavelets"** The number of wavelets at anytime depends on refractory period, mass conduction velocity in different part of atria.

A large atrial mass with short refractory period and delayed conduction increases the number of wavelets, favouring sustained AF.

For many years, the multiple wavelet hypotheses were the dominant theory explaining the mechanism of AF.

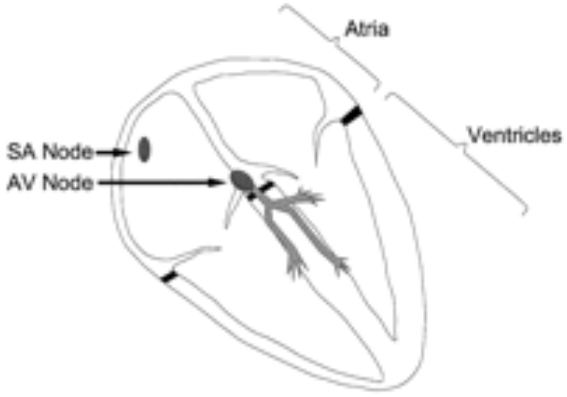


Figure 1. The atria contain the heart's natural pacemaker, the SA node, and are the part of the heart affected by atrial fibrillation. The ventricles are the muscular part of the heart that actually pumps the blood. They are electrically isolated from the atria, and the only way the electrical signal can reach them is via the AV node.

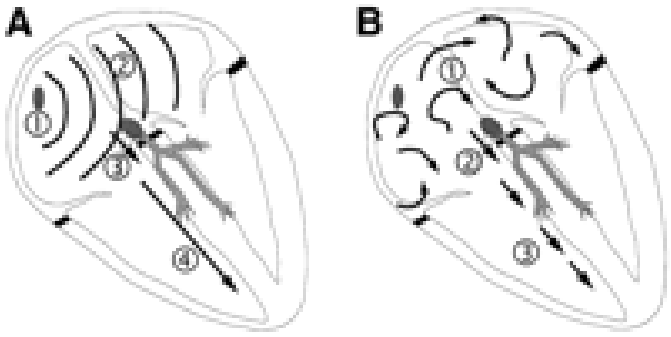


Figure 2. A, Sinus rhythm. During normal sinus rhythm, the heartbeat is a single carefully coordinated process beginning in the SA node (1). The electrical signal spreads across the atria (2) and via the AV node (3) to the ventricles (4). B, Atrial fibrillation. When patients are in AF, the atria are constantly activating in a chaotic

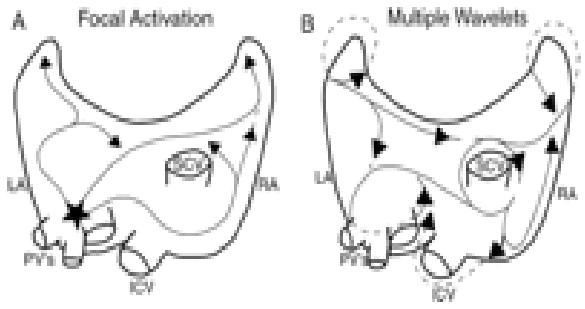


Figure 3. Posterior view of principal electrophysiological mechanisms of atrial fibrillation. Focal activation. The initiating focus (indicated by the star) often lies within the region of the pulmonary veins. The resulting wavelets represent fibrillatory conduction, as in multiple-wavelet re-entry. B, Multiple-wavelet re-entry. Wavelets (indicated by arrows) randomly renter tissue previously activated by the same or another wavelet. The routes the wavelets travel vary.

ATRIAL ELECTRICAL REMODELING:

After a period of rapid atrial rate, electrical remodelling of atria occurs. Which stimulates progressive intra cellular calcium current in terms shortens the action potential duration and atrial refractory period. This may promote sustained AF.Described by Phrase.

"Atrial fibrillation begets atrial fibrillation"

Tachycardia induced AF may result from AV node re-entry, an accessory pathway, atrial tachycardia, atrial flutter.

II. ATRIOVENTRICULAR CONDUCTION:

In the absence of accessory pathways or His- purkinge dysfunction, the AV node limits conduction during AF multiple atrial inputs to the AV node have been identified, two of which seem dominant, one directed posteriorly via the christa terminalis and the other aimed anteriorly via interatrial septum other factors affecting AV conduction are the intrinsic refractoriness of the AV node, concealed conduction, and autonomic tone. Concealed conduction which occurs when atrial impulses transverse part of the AV node but are not conducted to the ventricles and plays a prominent role in determining the ventricular response during AF.

These impulses after AV nodal refractoriness, slowing or blocking subsequent atrial impulses, and may explain the irregularity of ventricular response during AF. When the atrial rate is relatively slow during AF the ventricular rate is tend to rise, conversely a higher atrial rate is associated with slow ventricular rate.

Increased parasympathetic and reduced sympathetic tone exert negative dromotropic effects on AV nodal conduction, while the opposite is true in

states of decreased parasympathetic and increased sympathetic tone. Vagal tone is also enhances the negative chronotropic effects of

as exemplified by slow ventricular rate during sleep but accelerated ventricular response during exercise. Digitalis which slows the ventricular rate du concealed conduction in the AV node. Fluctuations in autonomic tone can produce disparate ventricular response to AF in a given patient ring AF predominantly by increasing vagal tone, is more effective for controlling heart rate at rest in AF but less effective during activity. Wide swings in rate due to variations in autonomic tone may create a therapeutic challenge.

III. Aberrant conduction with AF (*Ashman phenomenon)* [12]

The aberrant conduction was likely to complicate atrial flutter and fibrillation when a longer cycle was followed by a shorter cycle. Aberration produced by long- short sequence is sometimes referred as Ashman phenomenon. It is important to keep in mind that this cycle sequence cannot be used to differentiate aberration from ectopy because by the rate of bigemini, a lengthened cycle also tends to participate a ventricular extra systole therefore a long short sequence ending with wide QRS complex is as likely to represent [7]PB as an aberrant beat.

IV: AF with preexcitation: [7, 12]

(Atrioventricular conduction in patients with preexcitation)

Conduction across an accessory pathway during AF can result in a dangerously rapid ventricular rate. Whereas a substantial increase in sympathetic tone may increase e-existed ventricular response. Alterations in vagal tone have little effect on conduction over accessory pathways.

Transition of AV re-entry into AF in patients with **wolf-Parkinson-white syndrome** can produce a rapid ventricular response that degenerates into ventricular fibrillation, leading to death. Intravenous administration of drugs such as digitalis, verapamil, diltiazem, which lengthen refractoriness and slow conduction across the AV node, does not block conduction over the accessory pathway and may accelerate the ventricular rate. Hence these agents are contraindicated in this situation. It should be treated as emergency usually with DC cardio version.

V.Neurogenic Atrial Fibrillation [14, 15]

Coumel described a vagal and adrenergic form of AF.

Vagal origin of AF is characterized by

(1) Predominance in men rather than in women (approximately 4:1)

(2) Age at onset approximately 40 to 50 years

(3) Lone AF with minimal tendency to permanent AF

(4) Occurrence at night, during rest, after eating, or with absorption of alcohol; and

(5) AF usually preceded by progressive bradycardia. Importantly, both ß-adrenergic blocking drugs and digitalis may increase frequency of AF.

Adrenergic AF has the following features:

(1) Occurs less frequently than vagal AF;

(2) Onset is exclusively during daytime;

(3) Often preceded by exercise and emotional stress;

(4) Polyuria is common;

(5) Onset typically occurs with a specific sinus rate, often near 90 beats/min. In contrast to vagally induced AF, ß-adrenergic blockers are usually the treatment of choice.

Patients with "pure" vagally dependent or adrenergic AF are very uncommon. However, history taking may reveal a pattern of onset of AF that has elements of one of these syndromes. This is important because it allows the clinician to select agents that are more likely to prevent episodes of AF in these patients.

ETIOLOGY OF ATRIAL FIBRILLATION: [6, 7, 13]

I.CARDIAC

1. valvular heart disease (most often mitral valve disease)
2. Congestive cardiac failure
3. Coronary artery disease
4. Hypertension(particularly when LVH is present)
5. Hypertrophic cardiomyopathy
6. Dilated cardiomyopathy
7. Restrictive cardiomyopathy

   (Amyloidosis, Hemochromatosis, Endomyocardial fibrosis)
8. Congenital heart disease(ASD, MVP)
9. Myocarditis
10. Pericarditis
11. Cardiac tumours
12. WPW syndrome
13. After CABG

    (Occurs in up to 40% of patients primarily within 2-3 days) [6]
14. Calcification of mitral annulus
15. Idiopathic dilatation of RA

II.NON CARDIAC CAUSES: [16, 7, 13]

1. Thyrotoxicosis
2. Pheochromocytoma
3. Respiratory causes

   --Acute and chronic pulmonary disease (pneumonia, COPD)

   --pulmonary vascular disease (pulmonary embolism)
4. Electrolyte disturbances

   Hypokalemia,

   Hypocalcaemia

   Hypomagnesaemia
5. excessive alcohol intake

   --binge drinking or holiday heart syndrome
6. increase sympathetic tone

   --exercise, adrenergically mediated arrhythmia usually during daytime.
7. Increase parasympathetic tone

   --Vagally induced and post prandial arrhythmia.
8. Carbon monoxide poisoning
9. Hypothermia

10. Duel chamber pacemaker in the presence of normal AV conduction.
11. Caffeine
12. Smoking
13. Obesity

(Graded increase in LA size as BMI increases from normal to overweight and obese categories) 1,2,4,5

III.FAMILIAL: [6, 7, 9, 13]

Lone atrial fibrillation.

IV.OTHERS:

1. Duchene muscular dystrophy [6]
2. Myotonic dystrophy[6]
3. The fonton patient[6]

   (Palliative procedure that redirects the systemic venous return directly to the pulmonary arteries without passing through a sub pulmonary ventricle useful in tricuspid atresia, hypo plastic left heart syndrome, double inlet ventricle)
4. Electrocution

CLINICAL MANIFESTATIONS: [6, 11]

- Palpitations
- Chest pain
- Dyspnoea
- Fatigue
- Light headedness or syncope
- Polyuria(may be associate with the release of atrial natriuretic peptide[11] particularly as episodes of AF begin or terminate)
- Features of embolic complication (stroke or TIA)
- Features of exacerbation of heart failure.

PHYSICAL FINDINGS: [6, 8]

1. Irregular pulse with or without tachycardia, is typically described as irregularly irregular rhythm.
2. Significant pulse deficit.
3. Variation in intensity of first heart sound.
4. Absence of ‘a’ wave in JVP.
5. Hypotension and poor perfusion caused by decrease in atrial filling pressures and decrease in stroke volume are common

findings. This may be either rate related or because of lack of normal atrial kick.

6. Congestive heart failure, if present may be indicated by rales, jugular venous distension, peripheral edema, and gallop, which may be difficult to auscultate due to rapid rate.
7. Signs of embolisation, including TIA, stroke, and peripheral arterial embolisation may be indicated.

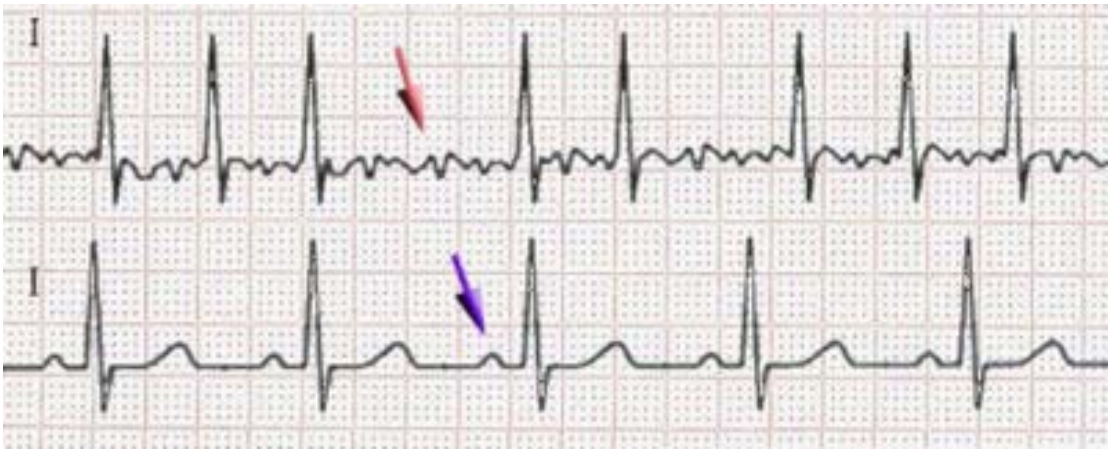
INVESTIGATIONS: [8, 16]

1. Complete blood count (looking for anaemia, infection)
2. Blood urea and serum creatinine, electrolytes
3. Cardiac enzymes—CK and /or troponin level

   (To investigate myocardial infarction as a primary or secondary event)
4. Thyroid function test. [15]
5. Digoxin level when appropriate

   (To look for sub therapeutic level and/ or toxicity). It is generally considered safe to administer Digoxin to a patient with AF on Digoxin for rare control without waiting for a level to return from the laboratory when patient presents with AF. [8]
6. Toxicology screening [8]

7. ECG –



- Absent ‘p’ waves
- Replace by irregular chaotic fibrillatory ‘F’ waves, in the setting of irregular QRS complexes.
- Look for aberrantly conducted beats after long – short R-R cycles(Ashman phenomenon)
- Evidence of LVH, preexcitation, bundle branch blocks, acute or prior myocardial infarction. And intervals (R-R, QRS, QT)

8. Holter monitoring or event monitoring may be considered for those discharged from emergency department.

If paroxysmal AF is suspected but the electrocardiogram shows a regular rhythm, episodes may be documented with the use of Holter monitoring (continuous ECG recording for 24 hours). If the symptoms are very infrequent, longer periods of continuous monitoring may be required.

9. Exercise testing might also be used in the outpatient setting to determine adequacy of rate-control, to reproduce exercise induced AF, and to exclude ischemic pathology.

10. Chest X- ray -- may be normal

Radiographic evidence of CHF, RHD, cardiomyopathy, lung or vascular pathology.

In particular, if an underlying pneumonia is suggested, and then treatment of the pneumonia may cause the atrial fibrillation to terminate on its own.

11. Echocardiography may be used to evaluate for

- Valvular heart disease
- Right and left atrial size and function.
- LV size and function

- LVH, RVH
- Pericardial disease
- Vegetations
- thrombus

12. Trans oesophageal echocardiography—

    High sensitivity in detecting LA thrombus.

DIFFERENTIAL DIAGNOSIS:

1. Atrial extra systole
2. Atrial tachycardia(multifocal)
3. Atrial flutter
4. WPW syndrome

COMPLICATION:

1. Stroke
2. Tachycardia induced cardiomyopathy
3. Heart failure

TREATMENT:

The main goals of treatment of atrial fibrillation are to prevent temporary circulatory instability and to prevent stroke. Rate and rhythm control are principally used to achieve the former, while anticoagulation may be required to decrease the risk of the latter. In emergencies, when circulatory collapse is imminent due to uncontrolled tachycardia, immediate cardioversion may be indicated. [18]

The primary factors determining atrial fibrillation treatment are duration and evidence of hemodynamic instability. Cardioversion is indicated with new onset AF (for less than 48 hours) and with hemodynamic instability. If rate and rhythm control can not be maintained by medication or cardioversion, electrophysiological studies with pathway ablation may be required. **[18]**

Prehospital Care [8]

- Care of hemodynamically unstable patients is guided by ACLS protocols, including direct current (DC) cardio version.
- Symptomatic patients may benefit from intravenous (IV) rate-controlling agents, either calcium-channel blockers or beta-adrenergic blockers.

Emergency Department Care [8]

The immediate role of the emergency medicine physician is to ascertain and ensure hemodynamic stability. Once this is done, the approach to atrial fibrillation is facilitated by generally accepted protocols.

- ***Urgently assess need for interventions, including the following:***
  - Airway and oxygenation (pulse oximetry); O_2 supplementation as needed
  - Blood pressure support (often difficult until rate is controlled)
  - A patient with hemodynamic instability, mental status changes, preexcitation, or angina will require urgent synchronized DC cardio version.
    - Obtain emergent laboratory and imaging studies (ECG, chest radiography).
    - Once the patient has been determined to be hemodynamically stable, often the first step to control ventricular tachycardia is the administration of a rate-controlling agent (a beta-blocker or a calcium-channel blocker). In a small percentage of cases, the patient may revert to sinus in response to a slowing down of the rate.

- If the patient persists in AF, anticoagulation should be initiated with either intravenous or subcutaneous heparin. The treatment branch then divides patients into duration of AF. Patients with duration of less than or equal to 48 hours (and without significant LV dysfunction, mitral valve disease, or prior embolism) may undergo immediate pharmacologic or DC cardio version. AF of duration of greater than 48 hours, or unknown duration, or high risk of embolisation can either undergo TEE-guided DC cardio version or be anticoagulant for 3 weeks, followed by DC cardio version. Multiple studies have shown the safety/efficacy of TEE-facilitated early cardio version versus conventional therapy. Although patients who have been on anticoagulation already for at least 3 weeks can be cardioverted without TEE, if any complicating factor (e.g., valvular disease, LV dysfunction) is present, TEE should be considered.
- Methods of cardio version: Cardio version may be achieved by means of drugs or electrical shocks. Electrical cardioversion is more effective than pharmacologic cardioversion, though it requires sedation/anesthesia.

ELECTRICAL CARDIOVERSION

- Direct-current cardio version involves an electrical shock synchronized with the intrinsic activity of the heart to ensure that electrical stimulation does not occur during the vulnerable phase of the cardiac cycle. The success rate of DC cardio version is 75-93%, with efficacy positively correlating to energy setting (360>200>100 J), and negatively correlating with duration of AF and left atrial size.
- Paddle positions include anterior-lateral (ventricular apex and right infraclavicular) and anterior-posterior (sternum and left scapular), with at least one study suggesting increased efficacy with the anterior-posterior method.
- Cardio version of patients with implanted pacemakers and defibrillator devices is safe when appropriate precautions are taken. Risks of electrical cardio version include embolic events and cardiac arrhythmias. Transient ST-segment elevations can be present after cardio version, as well as a bump in cardiac enzyme levels without apparent myocardial damage.
- The relapse rate after initial successful cardioversion is high: 25%-50% at 1 month and 70%-90% at 1 year.

- While successful cardioversion in AF of duration greater than 48 hours will require 6-12 weeks of anticoagulation due to atrial stunning, no clear consensus exists for anticoagulation after cardioversion for AF of less than 48 hours. One approach is to divide these patients into low-risk (treat with aspirin) and higher-risk (traditional anticoagulation).

PHARMACOLOGIC CARDIOVERSION

- Pharmacologic cardioversion appears to be most effective when initiated within 7 days after the onset of AF. .
- The risk factors for pharmacologic cardioversion include Brady arrhythmia, QT prolongation, and ventricular arrhythmias.

A study by Michael et al looked at 289 patients seen during an 18-month period in an emergency department setting. Sixty-two percent (180) underwent attempted chemical cardioversion with a 50% success rate, and 28% (80) had attempted electrical cardioversion with a 89% success rate. Ninety-three percent of electrical cardioversions were performed by emergency physicians. They concluded that cardioversion and immediate discharge of patients who present to the ED with acute atrial fibrillation appears to be both safe and effective.

Reasons for hospitalization would include but not be limited to the following:

- Presence of co morbidities
- For workup or treatment of underlying aetiology of AF, including evaluation for ACS or myocardial infarction
- For elderly patients
- For patients with underlying heart disease
- Patients at risk of complications from AF therapies

CONSULTATIONS [8]

- Patients with AF who are treated in the ED generally require consultation with a cardiologist. Those with new-onset AF and those with associated symptoms related to rate are generally admitted to rule out MI and to evaluate for possible elective cardioversion.
- A patient's cardiologist plays a vital role in determining the most appropriate long-term strategy for a patient with AF and provides crucial follow-up.
- A cardiologist may become involved emergently if complicating factors are present or if the patient is experiencing ongoing cardiac

ischemia or infarction not treatable with rate reduction measures and standard chest pain protocols. A patient with AMI and new-onset AF who is stable may benefit from simple [8, 16]e rate-control measures (e.g., intravenous beta-blockers, intravenous magnesium sulphate 2 g over 10 min) while being prepared for the catheterization laboratory and intravenous nitrates, heparin, and aspirin are begun.

MEDICATION:

Pharmacologic agents in AF fall into 1 of 2 classes: rate-controlling drugs and rhythm restoring/rhythm maintenance drugs (though some overlap exists with some drugs, such as amiodarone, exhibiting both qualities).

RATE-CONTROLLING DRUGS

In patients without ventricular preexcitation, rate is controlled most effectively with intravenous verapamil, diltiazem, or beta-adrenergic blockers. Beta-blockers are especially effective in the presence of Thyrotoxicosis and increased sympathetic tone or in patients with myocardial ischemia/AMI. The non-hydro pyridine calcium channel blockers may be chosen in patients lacking any history of heart failure and in patients with reactive airway disease.

Anecdotally, intravenous diltiazem has become many emergency medicine physicians' first-line rate-controlling drug in patients without a history of heart failure.

Digoxin is ineffective in controlling ventricular rate during acute episodes.

In patients with acute or chronic heart failure, Digoxin or amiodarone should be used. (Amiodarone does not currently have FDA approval for this intervention.)

ANTIARRHYTHMIC DRUGS

Antiarrhythmic drugs that can terminate AF include procainamide, disopyramide, propafenone, sotalol, flecainide, amiodarone, and ibutilide. The efficacy of antiarrhythmic drugs has been linked to the duration of AF.

The American College of Cardiology/American Heart Association / European Society of Cardiology (ACC/AHA/ESC) Guidelines make the following recommendations regarding pharmacologic conversion of AF:

-
-

- conversion of AF less than or equal to 7 days
    - Agents with proven efficacy include dofetilide, flecainide, ibutilide, propafenone, and to a lesser degree, amiodarone and quinidine.
    - Less effective or incomple udied agents in this scenario include procainamide, digoxin, and sotalol.

- Conversion of AF lasting greater than 7 days
    - Agents with proven efficacy include dofetilide, amiodarone, ibutilide, flecainide, propafenone, and quinidine.
    - Less effective or incompletely studied agents in this scenario include procainamide, sotalol, and digoxin.
- Conversion of AF lasting greater 90 days - Oral propafenone, amiodarone, and dofetilide have been shown to be effective at converting chronic AF to normal sinus rhythm (NSR).

A newer agent and recently approved class III antiarrhythmic is dofetilide. Another drug, azimilide, has been studied in the recent Azimilide Post-Infarct Survival Evaluation (ALIVE) trial, a post–heart attack survival study. Additional data from ALIVE further support the ongoing development of azimilide as a treatment for supraventricular

arrhythmias. Fewer patients in sinus rhythm at baseline developed AF/atrial flutter during the trial on azimilide compared with placebo.

Another newer drug is ***dronedarone***, a deiodinated derivative of amiodarone that has no organ toxicity. Its use extends to atrial and ventricular arrhythmias. At present, dronedarone is an experimental agent that has multiple actions (all 4 Von Williams class effects). Unlike amiodarone, it does not have the iodine moiety. The lack of iodination may offer a better adverse-effect profile. Dronedarone has been shown to (1) have antiadrenergic effects

(2) Prolong atrial and ventricular refractory periods, and

(3) prolong atrioventricular node conduction and the paced QRS complex.

In animal models, dronedarone has been shown to decrease ischemia-induced ventricular arrhythmias. The clinical effects of dronedarone are now being examined in patients with AF and in patients with internal cardioverter-defibrillators (ICDs).

When considering drug therapy for AF, remember the treatment caveat: "Electrical cardioversion is the preferred modality in the patient whose condition is unstable."

ANTICOAGULATION[25 ,26,27,28,29,30,31,32]

Patients with atrial fibrillation, even lone atrial fibrillation without other evidence of heart disease, are at increased risk of stroke during long term follow up. A systematic review of risk factors for stroke in patients with nonvalvular atrial fibrillation concluded that a prior history of stroke or TIA is the most powerful risk factor for future stroke, followed by advancing age, hypertension, and diabetes. The risk of stroke increases whether the lone atrial fibrillation was an isolated episode, recurrent, or chronic. The risk of systemic embolization (atrial clots migrating to other organs) depends strongly on whether there is an underlying structural problem with the heart (e.g. mitral stenosis) and on the presence of other risk factors, such as diabetes and high blood pressure. Finally, patients under 65 are much less likely to develop embolization compared with patients over 75. In young patients with few risk factors and no structural heart defect, the benefits of anticoagulation may be outweighed by the risks of hemorrhage (bleeding). Those at a low risk may benefit from mild (and low-risk) anticoagulation with aspirin (or clopidogrel in those who are allergic to aspirin). In contrast, those with a high risk of stroke derive most benefit from anticoagulant treatment with warfarin or similar drugs.

In the United Kingdom, the NICE guidelines recommend using a clinical prediction rule for this purpose. The *CHADS/CHADS2* score is the best validated clinical prediction rule for determining risk of stroke (and therefore who should be anticoagulated); it assigns points (totaling 0-6) depending on the presence or absence of co-morbidities such hypertension and diabetes. In a comparison of seven prediction rules, the best rules were the CHADS2 which performed similarly to the SPAF and Framingham [15] prediction rules. To compensate for the increased risk of stroke, anticoagulants may be required. However, in the case of warfarin, if a patient has a yearly risk of stroke that is less than 2%, then the risks associated with taking warfarin outweigh the risk of getting a stroke.

ACUTE ANTICOAGULATION

If anticoagulation is required urgently (e.g. for cardioversion), heparin or similar drugs achieve the required level of protection much quicker than warfarin, which may take several days to reach adequate levels.

In the initial stages after an embolic stroke, anticoagulation may be risky, as the damaged area of the brain is relatively prone to bleeding (hemorrhagic transformation). As a result, a clinical practice guideline by National Institute for Health and Clinical Excellence recommends that

anticoagulation should begin two weeks after stroke if no hemorrhage occurred.

CHRONIC ANTICOAGUATION

Among patients with "non-valvular" atrial fibrillation, anticoagulation can reduce stroke by 60% while antiplatelet agents can reduce stroke by 20%. There is evidence that aspirin and clopidogrel are effective when used together, but the combination is still inferior to warfarin.

Warfarin treatment requires frequent monitoring with a blood test called the international normalized ratio (INR); this determines whether the correct dose is being used. In atrial fibrillation, the usual target INR is between 2.0 and 3.0 (higher targets are used in patients with mechanical artificial heart valves, many of whom may also have atrial fibrillation). A high INR may indicate increased bleeding risk, while a low INR would indicate that there is insufficient protection from stroke.

ELDERLY PATIENTS

The very elderly (patients aged 75 years or more) may benefit from anticoagulation provided that their anticoaguation does not increase hemorrhagic complications, which is a difficult goal. Patients aged 80 years or more may be especially susceptible to bleeding complications,

with a rate of 13 bleeds per 100 person-years. A rate of 13 bleeds per 100 person years would seem to preclude use of warfarin; however, a randomized controlled trial found benefit in treating patients 75 years or over with a number needed to treat of 50. Of note, this study had very low rate of hemorrhagic complications in the warfarin group.

RATE CONTROL VERSUS RHYTHM CONTROL

AF can cause disabling and annoying symptoms. Palpitations, angina, lassitude (weariness), and decreased exercise tolerance are related to rapid heart rate and inefficient cardiac output caused by AF. Furthermore, AF with a persistent rapid rate can cause a form of heart failure called tachycardia induced cardiomyopathy. This can significantly increase mortality and morbidity, which can be prevented by early and adequate treatment of the AF.

There are two ways to approach these symptoms: rate control and rhythm control. *Rate control* treatments seek to reduce the heart rate to normal, usually 60 to 100 beats per minute. *Rhythm control* seeks to restore the normal heart rhythm, called normal sinus rhythm. Studies suggest that rhythm control is mainly a concern in newly diagnosed AF, while rate control is more important in the chronic phase. Rate control

with anticoagulation is as effective a treatment as rhythm control in long term mortality studies, the AFFIRM Trial.

The AFFIRM study showed no difference in risk of stroke in patients who have converted to a normal rhythm with anti-arrhythmic treatment, compared to those who have only rate control.

RATE CONTROL

Rate control is achieved with medications that work by increasing the degree of block at the level of the AV node, effectively decreasing the number of impulses that conduct down into the ventricles. This can be done with:

- Beta blockers (preferably the "cardioselective" beta blockers such as metoprolol, atenolol, bisoprolol)
- Cardiac glycosides (i.e. digoxin)
- Calcium channel blockers (i.e. diltiazem or verapamil)

In addition to these agents, amiodarone has some AV node blocking effects (particularly when administered intravenously), and can be used in individuals when other agents are contraindicated or ineffective (particularly due to hypotension).

CARDIOVERSION

Rhythm control methods include electrical and chemical cardioversion:

- *Electrical cardioversion* involves the restoration of normal heart rhythm through the application of a DC electrical shock.
- *Chemical cardioversion* is performed with drugs, such as amiodarone, dronedarone[26], procainamide, ibutilide, propafenone or flecainide.

The main risk of cardioversion is systemic embolization of a thrombus (blood clot) from the previously fibrillating left atrium. Cardioversion should not be performed without adequate anticoagulation in patients with more than 48 hours of atrial fibrillation. Cardioversion may be performed in instances of AF lasting more than 48 hours if a transesophogeal echocardiogram (TEE) demonstrates no evidence of clot within the he.

New understanding of the mechanism of atrial fibrillation

While it was once thought that atrial fibrillation was initiated by areas of electrical activity (foci) located all over the atria, research by Haissaguerre and co-workers proved that the majority of irregular foci (94%) come from the areas around the four pulmonary veins. Other less

common areas include the superior vena cava, right and left atrium, and the coronary sinus. This information has led to new ablation and surgical techniques to treat atrial fibrillation, including pulmonary vein ablation.

PULMONARY VEIN ISOLATION ABLATION

Ablation of the four pulmonary veins, using a circumferential mapping technique, is proving to be successful for many patients with atrial fibrillation.

The procedure involves the use of special catheters (soft wires) inserted into the left atrium. The catheters are used for mapping (searching for the electrical impulses that fire abnormally, causing atrial fibrillation) and the delivery of energy (ablation) to the area. Energy is delivered from one catheter into the area of the atria that connects to the pulmonary vein, producing a circular scar. The scar will then block any impulses firing from within the pulmonary vein, thus preventing atrial fibrillation from occurring. The process is repeated to all pulmonary veins.

Dr. Andrea Natale, department of Cardiovascular Medicine, section of Electrophysiology and Pacing, says, "This procedure has an 80% success rate with the first ablation. For those who have returned for

further ablation, the success rate has been 95%. After a period of recovery following the procedure, most patients can stop taking blood thinners."

Dr. Natale is investigating other methods of pulmonary vein ablation. The goal is to isolate the energy delivered to the ostium (opening) of the pulmonary veins. Circumferential Ultrasound Vein Ablation (CUVA) involves using a circular ultrasound catheter inserted into the pulmonary veins and delivering energy around the pulmonary vein ostium. Although this appeared promising, initial studies showed a high rate of pulmonary vein stenosis (narrowing) occurred. Future research is aimed at improving this technique.

SURGICAL PROCEDURES

Several factors have contributed to surgical approaches to treat atrial fibrillation:

- atrial fibrillation is very common and those with atrial fibrillation often have concurrent heart disease (coronary artery disease or valve disease) requiring surgery
- all patients with atrial fibrillation are not treatable by non-surgical methods
- new energy sources are available, increasing the safety and success rate

The Maze procedure (Cox-Maze procedure), developed by Dr. Jim Cox, began the surgical approach to treatment of atrial fibrillation. The surgery involves creating precise incisions in the right and left atria to interrupt the conduction of abnormal impulses and to direct normal sinus impulses to travel to the atrioventricular node (AV node) as they normally should. The Maze procedure has been very successful with a 95 % success rate. A number of surgeons have altered the traditional Cox-Maze procedure to focus mainly on the left atrium.

The success of the modified Maze procedures has supported the notion that the isolation of the pulmonary veins and portions of the left atrium can eliminate atrial fibrillation. This has encouraged surgeons to seek out other methods of isolation rather than cutting and sewing. Three alternative energy sources have been used surgically to treat atrial fibrillation: radiofrequency, microwave and cryothermy. The goal of all three is to produce lesions and ultimately scar tissue to block the abnormal electrical impulses from being conducted through the heart and promote the normal conduction of impulses through the proper pathway.

Radiofrequency ablation uses radiofrequency energy to heat the tissue and produce lesions on the heart, eliminating the incisions necessary in the Maze procedure. There is a variety of surgical techniques related to the type of catheter used, the dose of energy, and the types of

lesions created. Radiofrequency surgical ablation has proved to be successful in 80 % of cases. The greatest risk of this procedure is damage to surrounding structures, such as the oesophagus.

Cryothermy (also called cryoablation) uses very cold temperatures through a probe (called a cryoprobe) to create lesions. This technique is used commonly during arrhythmia surgery to replace the incisions made during the Cox-Maze procedure. This technique cures atrial fibrillation in close to 80% of people.

Microwave technology uses a special catheter (the Flex-4 catheter) to direct microwave energy to create several lesions on the heart. The lesions block the conduction of abnormal electrical beats and restore a normal heartbeat. The benefit of microwave radiation in comparison to other surgical ablation techniques, is that the depth and volume of heated tissue is more controlled and precise lesions are created, and less charring of the heart's surface occurs, decreasing the risk of blood clots that may travel to the brain or other organs. Microwave energy cures atrial fibrillation in about 80 % of people.

MORTALITY/MORBIDITY

- The rate of ischemic stroke among patients with nonrheumatic AF averages 5% a year, which is somewhere between 2-7 times the rate of stroke in patients without AF. The risk of stroke is not due solely to AF; it increases substantially in the presence of other cardiovascular disease.

- The attributable risk of stroke from AF is estimated to be 1.5% for those aged 50-59 years, and it approaches 30% for those aged 80-89 years.

- The total mortality rate is approximately doubled in patients with AF compared with patients in normal sinus rhythm and is linked with the severity of underlying heart disease.

- AF complicates acute myocardial infarction (AMI) in 5-10% of cases. The causes of AF in AMI are thought to be due to any number of factors, such as atrial infarction, atrial ischemic injury, atrial distension, or, perhaps, Pericarditis. According to Rathore et al, patients who developed new-onset AF during the course of myocardial infarction (MI) were at higher risk than patients who presented with chronic AF. Patients with AMI and AF tend to be

older, be less healthy, and have poorer outcomes during hospitalization and after discharge than individuals without AF. AF is independently associated with an increased mortality rate.

MATERIALS AND METHODS

MATERIALS AND METHODS

This study was conducted at Coimbatore medical college hospital, Coimbatore. This study was conducted during period of Jan 2006 to June 2007, 50 cases with atrial fibrillation were included in this study.50 consecutive cases were recorded. No patients had been counted twice if he or she got admitted again after discharge during this period.

Inclusion Criteria

Both male and female patients were included in this study.

Samples were collected from medical OP, medical ward, ICCU, cardiology OP.

Exclusion Criteria

Paediatric patients were not included in this study.

THE DIAGNOSIS OF AF:

The diagnosis was made on clinical grounds and then confirmed by ECC and Echo cardiogram.

Clinical Features:

The following symptoms were enquired from all the patients. Those include dyspnoea, palpitation, chest pain, fatigue, dizziness, neurological deficit, oliguria.

The presence of following signs was made out. That includes pedal edema, puffiness of face, cyanosis, anaemia, signs of hyperthyroidism.

Heart rate, pulse rate, pulse deficit, blood pressure monitoring, JVP-absent “a” wave, cardio vascular system examination were documented in all the patients.

ECG RECORDING:

A, 12, lead ECC was taken for all the cases. It was standardized to produce a deflection of 10 mm per 1MV input and the paper speed was set at 25 mm per second. The ECC features of AF were noted, it includes

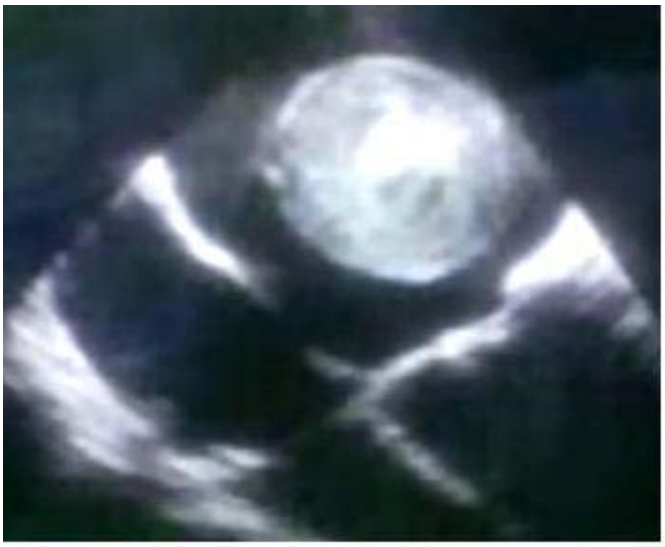
- Absent P wave
- Replaced by irregular chaotic fibrillatory F waves, in the setting of irregular QRS complex.
- Look for LVH, free excitation, bundle branch blocks, acute or prior myocardial infarction

ECHOCARDIOGRAPHY:

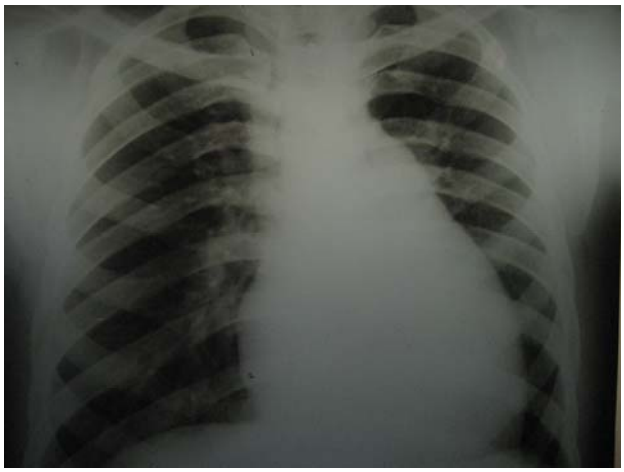
- ✓ M-mode, 2D echo was done in all the patients.
- ✓ The rhythm of heart was noted.
- ✓ The presence of valve thickening and calcification and regurgitation were noted.
- ✓ Size of valve orifice and chambers of heart were assessed.
- ✓ Presence of clot in the atrium and atrium appendages was identified.
- ✓ Vegetations were searched.
- ✓ Ejection fraction of ventricle was measured.

FEATURES ACCORDING TO SUSPECTED ETIOLOGY:

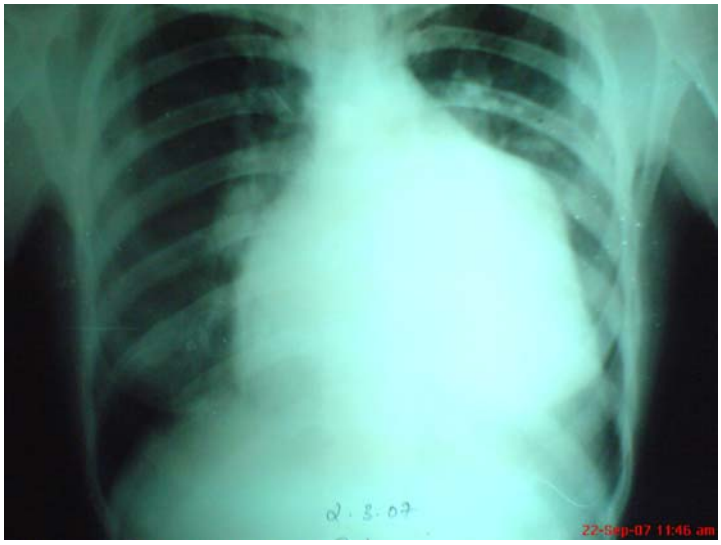
1. Rheumatic heart disease:
    - Features of rheumatic fever (as per updated Jones criteria)
    - Features of heart failure (as per Framingham criteria)
    - The presence of valvular heart disease
    - Features of infective endocarditis
    - Serum ASO titre, ESR, CRP



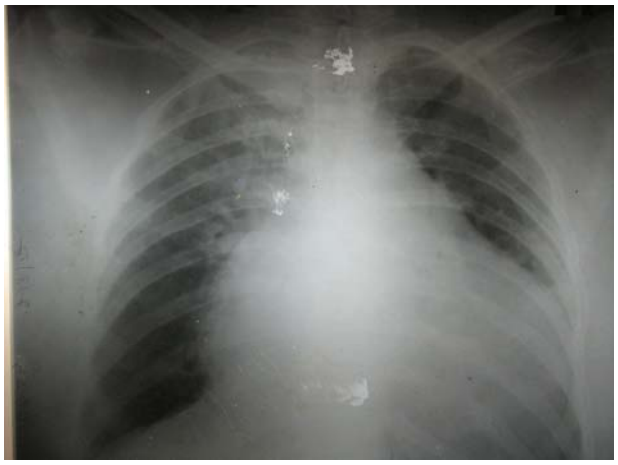
- X-ray chest PA view (chamber hypertrophy, cardiomegaly, mitralisation of heart)



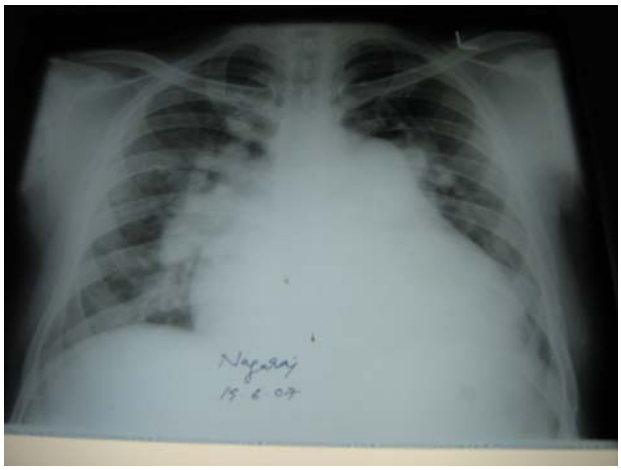
X-RAY SHOWS STRAIGHTENING OF LEFT BORDER OF HEART IN MITRAL STENOSIS



X-RAY SHOWS LEFT ATRIAL ENLARGEMENT IN A CASE OF MITRAL STENOSIS WITH REGURGITATION



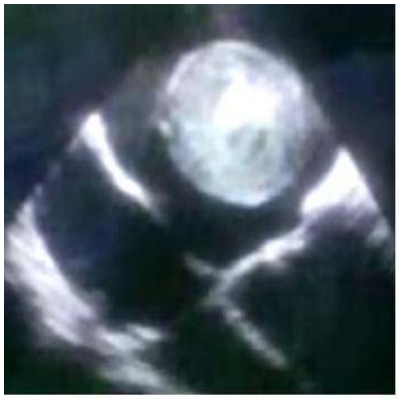
X-RAY SHOWS ISCHEMIC CARDIOMYOPATHY



X-RAY OF ATRIAL SEPTAL DEFECT [JUG HANDLE APPEARANCE]

ECHO CARDIOGRAPHY OF RHD PATIENT[MS+MR]

SHOWS LARGE CLOT

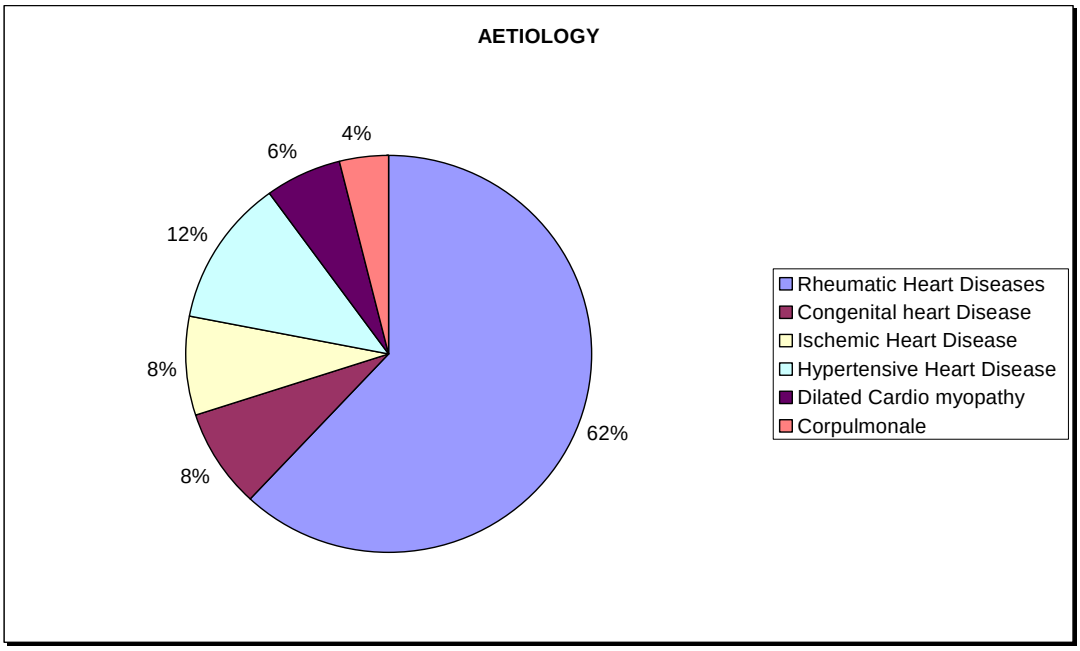


2. Coronary artery hypertensive disease:
    - History
    - Peripheral arterial thickening
    - Auscultation for S3,S4 (Which may denotes compliance of ventricle)
    - Fundus examination
3. Hypertensive heart disease
    - BP monitoring
    - Fundus examination
    - Urine analysis
    - Blood urea and creatinine level
    - If necessary other investigation to find out whether hypertension is primary or secondary
4. Chronic obstructive air way disease
    - History related to chronic lung disease
    - Chest wall deformities
    - Cardiac auscultation to find out pulmonary hypertension, pulmonary regurgitation, tricuspid regurgitation
    - ECC – P- pulmonale, RVH, RBBB, X- old PT, fibrosis, emphysematous chest, Bronchectasis

DATA ANALYSIS

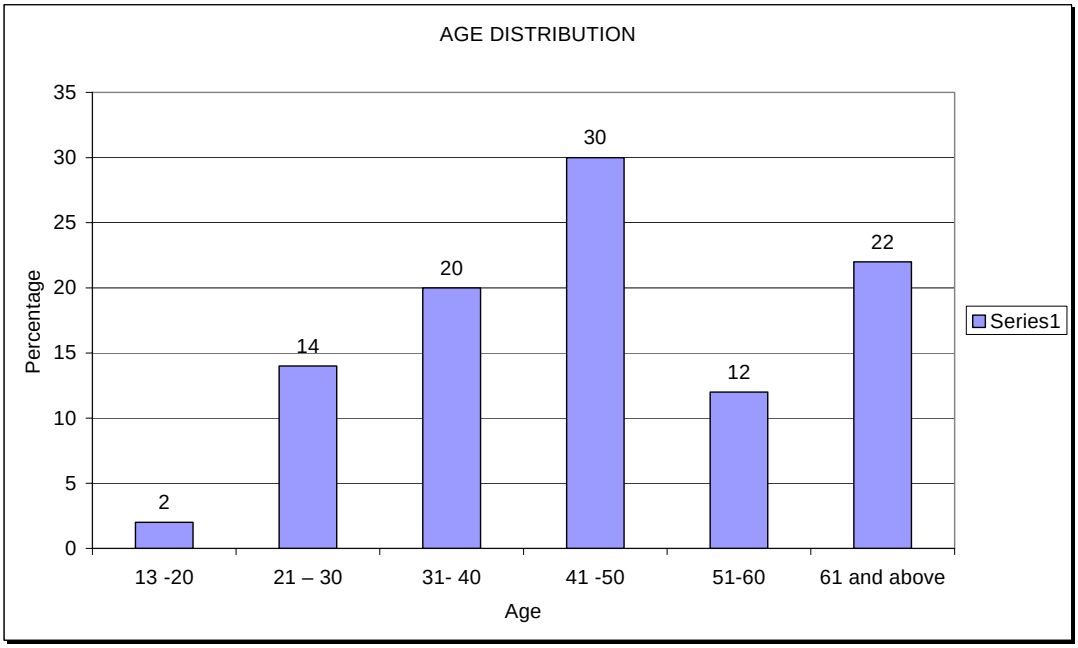
AETIOLOGY:

CAUSES	NO.OF CASES	PERCENTAGE
Rheumatic Heart Diseases	31	62
Congenital heart Disease	4	8
Ischemic Heart Disease	4	8
Hypertensive Heart Disease	6	12
Dilated Cardio myopathy	3	6
Corpulmonale	2	4



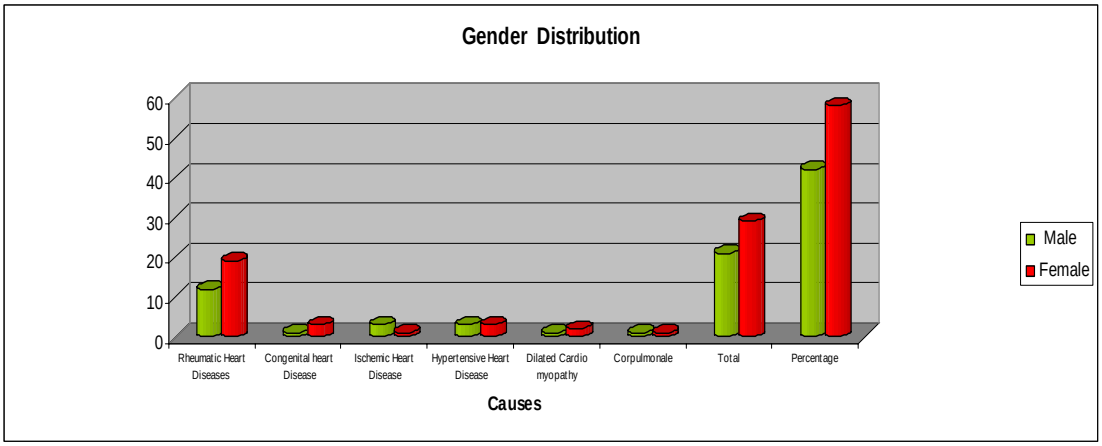
AGE DISTRIBUTION:

AGE IN YEARS	NO.OF CASES	PERCENTAGE
13 -20	1	2
21 – 30	7	14
31- 40	10	20
41 -50	15	30
51-60	6	12
61 and above	11	22



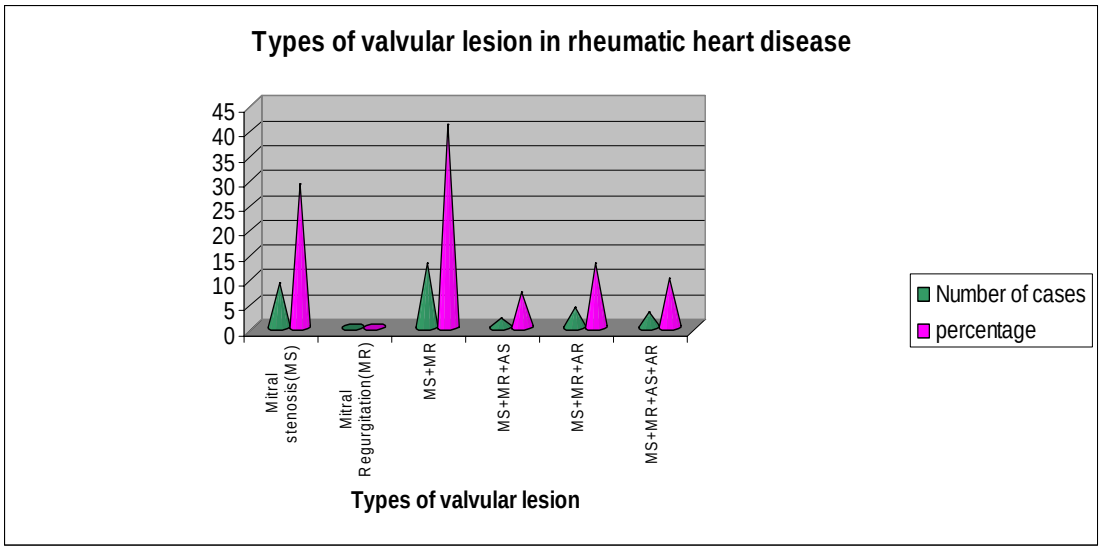
GENDER DISTRIBUTION

CAUSES	MALE	FEMALE
Rheumatic Heart Diseases	12	19
Congenital heart Disease	1	3
Ischemic Heart Disease	3	1
Hypertensive Heart Disease	3	3
Dilated Cardio myopathy	1	2
Corpulmonale	1	1
Total	21	29
Percentage %	42	58



TYPES OF VALVULAR LESION IN RHEUMATIC HEART DISEASE

Types of valvular lesions	Number of cases	Percentage
Mitral stenosis(MS)	9	29
Mitral Regurgitation(MR)	0	0
MS+MR	13	41
MS+MR+AS	2	7
MS+MR+AR	4	13
MS+MR+AS+AR	3	10

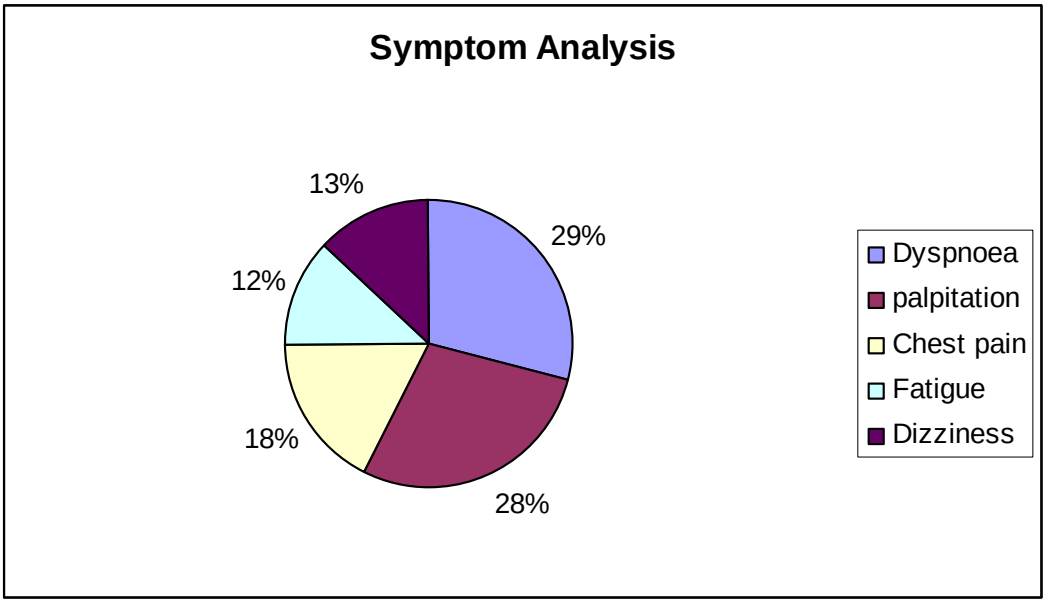


PREVIOUS HISTORY OF RHEUMATIC FEVER

No Of Cases Of RHD	History Of Rheumatic fever	Percentage
31	13	42

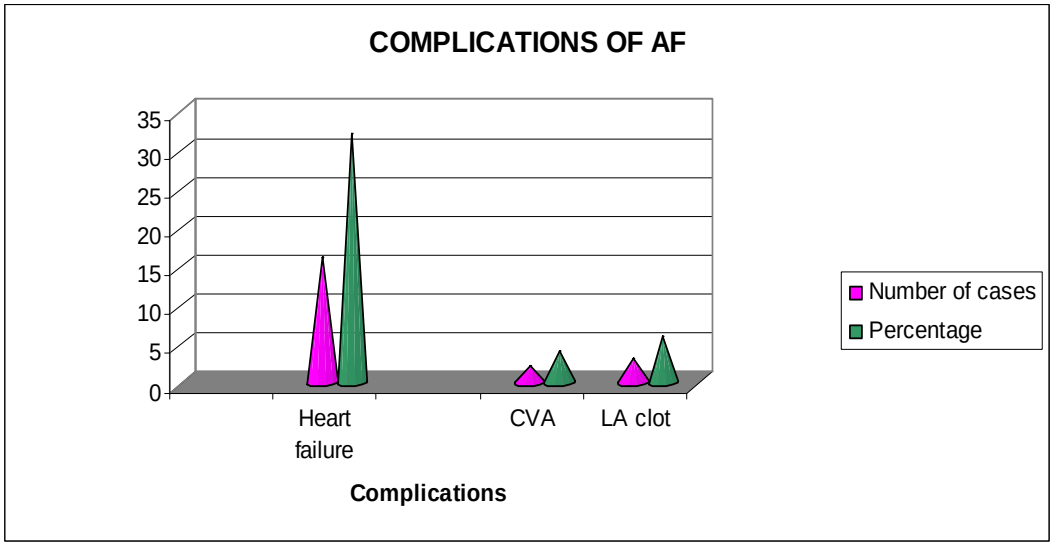
SYMPTOM ANALYSIS

SYMPTOMS	NUMBER OF CASES	PERCENTAGE
DYSPNOEA	45	90
PALPITATION	43	86
CHEST PAIN	27	54
FATIGUE	19	38
DIZZINESS	20	40



COMPLICATIONS

Complication	Number of cases	Percentage (%)
Heart failure	16	32
CVA	2	4
LA clot	3	6



DISCUSSION

DISCUSSION

An attempt has been made to study fifty cases of AF regarding aetiology, clinical manifestations, and complications.

AGE INCIDENCE:

In this study of atrial fibrillation cases, the occurrence of AF is maximum in age group 41-50 yrs. The incidence is about 30%. The next commonly affected age group is elderly people of 61yrs and above. The incidence is around 22%.

According to American heart association the cross sectional studies have found low prevalence in those below the age of 65yrs and, increasing to 8% in those older than 80 yrs.

According to AG shaper, HSR Huts zfezfar when 2 series of patients with AF were analysed they found mean age of patients seen in johanesberg was 38 yrs, where as in London it was 62 yrs.

The mean prevalence of AF of 0.5% for the group aged 50-59 years and rises to 8.8% in the group aged 80-89 years.

GENDER INCIDENCE:

Out of 50 cases 28 cases were female, 22 cases were male, the incidence in female is about 58% and incidence in male is about 42%.

The incidence of familial form of AF is unknown recent studies from department of health and human service- USA govt suggest that up to 30 % of all people with AF may have history of similar condition in their family.

According to Kannel.WB, AbbotR.D, savage, Mc.namara PM. epidemiologic features of chronic AF, The Framingham study N.Eng. J. Med 1982; 306; 1088-1122.

The prevalence of AF increases with age, and slightly more common in men than in women.

The incidence of development of AF over 22yrs in Framingham study was 2.2% in man and 1.7 in women.

AETIOLOGICAL ANALYSIS:

In the etiological analysis among 50 cases of AF, the most common aetiology was rheumatic heart disease, followed by Hypertensive heart disease, ischemic heart disease, congenital heart disease, DCM, corpulmonale.

RHEUMATIC HEART DISEASE:

Out of 50 cases 31 cases were Rheumatic heart disease. Incidence of RHD- 62%

R.Arora, G. Subramanian, M.Khalilullah and M.P Gupta from India reported the high incidence of RHD in India.

AG. Shaper, HSR Hutt, Zfejfar reported that the high incidence of RHD is common in tropical countries.

In western countries, coronary heart disease and hypertensive heart disease is common cause of AF than RHD.

A study was conducted in Govt medical college /Amrister Jan 2007 66 cases of AF analysed; they reported that RHD was the most common cause.

In this study of 50 cases of AF 31 cases were of rheumatic aetiology. In this group of 31 cases 42% of cases were presented with previous history of rheumatic fever. This study correlates well with many Indian studies. So the incidence of Rheumatic fever is still common in India.

In these 31 cases of RHD most of the cases were in age group between 31-50 years. Most commonly presented with valvular MS + MR, followed by Isolated MS.

According to R. arora, g. Subramanian M. Khaliullah and MG Gupta, in their study of 2500 cases of rheumatic heart disease 384 cases had atrial fibrillation. Among the AF cases the valvular lesion incident was MS 38%, MS + MR 30%.

The present study shows combination of MS + MR was the most common lesion.

HYPERTENSIVE HEART DISEASE:

In this study, the SHT with AF is detected 6 cases and incidence was 12%

According to Framingham study, hypertension accounted for about half of cases.

ISCHEMIC HEART DISEASE:

In this study old myocardial infarction was found in 4 cases. The incidence was 8%.

According to Greson VH lip, D. Gracth Beevas. AF may complicate Acute MI in 10-15% of cases. But in this study evidence of old MI was found in 8% of cases.

According to Krama RJ, Zelderson, hamby RJ of 1176 patients with coronary artery disease 10% had AF. This correlates well with the present study.

CONGENITAL HEART DISEASE:

In this study out of 50 cases of AF 4 cases were ASD, incidence was about 8%. Out of these 4 cases of ASD, 3 cases were in age group above 60 yrs.

DILATED CARDIOMYOPATHY:

In this study of 50 cases of AF, DCM found in 3 patients. Incidence was about 6%. Clinically, echocardiographically and ECG wise they had cardiomegaly without valvular lesion and ischemia.

According to Gurpal singh, Prem arora study of 66 patients of AF, they found 15 cases of DCM the incidence were 10.5% .

CORPULMONALE:

Out of 50 cases of 2 cases had features of COPD, incidence is about 4%.

SYMPTOM ANALYSIS:

In symptom analysis dyspnoea and palpitation were the most frequent symptoms found in almost all the patients, chest pain is the next frequent symptom found in 54% of cases, syncope found in 40% of cases. The most frequent symptomatic presentation in this study is dyspnea and palpitation. This study correlates well with common symptomatic presentation of AF.

COMPLICATIONS:

In this analysis atrial fibrillation cases, the most common complication documented is heart failure. The percentage is 36%, LA clot is found in 6% cases. Cerebro vascular accident is found in 4% of cases.

According to O.T. Samani and HB Chandalia congestive cardiac failure was percent in 64 % of cases with atrial fibrillation.

Cabin HS, Club KS, Hall C, pertmutter RA, had reported cerebral embolism in 85% of cases and peripheral embolism in 15% cases.

In this study pulmonary hypertension is found in 44 % cases. This is made out by clinical examination and echocardiography.

CONCLUSION

CONCLUSION

- The occurrence of atrial fibrillation was more common above the age of 40 years.
- AF was more common in females – 58%
- The incidence of AF in men increases with age.
- In this fifty cases the common aetiology of AF was RHD 62% followed by Hypertensive heart disease – 12 %, congenital heart disease (ASD) and Ischemic heart disease each carries 8%.
- In this 62% of rheumatic heart disease with atrial fibrillation. The mitral valve was involved in almost all the patients. The commonest clinical presentation was MS + MR – 41 %. Followed by isolated MS – 29 %. The combination of mitral and aortic valve lesion – 10%.
- The congenital heart disease (ASD) with AF was found in 8% of cases.
- The most common symptomatic presentations were dyspnoea and palpitation followed by chest pain and dizziness.
- The previous history of rheumatic fever was found in 42 % of cases.
- The commonest complication was noted in AF cases was heart failure – 32%. CVA with embolic stroke was found in 4% of cases.

- Left atrial clot was demonstrated by echo cardio graphically in 6% of cases.
- The pulmonary hypertension was found in 44 % of cases.

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PROFORMA
ANALYSIS OF AETIOLOGY CLINICAL FEATURES AND COMPLICATIONS OF ATRIAL FIBRILLATION

Name: **Age:** **Sex:** **Address**:

Symptoms	Signs
Dyspnoea	Pedal edema
Palpitation	Puffiness of face
Chest-pain	Cyanosis
Fatigue	Anemia
Dizziness	Signs of hyperthyroidism
Neurological deficit	Other signs
Oliguria	

Heart Rate	Pulse Rate	Pulse Deficit	BP	JVP	Carotid

CVS	Respiratory system
	Central nervous system
	Abdomen

Investigations:

Hb% -	CPK-MB -	X-ray chest –
ESR -	Trop-T -	ECG -
ASO Titre -	T3 –	
Blood Urea -	T4 –	
Blood sugar -	TSH -	ECHO –

Diagnosis: Complications: