“TO STUDY THE CLINICAL PROFILE OF ATRIAL FIBRILLATION IN ELDERLY PATIENTS A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL & MRC, BELGAUM”

REG NO. BG0112010

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. D.
in
GENERAL MEDICINE

DEPARTMENT OF MEDICINE,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
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ENDORSEMENT

This is to certify that the dissertation entitled **"TO STUDY THE CLINICAL PROFILE OF ATRIAL FIBRILLATION IN ELDERLY PATIENTS A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL & MRC, BELGAUM"** is a bonafide research work done by **CANDIDATE REG NO. BG0112010**.

Dr. Rekha Patil **MD**
Professor and Head,
Department of Medicine,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

Dr. N. S. Mahantshetti **MD**
Principal,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

LIST OF ABBREVIATIONS USED

ABCs	-	Airway, breathing, and circulation
ACC	-	American College of Cardiology
ACLS	-	Advanced Cardiac Life Support
AF	-	Atrial fibrillation
AFFIRM	-	Atrial Fibrillation Follow-up Investigation of Rhythm Management
AFI	-	Atrial Fibrillation Investigators
AHA	-	American Heart Association
APD	-	Action potential duration
ARIC	-	Atherosclerosis Risk in Communities
bpm	-	Beats per minute
CAD	-	Coronary artery disease
CCF	-	Congestive cardiac failure
CHF	-	Congestive heart failure
CI	-	Confidence interval
Cms	-	Centimeters
COPD	-	Chronic obstructive pulmonary disease
CVA	-	Cerebrovascular accident
Cx	-	Connexin
DADs	-	Delayed after Depolarizations
DBP	-	Diastolic blood pressure
DC	-	Direct current
ECG	-	Electrocardiogram
eg	-	For example

ERP	-	Effective Refractory Period
ESC	-	European Society of Cardiology
ESM	-	Ejection systolic murmur
FBS	-	Fasting blood sugar
GBD	-	Global Burden of Disease
gm	-	Grams
Hb	-	Haemoglobin
HDL	-	High density lipoprotein
HT	-	Hypertension
Hz	-	Hertz
i.e.	-	That is
I.P.	-	In patient number
IHD	-	Ischemic heart disease
IHRS	-	Indian Heart Rhythm Society
IV	-	Intravenous
JVP	-	Jugular venous pressure
Kgs	-	Kilograms
LA	-	Left atrium
LAE	-	Left atrial enlargement
LDL	-	Low density lipoprotein
LVH	-	Left ventricular hypertrophy
LVIDd	-	Left ventricular diastolic dimension
LVISd	-	Left ventricular systolic dimension
LVPWD	-	Left ventricular posterior wall dimension
MDM	-	Mid diastolic murmur

mg/dL	-	Milligram per deciliter
Min	-	Minutes
ml/min	-	Millimeters per minute
mm Hg	-	Millimeters of mercury
n	-	Total number
BNP	-	Brain natriuretic peptide
O.P	-	Out patient
PND	-	Paroxysmal nocturnal dyspnoea
PPBS	-	Post prandial blood sugar
PSM	-	Pansystolic murmur
RHD	-	Rheumatic valvular heart disease
RS	-	Respiratory system
RVH	-	Right ventricular hypertrophy
RVHD	-	Rheumatic valvular heart disease
SBP	-	Systolic blood pressure
SD	-	Standard deviation
SPAF	-	Stroke Prevention in Atrial Fibrillation
TC	-	Total cholesterol
TG	-	Triglycerides
TIA	-	Transient ischaemic attack
TSH	-	Thyroid stimulating hormone
UK	-	United Kingdom
Yr	-	Year

ABSTRACT

Background and objectives

The frequency of AF is increasing as the population ages, and therefore, knowledge of the clinical spectrum is essential. This study was aimed to explore the clinical profile of atrial fibrillation in elderly patients.

Methodology

The present one year cross-sectional study was done under the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 50 elderly patients (age > 65 years) who presented with atrial fibrillation were included in the study. Patients were subjected to clinical examination, electrocardiogram and 2D echocardiography. The annual stroke risk as assessed based on $CHADS_2$ score and CHA_2DS_2-VASc score.

Results Majority of the patients were males (78%). The male to female ratio was 3.54:1. Past history of hypertension, diabetes mellitus and HD were present in 64%, 62% and 58% of patients respectively. Palpitations were noted as commonest symptom (78%) followed by breathlessness (66%), chest pain (50%), swelling of feet (42%), fainting (20%) and PND (2%).

On examination 36% of the patients each had raised JVP and pedal oedema. The other signs included hepatojugular reflex (32%), left parasternal heave (22%) and murmurs (40%).

Lipid profile revealed maximum patients with abnormal LDL (84%) followed by abnormal HDL (54%), raised total cholesterol (54%) and triglycerides (28%). The biochemical profile revealed majority of the patients with raised blood urea nitrogen (90%) followed by raised serum creatinine (50%) and hence very low creatinine clearance was observed in patients (94%).

ECG showed most of the patients had abnormal rate (64%), ischaemic changes (62%),left axis deviation (38%) and left ventricular hypertrophy (26%). On X-ray, most of the patients had LVH (36%) followed by LVH with LAE (24%), LAE (14%) and LAE+RVH (14%).

The 2D echocardiographic findings revealed 50% of the patients had ejection fraction of < 60%, 39% of the patients had dilated left atrium, 31% of the patients had dilated left ventricle, Thickened mitral valve was noted in 20% and calcification in 26%. Aortic valve calcification was present in 58% of the patients.

Most of the patients had complications of CCF (36%) followed by CVA (6%) and cor pulmonale (4%). Hypertension is the commonest etiology(64%) and other etiologies or associations include Diabetes Mellitus(62%), cardiomyopathy(42%), ischaemic heart disease(30%), rheumatic heart disease(10%), COPD(6%), surgical stress(4%), chronic kidney disease(4%) and hypothyroidism(4%).

Majority of the patients presented were subjected to rate control (82%) than rhytm control(24%) and were being treated with antiplatelets (86%) and anticoagulants (50%). History of cardioversion was present in only (6%).

The annual stroke risk based on $CHADS^2$ score showed maximum patients (38%) with intermediate risk of thromboembolic event and CHA_2DS_2-VASc score revealed moderate to high risk of stroke in maximum patients (88%).

Conclusion and interpretation

Atrial fibrillation has differences in its clinical presentation and management across elderly age group.

Keywords

Annual stroke risk; Atrial fibrillation; Geriatric population;

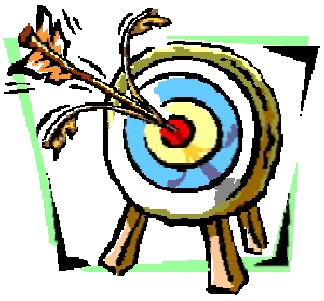
CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

Conclusion

Summary

Bibliography

Annexure-I

Annexure-II

Annexure-III

INTRODUCTION

Atrial fibrillation is the most common sustained arrhythmia worldwide, and it has a significant effect on morbidity and mortality rates.[1] it is characterized by irregular, disorganized and chaotic electrical activity of the atrium. It is a common arrhythmia and major cause of morbidity and mortality not only in the western world but in India as well.[2]

Currently, it is estimated that approximately 2.3 million adults in the United States have AF, and it is projected that this number will increase to 5.6 million to 15.9 million individuals by 2050.[3,4] The overall prevalence of AF in general population is estimated to be 0.4% to 1%.[5] The incidence of AF is 0.1% per year in the population below forty years and this increases to 2% in those over 80 years.[6] The incidence and prevalence of atrial fibrillation increases exponentially with aging. The adjusted incidence and prevalence of AF is roughly double for each advancing decade of life and at any given age, men have an ≈50% higher incidence of AF than women.[7]

Based solely on the aging of the population, the prevalence of AF in the united states has been projected to increase from ≈2 to 5 million in 2000 to ≈6 to 12 million in 2050, with estimates reaching almost 16 million if the increase in age-adjusted AF incidence continues.[3] From the Framingham data the lifetime risk of developing atrial fibrillation after age of 40 has been found to be 26% for men and 23% for women.[8]

Diabetes, hypertension, congestive heart failure and valvular heart disease are significantly associated with risk for atrial fibrillation in both sexes. Myocardial infarction is significantly associated with the development of atrial fibrillation in men. Women are significantly more likely than men to have valvular heart disease as a risk factor for atrial fibrillation.[8]

In addition to intrinsic cardiac causes such as valve disease and congestive heart failure, risk factors for cardiovascular disease also predispose to atrial fibrillation. Diabetes mellitus also increases risk of AF. Various mechanism of the same is autonomic remodeling, electrical remodeling, structural remodeling and insulin resistance.[9] Race and genetic variants have been suggested a novel susceptibility factor for developing AF.[10]

Rheumatic valvular heart disease (RVHD) continues to remain the most frequent cause of AF according to a recent survey in public and private institutes in India, however as the population is aging many patients with AF have associated hypertension (HTN) and ischemic heart disease (IHD).[11] In Western countries, IHD is the commonest cause of AF.[12]

Diagnosis of AF is based on rapid fibrillatory waves with changing morphology and ventricular rhythm that is irregularly irregular.[13] This is clinically identified as irregularly irregular pulse with rate varying from normal to 200 and pulse deficit >10 beats.

Atrial fibrillation has been classified by American Heart Association/ American college of cardiology/European Society of cardiology into first detected episode, recurrent (two or more episode), paroxysmal (terminates within 7 days),

persistent (persists for more than 7 days) and permanent (sustained for more than 1 year or has failed cardio version).[14]

About 30% of people with AF have no symptoms but some people can experience a combination of the symptoms like palpitation, shortness of breath, lethargy, dizzy spells or fainting, chest pain and symptoms of stroke. AF predisposes patients to the development of thrombi and a markedly increased risk of ischemic stroke.[15]

Overall, AF is the most common sustained cardiac arrhythmia and accounts for more physician visits and hospital days than any other cardiac rhythm disturbance. Further, age is the significant risk for the development of atrial fibrillation. The frequency of AF is increasing as the population ages, and therefore, knowledge of the clinical spectrum is essential.[16] Most published epidemiologic studies focus on predominantly white populations in North America or Europe, and information on AF in non white populations is scarce.[2] There is paucity of data in Indian region especially in elderly population. On extensive review of literature there is not much data on atrial fibrillation in the elderly individuals worldwide, all the studies conducted are on the general population. Hence this study was planned to analyze the various aspects of atrial fibrillation in elderly patients in detail.

OBJECTIVES

The objective of the present study was to assess the clinical profile of atrial fibrillation in elderly patients.

REVIEW OF LITERATURE

Atrial fibrillation is a condition in which control of heart rhythm is taken away from the normal sinus node pacemaker by rapid activity in different areas within the upper chambers (atria) of the heart. This result in rapid and irregular atrial activity and, instead of contracting, the atria only quiver. It is the most common cardiac rhythm disturbance and contributes substantially to cardiac morbidity and mortality. For over 50 years, the prevailing model of atrial fibrillation involved multiple simultaneous re-entrant waves but, in light of new discoveries this hypothesis is now undergoing re-evaluation.[17]

Historical perspectives

In 1685 Abercromby believed that "the origin of the pulse is as mysterious as the source of the Nile". However, in 1883 the tall, indefatigable Scotsman and general practitioner Sir James Mackenzie was determined to unravel the mystery.[18]

After the unexpected death during childbirth of one of his patients due to a rhythm disorder and congestive heart failure he could not help but ask himself "Would this death have occurred if I had better knowledge of heart afflictions?"[18]

At that time little was known about "fibrillation of the auricles". The ancient disease was probably first described by the legendary emperor physician **Huang Ti** in China between 1696 and 2598 BC; "When the pulse is irregular and tremulous and the beats occur at intervals, then the impulse of life fades".[18]

In 1628 the meticulous observer **William Harvey** was the first to notice the "undulation/palpitation" of the "auricle" of the dying animal heart. "…but I noticed

that after the heart proper, and even the right auricle were ceasing to beat and appeared on the point of death, an obscure movement undulation or palpitation had clearly continued in the right auricular blood itself for as long as the blood was perceptibly imbued with warmth and spirit".[18]

Jean-Baptiste de Senac (1693–1770), the physician to Louis XV, devoted much attention to palpitations and in his necropsy reports related them to mitral valve disease, noting that the "reflux of blood into the auricles causes contractions of greater force, necessarily precipitating palpitations" and that "if the auricles are strained and increased in volume they cause palpitations". He almost prophetically rationalized the use of quinidine for atrial fibrillation, and he insightfully hinted at an ectopic origin: "the causes of palpitation are not the causes of the natural heart beat".[18]

The discovery of the therapeutic properties of the 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 treatment with digitalis.[18]

René Laennec's experience with irregular heartbeats led him to disparage reliance on palpation of the pulse, considering it an unreliable guide to the state of circulation. Soon after, **Robert Adams** recognized atrial fibrillation clinically as a "sign of mitral stenosis" (1827): "Extremely irregular action of the heart is almost pathognomonic of mitral stenosis".[18]

In 1830 **Jean Baptiste Bouillaud** considered digitalis as a "sort of opium for the heart". Digitalis decreased the ventricular rate in patients even though the irregularity of pulse persisted.[18]

Using heart sounds, **Hope** (1839) noted that exercise induced an increase in irregularity and a decrease in the "intermittent pulse".[18]

Marey and Chauveau passed metal sounds through the jugular veins of horses and interpreted their observations, and in 1863 were the first to publish pulse tracings of atrial fibrillation from humans with mitral valve stenosis.[18]

In light of Marey and Chauveau's observation, **Potain** was able to interpret the pulsations of the neck in 1867.[18]

Around 1874 **Vaulpian** in France and **Hoff and Ludwig** in Germany observed atrial fibrillation in experiments with animals. **Theodor Wilhelm Engelmann** (1843–1909) proposed in 1894 that atrial fibrillation is caused by multiple foci, a theory that was further developed by **Winterberg** in 1906 and by Lewis.[18]

Around 1900, a few clinical investigators, notably **James Mackenzie** in Scotland and **Karel Wenckebach** in Holland, studied cardiac arrhythmias with the use of arterial and venous pulse tracings. Mechanically, Mackenzie (1853–1925) noted the absence of the presystolic 'a' wave seen in the jugular phlebogram during "pulsus irregularis perpetua".[18]

He described his findings as the "most Talal Moukabary, Understanding AF: A historical perspective puzzling of all forms of irregularity of the heart, where the

heart is never regular in its action, where seldom or never two beats of the same character follow one another".[18]

Electrically, **Einthoven** published in 1906 an ECG tracing of atrial fibrillation without knowing its nature. He called it "pulsus inequalis et irregularis".[18]

In 1906 **Cushing and Edmunds**, two pharmacologists from the University of Michigan, convinced Mackenzie that the so-called "nodal rhythm" was atrial fibrillation. They studied atria in the open chest of anesthetized dogs and noticed that they were dilated and motionless when a "myograph" was placed directly on the atrium. They also used the term "fibrillary state" to describe the atrial walls.[18]

Sir Thomas Lewis (1881–1945), sometime around 1909, captured atrial fibrillation on an ECG and studied the mechanism of conduction, noting that atrial fibrillation was "continuously and extremely irregular". He called it a "common clinical condition", establishing it as a clinical entity.[18]

In 1909 **Viennse Rothenberger and Winterberger** identified a direct connection between the "arrhythmia perpetua" and "fibrillation of the auricles". Their paper from 1915 proposed that atrial fibrillation resulted from rapidly discharging spontaneously active ectopic foci. They postulated that the irregularity of the rhythm resulted from the interaction between the wave fronts produced by the focal generator and the variable refractory periods of the atrial tissue.[18]

The theory of multiple-circuit re-entry was introduced by **Garrey,** and advanced later by **Moe and Abildskov**. This was later refined by **Allessie** in the

1970s and 1980s.[18] Garrey also provided experimental evidence that a "critical mass" of atrium was required to sustain atrial fibrillation.[18]

Our understanding of the basic mechanism of atrial fibrillation has improved with the observations of many brilliant scientists, especially noting the modern work of **Jalife et al**. "This history of atrial fibrillation underscores the importance of collaboration of scientists, clinical investigators and physicians in the discovery, refinement and application of new knowledge". "I know that the truth will prevail, when if I am wrong my views will be forgotten and if I am right, time will vindicate them." — James Mackenzie.[18]

Definition

Atrial fibrillation is defined as a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of mechanical atrial function.[19] Electrocardiographic findings include the replacement of the normal consistent P waves (which represent synchronous atrial activation) with oscillatory or fibrillatory waves of different sizes, amplitudes, and timing (Figure 1). The QRS complex remains narrow unless other conduction abnormalities exist (e.g., bundle branch block, accessory pathways). The ventricular response is often rapid, between 90 and 170 beats per minute.[20]

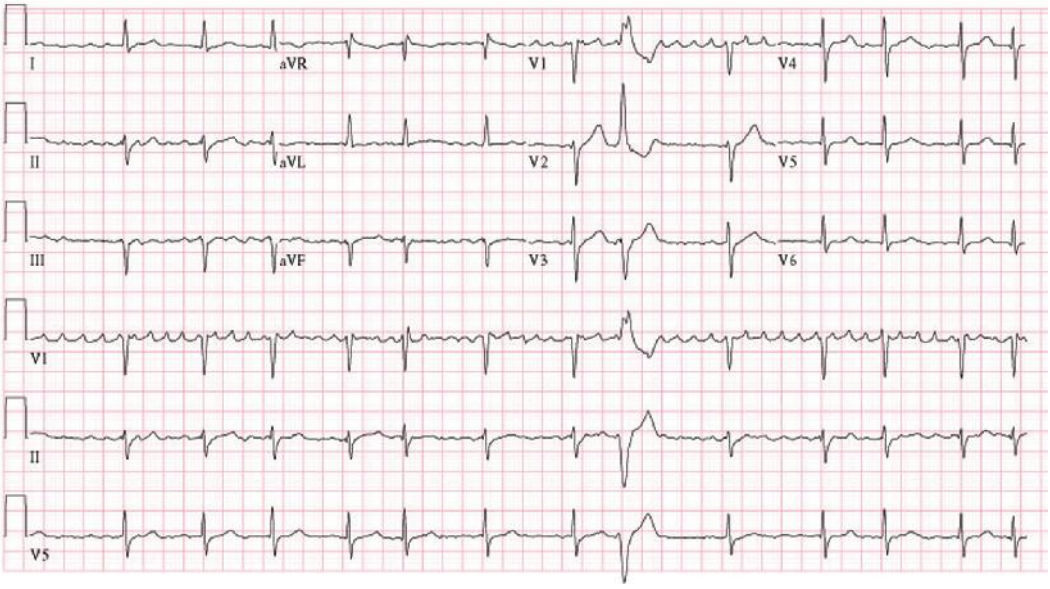


Figure 1. Electrocardiogram showing atrial fibrillation. P waves are absent and replaced by irregular electrical activity. The ventricular rate is irregular and chaotic.[21]

Classification of atrial fibrillation[19]

Type	Characteristics
Chronic/permanent	Continuous atrial fibrillation that is unresponsive to cardioversion; cardioversion will not be reattempted
Lone	Occurs in persons younger than 60 years and in whom no clinical or echocardiography causes are found
Nonvalvular	Not caused by valvular disease, prosthetic heart valves, or valve repair
Paroxysmal	Episodes that terminate spontaneously
Persistent	Paroxysmal atrial fibrillation sustained for more than seven days, or atrial fibrillation that terminates only with cardioversion

Chronic atrial fibrillation is continuous and either cannot be converted back to normal sinus rhythm or a decision has been made not to attempt cardioversion.[21]

Lone atrial fibrillation occurs in patients younger than 60 years who have no underlying cardiac disease and no identifiable cause. The prognosis is very good in patients with lone atrial fibrillation.[21]

Paroxysmal atrial fibrillation refers to episodes of intermittent atrial fibrillation that terminate spontaneously.[21]

Persistent atrial fibrillation does not self-terminate, but may be terminated by electrical or pharmacologic cardioversion.[21]

Clinical implications

Atrial fibrillation is a source of significant morbidity and mortality because it impairs cardiac function and increases the risk of stroke. Its most important clinical implications are shown in Figure 2. The cost of caring for patients with atrial fibrillation is about five times greater than caring for patients without it. Atrial fibrillation is an independent risk factor for mortality; it can also lead to or worsen heart failure and increase mortality rates in patients who have had myocardial infarction.[21]

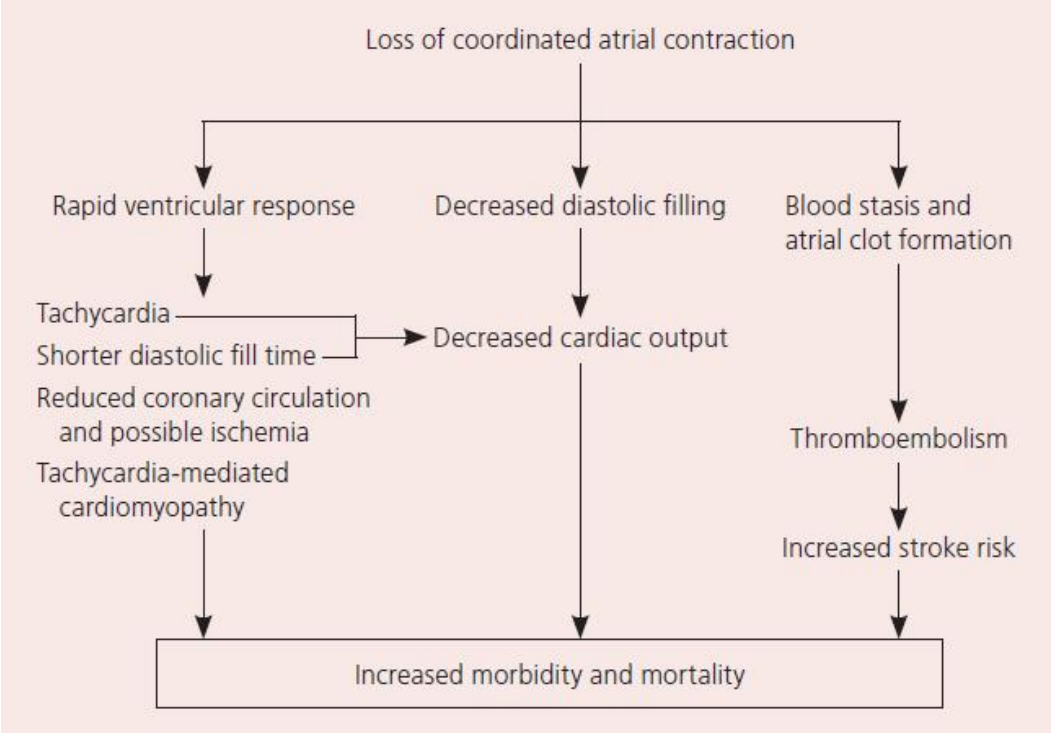


Figure 2. Flowchart for clinical implications of atrial fibrillation[21]

Different types of atrial fibrillation have different prognosis, morbidity rates, mortality rates, and treatment options.[19] For example, valvular atrial fibrillation, which is caused by structural changes in the mitral valve or congenital heart disease, carries the highest risk of stroke (i.e., 17 times that of the general population and

five times the risk of stroke with nonvalvular atrial fibrillation). Secondary atrial fibrillation is caused by an underlying condition and is reversible if the condition is treated. The most common underlying conditions are listed below.

Causes[21]

Cardiac

- Valvular disease
- Heart failure
- Cardiothoracic surgery
- Congenital heart disease
- Infiltrative disease (e.g., amyloid heart disease)
- Longstanding hypertension
- Myocardial infarction
- Myocarditis
- Pericarditis
- Wolff-Parkinson-White syndrome

Noncardiac

- Alcoholism
- Cor pulmonale
- Drug abuse
- Hyperthyroidism
- Pneumonia
- Pulmonary embolism
- Sleep apnea

Epidemiology

Worldwide

Estimated Age-Adjusted Prevalence Rates With 95% Uncertainity Intervals of Atrial Fibrillation (per 100 000 Population) for Men and Women[22]

	1990	1995	2000	2005	2010
Men					
Global, all ages	569.5 (532.8–612.7)	578.1 (541.2–620.9)	586.8 (549.8–629.5)	595.1 (557.3–639.0)	596.2 (558.4–636.7)
Age ≥35 y	1307.4 (1222.5–1407.3)	1327.3 (1243.2–1425.7)	1347.6 (1263.4–1445.8)	1366.6 (1281.0–1467.1)	1368.5 (1280.8–1462.7)
Developed countries	608.2 (547·0–693.5)	625.6 (564.0–712.5)	643.1 (580.3–730.2)	660.0 (594.5–740.8)	660.9 (597.1–738.2)
Developing countries	546.6 (503.0–599.6)	551.1 (506.6–604.8)	555.8 (511.0–610.1)	561.3 (517.5–618.4)	565.7 (522.9–617.6)
Women					
Global, all ages	359.9 (334.7–392.6)	363.4 (338.5–395.3)	366.7 (342.0–397.8)	369.6 (345.5–399.9)	373.1 (347.9–402.2)
Age ≥35 y	826.5 (768.4–902.3)	834.7 (776.6–909.2)	842.3 (784.7–915.5)	849.0 (792.4–919.6)	856.8 (797.7–923.5)
Developed countries	362.5 (319.3–422.3)	370.1 (326.7–429.5)	377.5 (334.0–436.8)	385.1 (340.1–446.8)	387.7 (343.8–450.0)
Developing countries	358.2 (329.8–393.0)	359.0 (330.8–394.0)	359·8 (331.5–395.0)	360.9 (331.6–396.0)	366.1 (337.4–400.8)

In 1990, the estimated global prevalence rates (per 100 000 population) were 569.5 (95% UI, 532.8–612.7) in men and 359.9 (95% UI, 334.7–392.6) in women. In 2010, prevalence rates were 596.2 (95% UI, 558.4–636.7) in men and 373.1 (95% UI, 347.9–402.2) in women. The prevalence rates showed a modest increase between 1990 and 2010 across both sexes. Developed countries had higher prevalence rates compared with developing countries; however, this difference was more pronounced in men than in women. For all time, points the prevalence was higher in men compared to women. There was significant variation in prevalence between Global burden of disease regions. The lowest prevalence rates (2010) were estimated in the Asia-Pacific region for both men and women (340.2 and 196.0,

respectively). The highest rates were estimated in North America (925.7 for men and 520.8 for women). The prevalence and incidence for Sub- Saharan Africa were lower compared with a developed region such as North America.[22]

Overall, for the Sub-Saharan Africa super-region, in 2010, the prevalence of AF (age-adjusted, per 100 000 population) was 659.8 (95% UI, 511.0–850.4) for men and 438.1 (95% UI, 340.2–561.0) for women. The median change in prevalence was higher in developed countries, with the largest increase noted in North America (40.1%) and the least change in Sub-Saharan Africa, East (3.4%;). Prevalence rates increased significantly with increasing age, with rates in the ≥35-year-old population observed to be more than double the overall prevalence. With the DisMod MR-estimated prevalence rates applied to the world population of 2010, the estimated number of individuals with AF globally is 20.9 million men (95% UI, 19.5–22.2 million) and 12.6 million women (95% UI, 12.0–13.7 million).[22]

In 1990, the overall incidence rates of the world population were 60.7 (95% UI, 49.2–78.5) per 100 000 person-years in men and 43.8 (95% UI, 35.9–55.0) in women. In 2010, the estimated incidence rates were higher, 77.5 (95% UI, 65.2–95.4) in men and 59.5 (95% UI, 49.9–74.9) in women. There were significantly higher (≈2-fold) incidence rates in developed regions compared with developing countries. For both time periods, similar to the observations for prevalence, AF incidence rates were higher in men compared with women. Again, there was great variation between GBD regions. The lowest incidence rates (2010) were estimated in the Asia-Pacific region for both men and women (33.8 and 19.8, respectively). The highest rates were estimated in North America (264.5 for men and 196.3 for women). As for prevalence, the incidence rates were also lower in the Sub-Saharan

region, reported as 58.4 (95% UI, 43.7–78.5) and 42.7 (95% UI, 31.1–60.5) in men and women, respectively. Incidence rates were also higher in the older age groups.[22]

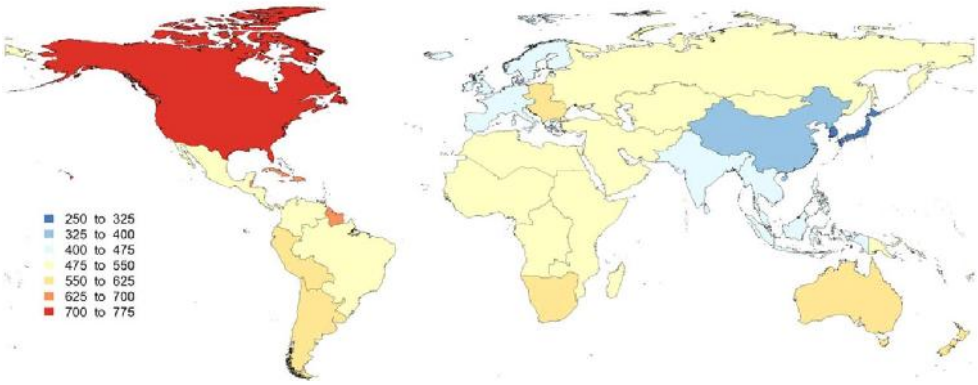


Figure 3. World map showing the age-adjusted prevalence rates (per 100 000 population) of atrial fibrillation in the 21 Global Burden of Disease regions, 2010[22]

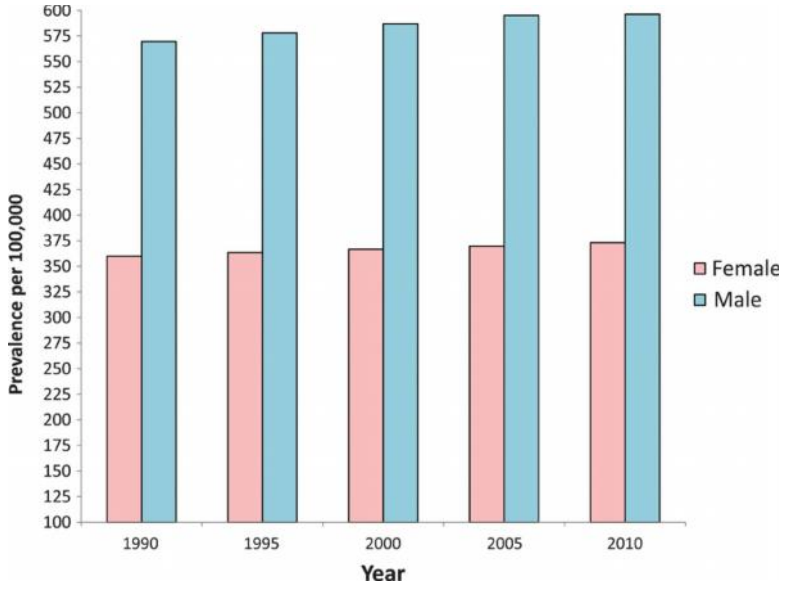


Figure 4. Prevalence of atrial fibrillation: 1990 to 2010. Estimated age-adjusted global prevalence of atrial fibrillation (per 100 000 population) for men and women from 1990 to 2010[22]

Age predilection

The prevalence of AF is 0.1% in persons younger than 55 years, 3.8% in persons 60 years or older, and 10% in persons 80 years or older. With the projected increase in the elderly population in the United States, the prevalence of AF is expected to double by the year 2050. AF is uncommon in childhood except after cardiac surgery.[3]

Sex predilection

The incidence of AF is significantly higher in men than in women in all age groups. AF appears to be more common in whites than in blacks, with blacks have less than half the age-adjusted risk of developing AF.[8,20]

Cormorbidities

In 10-15% of cases of AF, the disease occurs in the absence of comorbidities (lone atrial fibrillation). However, AF is often associated with other cardiovascular diseases, including hypertension; heart failure; diabetes-related heart disease; ischemic heart disease; and valvular, dilated, hypertrophic, restrictive, and congenital cardiomyopathies.[20] The Atherosclerosis Risk in Communities (ARIC) Study suggests reduced kidney function and presence of albuminuria are strongly associated with AF.[23]

The rate of ischemic stroke in patients with nonrheumatic AF averages 5% a year, which is somewhere between 2 and 7 times the rate of stroke in patients without AF. The risk of stroke is not solely due to AF; it increases substantially in the presence of other cardiovascular diseases.[24] The prevalence of stroke in patients

younger than 60 years is less than 0.5%; however, in those older than 70 years, the prevalence doubles with each decade.[25] 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. Women are at a higher risk of stroke due to AF than men and some have suggested this may be due to undertreatment with warfarin. However, one study of patients 65 years or older with recently diagnosed AF found warfarin use played no part in the increased risk of stroke among female patients.[26]

Mortality and Disease Burden Associated With AF

The age-adjusted mortality rate (per 100 000 population) for AF in 1990 was 0.8 (95% UI, 0.5–1.1) for men and 0.9 (95% UI, 0.7–1.2) for women. The age-adjusted mortality rate increased to 1.6 (95% UI, 1.0–2.4) and 1.7 (95% UI, 1.4–2.2) in 2010, representing 2-fold (95% UI, 2.0–2.2) and 1.9-fold (95% UI, 1.8–2.0) increases, for men and women, respectively, especially in the developed world. Mortality associated with AF was higher in women overall; this was driven mainly by comparatively higher mortality in women (compared with men) in developing countries. In 2010, the estimated numbers of total deaths (men and women) represented <1% of the global mortality in the vast majority of the 21 GBD regions.[21]

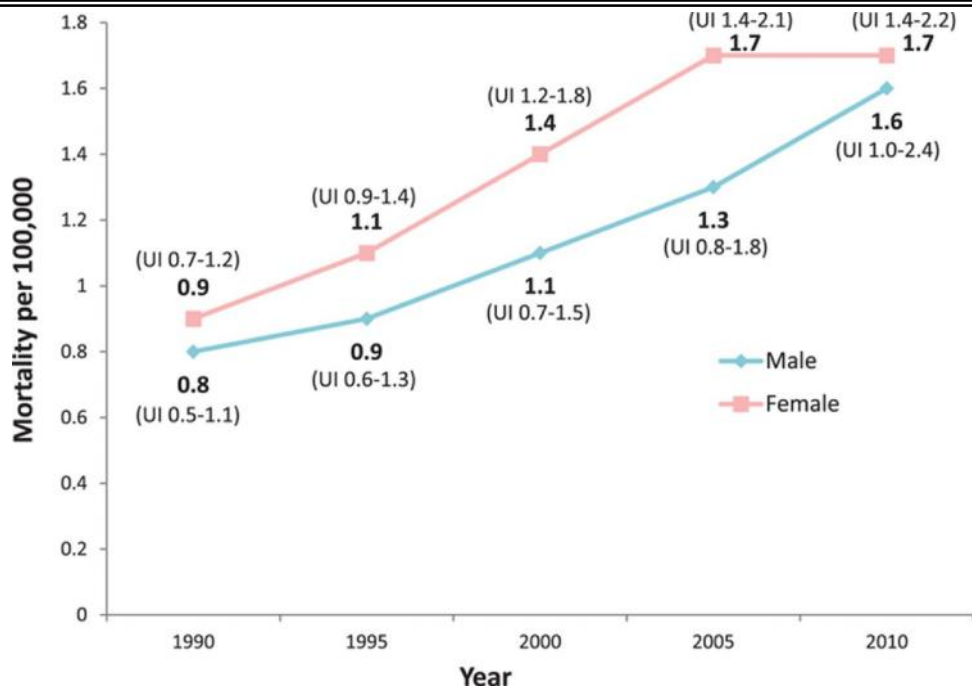


Figure 5. Mortality associated with atrial fibrillation: 1990 to 2010. Estimated age-adjusted mortality (per 100 000 population) associated with atrial fibrillation from 1990 to 2010. UI indicates uncertainty interval[21]

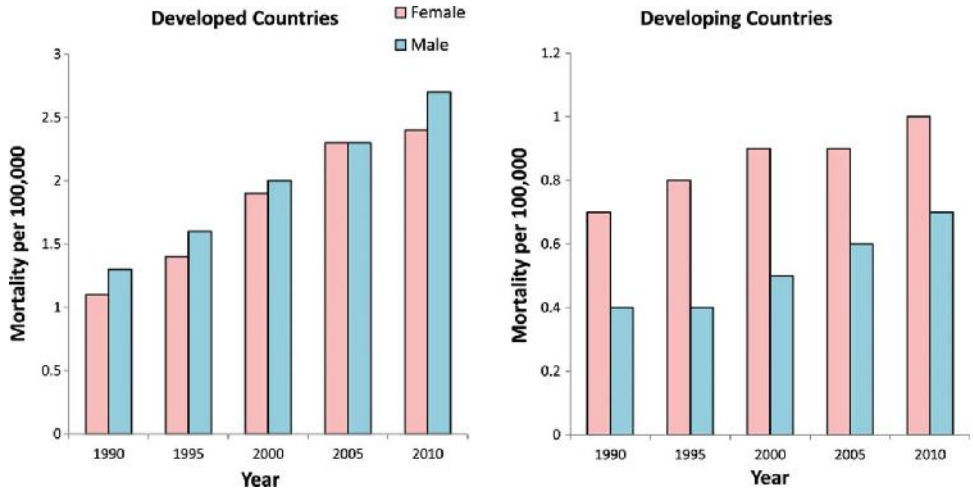


Figure 6. Mortality associated with atrial fibrillation (AF) stratified by sex and type of region (developed vs developing). Mortality associated with AF was higher in men and women in the developed regions. The significantly higher mortality in women in the developing regions is responsible for the overall higher AF-related mortality among women compared with men[21]

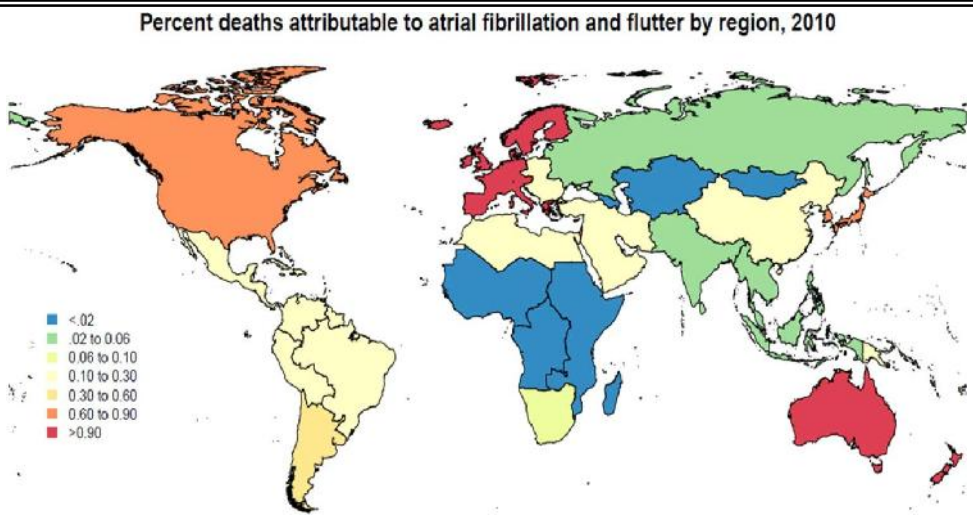


Figure 7. Proportion of global deaths associated with atrial fibrillation in 2010. The map shows color-coded proportions (in percentages) of global deaths in 2010 associated with atrial fibrillation[21]

There has been a worldwide increase in the ageing population, and as age is the most significant risk factor for AF, AF cases are predicted to reach nearly 16 million in the USA and 25 to 30 million in Europe by 2050.[4] The prevalence of AF shows a strong age dependence varying from 0.5% in patients aged <40 years to 5% in patients aged >65 years and nearly 10% amongst octogenarians.[3,8] Both the Framingham Heart Study and the Rotterdam Study estimated that the lifetime risk for development of AF in adults >40 years and at the age of 55 years respectively to be approximately 1 in 4.[20,27]

The Cardiovascular Health Study which was a large population study of 5201 elderly adults showed an incidence of 17.6 and 42.7 events per 1000 person-years amongst men aged 65 to 74 years and 75 to 84 years, respectively, whereas amongst women in the corresponding age groups, the incidences were 10.1 and 21.6.[28]

The SAFE study which was a UK-based multicentre randomised control trial of elderly (≥65 years) patients with AF showed an overall prevalence of 7.2% and 10.3% in those aged 75 years and older, with a 1.6% yearly incidence of new AF.[29]

AF in the young in the absence of identifiable causes can be idiopathic or termed as "lone AF," that is, not associated with comorbidities or obvious cardiac disease. The term "lone AF," itself was first described nearly sixty years back by Evans and Swann.[30]

Traditionally the cut-off age of 60 years has also been included in the diagnosis of lone AF as suggested by ACC/AHA/ESC guidelines[19] and used previously in several landmark epidemiological studies such as the Framingham Heart Study[31] and the Mayo Clinic Study.[32]

Old age is indeed one of the strongest risk factors for AF but terming adults over 60 years as "elderly" seems rather controversial in this modern age of improving longevity. The rationale for this "threshold age" therefore has to be questioned.[33] At variance with this age-based definition of lone AF, studies such as that by Kopecky et al. have analysed the outcome of lone AF in patients aged over 61 years, mean age 74 years (range, 61–97 years).[34]

As newer etiologies are uncovered and the existence of "truly lone AF" becomes increasingly controversial, the prevalence of "lone AF" also seems to be steadily "decreasing" in modern studies.[33]

A recent study of 3978 AF patients from the Euro Heart Survey by Weijs et al. that excluded age or left atrial size from the definition reported a prevalence of idiopathic AF of 3% out of 3978 patients.[35] The mean age of patients with idiopathic

AF in this study was 58 (SD 14) years and nearly half of these (48%) were older than 60 years.

Similarly a 30-year follow-up study reported by the Mayo Clinic reported that lone AF constituted only 2% of the total proportion of patients with AF.[32]

In contrast, earlier studies showed that lone AF occurs in 1.6% to 30% of all cases of AF, depending on the definition of idiopathic AF or inclusion criteria used.[31,33]

Indian Scenario

The first population based study from India was in 1995 and included 984 Himalayan, village residents. They found AF prevalence of only 0.1% (n=1). This low prevalence is because the participants were healthy, received only a single ECG, and only 6% were >65 years of age.[36] The West Birmingham Atrial Fibrillation Project, showed 0.6% prevalence of AF among Indians.[37] The PANARM study revealed AF to be the commonest arrhythmia seen by clinicians (66% of all arrhythrnias).[38] The average age of the Indian AF patient is, however, more than a decade young, REALIZE study and in the IHRS AF registry the average age was 60 and 54 years, respectively.[38] The lower age is because of the prevalence of rheumatic valvular heart disease (RVHD). The CRRAFT study on AF had a mean age of 38 years. There is almost an equal gender distribution in RELY and REALIZE AF Indian subset (55% men). AF, in fact has 51% females (likely because of more RVHD). The general population data, to identify the incidence and prevalence of AF in India is lacking. Importantly, community based longitudinal studies (world-over) have shown that there has been a steady increase in AF

incidence over the last two decades. This will continue over the next few decades with an aging population and higher occurrence of the associated risk factors.[38]

Types of AF

Paroxysmal AF was more often seen in the RELY and REALIZE (Indian subset) of 38% and 43% as opposed to only 19.5% in the IIIRS-AF study. Permanent AF was similar in the REALIZE and IIIRS-AF at 34.3% and 33.7% vs. only 18.6% in RELY study. This discrepancy is likely because there were more private hospital centres in the RELY as compared to IIIRS-AF which has more equal distribution of private and public hospital centres (RVHD and permanent AF being more prevalent in the public hospitals).[38]

Pathophysiology

Atrial fibrillation (AF) shares strong associations with other cardiovascular diseases, such as heart failure, coronary artery disease (CAD), valvular heart disease, diabetes mellitus, and hypertension.[39] These factors have been termed upstream risk factors, but the relationship between comorbid cardiovascular disease and AF is incompletely understood and more complex than this terminology implies. The exact mechanisms by which cardiovascular risk factors predispose to AF are not understood fully but are under intense investigation. Catecholamine excess, hemodynamic stress, atrial ischemia, atrial inflammation, metabolic stress, and neurohumoral cascade activation are all purported to promote AF.

Because diabetes mellitus and obesity are increasing in prevalence and are associated with an elevated risk of AF, Fontes et al examined whether insulin resistance is an intermediate step for the development of AF. In a community-based

cohort that included 279 patients who developed AF within 10 years of follow-up, no significant association was observed between insulin resistance and incident AF.[40]

Although the precise mechanisms that cause atrial fibrillation are incompletely understood, AF appears to require both an initiating event and a permissive atrial substrate. Significant recent discoveries have highlighted the importance of focal pulmonary vein triggers, but alternative and nonmutually exclusive mechanisms have also been evaluated. These mechanisms include multiple wavelets, mother waves, fixed or moving rotors, and macro-reentrant circuits. In a given patient, multiple mechanisms may coexist at any given time. The automatic focus theory and the multiple wavelet hypothesis appear to have the best supporting data.[41]

Automatic focus

A focal origin of AF is supported by several experimental models showing that AF persists only in isolated regions of atrial myocardium. This theory has garnered considerable attention, as studies have demonstrated that a focal source of AF can be identified in humans and that isolation of this source can eliminate AF.[41]

The pulmonary veins appear to be the most frequent source of these automatic foci, but other foci have been demonstrated in several areas throughout the atria. Cardiac muscle in the pulmonary veins appears to have active electrical properties that are similar, but not identical, to those of atrial myocytes. Heterogeneity of electrical conduction around the pulmonary veins is theorized to promote reentry and sustained AF. Thus, pulmonary vein automatic triggers may

provide the initiating event, and heterogeneity of conduction may provide the sustaining conditions in many patients with AF.[41]

Multiple wavelet

The multiple wavelet hypothesis proposes that fractionation of wave fronts propagating through the atria results in self-perpetuating "daughter wavelets." In this model, the number of wavelets is determined by the refractory period, conduction velocity, and mass of atrial tissue. Increased atrial mass, shortened atrial refractory period, and delayed intra-atrial conduction increase the number of wavelets and promote sustained AF. This model is supported by data from patients with paroxysmal AF demonstrating that widespread distribution of abnormal atrial electrograms predicts progression to persistent AF.[42] Intra-atrial conduction prolongation has also been shown to predict recurrence of AF.[43] Together, these data highlight the importance of atrial structural and electrical remodeling in the maintenance of AF hence the phrase "atrial fibrillation begets atrial fibrillation."

Etiology

A. Etiology in the Young

Lone AF is a diagnosis of exclusion for which any clinical features of comorbidities or structural cardiac abnormalities that could cause AF must be ruled out. Novel risk factors for AF are increasingly being discovered such as genetic causes, lifestyle factors (such as alcohol consumption, personality traits, and smoking), body mass index, and physical activity, thereby all refuting the diagnosis of “lone AF.” In addition, it can also be argued that occult cardiac pathologies such

as hypertension or ischaemic heart disease may well be diagnosed in these patients if they are investigated thoroughly.[33]

1. Familial

Nearly seven decades ago, Wolff described a case of familial AF in three brothers and since then studies have found a positive family history of AF in up to a third of AF patients. Having a positive family history especially in younger patients nearly doubles the risk of developing AF.[33]

In 1997, Brugada et al.[44] described the first genetic locus on chromosome 10q22-24 in a family with AF segregated as an autosomal dominant trait.

In the last few years, genomewide association studies for AF have shown SNPs (small nuclear polymorphisms) at three genetic loci—4q25, 16q22, and 1q21. Monogenic forms of AF have also been described due to mutations of genes encoding for potassium channels (KCNQI, KCNE2, KCNJ2, and KCNA5), sodium channel gene SCN5A, K(ATP) gene, the ABCC9 gene, and the connexin 40 gene GJA5. Attempts to further unravel the interplay of genomics and AF have shown that the genetic basis of AF is both complex and heterogeneous.[33]

2. Alcohol

The "holiday heart syndrome" was described in 1978 by Ettinger to explain the association between supraventricular tachyarrhythmias particularly AF and episodes of increased alcohol consumption during weekends and holiday binge drinking by people without structural heart disease.[45]

Overall chronic heavy alcohol consumption (>36 g/day) has been shown to increase risk of AF in several studies including the Framingham cohort.[46] Mechanisms of acute alcohol-induced AF include metabolic acidosis, catecholamine release, and electrolyte disturbances whereas chronic overconsumption leads to myocardial fibrosis, dilatation, and autonomic changes.[47] Binge drinking is particularly prevalent amongst young people and alcohol has been identified to potentiate paroxysmal AF in up to two-thirds of cases.

In the elderly, the relationship between AF and alcohol intake is more complex however.[33] The Cardiovascular Health Study showed an inverse association between alcohol consumption and risk of AF in patients over 60 years old, with a 4% lower risk for each additional drink per week.[28] Other studies have also shown a lack of association between risk of developing AF and moderate alcohol consumption.[48,49]

3. Obesity

A meta-analysis by Wanahita et al[50] looking at 16 studies that enrolled 123249 individuals (mean age years) demonstrated a 49% increased risk of developing AF due to obesity (relative risk 1.49, 95% CI 1.36–1.64). Obesity-associated left ventricular hypertrophy and left atrial dilation are postulated to be important causes which lead to AF. A 3–8% increased risk of developing AF has been associated with each unit increase in body mass index. Obstructive sleep apnoea which is also associated with obesity has been shown to portend risk of AF development in individuals <65 years of age. The association between obesity and

lone AF is not robust however and in fact taller and leaner adults are reported to be more prone to develop lone AF.[33]

4. Sports and Physical Activity

AF has been recognised to be the commonest cause of palpitations amongst young athletes. Endurance athletes such as marathon runners have been shown to have a greater predisposition to develop AF when compared to nonathletes.[33]

Karjalainen et al.[51] diagnosed lone AF in 5.3% out of a cohort of 228 male cross-country runners (mean age 47.5 years).

Mont et al.[52] reported a four times higher proportion of sports enthusiasts who had lone AF (aged <65 years) in comparison to controls in a Catalonian population (63% versus 15%). The same group reported that >1500 lifetime hours is the threshold for increased AF propensity. In more than half of the sportsmen with lone AF, a likely vagal precipitant was identified (postprandial, postexercise or at rest).

Interestingly, in the recent GIRAFA study, cumulative work-related moderate-to-heavy physical activity has also been shown to predict risk of developing lone AF in middle aged men aged <65 years.[53]

A meta-analysis of six case control studies, including 655 athletes and 895 controls (predominantly men), with a mean age of years showed a significantly higher risk of AF in athletes.[54]

Increased left atrial volume was shown to strongly predict AF in athletes. Mechanisms for sports-induced AF postulated include enlarged left atrium and left

ventricular mass, increased vagal tone leading to bradycardia as well as shorter atrial refractory period and hypovolemia.[33]

5. Cardiac Pathologies

There are a variety of cardiac pathologies associated with AF in the young. These include hypertrophic cardiomyopathy which confers a four-to sixfold greater risk of AF. Prevalence of AF in these patients is relatively high at about 22% and incidence is 2% annually. Even in this pathology, prevalence increases with age and is seen predominantly in the elderly (>60 years age).[33]

Congenital Heart Disease is another risk factor for AF and with improved surgical outcomes, increasing numbers of infants and children are surviving into adulthood.[33]

A large Quebec-based population study of about 38000 adult congenital heart disease patients, with a median age of 42 years, showed a prevalence of atrial arrhythmias of 15.1% (three times greater than that seen in the general population).[55]

Wolff-Parkinson-syndrome, myocarditis, pericarditis, and dilated cardiomyopathy are some of the other causes of AF in the young. Valvular heart disease secondary to rheumatic fever is also a significant cause of AF in the young in the developing world.[33]

6. Other Risk Factors

Behavioural or emotional triggers such as Type A personality, stress, anger, and hostility in men have also been shown to predispose to development of AF. Other risk factors associated with AF include increased coffee and nicotine

consumption. The association with caffeine intake is debatable however as a canine study showed inverse association between risk of AF and intravenous caffeine and no causative role was found in the Danish diet, cancer, and health study.[33,56] Smoking has been shown to lead to atrial fibrosis which is well recognised to portend AF. Endocrine causes of AF in the young include hyperthyroidism and phaeochromocytoma.[33]

B. Etiology in the Elderly

As shown in the Framingham Heart Study, AF is usually secondary to a variety of cardiac pathologies (ischaemic heart disease, heart failure, and valvular heart disease) as well as systemic disorders (hypertension, diabetes, hyperthyroidism). This is particularly true in the elderly who also have an increased predisposition of these conditions. However even accounting for other comorbidities, ageing is the strongest independent risk factor that predisposes to AF.[33]

The Cardiovascular Health Study[57] showed that the prevalence of AF was 9.1% in the subgroup with clinical cardiovascular disease, 4.6% in the subgroup with only subclinical cardiovascular disease, and 1.6% in the absence of clinical or sub-clinical cardiovascular disease (i.e., lone AF). Independent risk factors for AF in the elderly included age, treated systemic hypertension, congestive cardiac failure, valvular heart disease, stroke, enlarged left atria size, mitral or aortic valve dysfunction, echocardiographic features of diastolic dysfunction, and raised serum levels of NT-proBNP.

Hyperthyroidism is another cause of atrial fibrillation in the elderly and one study reports an AF incidence of 25% amongst hyperthyroid patients older than 60 years in comparison to 5% in those aged less than 60 years.[33]

Electrophysiological and Structural Alterations in Aged Atria

Initiation of AF in the Aged Atria

A number of attempts have been made to unravel the atrial electrophysiological characteristics of aged atria in relation to AF. Whilst most animal and human studies have shown that aged atria have an increased propensity to develop AF, some studies in elderly AF patients have yielded conflicting results although these could have been influenced by underlying pathology or treatment in patients. Increased atrial ERP has been noted in these studies which could be sufficient to overcome any other arrhythmogenic remodelling. There is therefore a need to understand, in the absence of underlying disease, how AF is initiated and maintained in the aged atria.[33]

Initiation of AF

Why the aged atria are more susceptible to the development of AF in the absence of other risk factors remains poorly understood. We shall describe below how ageing may increase the propensity of both (i) triggered activity in the form of DADs and (ii) reentrant circuits.[33]

Delayed after Depolarizations (DADs) in the Aged Atria

Potential contributory factors that may predispose to DAD formation and indeed the increased incidence of DADs have been demonstrated in some models of ageing.[33]

Wongcharoen et al.[58] observed DADs of greater amplitude in aged rabbit LA pulmonary vein sleeve tissue sections associated with an increase in NCX protein which would provide an additional means to facilitate triggered activity as more depolarising current would flow during any spontaneous Ca^{2+} release and therefore, all things being equal, a smaller spontaneous release would produce a bigger DAD [90].

There is also evidence of atrial calcium mishandling in AF. This has been attributed to protein kinase A-induced hyperphosphorylation leading to dysfunctional ryanodine receptor. While decreased SERCA and increased RyR protein expression give us clues to SR function, it would also be of interest to understand how SR Ca2+ content responds to age in the pulmonary vein. One study using human atrial tissue from elderly patients (mean age 68 years) has suggested that hyperphosphorylation of phospholamban could be contributory to leaky ryanodine receptors and thus abnormal calcium handling in chronic AF patients. Clearly there is a pressing need to understand how Ca2+ homeostasis is achieved in the aged atria and how it is subsequently remodelled in the aged atria in AF.[33]

Reentrant Circuits in the Aged Atria

A reentrant substrate can result from altered functional (electrical) properties or structural changes to the atrium and these are discussed below with reference to ageing.[33]

Effective Refractory Period (ERP)

Whilst conflicting results have been noted when analysing the effects of ageing on effective refractory period in both human as well as animal right atria,[33] review of various studies indicates that the right atrial ERP is prolonged with ageing.[59]

Inconsistency in the literature may relate to variation in anatomical sites studied and, in terms of patient studies, the effects of underlying disease, arrhythmias, drug treatment, or a general lack of studies including very elderly patients as highlighted by Dun and Boyden.[59]

The few studies investigating action potential duration (APD) or ERP in aged left atrium also show conflicting results. Furthermore in rabbit pulmonary vein sleeves and left atrial posterior wall APD was prolonged with age. Electrical characteristics and age-associated remodelling of the atrium appear to be region specific and this may underlie differences between the above studies. Interestingly while the prolonged action potential in aged rabbit left atrial posterior wall may be expected to be anti-arrhythmogenic, this gave rise to an increase in APD dispersion (discussed below) which was suggested to potentiate AF.[33]

Many ion channels have yet to be studied in the aged atria and work mainly performed in the dog has shown that there is no age-related change in sodium current density. However is depressed, which would be expected to depress the plateau of the action potential, and since repolarising currents activate more strongly at positive potentials, this may slow activation and prolong repolarisation, thus prolonging ERP. Of the repolarising currents only has been assessed and peak as well as sustained was increased with age in the right atrium but not the left atrium. Indirect evidence suggests may be enhanced in the right atrium with age; however there are no studies investigating age-associated changes in either atria.[33]

Conduction Velocity

It is reported that, age-associated general or directional conduction slowing and resultant spiral-reentrant waves in the right atria mainly in tissue strips but also in vivo in various species. In the aged dog, conduction of normal beats was unaltered but premature impulses were slowed suggesting a certain amount of depolarizing current is needed to overcome conduction discontinuities in age.[33]

By calculating conduction in the direction of the wavefront, Kojodjojo et al.[60] have shown in human atria that increasing age is associated with decreased propagation velocity in both atria during sinus rhythm and also during pacing.

In this respect the reduction of noted in AF is potentially significant as it will slow conduction velocity and reduces the excitation wavelength. Based on a very limited number of studies the effects of ageing on peak are inconsistent, showing either no change at low stimulation frequencies but reduced at stimulation frequencies relevant to AF (10 Hz) or a decrease.[33]

Changes in connexin expression (especially Cx40 and Cx43) have been noted in AF-related remodelling and are also noted in ageing. A single study has shown an age-associated decrease in connexin 43 in the sinoatrial node but unaltered expression of connexins in the right atria.[61]

However a change in the distribution of connexins away from the lateral cell edges to the intercalated discs has also been noted in ageing. This is potentially significant, as it will result in anisotropic propagation of excitation and the formation of reentrant circuits.[33]

Increased or heterogeneous fibrosis, often associated with advancing age, can disrupt the coupling between individual myocytes and result in non homogenous conduction or conduction slowing which can lead to re-entry. Recently it is demonstrated that, increased AF stability in long-term AF in goats due to "microfibrosis" separating myocyte bundles. Conduction abnormalities can occur following redistribution without altered expression.[33]

Dispersion of Conduction Velocity and ERP

ERP dispersion and conduction heterogeneity correlate with AF inducibility and both have been shown to increase with ageing.[33]

In summary, what induces and sustains AF in the young may be different to that in the old. In old dogs atrial tissue was depolarised with longer APD and a slower max upstroke and greater variability in APD. In chronic AF both young and old, the atrial cell membrane was hyperpolarised, with slowed upstroke and decreased APD. But chronic AF led to an increase of APD dispersion in adults and a decrease in old dogs. Thus, AF was sustained in two different substrates: one with

short AP duration and with expanded heterogeneity of AP parameters (adult) and one with short AP duration but limited heterogeneity (old). These data also suggest that the increased dispersion in atrial electrophysiology that occurs in adults may be an important additional contributory factor for AF stabilization at this age, while the occurrence of fibrosis and slowed conduction of premature beats that has been demonstrated previously may be more contributory in the old.[33]

Histological changes in healthy elderly patients with AF include increased deposition of collagen, adipose tissue and amyloid, atrophy and vacuolar myocyte degeneration and fibrofatty substitution of the sinoatrial node. Aging-related oxidative damage has been shown to portend atrial fibrillation through mitochondrial bioenergetic dysfunction. Imaging has also shown age-related dilatation of the pulmonary veins and the atrium, thereby potentiating pulmonary vein triggers as well as substrate-induced AF maintenance through mechanoelectric feedback.[33]

Clinical Features

Clinical presentation

Clinical presentation spans the entire spectrum from asymptomatic atrial fibrillation (AF) with rapid ventricular response to cardiogenic shock or devastating cerebrovascular accident (CVA).

Initial evaluation of the patient with new-onset atrial fibrillation should focus on the patient's hemodynamic stability. Care of hemodynamically unstable patients is guided by Advanced Cardiac Life Support (ACLS) protocols, including immediate direct current (DC) cardioversion.[62] Symptomatic patients may benefit

from intravenous (IV) rate-controlling agents, either calcium-channel blockers or beta-adrenergic blockers.

While up to 90% of AF episodes may not cause symptoms,[63] many patients experience a wide variety of symptoms, including palpitations, dyspnea, fatigue, dizziness, angina, and decompensated heart failure. In addition, AF can be associated with hemodynamic dysfunction, tachycardia-induced cardiomyopathy, and systemic thromboembolism.

Unstable patients requiring immediate DC cardioversion include patients with decompensated congestive heart failure (CHF); patients with hypotension; patients with uncontrolled angina/ischemia.[41]

Less severe symptoms and patient complaints include palpitations; fatigue or poor exercise tolerance; presyncope or syncope; generalized weakness, dizziness, fatigue.[41]

In addition to eliciting the symptoms above, history taking of any patient presenting with suspected AF should include questions relevant to temporality, precipitating factors (including hydration status, recent infections, alcohol use), history of pharmacologic or electric interventions and responses, and presence of heart disease. An effort should also be made to evaluate for potential comorbid diseases that contribute to initiation or maintenance of AF. Occasionally, a patient may have a clear and strong belief about the onset of symptoms that may be helpful in determining a course of action.[41]

Initial history includes documentation of clinical type of AF (paroxysmal, persistent, or permanent); assessment of type, duration, and frequency of symptoms;

assessment of precipitating factors (eg, exertion, sleep, caffeine, alcohol use); assessment of modes of termination (eg, vagal maneuvers); documentation of prior use of antiarrhythmics and rate-controlling agents and assessment of presence of underlying heart disease.[41]

Physical examination findings

Atrial fibrillation has a wide spectrum of clinical presentations. Some patients may be asymptomatic. Others may present with stroke, overt heart failure, or cardiovascular collapse. Patients most commonly report palpitations, dyspnea, fatigue, lightheadedness, and chest pain. Because symptoms are nonspecific, they cannot be used to diagnose and determine the onset of atrial fibrillation. If electrocardiography does not demonstrate atrial fibrillation and a strong suspicion persists, a Holter or cardiac event monitor may be needed to document the arrhythmia.[21]

Physical examination always begins with airway, breathing, and circulation (ABCs) and vital signs, as these guide the pace of the intervention. The physical examination also provides information on underlying causes and sequelae of atrial fibrillation.[21]

General examination

Vital signs[41]

- Heart rate, blood pressure, respiratory rate, and oxygen saturation are particularly important in evaluating hemodynamic stability and adequacy of rate control in AF.

- Patients will have an irregularly irregular pulse and will commonly be tachycardic, with heart rates typically in the 110- to 140-range, but rarely over 160-170. Patients who are hypothermic or who have cardiac drug toxicity may present with bradycardic atrial fibrillation.

Head and Neck[41]

- Examination of the head and neck may reveal exophthalmos, thyromegaly, elevated jugular venous pressures, or cyanosis.

- Carotid artery bruits suggest peripheral arterial disease and increase the likelihood of comorbid coronary artery disease

Lower extrimities[41]

- Examination of the lower extremities may reveal cyanosis, clubbing, or edema.

- A cool or cold pulseless extremity may suggest peripheral embolization, and assessment of peripheral pulses may lead to the diagnosis of peripheral arterial disease or diminished cardiac output.

Cardiac[41]

The cardiac examination is central to the physical examination of the patient with AF. Thorough palpation and auscultation are necessary to evaluate for valvular heart disease or cardiomyopathy. A displaced point of maximal impulse or S_3 suggests ventricular enlargement and elevated left ventricular pressure. A prominent P_2 points to the presence of pulmonary hypertension.

Pulmonary[41]

The pulmonary examination may reveal evidence of heart failure (eg, rales, pleural effusion). Wheezes or diminished breath sounds are suggestive of underlying pulmonary disease (eg, chronic obstructive pulmonary disease [COPD], asthma).

Abdomen[41]

The presence of ascites, hepatomegaly, or hepatic capsular tenderness suggests right ventricular failure or intrinsic liver disease. Left upper quadrant pain may suggest splenic infarct from peripheral embolization.

Neurologic[41]

Signs of a transient ischemic attack or cerebrovascular accident may be discovered. Evidence of prior stroke and increased reflexes is suggestive of hyperthyroidism.

The clinical presentation of AF varies significantly depending on age and comorbidities. In the young, the initial presentation is usually with paroxysmal AF; persistent AF under the age of 50 is often associated with identifiable causes like structural heart disease, hyperthyroidism, or alcohol excess. Whilst the incidence of both paroxysmal and persistent AF increases dramatically over the age of 60, there is a disproportionate increase in chronic forms, with the result that 80% of newly diagnosed AF in octogenarians is of a persistent or permanent form, even in the absence of structural heart disease. Moreover, advanced age is a risk factor for early recurrence after first AF presentation and of rapid progression from paroxysmal to persistent AF.[33]

AF is classically associated with “typical” symptoms of irregular palpitations, with or without chest pain, breathlessness, or dizziness. Palpitations are reported in 80% of young patients with paroxysmal AF. In contrast, less than 10% of AF patients over the age of 80 years have palpitations and up to 40% of elderly hospital inpatients found to have AF are entirely asymptomatic. Whilst atypical chest pain is relatively common in young AF patients, in elderly patients anginal chest pain during AF episodes strongly suggests the presence of significant concurrent coronary disease and might be sufficient to warrant investigation for coronary ischaemia even in the absence of typical symptoms of angina.[33]

AF in elderly patients is frequently diagnosed coincidentally during general health assessment, hospital admission for nonrelated illnesses, or as a result of its complications.[33]

A recent randomized controlled study in primary care suggests that implementing targeted opportunistic screening of over 65-year olds, based on a simple annual pulse assessment, is likely to be cost-effective in improving AF detection.[29]

Common differences between AF in the young versus elderly.

	AF in the young	AF in elderly patients
Causes	(i) Idiopathic	(i) Ischaemic heart disease
	(ii) Genetic	(ii) Heart failure
	(iii) Alcohol, smoking	(iii) Valvular heart disease
	(iv) Personality traits	(iv) Hypertension
	(v) Body mass index	(v) Diabetes Mellitus
	(vi) Endurance sports	(vi) Cardiomyopathies
	(vii) Cardiac pathologies	(vii) Hyperthyroidism
	(viii) Endocrine disorders	(viii) Secondary causes such as post operative, infection, pulmonary embolism
		(ix) Idiopathic
Pathogenesis	Triggers/pulmonary vein Repetitive activity +++	Pulmonary vein repetitive activity ++
	Substrate/atrial abnormalities +	Atrial Abnormalities +++
Clinical features	Usually typical symptoms	Atypical symptoms or asymptomatic
Management	Rhythm control preferred	Rate control preferred
	Thromboprophylaxis usually not required unless based on CHADS2VASC	Thromboprophylaxis usually required unless contraindicated

Evaluation

The first goal is to determine the patient's cardiac stability and provide emergency stabilization if needed. If the patient is unstable because of hypotension, ongoing ischemia, severe heart failure, or cerebrovascular events, emergency electrical cardioversion is warranted. If the patient is clinically stable, the history, physical examination, and diagnostic testing should focus on potential causes, triggers, and comorbid conditions. Standard tests used to evaluate cardiac function and identify common comorbid conditions include electrocardiography, complete blood count, complete metabolic profile, thyroid-stimulating hormone measurement, chest radiography, and echocardiography.[21]

Echocardiography provides information about heart size, chamber sizes, valvular anatomy and function, wall motion abnormalities, systolic and diastolic function, and pericardial disease. If there is clinical suspicion of myocardial ischemia, creatine kinase isoenzyme and troponin levels should be obtained. Select patients may need additional tests, such as stress testing and electrophysiology studies.[21]

METHODOLOGY

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2013 to December 2013.

Study design and duration

The study design was a one year cross-sectional study.

Study period

The present study was carried out from January 2013 to December 2013.

Source of Data

Patients with atrial fibrillation aged above 65 years attending Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were studied.

Sample size

A total of 50 patients with atrial fibrillation were included in the study.

Sampling procedure

Based on the universal sample, a minimum sample size of 50 patients was considered.

Selection criteria

Inclusion Criteria

- Patients with age group 65 yrs and above.
- Patients having atrial fibrillation who fulfill the European society of cardiology definition for atrial fibrillation[19]
    - The surface ECG shows 'absolutely' irregular RR intervals (AF is therefore sometimes known as arrhythmia absoluta), i.e. RR intervals that do not follow a repetitive pattern.
    - There are no distinct P waves on the surface ECG. Some apparently regular atrial electrical activity may be seen in some ECG leads, most often in lead V1.
    - The atrial cycle length (when visible), i.e. the interval between two atrial activations, is usually variable and 200 ms (.300 bpm).

Exclusion Criteria

- Transient atrial fibrillation (atrial fibrillation lasting for less than 24 hours).

Ethical clearance

Prior to the beginning the study was approved by the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed consent

The patients who fulfilled the selection criteria were informed about the nature of study in detail and a written informed consent was obtained (Annexure–I).

Data collection

Patients were interviewed and demographic data, history of present illness, other comorbid conditions, personal and treatment history were obtained. Further these patients underwent clinical examination followed by systemic examination. These findings were noted on a predesigned and pretested proforma (Annexure-II).

Investigations

Patients were subjected to following investigations.

- Complete blood count
- Serum TSH
- Blood urea nitrogen
- Serum creatinine
- Lipid profile – Cholesterol, LDL, HDL and triglycerides.
- Random blood sugar
- 12 lead ECG
- Chest X-ray
- 2D echocardiography

Outcome variables

Based on clinical presentation, examination and investigations, patients were evaluated for;

- Symptom profile
- Risk factor

- Clinical signs
- Lipid abnormalities
- Haematological and biochemical variations
- Complications
- Etiology
- Stroke risk assessment, and antithrombotic therapy
- Creatinine clearance

Stroke risk assessment, and antithrombotic therapy

The stroke risk assessment and antithrombotic therapy was determined using $CHADS_2$ score.

	Condition	Points
C	Congestive heart failure	1
H	Hypertension: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication)	1
A	Age ≥75 years	1
D	Diabetes mellitus	1
S_2	Prior Stroke or TIA or Thromboembolism	2

It is a clinical prediction rule for estimating the risk of stroke in patients with non-rheumatic atrial fibrillation (AF), a common and serious heart arrhythmia associated with thromboembolic stroke. It is used to determine whether or not treatment is required with anticoagulation therapy or antiplatelet therapy,[64] since AF can cause stasis of blood in the upper heart chambers, leading to the formation of a mural thrombus that can dislodge into the blood flow, reach the brain, cut off supply

to the brain, and cause a stroke. A high $CHADS_2$ score corresponds to a greater risk of stroke, while a low $CHADS_2$ score corresponds to a lower risk of stroke. The $CHADS_2$ score is simple and has been validated by many studies.[65]

To complement the $CHADS_2$ score, by the inclusion of additional 'stroke risk modifier' risk factors, the CHA_2DS_2-VASc-score has been proposed.[66] These additional non-major stroke risk factors include age 65-74, female gender and vascular disease. In the CHA_2DS_2-VASc score, 'age 75 and above' also has extra weight, with 2 points.

$CHADS_2$ scoring interpretation

$CHADS_2$ Score	Stroke Risk %	95% CI
0	1.9	1.2–3.0
1	2.8	2.0–3.8
2	4.0	3.1–5.1
3	5.9	4.6–7.3
4	8.5	6.3–11.1
5	12.5	8.2–17.5
6	18.2	10.5–27.4

The $CHADS_2$ scoring table is shown above:[65] adding together the points that correspond to the conditions that are present results in the $CHADS_2$ score, that is used to estimate stroke risk.

CHA_2DS_2-VASc score

In clinical use, the $CHADS_2$ score has been superseded by the CHA_2DS_2-VASc score that gives a better stratification of low-risk patients.

	Condition	Points
C	Congestive heart failure (or Left ventricular systolic dysfunction)	1
H	Hypertension: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication)	1
A_2	Age ≥75 years	2
D	Diabetes Mellitus	1
S_2	Prior Stroke or TIA or thromboembolism	2
V	Vascular disease (e.g. peripheral artery disease, myocardial infarction, aortic plaque)	1
A	Age 65–74 years	1
Sc	Sex category (i.e. female sex)	1

The CHA_2DS_2-VASc[67] score is a refinement of $CHADS_2$ score[65,66] and extends the latter by including additional common stroke risk factors. The maximum $CHADS_2$ score is 6, whilst the maximum CHA_2DS_2-VASc score is 9 (for age, either the patient is ≥75 years and gets two points, is between 65-74 and gets one point, or is under 65 and does not get points). Female gender only scores one point if the patient has at least one other risk factor, and does not score any points in isolation.

Interpretation of CHA_2DS_2-VASc score

Score	Risk	Anticoagulation Therapy	Considerations
0	Low	None or Aspirin	Aspirin daily
1	Moderate	Aspirin, Warfarin, or other oral anti-coagulant	Aspirin daily or raise INR to 2.0-3.0, depending on patient preference
2 or greater	Moderate or High	Warfarin or other oral anti-coagulant	Raise INR to 2.0-3.0, unless contraindicated

Treatment strategies recommended based on the $CHADS_2$ score are shown in the table.[65,66]

How the treatment recommendations based on the $CHADS_2$ score are modified by considering additional 'stroke risk modifier' risk factors using the CHA2DS2-VASc score.[68]

Creatinine clearance

The creatinine clearance was calculated using the formula as below.[69]

Est. Creatinine Clearance = [[140 - age(yr)]*weight(kg)]/[72*serum Cr(mg/dL)]

In case of women the value obtained was multiplied by 0.85.

Interpretation

Creatinine clearance values were interpreted as normal if found in the range as below.[69]

- Male: 97 to 137 ml/min.
- Female: 88 to 128 ml/min.

Statistical methods

The data obtained was coded and entered into the Microsoft Excel Spreadsheet (Annexure III). The categorical data was expressed in terms of rates, ratios and percentages and continuous data was expressed as mean ± standard deviation.

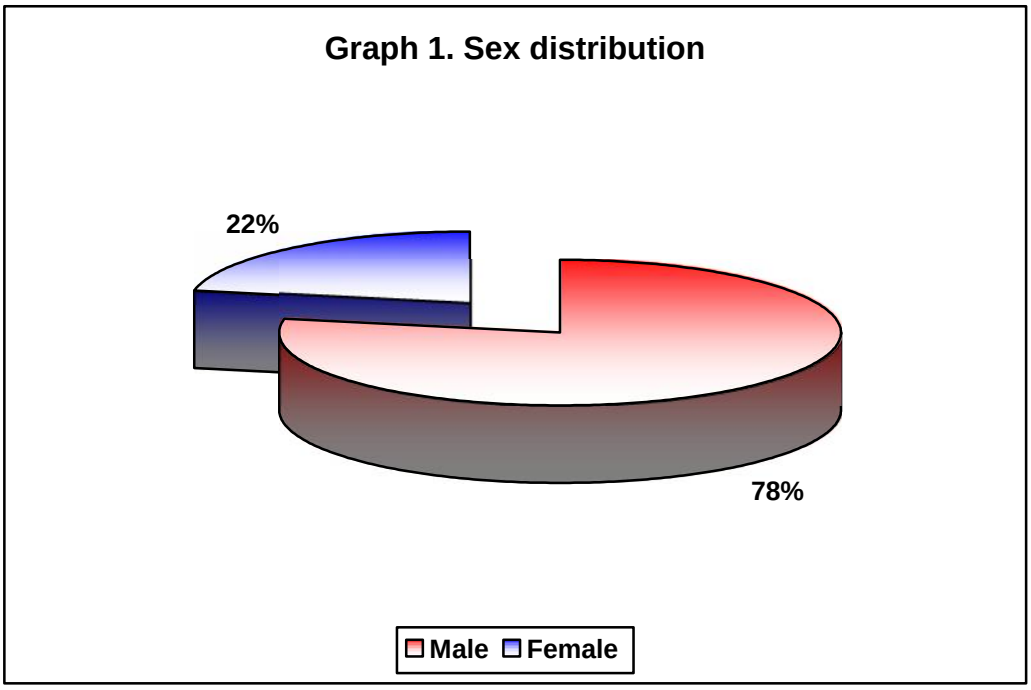
RESULTS

This one year cross-sectional study was done at Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 50 elderly patients (age > 65 years) who presented with atrial fibrillation were included in the study.

The data obtained was coded and entered into the Microsoft Excel Spreadsheet (Annexure III). The data was analysed and the final results and observations were tabulated as below.

Table 1. Sex distribution

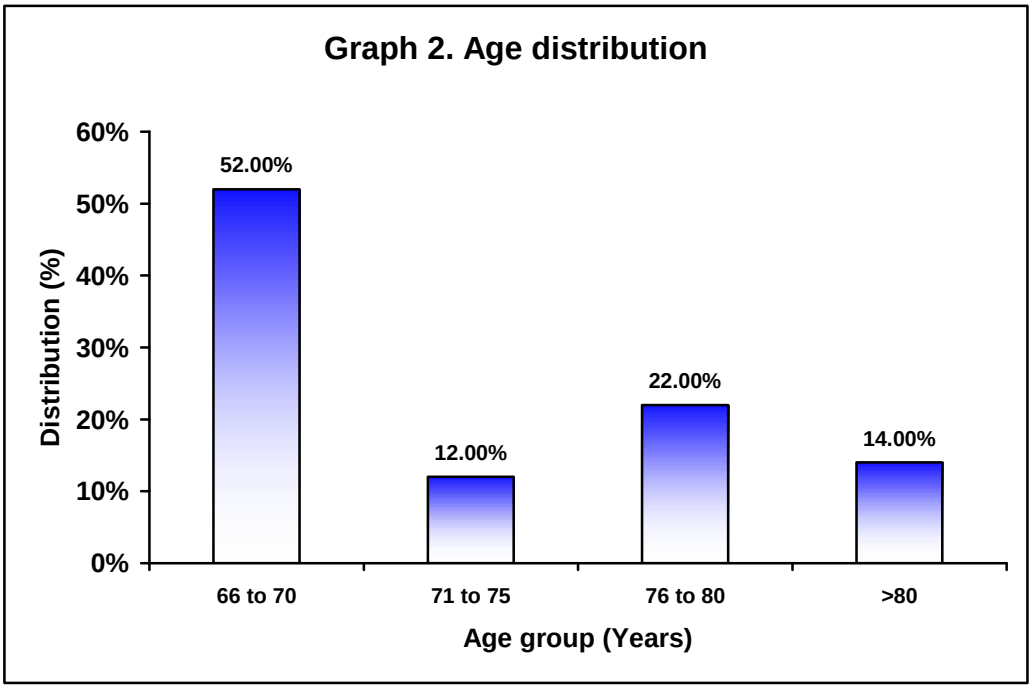
Sex	Distribution (n=50)	
	Number	**Percentage**
Male	39	78.00
Female	11	22.00
Total	**50**	**100.00**



In the present study majority of the patients were males (78%). The male to female ratio was 3.54:1.

Table 2. Age distribution

Age group (Years)	Distribution (n=50)	
	Number	**Percentage**
66 to 70	26	52.00
71 to 75	6	12.00
76 to 80	11	22.00
> 80	7	14.00
Total	**50**	**100.00**

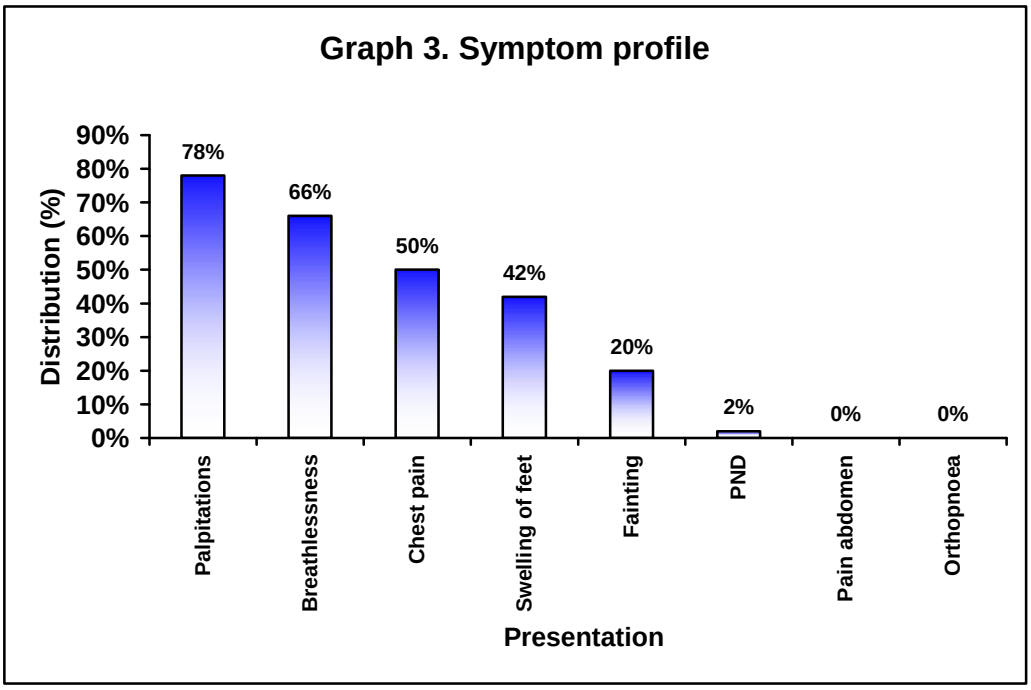


In this study 52% of the patients presented with age between 66 to 70 years, 22% between 76 to 80 years, 14% with > 80 years and 12% between 71 to 75 years. The mean age was noted as 72.32 ± 6.54 years.

Table 3. Symptom profile

Presentation	Distribution (n=50)	
	Number	Percentage
Palpitations	39	78.00
Breathlessness	33	66.00
Chest pain	25	50.00
Swelling of feet	21	42.00
Fainting	10	20.00
PND	1	2.00
Pain abdomen	0	0.00
Orthopnoea	0	0.00

Multiple presentations hence total not shown

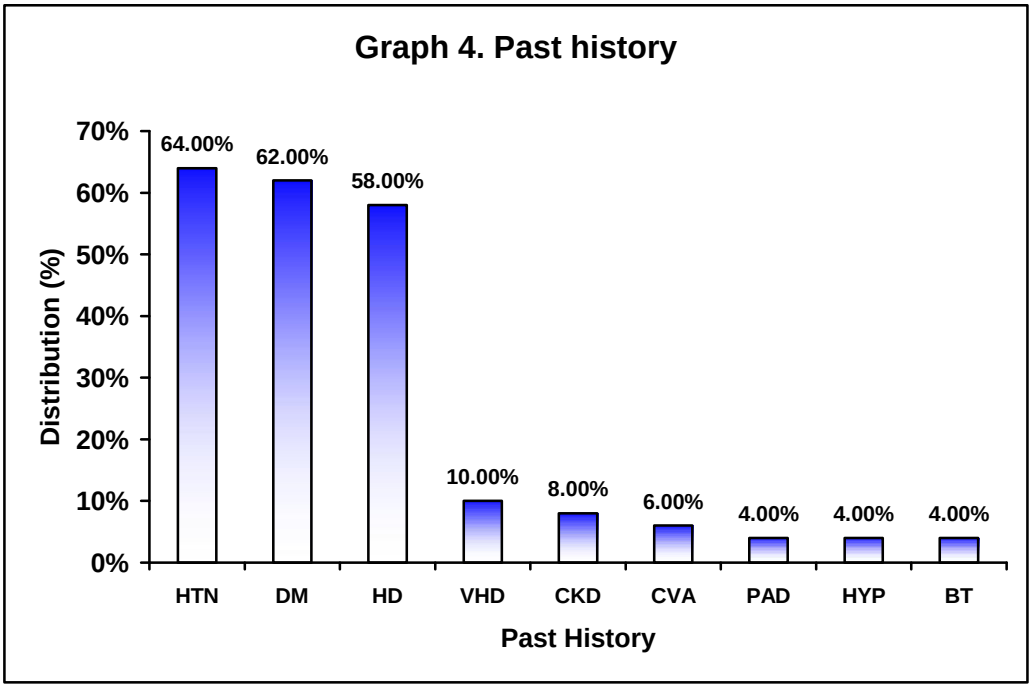


In the present study palpitations were noted as commonest symptom (78%) followed by breathlessness (66%), chest pain (50%), swelling of feet (42%), fainting (20%) and PND (2%).

Table 4. Past history

Past history	Distribution (n=50)	
	Number	Percentage
Hypertension	32	64.00
Diabetes mellitus	31	62.00
Heart disease	29	58.00
Valvular heart disease	5	10.00
Chronic kidney disease	4	8.00
Cerebrovascular accident	3	6.00
Peripheral artery disease	2	4.00
Hypothyroidism	2	4.00
Bleeding tendency	2	4.00

Multiple presentations hence total not shown

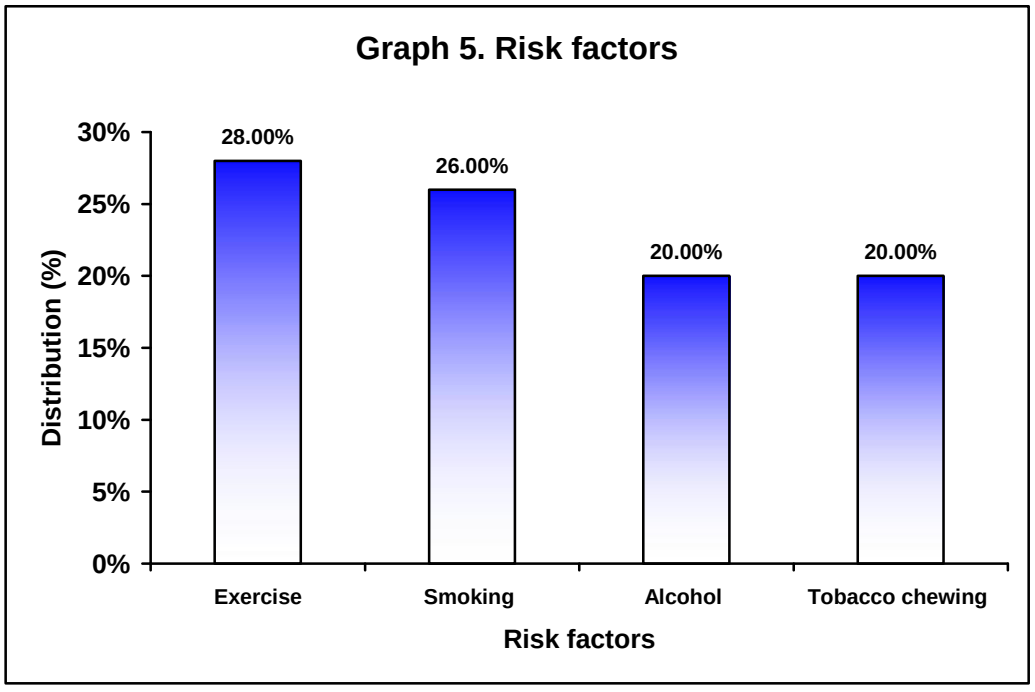


In this study past history of hypertension, diabetes mellitus and Heart Disease were present in 64%, 62% and 58% respectively. The other comorbid conditions are as shown in table 4 and graph 4.

Table 5. Risk factors

Risk factors	Distribution (n=50)	
	Number	Percentage
Exercise	14	28.00
Smoking	13	26.00
Alcohol	10	20.00
Tobacco chewing	10	20.00

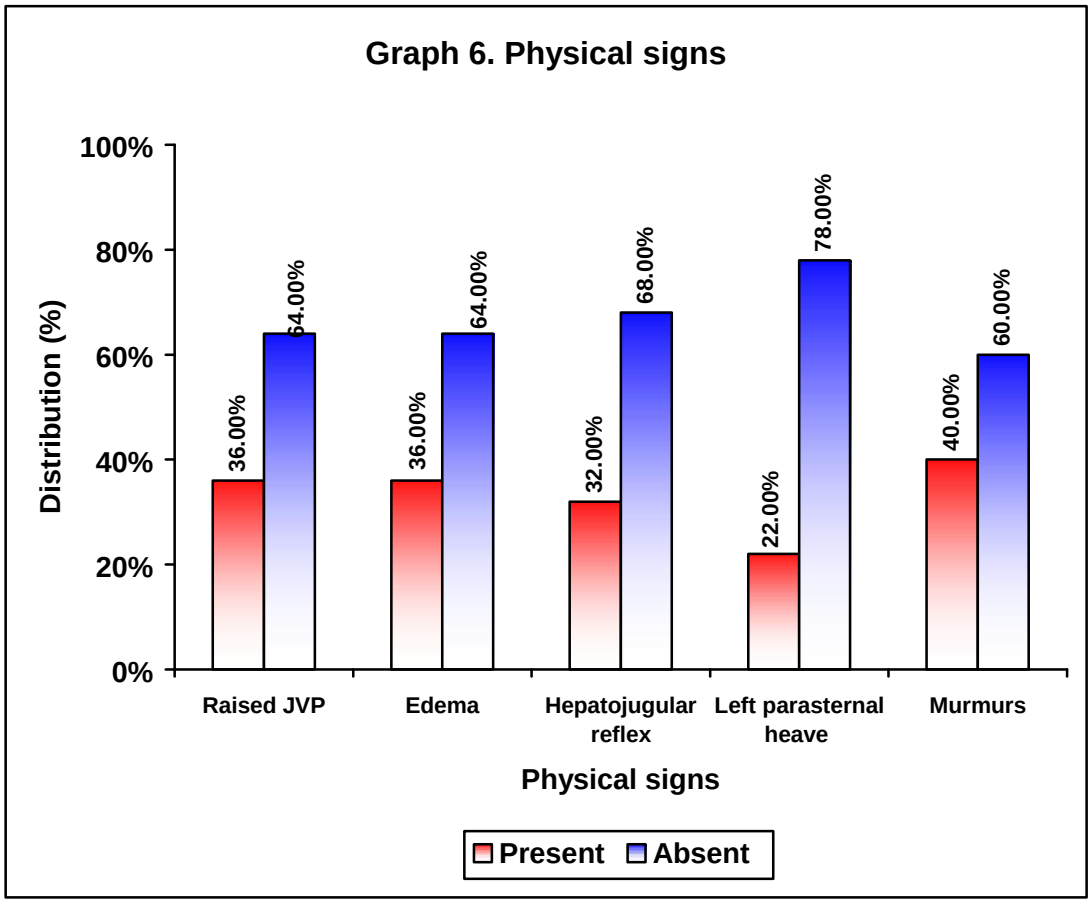
Multiple presentations hence total not shown



In this study in 28% of the patients history of exercise was present

Table 6. Physical signs

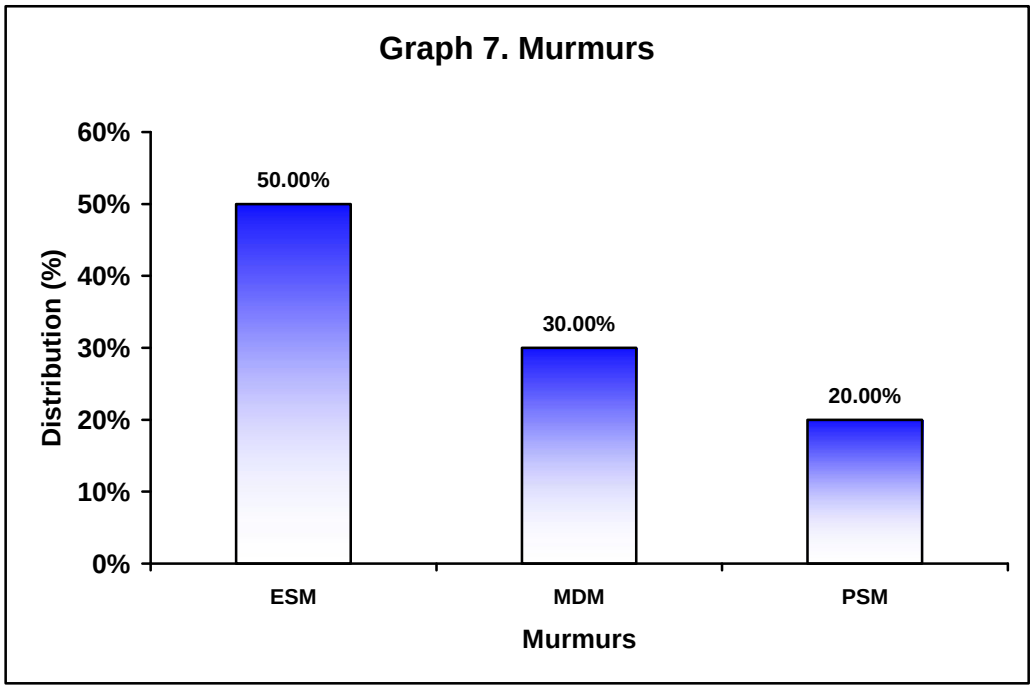
Signs	Findings	Distribution (n=50)	
		Number	**Percentage**
Raised JVP	Present	18	36.00
	Absent	32	64.00
	Total	**50**	**100.00**
Edema	Present	18	36.00
	Absent	32	64.00
	Total	**50**	**100.00**
Hepatojugular reflex	Present	16	32.00
	Absent	34	68.00
	Total	**50**	**100.00**
Left parasternal heave	Present	11	22.00
	Absent	39	78.00
	Total	**50**	**100.00**
Murmurs	Present	20	40.00
	Absent	30	60.00
	Total	**50**	**100.00**



In this study on examination 36% of the patients each had raised JVP and oedema. The other signs included hepatojugular reflex (32%), left parasternal heave (22%) and murmurs (40%).

Table 7. Murmurs

Murmurs	Distribution (n=50)	
	Number	**Percentage**
ESM	10	50.00
MDM	6	30.00
PSM	4	20.00
Total	**20**	**100.00**



In the present study murmurs were noted in 20 patients. Among them, ESM, MDM and PSM were noted in 50%, 30% and 20% of the patients respectively.

Table 8. Clinical examination findings

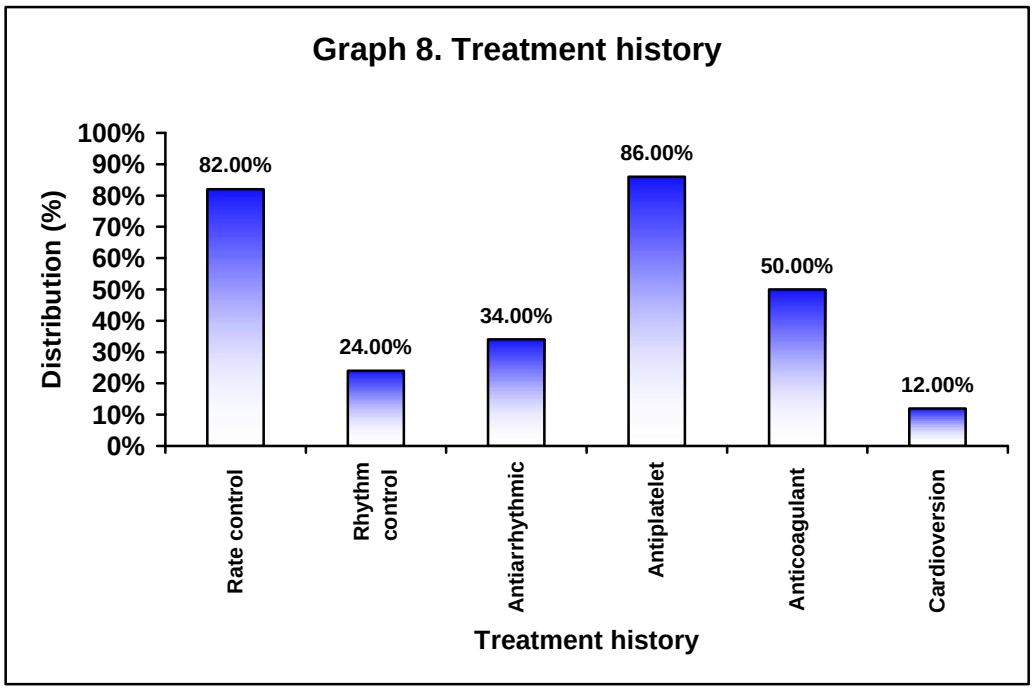
Variables	Distribution (n=50)	
	Mean	**SD**
Pulse rate (/Minute)	110.44	24.75
SBP (mm Hg)	111.76	21.06
DBP (mm Hg)	71.36	9.19
Height (Cms)	177.68	18.10
Weight (Kgs)	68.01	19.67

The clinical examination findings including vitals that is, pulse rate, systolic and diastolic blood pressure, and anthropometry that is, height and weight are as shown in table 8.

Table 9. Treatment history

History	Distribution (n=50)	
	Number	Percentage
Rate control	41	82.00
Rhythm control	12	24.00
Antiarrhythmic	17	34.00
Antiplatelet	43	86.00
Anticoagulant	25	50.00
Cardioversion	6	12.00

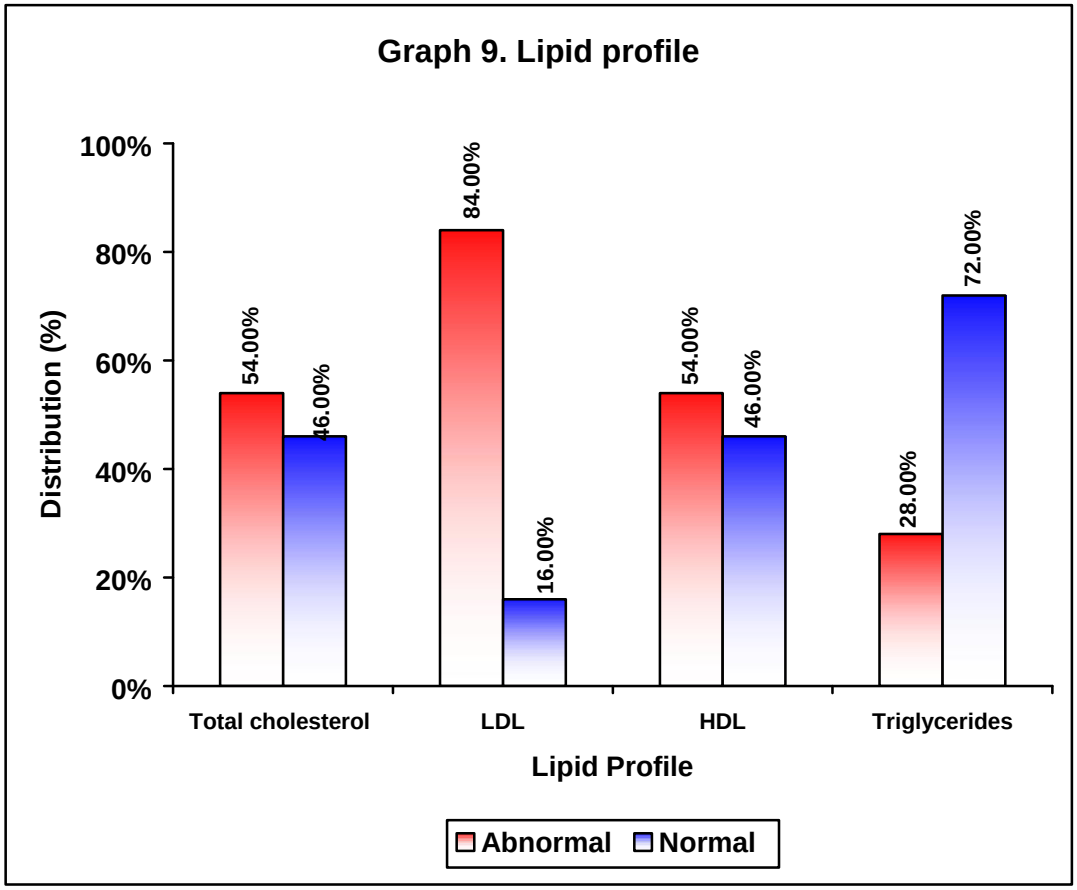
Multiple presentations hence total not shown



In this study. majority of the patients presented were subjected to rate control (82%) than rhythm control (24%) and were being treated with antiplatelets (86%) and anticoagulants (50%). History of cardioversion was present in only (6%).

Table 10. Lipid profile

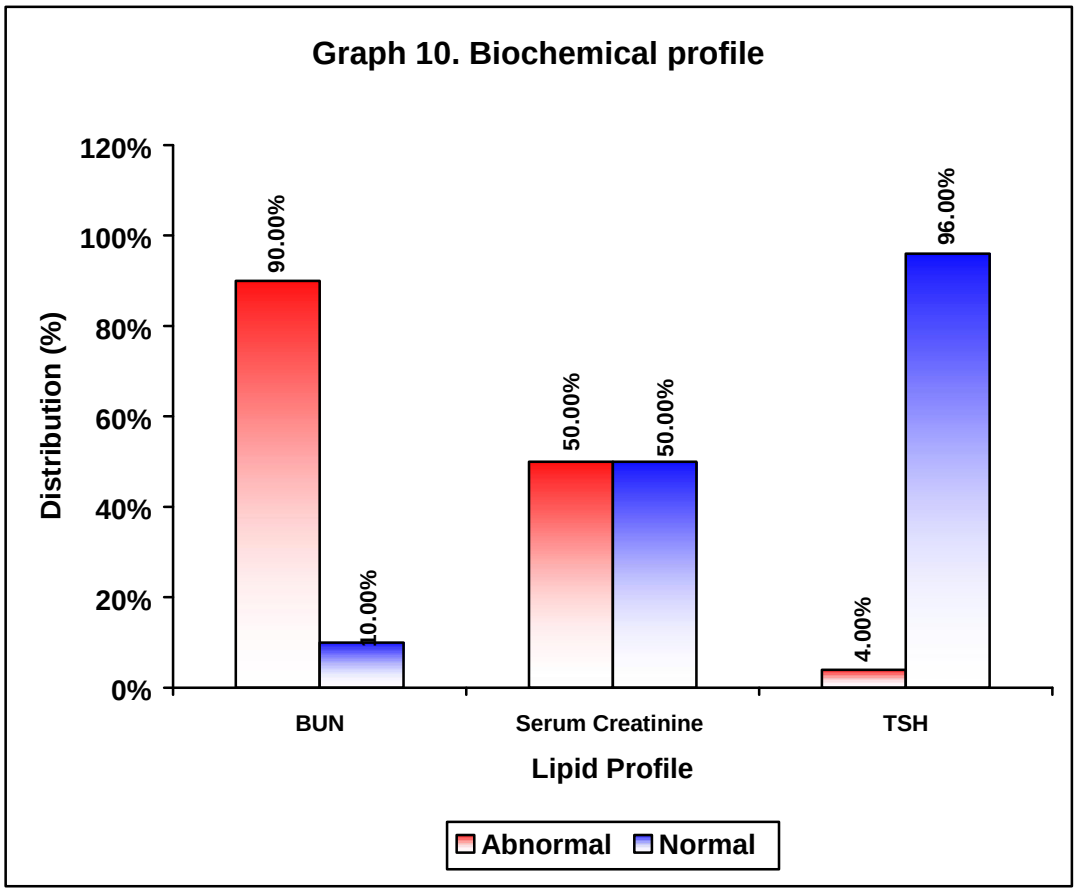
Lipids	Findings	Distribution (n=50)	
		Number	**Percentage**
Cholesterol levels	Raised (≥200 mg/dL)	27	54.00
	Normal (<200 mg/dL)	23	46.00
	Total	**50**	**100.00**
	Mean ± SD	**200.76**	**55.63**
Low density lipoprotein	Raised (≥100 mg/dL)	42	84.00
	Normal (<100 mg/dL)	8	16.00
	Total	**50**	**100.00**
	Mean ± SD	**152.84**	**92.42**
High density lipoprotein	Abnormal (≤ 40 / ≤ 50 mg/dL)	27	54.00
	Normal (>40 / >50 mg/dL)	23	46.00
	Total	**50**	**100.00**
	Mean ± SD	**52.38**	**32.91**
Triglycerides	Abnormal (≥ 150 mg/dL)	14	28.00
	Normal (<150 mg/dL)	36	72.00
	Total	**50**	**100.00**
	Mean ± SD	**127.18**	**85.64**



In the present study lipid profile revealed maximum patients with abnormal LDL (84%) followed by abnormal HDL (54%), raised total cholesterol (54%) and triglycerides (28%). The mean total cholesterol, LDL, HDL and triglycerides are as shown in table 10.

Table 11. Biochemical profile

Variables	Findings	Distribution (n=50)	
		Number	**Percentage**
Blood Urea Nitrogen	Abnormal (≥ 21 mg/dL)	45	90.00
	Normal (3 to 20 mg/dL)	5	10.00
	Total	**50**	**100.00**
	Mean ± SD	**66.52**	**53.29**
Serum creatinine	Raised (≥ 1.4 mg/dL)	25	50.00
	Normal (< 1.4 mg/dL)	25	50.00
	Total	**50**	**100.00**
	Mean ± SD	**2.20**	**2.25**
TSH	Raised (≥4.8)	2	4.00
	Normal (0.4-4.8)	48	96.00
	Total	**52**	**104.00**
	Mean ± SD	**1.62**	**1.17**



In this study the biochemical profile revealed majority of the patients with raised blood urea nitrogen (90%) followed by raised serum creatinine (50%). However, few that is 4% of the patients presented with raised TSH levels.

Table 12. Haematological profile

Variables	Distribution (n=50)	
	Mean	**SD**
Haemoglobin	10.93	2.31
Total count	9743.44	3173.39
Neutrophils	76.2	11.57
Eosinophils	9.14	9.67
Monocytes	4.38	6.14
Lymphocytes	10.20	2.80
INR	1.83	1.30

The Haematological profile of the patients that is, mean haemoglobin, total count, neutrophils, eosinophils, monocytes, lymphocytes and INR are as shown in table 12.

Table 13. Electrocardiogram findings

Variables	Findings	Distribution (n=50)	
		Number	**Percentage**
Rate	< 100	19	38.00
	100 - 130	18	36.00
	> 130	13	26.00
	Total	**50**	**100.00**
Axis	Normal axis	31	62.00
	Left axis deviation	14	28.00
	Right axis deviation	5	10.00
	Total	**50**	**100.00**
Blocks	Absent	48	96.00
	LBBB	2	4.00
	Total	**50**	**100.00**
Ischemic changes	Absent	19	38.00
	Present	31	62.00
	Total	**50**	**100.00**
Hypertrophy	Absent	37	74.00
	LVH	13	26.00
	Total	**50**	**100.00**

Table 13 shows electrocardiogram findings. It was observed that, most of the patients had abnormal rate (62%), ischaemic changes (62%), axis deviation (38%) and left ventricular hypertrophy (26%)

Table 14. X-ray changes

Findings	Distribution (n=50)	
	Number	**Percentage**
LVH	18	36.00
LVH+LAE	12	24.00
LAE	7	14.00
LAE + RVH	7	14.00
Emphysema + RAE	3	6.00
RAE+RVH	2	4.00
Normal	1	2.00
Total	**50**	**100.00**

In the present study on X-ray, most of the patients had LVH (36%) followed by LVH with LAE (24%), LAE (14%) and LAE+RVH (14%)

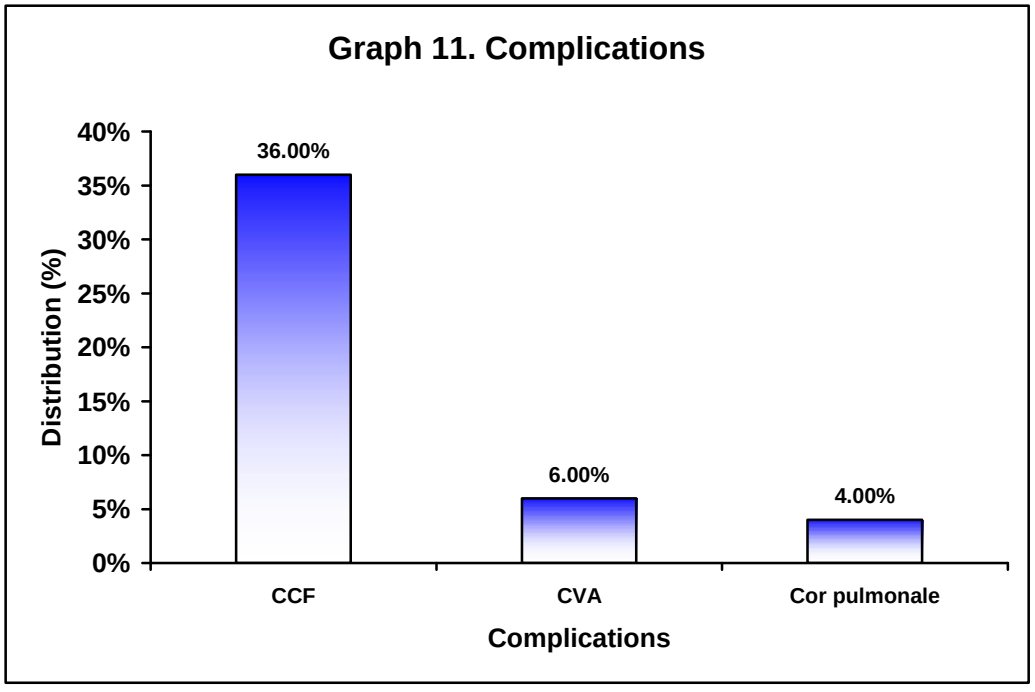
Table 15. 2D Echocardiography findings

Variables	Findings	Distribution (n=50)	
		Number	**Percentage**
LVISd	Normal (2.4-3.2)	19	38.00
	Dilated	31	62.00
	Total	**50**	**100.00**
LVIDd	Normal (3.9-5.3)	28	56.00
	Dilated	22	44.00
	Total	**50**	**100.00**
LA AP	Normal (3.0-4.0)	11	22.00
	Dilated	39	78.00
	Total	**50**	**100.00**
Ejection fraction	≥ 60	25	50.00
	45 to 59	12	24.00
	< 45	13	26.00
	Total	**50**	**100.00**
Mitral valve	Calcification	13	26.00
	Normal	27	54.00
	Thick	10	20.00
	Total	**50**	**100.00**
Aortic valve	Calcified	29	58.00
	Normal	21	42.00
	Total	**50**	**100.00**

The 2D echocardiographic findings are as shown in table 15. It was observed that 50% of the patients had ejection fraction of < 60%, 78% of the patients had dilated left atrium, 62% of the patients had dilated left ventricle, Thickened mitral valve was noted in 20% and calcification in 26%. Aortic valve calcification was present in 58% of the patients.

Table 16. Complications

Complications	Distribution (n=50)	
	Number	**Percentage**
CCF	18	36.00
CVA	3	6.00
Cor pulmonale	2	4.00
Absent	27	54.00
Total	**50**	**100.00**



In this study most of the patients had complications of CCF (36%) followed by CVA (6%) and cor pulmonale (4%).

Table 17. Etiology

Etiology	Distribution (n=50)	
	Number	**Percentage**
Hypertension	32	64.00
Diabetes mellitus	31	62.00
Cardiomyopathy	21	42.00
Ischaemic heart disease	15	30.00
Rheumatic heart disease	5	10.00
COPD	3	6.00
Surgical stress	2	4.00
Chronic kidney disease	2	4.00
Hypothyroidism	2	4.00
Total	**50**	**100.00**

In the present study hypertension and diabetes mellitus were the etiologies in 64% and 62% of the patients respectively. The other etiologies noted were cardiomyopathy (42%) ischaemic heart disease (30%), rheumatic heart disease (10%), COPD (6%), surgical stress (4%), Chronic kidney disease (4%) and hypothyroidism (4%).

Table 18. Annual stroke risk based on $CHADS_2$ score

$CHADS_2$ score	Stroke risk (%)	Distribution (n=50)	
		Number	Percentage
0	1.9	6	12.00
1	2.8	7	14.00
2	4.0	19	38.00
3	5.9	10	20.00
4	8.5	7	14.00
5	12.5	1	2.00
6	18.2	0	0.00
Total		**50**	**100.00**

In the present study Annual stroke risk based on $CHADS_2$ score showed maximum patients (38%) with intermediate risk of thromboembolic event and 4.0% risk of event per year if no Coumadin while 20% of the patients and 14% of the patients with CHADS2 score of 3 and 4 had high risk of thromboembolic event (5.9% and 8.5% risk of event per year if no Coumadin respectively).

Table 19. Requirement of anticoagulation based on CHA_2DS_2-VASc score

CHA_2DS_2-VASc score	Risk	Distribution (n=50)	
		Number	Percentage
0	Low	0	0.00
1	Moderate	6	12.00
2 or more	Moderate or High	44	88.00
Total		**50**	**100.00**

In the present study CHA_2DS_2-VASc score revealed moderate to high risk of stroke in maximum patients (88%).

Table 20. Creatinine clearance

Creatinine clearance	Distribution (n=50)	
	Number	Percentage
Normal	3	6.00
Low	47	94.00
Total	**50**	**100.00**

In the present study creatinine clearance was observed to be low in majority of the patients (94%).

DISCUSSION

With aging, the prevalence of atrial fibrillation (AF) increases. In individuals aged 65 and older, the prevalence of AF is about 5%, and doubles to 10% in those aged 80 and older.[70] Additionally, as the population ages, the number of people with AF is rising dramatically, with 5.6 million people in the United States projected to have AF by the year 2050. In this population, the risk of ischemic stroke is of primary concern; nonvalvular AF increases the risk of stroke by 5-fold and is responsible for approximately 24% of strokes in patients aged 80 to 89.[70]

AF is a heterogeneous condition, with significant differences in its epidemiology, pathogenesis, clinical presentation and management across age groups. Older patients are more likely to have an abnormal substrate and present at an advanced stage with atypical symptoms and associated comorbidities. This necessitates for knowledge about the important differences between AF in the young and elderly so as to identify the condition and target management strategies to relieve symptoms as well as to prevent complications. However, data is scarce in elderly population. Hence this study was planned to explore the clinical profile of atrial fibrillation in elderly patients.

The present one year cross-sectional study was done from January 2013 to December 2013 at Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 50 elderly patients who were aged more than 65 years, presented with atrial fibrillation during the study period were included. Patients underwent clinical, haematological, biochemical examination. Electrocardiograph and 2D echocardiography was also done.

In this study the commonest age group was 66 to 70 years noted in 42% of the patients. The next common age group was between 76 to 80 years with 22% of the patients. The mean age was noted as 72.32 ± 6.54 years indicating majority of the patients above the age 70 years.

In the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM)[71] Study, a total of 4060 patients were enrolled in which the average age of patients with atrial fibrillation was 70 years.

In the Framingham study,[72] 2325 men and 2826 women, 30 to 62 years old at entry were followed biennially over 22 years for the development of chronic atrial fibrillation in relation to antecedent cardiovascular disease and risk factors. The incidence rose sharply with age. Overall there was a 2.0 percent chance that the disorder would develop in two decades. Similarly, the prevalence of atrial fibrillation was 0.5% for the group aged 50-59 years which rose to 8.8% in the group aged 80 to 89 years.[73]

The findings of the present study that the incidence of atrial fibrillation increases with increasing age is in agreement with the findings noted in the above two studies.

In the present study male preponderance was noted, as 78% of the patients were males, with male to female ratio of 3.54:1.

A study[74] from Bareily, India also reported higher proportion (54.54%) of the patients as males compared to females (45.46%). Similar observations were made by other studies also.[8,57,75] In contrast, Manyari DE et al[76] in their study on patients with AF reported 37 men vs 49 women out of 86 subjects. Gurupal Singh et al[77] in their

study of 64 patients in AF found the sex distribution to 29 males vs 35 females. In the Framingham study William B Kannel et al,[75] 2325 men and 2826 women were followed biennially over 22 years for the development of atrial fibrillation. The incidence of atrial fibrillation did not differ significantly between sexes.

However, the male preponderance observed in the present study would be attributed to the risk profile of the study population including exercise, smoking, alcohol and tobacco chewing and the smaller number of patients in the study population.

Atrial fibrillation has a wide spectrum of clinical presentations. Some patients may be asymptomatic. Others may present with stroke, overt heart failure, or cardiovascular collapse. Patients most commonly report palpitations, dyspnea, fatigue, lightheadedness, and chest pain. Because symptoms are nonspecific, they cannot be used to diagnose and determine the onset of atrial fibrillation.[21]

In this study more than three fourth of the patients presented with palpitations (78%). The next common compliant was breathlessness noted in 66%, followed by chest pain in 50%, swelling of feet in 42% and fainting in 20%. On examination, most of the patients had raised JVP and oedema (36% each) followed by hepatojugular reflex (32%), left parasternal heave (22%) and murmurs (40%). Among the patients with murmurs 50% of the patients had ESM in aortic area, 30% had MDM in mitral area and 20% had PSM in mitral area. Chatap G et al[78] reported that, clinical symptoms of atrial fibrillation are varied and include palpitations, syncope, dizziness or embolic events.

To-date despite of higher prevalence of AF in elderly population the clinical profile in this subset remains least explored. However studies in general population have showed vide variation in clinical presentation. A study by Kumar B et al[79] on 100 patients in general population reported, 74% of patients with history of dyspnoea, followed by palpitation in 57%. Mild to moderate chest pain was present in 11% of patients, 17% patients had syncope /dizzy spells, fatigue was noticed in 19% patients. Tischler et al[80] reported dyspnoea in 62% of patients, palpitation in 33% patients, and syncope in 12% patients in a similar study. Flaker et al[81] in their study observed that 78% patients had dyspnoea and 11 % had chest pain at presentation whereas Levey et al[82] reported that 54.1% patients had palpitation, 44.4% patients had dyspnoea and 10.1% patients had chest pain.

AF is classically associated with "typical" symptoms of irregular palpitations, with or without chest pain, breathlessness, or dizziness. Palpitations are reported in 80% of young patients with paroxysmal AF. In contrast, less than 10% of AF patients over the age of 80 years have palpitations and up to 40% of elderly hospital inpatients found to have AF are entirely asymptomatic. Whilst atypical chest pain is relatively common in young AF patients, in elderly patients anginal chest pain during AF episodes strongly suggests the presence of significant concurrent coronary disease and might be sufficient to warrant investigation for coronary ischaemia even in the absence of typical symptoms of angina.[33]

Suspicion or confirmation of atrial fibrillation necessitates investigation and, as far as possible, appropriate treatment of underlying causes such as hypertension, diabetes mellitus, hypoxia, hyperthyroidism and congestive heart failure.

In the present study hypertension and diabetes mellitus were the commonest comorbid conditions documented in nearly two third of the study population. The next common condition was heart disease noted in 58% of the patients. According to Davis et al,[83] IHD and hypertension are the most commonly found condition in patients of AF. In India, a study conducted by Singh et al[84] reported RHD in 37.87%, HTN in 3% and IHD in 3.03%. Kumar et al[85] reported RHD in 39%, IHD in 29% and HTN in 54%.

The two studies quoted by Kumar et al[84] and Singh et al[85] by Indian authors had an increased incidence of rheumatic heart disease, but in the present study the lower incidence of rheumatic heart disease is possibly because of the lower incidence of rheumatic valvular heart disease in the elderly population as RVHD occurs at an younger age group amongst Indians.

Also the findings of the present study were consistent with a study by Davis et al[83] and Kumar et al[85] who reported IHD and hypertension as most commonly found conditions in patients with AF, these studies were carried out on general population.

It is noteworthy that the prevalence of hypertension in patients with AF also increased in the last decade. On the other hand, there has been progress in the rate of hypertension control (relative increase of 38.5%). This is important because of the emphasis on upstream therapies to slow or halt the progression of AF due to underlying cardiovascular disease and to AF itself.[86]

Triglyceride levels or several lipid ratios that have been proposed as biomarkers for atherosclerotic diseases were not associated with AF. In the present

study maximum patients had lipid profile abnormalities. Low density LDL was abnormal in 84%, abnormal HDL in 54%, raised total cholesterol in 54% and triglycerides 28%. These findings suggest that, elderly patients with atrial fibrillation are likely to present with dyslipidemia. It is reported that dyslipidemia is associated with atherosclerosis and is a well established independent risk factor for cardiovascular diseases, such as coronary artery disease and stroke, as well as heart failure. Several factors, including age, gender, obesity, metabolic syndrome, and hypertension, have been associated with an increased risk of developing AF, suggesting a strong association between AF and atherosclerosis.

The association between dyslipidemia and AF, however, has been controversial. In one cross-sectional study, total cholesterol, triglycerides, and HDL cholesterol levels were lower in patients with paroxysmal AF than in those without AF.[88] In another study, total cholesterol levels were lower in patients with AF than in those without AF, but HDL cholesterol levels were not different between the groups.[89] In another cross-sectional study, there was no relationship between the lipid levels and AF.[90] The inconsistent results for the association between lipid levels and AF in previous studies may have been driven by the cross-sectional study design;[89,90] and the recruitment of general population.

The results of the present study are consistent with those of a previous longitudinal study of patients with hypertension, which were that there was a higher incidence of AF in individuals with low HDL cholesterol than in those with high HDL cholesterol.[91]

In the present study there is an increase in LDL cholesterol in 84% and decreased HDL amongst 54%, this can be attributed to the study population being elderly, and 62% of the study population have either diabetes, hypertension or ischemic heart disease, this was not found amongst most of the other studies done, as they were carried out in general population.

In this study, X-ray findings showed nearly one third of the patients had Left ventricular enlargement (36%) and next common finding was Left ventricular enlargement along with left atrial enlargement (24%). This is possibly due to increased incidence of IHD in the present study.

In the present study, ECG findings revealed 64% of the patients with abnormal rate and ischaemic changes were noted in 62% of the patients. However left axis deviation and left ventricular hypertrophy were documented among 38% and 26%.

Substantial evidence suggests that, the RR intervals follow no repetitive pattern in patients with AF and they have been labeled as “irregularly irregular.” The ventricular rate usually ranges from 90 to 170 beats/min and the QRS complexes are narrow unless AV conduction is abnormal due to functional (rate-related) aberration, pre-existing bundle branch or fascicular block, or ventricular preexcitation. Although ECG findings described above usually allow the diagnosis of AF to be made easily, there are several pitfalls in correct identification of the rhythm. Errors in the diagnosis of AF are especially common with computerized ECG interpretation.

Routine performance of transthoracic echocardiography is suggested for all patients presenting with their first episode of atrial fibrillation (AF) to obtain information regarding atrial size, ventricular function, possible pericardial effusion, and valvular function. Repeated transthoracic echocardiographic examinations for recurrent AF are not necessary unless the clinical presentation has changed.

In the present study, on 2D echocardiography, it was observed that 50% of the patients had ejection fraction of < 60%, 78% of the patients had dilated left atrium, 62% of the patients had dilated left ventricle, Thickened mitral valve was noted in 20% and calcification in 26%. Aortic valve calcification was present in 58% of the patients.

The Framingham Study has examined the echocardiographic predictors of AF.[75] The echocardiographic characteristics of persons who developed AF included larger left atrial, and left ventricular dimensions and ventricular wall thickness and more mitral annular calcification. Each 5mm increment in left atrial dimension increased AF risk by 39%. A 5% decrement in left ventricular fractional shortening increased the risk by 34%. A 4 mm increment in left ventricular wall thickness increased AF risk by 28%. Mitral annular calcification doubled the risk. Those with two or more of the foregoing echocardiographic abnormalities had a 4-fold greater risk than those free of them all.

Henry et al in their study of 85 patients with isolated mitral valve disease, 50 patients with isolated aortic valve disease, and 130 patients with asymmetric septal hypertrophy. In all three groups of patients, atrial fibrillation was rare when left

atrial dimension was below 44 mm (3 of 117 or 3%) but common when this dimension exceeded 40 mm (80 of 148 or 54%).

In addition, when left atrial dimension exceeds 45 mm, cardioversion, while initially successful, is unlikely to produce sinus rhythm that can be maintained at least six months. The echocardiographic findings in the present study are in agreement with the studies quoted above.

In this study nearly one third of the study population had complications of CCF. These findings were consistent with ALFA study[92] which showed 38% of patients with congestive cardiac failure. Similarly Sharma et al[93] reported 30% of patients were having symptomatic heart failure. In the present study few patients had CVA (6%) and cor pulmonale (4%). In the ALFA study[92] 10.8% cases presented with history of embolic episodes which is comparable with the present study. Hinton et al[94] reported a high incidence of stroke in mitral valve lesions and observed that stroke occurred in 41% of patients with rheumatic heart disease, 35% of patients with ischaemic heart disease and 35% of those with coexisting rheumatic heart disease and ischaemic heart disease.

According to Framingham study[75] the risk of stroke was 17 times in those with rheumatic AF when compared to general population. The attributable risk to stroke from atrial fibrillation is estimated to be 1.5% for 50 to 59 years age group and approaches 30% for those aged 80 to 89. Wolf PA et al[95] reported that rate of ischemic stroke among elderly people with atrial fibrillation averages 5% per year, about six times that of people without atrial fibrillation. Most ischemic strokes associated with atrial fibrillation are probably due to embolism of stasis - induced

thrombi in the left atrium and particularly its appendage. About 50% of the elderly patients with atrial fibrillation who have chronic hypertension have a major risk factor for primary cerebrovascular disease.

The risk of thromboembolism is well known; other outcomes of atrial fibrillation are less well recognised, such as its relationship with dementia, depression and death. Such consequences are responsible for diminished quality of life and considerable economic cost.

Atrial fibrillation is characterised by rapid and disorganised atrial activity, with a frequency between 300 and 600 beats/minute. The ventricles react irregularly, and may contract rapidly or slowly depending on the health of the conduction system. Clinical symptoms are varied, including palpitations, syncope, dizziness or embolic events. Atrial fibrillation may be paroxysmal, persistent or chronic, and a number of attacks are asymptomatic. Suspicion or confirmation of atrial fibrillation necessitates investigation and, as far as possible, appropriate treatment of underlying causes such as hypertension, diabetes mellitus, hypoxia, hyperthyroidism and congestive heart failure.[78]

In elderly patients, the management of atrial fibrillation varies; it requires an individual approach, which largely depends on comorbid conditions, underlying cardiac disease, and patient and physician preferences. This management is essentially based on pharmacological treatment, but there are also nonpharmacological options. Two alternatives are possible: restoration and maintenance of sinus rhythm, or control of ventricular rate, leaving the atria in arrhythmia.

Pharmacological options include antiarrhythmic drugs, such as class III agents, beta-blockers and class IC agents. These drugs have some adverse effects, and careful monitoring is necessary. The nonpharmacological approach to atrial fibrillation includes external or internal direct-current cardioversion and new methods, such as catheter ablation of specific foci, an evolving science that has been shown to be successful in a very select group of atrial fibrillation patients.[78]

In this study history of cardioversion was present in 12% of the study population whereas rate control was initiated in 82% of the patients and rhythm control was initiated in 24%.

It is postulated that, most patients who present with AF will require slowing of the ventricular rate to improve symptoms. Once sinus rhythm is achieved, a decision regarding the long-term approach to the management of the rhythm disturbance (rhythm versus rate control) should be made. A rhythm control strategy uses either antiarrhythmic drug therapy, percutaneous catheter ablation, and/or a surgical procedure. Electrical cardioversion may be necessary prior to an attempt to maintain sinus rhythm. Rate slowing drugs are generally started before rhythm control and continued in many patients who remain in sinus rhythm (in the event of return to AF).[96]

Current data suggest that rhythm and rate control strategies are associated with similar rates of mortality and serious morbidity, such as embolic risk. However, there are several reasons why pursuing a rhythm-control strategy would be preferred, including symptom improvement, younger patient age, and irreversible structural and electrical remodeling that occurs with longstanding persistent AF.

The decision to adopt a rhythm or rate control strategy is often dictated by the (1) presence of symptoms associated with atrial fibrillation and/or (2) presence of diminutions in left ventricular systolic function thought secondary to the arrhythmia.[96]

Another serious challenge in the management of chronic atrial fibrillation in older individuals is the prevention of stroke, its primary outcome, by choosing an appropriate antithrombotic treatment (aspirin or warfarin). Several risk-stratification schemes have been validated and may be helpful to determine the best antithrombotic choice in individual patients.[78]

In our study majority of the patients presented with treatment history of antiplatelets (86%) and anticoagulant (50%). Similar findings were reported in CARDIOTENS 1999[97] and CARDIOTENS 2009[98] which were two observational, cross-sectional, multicenter studies. A total of 32051 and 25137 subjects were analyzed in the two 1540 and 1524 of them, respectively, diagnosed with AF.[86]

The CHADS2 score, a stroke risk stratification scheme, was derived from the Atrial Fibrillation Investigators (AFI) and the SPAF Investigators risk stratification schemes. The CHADS2 score is based on a point system: 1 point each for, age 75 years and over, history of hypertension, diabetes or congestive heart failure and 2 points for a history of stroke or transient ischaemic attack. The AFI, SPAF and CHADS2 stratification schemes were evaluated and the predictive value of the CHADS2 score was confirmed in 1733 Medicare beneficiaries with non-valvular AF aged 65 to 95 years.[65]

In the present study Annual stroke risk based on $CHADS_2$ score showed as high as 38% of the patients with intermediate risk of thromboembolic event having 4.0% risk of a thromboembolic event per year without Coumadin. In 20% of the patients and 14% of the patients with $CHADS_2$ score of 3 and 4 was suggestive of high risk of thromboembolic event (5.9% and 8.5% risk of a thromboembolicevent per year without Coumadin respectively). Further CHA_2DS_2-VASc score revealed moderate to high risk of stroke (a score of 2 or more) in majority of the patients that is 88% indicating Warfarin or other oral anti-coagulant.

The Birmingham 2009 risk schema (CHA2DS2-VASc) was used to refine and improve stroke risk stratification in patients with AF by reclassifying and incorporating additional risk factors.[66] One point is assigned for congestive heart failure, hypertension, diabetes, vascular disease, age 65 to 74 years and female gender, and 2 points for age 75 years and over, and history of stroke or transient ischaemic attack. A high score in the CHA2DS2- VASc and CHADS2 stroke risk stratification schemes is associated with a high annualised stroke risk.[66,99]

As the population with AF is heterogeneous and includes a range of ischaemic stroke risks, the absolute benefit of anticoagulation depends on the underlying risk of stroke. A retrospective population-based study in Minnesota evaluated 97 patients with lone AF who were less than 60 years of age at the time of diagnosis (mean age 44 years) and found that lone AF was associated with a low risk of stroke with a cumulative stroke risk of 1.3% after 15 years.[100]

The Stroke Prevention in Atrial Fibrillation (SPAF) trials (n = 2012; mean age 69 years) excluded patients less than 60 years of age or with lone AF. It reported

that the six independent predictors strongly associated with increased stroke risk were: age, female gender, history of hypertension, systolic blood pressure above 150 mmHg, alcohol intake over 14 units per week and history of stroke or transient ischaemic attack.[101] The rate of ischaemic stroke was similar in those with paroxysmal or permanent AF.[102]

The SPAF, Framingham, AFI and CHADS2 stroke risk stratification schemes were conflicting as to whether female gender was an independent predictor for increased risk of stroke in AF. The SPAF and Framingham schemes considered women to be at high risk, especially women over 75 years of age, while the AFI and CHADS2 schemes did not include gender as a risk factor for stroke. Analysis of the Anticoagulation and Risk Factors in Atrial Fibrillation study population (n=13559) concluded that female gender was an independent predictor of risk at all age groups and across all stroke risk categories.16

Peripheral vascular disease, coronary artery disease, left atrial appendage thrombus, dense spontaneous echocardiographic contrast and the presence of complex aortic plaque all contribute to increased stroke risk in AF. However, these are not taken into account in current stroke risk stratification schemes, which aim to provide a simple clinician-friendly tool.[103-105]

The Birmingham 2009 risk schema (CHA2DS2-VASC) was compared with existing stroke risk stratification schemes in 1084 patients from the Euro Heart Survey for AF. The CHADS2 score classified over 60% of the cohort into an intermediate-risk group, while the CHA2DS2-VASc score improved predictive

value for thromboembolism risk, with only a small number of subjects classified in the intermediate-risk category.[66]

Both the CHADS2 and CHA2DS2-VASc scores have one of the best predictive values for thromboembolism in a 'real-world' elderly AF cohort.[106] Based on these stroke risk stratification schemes, all patients with AF aged over 75 years are at high risk and ideally should receive anticoagulants. The CHA2DS2-VASc risk scheme has been incorporated into the European Society of Cardiology's guidelines for management of AF.[19]

Atrial fibrillation is in most patients (approximately 70%) associated with chronic organic heart disease including valvular heart disease, coronary artery disease, hypertension, particularly if left ventricular hypertrophy is present, hypertrophic cardiomyopathy, dilated cardiomyopathy and congenital heart disease and most commonly in adults, atrial septal defect. As in many chronic conditions, determining whether AF is the result or is unrelated to the underlying heart disease, remains unclear. The list of possible etiologies also include cardiac amyloidosis, hemochromatosis and endomyocardial fibrosis. Other heart diseases, such as mitral valve prolapse (with or without mitral regurgitation), calcification of the mitral annulus, atrial myxoma, pheochomocytoma and idiopathic dilated right atrium, present a higher incidence of AF. The relationship between these findings and the arrhythmia are still unclear. Atrial fibrillation may occur in the absence of detectable organic heart disease, the so-called "lone AF", in about 30% of cases. The term "lone AF" or "idiopathic AF" implies the absence of any detectable etiology including hyperthyroidism, chronic obstructive lung disease, overt sinus node dysfunction, and overt or concealed preexcitation (Wolf-Parkinson-White

syndrome), only to mention a few of other rare causes of AF. In every instance of recently discovered AF, thyrotoxicosis should be ruled out. The autonomous nervous system may contribute to the occurrence of AF in some patients. Atrial fibrillation occurs commonly in patients with valvular heart disease, particularly when it involves the mitral valve. The occurrence of AF is unrelated to the severity of mitral stenosis but is more common in patients with enlarged left atrium and congestive heart failure. In patients with coronary artery disease, Af occurs predominantly in older patients, males and patients with left ventricular dysfunction. Important predictive factors of AF include hypertension, left ventricular hypertrophy and diabetes. However, the relation between AF and hypertension remains unclear. The risk of the development of AF, in an individual patient, is often difficult to assess but increasing age, presence of valvular heart disease and congestive heart failure, increase the risk of AF.[107]

In the present study cardiomyopathy was the etiology in 42% of the patients. The other etiologies noted were ischaemic heart disease (30%), rheumatic heart disease (10%), COPD (6%), surgical stress (4%), chronic kidney disease(4%) and hypothyroidism (4%).

A similar hospital based study by Prakash SK[108] reported 91.61% of atrial fibrillation to be secondary to chronic rheumatic valvular heart disease, 5.94% due to coronary artery disease and the rest due to miscellaneous causes. Low incidence of thyrotoxicosis and hypertensive heart disease causing atrial fibrillation in the Indian study on female population is noteworthy.

The Framingham study[75] identified rheumatic heart disease and cardiac failure as the most predictive precursor of atrial fibrillation. Hypertensive heart disease was the most common precursor, but the risk ratio for this disorder was not as great as for chronic rheumatic heart disease or cardiac failure. Coronary artery disease was found to be less striking and more inconsistent risk factor for the arrhythmia, except for the paroxysmal form of atrial fibrillation which showed strong relationship with newly developed coronary events.

An extensive retrospective study done by Davidson et al[109] on 704 consecutive cases of atrial fibrillation reported, atherosclerotic cardiovascular disease (55%) including diagnosed cases of myocardial infarction, hypertensive heart disease and coronary artery disease as the most frequent cause associated with this arrhythmia. Chronic rheumatic valvular heart disease (22.8%), chronic obstructive pulmonary disease (2.8%), WPW syndrome (2.6%) and thyrotoxicosis (2.6%) were also found to be associated. Rare causes of atrial fibrillation included cardiomyopathy (0.9%), mitral valve prolapse (0.9%), sick sinus syndrome (0.7%) myocarditis (0.6%), pulmonary embolism (0.3%) and atrial septal defect (0.3%). There was a relatively large group of idiopathic atrial fibrillation (4.5%).

The incidence of rheumatic fever is decreasing in developed countries, while in India it still accounts for 30-45% of all cardiac cases in hospital practice as reported by Padmavati S.[110] Levy et al[111] and ALFA study[92] had observed that valvular heart disease was present in about 20-23% of AF patients.

The Framingham study[75] reported hypertensive heart disease as the most common cardiac precursor for atrial fibrillation, which was noted in 47.5% of males

and 51.2% females in their study population. Gajewski J et al[112] were of the opinion that 16% cases of atrial fibrillation had hypertension as the predominantly associated factor. The incidence of hypertension was 38% in the ALFA study.[92] It is suggested that long standing hypertension can cause increase in left atrial pressure and dilatation which in turn can initiate and perpetuate the arrhythmia.

Hypertension in the present study is 64% which is much higher than the other studies which I have quoted above this is probably because of the selective inclusion criteria of the elderly population.

It is of interest to note that Diabetes has been found in 62% of the patients in the present study. None of the studies have made a mention of Diabetes as an association or a cause, since the study population is elderly, and since amongst Indians the incidence and prevalence of diabetes being very high, the study findings can be justified.

In the present study it was noted that Rheumatic heart disease was noted in 10% of the study population and IHD was noted in 30% of the study population, this discrepancy on comparison with the other studies is possibly because of the selective inclusion criteria. And also rheumatic heart disease amongst Indians occurs at a much younger age group and most of the Indian patients do not undergo valvular intervention because of financial reasons and may not reach the elderly age group.

Cardiomyopathy was an important underlying condition to cause atrial fibrillation in 42% cases in this study which was very high compared to ALFA study[92] where 17.5% of the patients had cardiomyopathy who presented with atrial

fibrillation. This difference may be explained by the fact that the present study included elderly patients and study was carried out at a tertiary care centre.

Another interesting observation in the present study is hypothyroidism which was seen in 2 patients (4%) which has not been mentioned in any of the studies, hyperthyroidism was not observed in the present study.

In the present study, chronic obstructive pulmonary disease accounted for 6% of the patients. Similar findings were described by Davidson et al[71] who reported 2.8% cases with chronic obstructive pulmonary disease.

In the present study surgical stress was detected to cause atrial fibrillation in 1 (2%) of the patients. These findings were consistent with a study by Davidson et al[71] who reported and 4.6% of cases with surgical stress.

In the present study majority of the patients (94%) had lower creatinine clearance suggestive of renal dysfunction. Renal dysfunction continues to emerge as a major risk factor for adverse cardiovascular outcomes.[113,114] The impact of renal dysfunction and AF remains less understood. However, Auer J et al[115] in the setting of cardiac surgery, concluded that, impaired calculated GFR is associated with increased risk for development of postoperative AF. These data provide additional evidence supporting the association between renal dysfunction and adverse cardiovascular outcomes including AF.

Recently Paccini JP et al[116] to define the factors associated with the occurrence of stroke and systemic embolism in a large, international atrial fibrillation (AF) trial concluded that, impaired renal function is a potent predictor of

stroke and systemic embolism. Stroke risk stratification in patients with AF should include renal function.

However, in the present study the history of CVA was noted in only 6% of the patients. This could be explained by the fact that, majority of the study population was already on prophylactic antiplatelet and anticoagulation therapy (90% and 50% respectively) while the indication of antiplatelet therapy based on $CHADS_2$ score was present in 88% of the patients and anticoagulation therapy based on $CHADS_2$ was 74%.

Many of the findings of the present study differ from the other studies quoted above, due to the inclusion criteria of selectively including patients above the age of 65 yrs. All the other studies quoted above were studied in a general population and hence the difference.

The problems of atrial fibrillation are different in younger individuals compared to the elderly. Even on extensive search of literature I have failed to come across any study which has exclusively studied atrial fibrialltion in the elderly population. Most of the studies have been done on general population and subsequently they have analyzed and have extrapolated their findings to the elderly population.

This study is unique in that we have studied selectively and extensively on the elderly age group. The shortcomings of this study could be the smaller sample size and a smaller duration of the study.

CONCLUSION

Based on the findings of the present study the prominent features of atrial fibrillation among elderly are;

- Males are more commonly affected compared to females.
- The commonest presentation is palpitations and breathlessness.
- Patients with history of hypertension, diabetes and Heart Disease are more likely to develop AF.
- The commonest signs of presentation were raised JVP and pedal oedema.
- Patients are likely to have abnormal lipd profile, with a raised total cholesterol and LDL and low HDL.
- The biochemical profile may Patients are likely to have raised blood urea nitrogen and serum creatinine, and hence a very low creatinine clearance.
- The commonest abnormality on ECG includes, abnormal rate, ischaemic changes, left axis deviation and left ventricular hypertrophy.
- The most common Echocardiographic findings may be low (<60%) ejection fraction, dilated left atrium, dilated left ventricle, Thickened mitral valve and aortic valve calcification.
- The common complications include CCF, CVA and cor pulmonale .

- Hypertension is the commonest etiology and other etiologies or associations include Diabetes Mellitus, cardiomyopathy, ischaemic heart disease, rheumatic heart disease, COPD, surgical stress, chronic kidney disease and hypothyroidism.

- Patients with AF are at high risk of thromboembolic events based on $CHADS_2$ and CHA_2DS_2-VASc score indicating Warfarin or other oral anti-coagulant therapy.

Overall, AF is a heterogeneous condition, with significant differences in its epidemiology, pathogenesis, clinical presentation and management across age groups. The important differences between AF in the young and that in the elderly necessitate clearly defined diagnostic and targeted management strategies to relieve symptoms as well as to prevent complications.

SUMMARY

The frequency of AF is increasing as the population ages, and therefore, knowledge of the clinical spectrum is essential. This study was aimed to explore the clinical profile of atrial fibrillation in elderly patients.

The present one year cross-sectional study was done under the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 50 elderly patients (age > 65 years) who presented with atrial fibrillation were included in the study. Patients were subjected to clinical examination, necessary investigations including electrocardiogram and 2D echocardiography. The annual stroke risk as assessed based on $CHADS_2$ score and CHA_2DS_2-VASc score.

Majority of the patients were males (78%). The male to female ratio was 3.54:1. Past history of hypertension, diabetes mellitus and HD were present in 64%, 62% and 58% of patients respectively. Palpitations were noted as commonest symptom (78%) followed by breathlessness (66%), chest pain (50%), swelling of feet (42%), fainting (20%) and PND (2%).

On examination 36% of the patients each had raised JVP and pedal oedema. The other signs included hepatojugular reflex (32%), left parasternal heave (22%) and murmurs (40%).

Lipid profile revealed maximum patients with abnormal LDL (84%) followed by abnormal HDL (54%), raised total cholesterol (54%) and triglycerides (28%). The biochemical profile revealed majority of the patients with raised blood

urea nitrogen (90%) followed by raised serum creatinine (50%) and hence very low creatinine clearance was observed in patients (94%).

ECG showed most of the patients had abnormal rate (64%), ischaemic changes (62%),left axis deviation (38%) and left ventricular hypertrophy (26%). On X-ray, most of the patients had LVH (36%) followed by LVH with LAE (24%), LAE (14%) and LAE+RVH (14%).

The 2D echocardiographic findings revealed 50% of the patients had ejection fraction of < 60%, 39% of the patients had dilated left atrium, 31% of the patients had dilated left ventricle, Thickened mitral valve was noted in 20% and calcification in 26%. Aortic valve calcification was present in 58% of the patients.

Most of the patients had complications of CCF (36%) followed by CVA (6%) and cor pulmonale (4%). Hypertension is the commonest etiology(64%) and other etiologies or associations include Diabetes Mellitus(62%), cardiomyopathy(42%), ischaemic heart disease(30%), rheumatic heart disease(10%), COPD(6%), surgical stress(4%), chronic kidney disease(4%) and hypothyroidism(4%).

Majority of the patients presented were subjected to rate control (82%) than rhytm control (24%) and were being treated with antiplatelets (86%) and anticoagulants (50%). History of cardioversion was present in only (6%).

The annual stroke risk based on $CHADS^2$ score showed maximum patients (38%) with intermediate risk of thromboembolic event and CHA_2DS_2-VASc score revealed moderate to high risk of stroke in maximum patients (88%).

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ANNEXURE I – CONSENT FORM

"To study the clinical profile of atrial fibrillation in elderly patients - A one year cross sectional study"

Objective and purpose of the study

This research is intended to study the clinical profile of atrial fibrillation in elderly patients. The principal investigator of the study is Dr. *** ***** under the guidance of Dr. **** ******. My co-operation will be of great help to Elderly patients with Atrial fibrillation.

Procedure

If you agree to be part of the research study you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood samples and undergo other necessary investigations

Risk and Benefits

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations which may cause swelling, pain, redness, bruising or infection (rarely happens) at the site from where the blood is drawn.

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or

other services that you receive. The study doctor or sponsor may stop your participation in this study any time. If you choose not to take part in the study you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy

Does not apply to this research

Financial incentives for participation

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

If you have any questions about my rights as a participant you may call Dr. **** ******, Principal and Chairman, J.N.M.C Ethical Committee for Human Research phone number **** ******.

In case of the queries during study or in future you may contact following person

Principal investigator	:	Dr. **** ****	Mob No: **** *****
Guide	:	Dr. ***** *****	Ph. No: **** *****

Consent Statement

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant: _____ Signature _____

Thumb print

Name of the Witness; _____ Signature_____

Investigator Name _____ Signature_____

ANNEXURE II – PROFORMA

Patient Name: I.P/O.P number:

Age: Sex:

Date of admission: Case NO.

Occupation

Address:

Presenting complaints

- Chest pain
- Shortness of breath
- Palpitations
- Fainting
- Edema of feet
- Distended neck veins
- Pain abdomen
- others

Present History

Past History

- Valvular heart disease
- Ischemic heart disease
- Hypertension
- Diabetes mellitus
- Intra cardiac thrombus
- Peripheral artery disease

- Cerebro vascular accident
- Other thromboembolism
- Chronic kidney disease
- Hypothyroidism / Hyperthyroidism
- Any history of bleed anywhere

Family History

Personal history

Diet

Appetite

Sleep

Bowel / Bladder

Habits

Smoking :

Exercise :

Alcohol intake :

Tobacco chewing :

Any other :

Treatment History:

Atrial fibrillation:

1. History of cardio version :
2. Strategy initiated at diagnosis is for

- Rhythm control
- Rate control

3. Treatment initiated at diagnosis

- Antiplatlet Drug: Class:

  Duration: Complications if any

- Anticoagulant Drug: Class:

  Duration Complications

  Frequency of monitoring & method

- Anti arrhythmic Drug: Class:

  Duration Complicaions

General Physical Examination

Pulse	:	Blood pressure	:
Anthropometry	**:**		
Height (Cms)	:	Weight(Kg)	:
Hip girth (Cms)	:	Waist girth (Cms)	:
Waist hip ratio	:	BMI	:

Systemic examination

Cardiovascular system Examination :

Peripheral CVS

Pulse

a) Rate
b) Rhythm
c) Volume
d) Tension
e) Force

f) Condition of vessel wall

g) Character

h) Radio radial comparison

i) Radio- femoral comparison

j) Other peripheral artery pulsations rt

left

Dorsalis pedis

Popliteal

Femoral

Brachial

Carotid

Signs of congestive cardiac failure

a) Juglar venous pulse

b) Liver

c) Dependent edema

d) Hepatojuglar reflux

Blood pressure : Upper limb

Lower limb

Standing B.P

Signs of infective Endocarditis

Central Cardiovascular Examination

Inspection

1) Precordial Bulge

2) Apical Impulse location

3) Pulsation other than apical impulse

a) Epigastric

b) Left parasternal

c) Pulmonary edema'

d) Supra sterna

e) Supraclvicular

Palpation

1) Apical impulse : location character

2) Left parasternal heave

3) Epigastric pulsation

4) Diastolic shock

5) Supraclavicular pulsation

6) Thrills

7) Any other pulsation

8) Tracheal tug

Percussion

1) Right border

2) Left border

3) Rt 2^{nd} space

4) Left 2^{nd} space

5) Sternum : Upper

Lower

Auscultaion

1) Heart sounds, Splits, added sounds
2) Murmurs
3) Pericardial rub

Respiratory system Examination :

Per abdomen examination :

Central Nervous system Examinaion :

Investigations

Haemogram

Hb% :

TC :

DC : N - M - E - B -

Blood Sugar

FBS : PPBS :

Lipid profile

Cholesterol :

LDL :

HDL :

TG :

Renal profile

Serum Urea

Serum Creatinine

Thyroid Profile

Serum TSH

Chest X-ray:

ECG

- Rhythm
- Rate
- Axis
- any blocks
- QT interval
- ST- T changes
- Significant Q waves
- QRS complexes
- Hypertrophy
- others

Echocardiography

Others:

ANNEXURE III – KEY TO MASTER CHART

-	-	Absent
+	-	Present
ACT	-	Artery systolic flow
BP	-	Blood pressure
CALC	-	Calcified
Cms	-	Centimeters
ESM	-	Ejection systolic murmur
F	-	Female
gm	-	Gram
IRIR	-	Irregularly irregular
Kgs	-	Kilograms
L	-	Left
LA (AP)	-	Left atrium (Anteroposterior)
LBBB	-	Left bundle branch block
LV	-	Low volume
LVH	-	Left ventricular hypertrophy
LVIDd	-	Left ventricular diastolic dimension
LVISD	-	Left ventricular systolic dimension
LVPWD	-	Left ventricular posterior wall dimension
M	-	Male
MDM	-	Middiastolic murmur
mg/dL	-	Milligram per deciliter
Min	-	Minute

mIU/L	-	Million international units per liter
mL/Min	-	Milliliter per minute
mm Hg	-	Millimeters of mercury
MOD	-	Moderate
N	-	Normal
PAH	-	Pulmonary arterial hypertension
PSM	-	Pansystolic murmur
R	-	Right
R-F	-	Radio-femoral delay
R-R	-	Radio-radial delay
RV	-	Right ventricle
SCL	-	Sclerotic
THK	-	Thickened
TSH	-	Thyroid stimulating hormone
VR	-	Variable volume

ANNEXURE III - MASTER CHART

				Presenting complaints								History																						Examination																				
												Past history										Personal history					Treatment							BP												Inspection			Palpation				
Serial number	In patient number	Age (Years)	Sex	Chest pain	Breathlessness	Palpitations	Fainting	Swelling of feet	Pain abdomen	Paroxysmal nocturnal dyspnoea	Orthopnoea	Valvular heart disease	Ischaemic heart disease	Hypertension	Diabetes mellitus	Peripheral artery disease	Cerebrovascular accident	Chronic kidney disease	Hypothyroidism	Bleeding tendency	Family history	Smoking	Alcohol	Exercise	Tobacco	Cardioversion	Rate control	Rhythm control	Antiplatelet	Anticoagulant	Antiarrhythmic	Pulse (/Min)	Systolic (mm Hg)	Diastolic (mm Hg)	Height (Cms)	Weight (Kgs)	Rhythm	Volume	R-R delay	R-F delay	Peripheral pulses	Jugular venous pulse	Edema	Hepatojuglar reflex	Precordial bulge	Apical pulse location	Other pulsations	Apical impulse location	Left parasternal heave	Pulsations	Thrills	Diastolic shock
1	484869	67	F	+	+	+	-	+	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	132	110	70	166	68	IRIR	VR	-	-	+	+	+	+	+	5	-	5	+	+	-	-
2	485955	82	M	-	+	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	130	150	90	156	80	IRIR	VR	-	-	+	-	-	-	-	5	+	5	-	+	-	-
3	486660	74	M	-	-	+	-	-	-	-	-	-	+	+	+	-	+	-	-	-	+	+	-	-	-	-	+	-	+	+	-	70	140	60	160	70	IRIR	VR	-	-	+	-	-	-	-	5	+	5	-	+	-	-
4	490804	68	M	-	+	+	+	+	-	-	-	-	+	+	-	-	-	-	-	+	-	+	-	-	+	-	+	-	+	+	-	110	90	60	170	56	IRIR	VR	-	-	+	+	+	+	-	6	+	6	-	+	+	+
5	497562	65	M	+	-	+	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	84	130	70	162	84	IRIR	VR	-	-	+	-	-	-	-	5	+	5	-	+	-	-
6	499283	78	M	-	+	+	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	82	120	70	172	68	IRIR	VR	-	-	+	-	-	-	+	5	-	5	-	-	-	-
7	504618	69	M	+	+	+	+	+	-	-	-	-	+	+	+	-	-	+	+	-	+	+	+	+	+	-	+	+	+	+	+	100	90	60	181	96	IRIR	VR	-	-	+	+	+	+	-	6	+	6	-	+	-	-
8	510366	66	F	+	+	+	+	+	-	+	-	-	+	+	+	-	-	-	-	-	-	-	-	+	-	-	+	+	+	-	+	106	130	70	152	46	IRIR	VR	-	-	+	+	+	+	+	6	+	6	+	+	+	-
9	522823	81	M	+	+	+	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	88	120	70	172	94	IRIR	VR	-	-	+	-	-	-	-	6	-	5	-	-	-	-
10	524067	68	M	+	+	+	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	108	140	70	160	83	IRIR	VR	-	-	+	+	+	+	-	5	+	6	+	+	-	+
11	533357	70	M	+	-	+	+	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	+	-	-	+	+	+	+	+	64	90	60	163	60	IRIR	VR	-	-	+	-	-	-	-	6	+	6	-	+	-	-
12	533680	88	M	+	-	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	+	-	-	+	-	+	-	+	+	-	130	100	90	160	50	IRIR	VR	-	-	+	-	+	+	-	5	+	5	-	+	+	+
13	541549	77	M	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	132	130	80	160	60	IRIR	VR	-	-	+	-	-	-	-	5	+	5	-	+	+	-
14	542182	72	M	-	+	+	-	+	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	+	108	110	70	168	63	IRIR	VR	-	-	+	+	+	+	-	6	+	6	+	+	+	-
15	543874	72	M	-	+	+	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	140	110	70	155	55	IRIR	VR	-	-	+	+	+	+	-	6	+	6	-	+	-	-
16	548178	80	M	-	+	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	148	96	70	164	72	IRIR	LV	-	-	+	-	-	-	-	5	+	5	+	+	-	-
17	548894	81	M	-	+	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	+	-	+	-	+	-	+	-	-	124	100	70	65	164	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
18	549269	80	M	+	+	+	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	104	130	84	143	48	IRIR	VR	-	-	+	+	+	+	-	5	+	4	+	+	-	-
19	550338	68	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	+	+	+	140	100	70	160	59	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
20	550741	69	M	+	+	+	-	+	-	-	-	-	+	--	+	-	-	-	-	-	-	-	+	-	-	-	+	-	+	+	-	100	80	64	170	65	IRIR	VR	-	-	+	+	+	+	-	6	+	6	+	+	-	-
21	550774	71	M	+	+	+	-	+	-	-	-	+	+	-	+	+	+	-	-	-	-	+	-	-	+	-	+	+	+	+	+	130	90	60	171	48	IRIR	VR	-	-	+	+	+	+	-	6	+	6	+	+	-	-
22	550958	73	M	+	-	-	-	-	-	-	-	-	+	+	+	-	-	-	-	-	+	+	-	-	+	-	+	-	+	+	-	110	100	70	165	65	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
23	551356	81	M	-	+	+	+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	160	90	60	160	47	IRIR	LV	-	-	-	-	-	-	-	5	-	5	+	-	-	-
24	552556	66	F	+	+	+	-	+	-	-	-	-	+	+	+	-	+	-	-	-	-	-	-	-	-	+	+	+	+	+	-	88	160	90	171	86	IRIR	VR	-	-	+	+	+	+	-	6	-	6	-	-	-	-
25	552758	67	M	-	++	+	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	+	+	-	+	-	+	-	+	+	-	140	160	90	177	84	IRIR	VR	-	-	+	+	-	-	-	5	-	5	-	-	-	-
26	553093	78	F	-	+	+	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	140	100	70	105	65	IRIR	LV	-	-	+	+	+	+	-	5	-	5	-	-	-	-
27	554768	67	F	-	+	+	+	+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	+	-	-	+	+	-	+	+	+	150	80	70	160	102	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-

ANNEXURE III - MASTER CHART

Serial number	Examination			Investigations																																	
	Auscultation			Haemogram					Lipid profile (mg/dL)				Renal profile		Serum TSH (mIU/L)	Electrocardiogram								Echocardiography													
																								Measurements								Valves					
	Heart sounds	Murmurs	Pericardial rub	Haemoglobin (gm%)	Total count	Neutrophils	Eosinophils	Monocytes	Cholesterol	Low density lipoprotein	High density lipoprotein	Triglycerides	Urea (mg/dL)	Creatinine (mg/dL)		Rate	Rhythm	Axis	Any blocks	ST-T changes	Hypertrophy	Q waves	Other	LVPWD	LA (AP)	LVIDd	LVISd	Ejection fraction	RV Free wall	Aorta	Pulmonary artery	Mitral	Aortic	Tricuspid	Pulmonary
1	S1 VARIABLE	ESM	-	7.60	7000	85	2	0	190	106	44	324	15	0.8	0.9	132	IRIR	L	-	-	-	-	-	1.00	5.50	6.20	4.90	25.00	0.70	2.10	2.80	THK	THK	N	N
2	S1 VARIABLE	-	-	12.60	10100	84	4	0	210	146	46	92	25	1.1	0.8	130	IRIR	L	-	-	-	-	-	1.00	3.80	4.50	3.10	60.00	0.50	2.60	2.00	CALC	THK	N	N
3	S1 VARIABLE	ESM	-	11.90	7062	80	5	2	155	720	46	133	46	1.3	1.4	70	IRIR	N	-	-	-	-	-	1.17	5.00	4.86	3.29	55.00	0.60	2.60	2.20	N	N	N	N
4	S1 VARIABLE	PSM	-	8.60	5600	78	2	2	230	140	36	180	97	2.7	1.4	80	IRIR	N	-	-	LVH	-	-	1.00	5.50	5.40	3.90	60.00	0.50	2.80	3.00	THK	THK	N	N
5	S1 VARIABLE	-	-	10.10	7200	72	6	3	160	30	107	114	21	0.74	0.4	75	IRIR	N	-	+	-	-	-	1.00	4.80	4.30	3.10	50.00	0.60	2.10	2.10	N	N	N	N
6	S1 VARIABLE	-	-	4.20	5600	84	9	-	180	160	94	38	104	6	1.3	2.4	IRIR	L	-	-	-	+	-	0.90	4.90	4.30	2.78	50.00	0.50	2.30	2.30	N	N	N	N
7	S1 VARIABLE	ESM	-	9.70	4400	57	30	3	310	140	32	288	83	2.84	1.62	100	IRIR	R	-	-	-	-	-	0.90	5.40	5.80	4.50	30.00	0.80	2.80	1.80	N	THK	N	N
8	S1 VARIABLE	MDM	-	10.60	9100	76	14	4	200	140	40	98	27	0.9	0.2	100	IRIR	N	-	+	+	-	-	1.10	5.50	6.20	4.90	25.00	0.70	2.10	2.80	THK	THK	N	N
9	S1 VARIABLE	-	-	13.20	10100	71	4	4	300	220	32	140	74	2.28	0.9	140	IRIR	N	-	-	-	-	-	0.90	4.10	4.80	3.10	50.00	0.60	2.10	2.00	N	N	N	N
10	S1 VARIABLE	PSM	-	13.00	6900	72	9	0	280	220	36	150	33	1.29	0.6	105	IRIR	N	-	-	-	-	-	1.00	3.80	4.50	3.10	60.00	0.50	2.60	2.00	CALC	THK	N	N
11	S1 VARIABLE	ESM	-	13.60	5700	57	35	7	115	79	27	48	34	0.4	0.9	60	IRIR	L	-	+	LVH	-	-	1.10	3.60	5.70	4.40	60.00	0.50	2.70	2.70	CALC	THK	N	N
12	S1 VARIABLE	PSM	-	8.60	5100	90	4	8	210	146	46	82	25	1.1	4.2	130	IRIR	L	-	-	+	-	-	1.00	5.50	4.80	3.10	60.00	0.60	2.60	3.00	CALC	THK	THK	N
13	S1 VARIABLE	PSM	-	14.50	13000	84	8	10	220	180	40	96	30	1.67	0.6	110	IRIR	N	-	-	-	-	-	0.90	4.20	4.00	2.80	60.00	0.50	2.10	2.80	N	N	N	N
14	S1 VARIABLE	ESM	-	13.80	9800	79	0	0	210	190	36	316	100	2.03	0.9	140	IRIR	R	-	+	-	-	-	0.90	5.50	4.30	2.78	60.00	0.70	2.90	3.00	N	N	THK	N
15	S1 VARIABLE	-	-	11.40	8400	70	3	1	220	140	32	156	89	2.1	2.24	140	IRIR	L	-	-	LVH	-	-	1.00	3.60	5.20	3.40	45.00	0.50	2.40	2.60	N	CALC, THK	N	N
16	S1 VARIABLE	-	-	12.60	9000	52	38	5	4.04	91	42	376	61	5.6	1.51	1.96	IRIR	L	-	-	LVH	-	-	1.20	4.00	3.90	2.60	60.00	0.50	2.30	1.90	N	N	N	N
17	S1 VARIABLE	-	-	12.50	9700	88	10	2	146	88	34	94	88	2.39	2.26	90	IRIR	N	-	-	-	-	-	1.10	4.60	4.10	2.80	60.00	0.50	2.20	2.10	CALC	THK	N	N
18	S1 VARIABLE	-	-	11.30	7600	83	0	0	190	164	40	113	96	1.23	3.8	140	IRIR	N	-	-	-	-	-	1.10	5.06	4.20	3.90	60.00	0.70	2.60	1.90	THK	SCL	N	N
19	S1 VARIABLE	-	-	11.50	10600	78	14	2	230	208	117	33	290	0.6	1.38	0.79	IRIR	N	-	+	-	-	-	1.10	4.60	5.50	4.60	40.00	0.60	3.00	2.30	THK	THK	N	N
20	S1 VARIABLE	-	-	10.40	5500	74	18	6	212	212	109	32	128	9.2	2.72	0.9	IRIR	N	-	-	-	-	-	1.10	5.60	5.90	4.60	30.00	0.80	2.80	2.30	N	N	N	N
21	S1 VARIABLE	MDM	-	13.90	14900	78	1	2	150	110	23	61	57	2.22	0.84	140	IRIR	N	-	+	-	-	-	1.30	4.10	5.60	4.20	45.00	0.90	2.10	2.80	THK	CALC	N	N
22	S1 VARIABLE	-	-	10.20	4800	92	6	4	115	79	27	48	43	2.15	1.8	130	IRIR	N	-	+	-	-	-	1.10	4.30	4.10	3.40	60.00	0.40	2.30	2.10	N	CALC, THK	N	N
23	S1 VARIABLE	ESM	-	12.40	13700	89	6	0	348	210	120	38	108	13	4.17	0.48	IRIR	N	-	-	-	-	-	9.80	5.00	5.60	4.30	45.00	11.10	2.40	1.90	N	N	N	N
24	S1 VARIABLE	-	-	11.30	11110	76	13	2	143	143	84	45	70	4	1.1	2.18	IRIR	N	-	+	-	-	-	0.90	5.40	5.60	4.30	30.00	0.60	2.60	2.10	N	N	N	N
25	S1 VARIABLE	-	-	12.60	13700	73	15	5	165	165	139	35	95	3.6	1.09	0.9	IRIR	N	-	+	-	-	-	3.60	4.30	4.90	4.10	60.00	0.80	3.00	2.50	CALC	CALC	N	N
26	S1 VARIABLE	-	-	12.10	12800	73	0	9	115	83	18	76	110	1.57	2.79	140	IRIR	N	-	-	-	-	-	0.90	4.20	4.10	2.70	60.00	0.50	2.30	2.00	N	N	N	N
27	S1 VARIABLE	-	-	10.70	14900	72	22	2	220	202	139	39	122	2.1	0.87	2.86	IRIR	N	-	-	-	-	-	1.30	5.02	5.07	3.32	35.00	0.90	2.90	2.60	N	N	N	N

ANNEXURE III - MASTER CHART

Serial number	Investigations														
	Echocardiography							Doppler study				CHA2DS2 VASc Score	CHADS2 Score	Creatnine clearance (mL/min)
	Chambers				Septae	Great artery								
	Ventricle		Atrium											
	Left	Right	Left	Right		Aorta	Pulmonary artery	Mitral valve	Aortic valve	Tricuspid valve	Pulmonary valve (ACT)			
1	DILATED	DILATED	DILATED	DILATED	N	N	DILATED	0.7	2.1	N	85	4	2	73.25
2	N	DILATED	N	DILATED	N	N	N	1.1	0.9	1.6	71	4	3	58.59
3	DILATED	DILATED	DILATED	N	N	N	N	0.83	0.94	0.62	87	5	4	49.36
4	DILATED	N	DILATED	DILATED	N	N	DILATED	1.7	1.3	2.8	80	3	2	20.74
5	N	N	DILATED	N	N	N	N	0.9	1.2	0.8	65	3	2	118.24
6	N	N	DILATED	N	N	N	N	1.3	0.8	0.7	80	2	1	9.82
7	DILATED	N	DILATED	DILATED	N	N	MILD PAH	0.8	0.9	0.6	80	4	3	33.33
8	DILATED	DILATED	DILATED	DILATED	N	N	DILATED	0.7	1.6	N	85	5	3	44.65
9	N	N	N	DILATED	N	N	N	0.8	1	N	65	4	3	33.78
10	N	DILATED	N	DILATED	N	N	N	1.1	0.9	2.1	71	3	2	64.34
11	DILATED	N	DILATED	N	N	N	DILATED	0.6	3.8	0.8	40	3	2	145.83
12	DILATED	N	DILATED	N	N	N	DILATED	0.6	3.8	0.9	44	5	4	32.83
13	N	N	DILATED	N	N	N	N	0.7	0.9	N	80	2	1	31.44
14	DILATED	N	DILATED	N	N	N	DILATED	0.9	1.2	N	165	3	2	29.31
15	DILATED	DILATED	N	DILATED	N	N	DILATED	1.1	1.2	2.8	70	2	1	24.74
16	DILATED	N	DILATED	N	N	N	N	0.6	2.2	N	80	3	2	10.71
17	LVH	N	DILATED	DILATED	N	N	N	0.8	2.4	N	78	4	3	56.23
18	LVH	N	DILATED	N	N	N	N	1.4	1.4	0.8	20	5	4	32.52
19	DILATED	N	DILATED	N	N	N	N	1.2	1.1	1.6	81	1	0	98.33
20	DILATED	DILATED	DILATED	DILATED	N	N	MILD PAH	1.4	1.4	N	78	3	2	7.85
21	N	N	DILATED	N	N	N	N	1.6	1.8	N	59	6	4	20.72
22	LVH	N	DILATED	N	N	N	N	0.9	1.3	N	64	3	2	28.13
23	DILATED	N	DILATED	DILATED	N	N	SEVERE PAH	0.8	1.2	0.6	80	3	2	2.96
24	N	N	N	N	N	N	N	0.6	1.6	1.4	30	7	5	18.78
25	N	N	DILATED	N	N	N	N	2.8	0.6	N	30	3	2	23.66
26	N	N	DILATED	N	N	N	N	0.8	1.3	N	25	6	4	30.30
27	N	N	DILATED	N	N	N	MILD PAH	2.4	1.7	N	90	4	2	41.86

ANNEXURE III - MASTER CHART

				Presenting complaints								History																				Examination																			
												Past history										Personal history				Treatment							BP												Inspection			Palpation				
Serial number	In patient number	Age (Years)	Sex	Chest pain	Breathlessness	Palpitations	Fainting	Swelling of feet	Pain abdomen	Paroxysmal nocturnal dyspnoea	Orthopnoea	Valvular heart disease	Ischaemic heart disease	Hypertension	Diabetes mellitus	Peripheral artery disease	Cerebrovascular accident	Chronic kidney disease	Hypothyroidism	Bleeding tendency	Family history	Smoking	Alcohol	Exercise	Tobacco	Cardioversion	Rate control	Rhythm control	Antiplatelet	Anticoagulant	Antiarrhythmic	Pulse (/Min)	Systolic (mm Hg)	Diastolic (mm Hg)	Height (Cms)	Weight (Kgs)	Rhythm	Volume	R-R delay	R-F delay	Peripheral pulses	Jugular venous pulse	Edema	Hepatojugular reflex	Precordial bulge	Apical pulse location	Other pulsations	Apical impulse location	Left parasternal heave	Pulsations	Thrills	Diastolic shock
28	556693	68	M	-	+	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	148	96	70	164	72	IRIR	LV	-	-	+	-	-	-	-	5	+	8	+	+	-	-
29	558031	80	F	-	+	-	-	-	-	-	-	-	-	+	+	-	-	+	-	-	-	-	-	+	-	+	+	-	+	+	-	140	90	60	150	65	IRIR	VR	-	-	+	-	-	-	-	5	+	5	-	+	-	-
30	560518	79	F	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	100	120	80	160	70	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
31	561164	65	F	+	+	+	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	100	90	60	150	56	IRIR	VR	-	-	+	+	-	-	-	5	+	5	-	+	-	-
32	561367	69	M	-	+	+	-	-	-	-	-	-	+	+	+	-	-	+	-	-	-	+	-	+	-	-	+	+	+	+	+	64	120	70	151	80	IRIR	N	-	-	+	-	-	-	-	5	-	5	-	-	-	-
33	562156	66	M	-	-	+	-	-	-	-	-	-	+	+	+	+	-	-	-	-	-	+	-	-	-	-	+	-	+	-	-	100	110	80	177	60	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
34	778125	77	F	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	100	90	70	140	50	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
35	1E+06	67	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100	120	70	163	65	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
36	2E+06	79	F	+	+	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	88	130	90	157	78	IRIR	VR	-	-	+	-	-	-	-	5	+	5	-	+	-	-
37	544894	81	M	-	+	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	124	100	70	164	65	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
38	553088	68	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	+	+	+	140	100	70	160	59	IRIR	LV	-	-	+	-	-	-	-	4	-	4	-	-	-	-
39	506786	69	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	++	-	+	-	+	-	-	88	146	90	162	63	IRIR	VR	-	-	+	-	-	-	-	5	+	5	-	-	-	+
40	589321	69	M	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	++	+	-	++	-	-	-	-	-	-	106	140	70	170	46	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
41	569485	68	M	-	-	+	-	-	-	-	-	-	+	+	+	-	-	+	-	-	-	-	-	+	-	-	+	-	+	-	-	82	120	70	172	69	IRIR	VR	-	-	+	-	-	-	+	5	-	5	-	-	-	-
42	574698	69	M	-	+	+	+	+	-	-	-	-	+	+	-	-	-	-	-	+	-	+	-	-	+	-	+	-	+	+	-	110	90	60	170	56	IRIR	VR	-	-	-	+	+	+	-	6	+	6	-	+	+	+
43	584713	70	M	+	+	+	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	132	110	70	166	68	IRIR	VR	-	-	+	+	+	+	+	5	+	5	+	-	-	-
44	584437	74	M	+	+	+	+	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	+	-	-	+	+	+	-	+	106	130	70	152	46	IRIR	VR	-	-	+	+	+	+	+	5	+	6	-	-	+	-
45	484464	77	M	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	132	130	80	160	64	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-
46	583998	80	M	+	-	-	+	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	+	-	-	+	+	+	+	+	64	90	60	163	60	IRIR	VR	-	-	+	-	+	-	+	6	+	6	-	+	-	-
47	583467	81	M	+	+	-	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	80	120	70	170	94	IRIR	VR	-	-	+	-	+	-	-	5	-	5	-	-	-	-
48	582544	68	F	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	100	120	80	160	70	IRIR	VR	-	-	+	-	-	-	-	4	-	4	-	-	-	-
49	583334	66	M	+	+	+	-	+	-	-	-	-	+	+	+	-	-	-	-	-	+	-	-	-	-	-	+	-	+	+	-	100	90	60	150	56	IRIR	LV	-	-	+	+	-	-	-	5	+	5	-	+	-	-
50	584460	70	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	100	90	70	140	50	IRIR	VR	-	-	+	-	-	-	-	5	-	5	-	-	-	-

ANNEXURE III - MASTER CHART

Serial number	Examination			Investigations																																	
	Auscultation			Haemogram					Lipid profile (mg/dL)				Renal profile		Serum TSH (mIU/L)	Electrocardiogram								Echocardiography													
																								Measurements								Valves					
	Heart sounds	Murmurs	Pericardial rub	Haemoglobin (gm%)	Total count	Neutrophils	Eosinophils	Monocytes	Cholesterol	Low density lipoprotein	High density lipoprotein	Triglycerides	Urea (mg/dL)	Creatinine (mg/dL)		Rate	Rhythm	Axis	Any blocks	ST-T changes	Hypertrophy	Q waves	Other	LVPWD	LA (AP)	LVIDd	LVISd	Ejection fraction	RV Free wall	Aorta	Pulmonary artery	Mitral	Aortic	Tricuspid	Pulmonary
28	S1 VARIABLE	-	-	12.60	8400	64	28	4	190	164	140	113	96	1.23	2.4	140	IRIR	N	-	-	LVH	-	-	1.20	4.00	5.90	2.60	60.00	0.50	2.30	1.90	N	N	N	N
29	S1 VARIABLE	-	-	8.10	16500	91	7	1	130	130	90	34	90	1.6	5.39	0.54	IRIR	N	-	-	-	-	-	1.20	4.80	4.50	3.00	60.00	0.50	2.40	2.40	N	N	N	N
30	S1 VARIABLE	-	-	10.8	15400	84	10	8	124	106	38	90	20	1.37	0.9	100	IRIR	R	-	-	-	-	-	1.20	4	3.9	2.6	60	0.6	2.2	3.1	CALC	THK	N	N
31	S1 VARIABLE	MDM	-	12.6	7900	51	38	1	232	130	40	210	29	0.92	0.74	100	IRIR	L	LBBB	-	LVH	-	-	0.90	5.4	5.6	4.3	30	0.7	2.1	2.3	N	N	N	N
32	S1 VARIABLE	-	-	8	14100	91	7	1	220	164	30	180	244	5.6	1.8	80	IRIR	L	-	-	-	-	-	1.00	4.6	5.4	4.5	35	0.8	2.8	2.4	N	N	N	N
33	S1 VARIABLE	-	-	10.2	12100	80	7	2	240	130	34	108	38	1.2	3	150	IRIR	R	-	+	-	-	-	1.00	2.7	5.3	3.8	35	0.9	2.7	2.8	N	N	N	N
34	S1 VARIABLE S3 GALLOP	-	-	10.2	12400	71	8	1	215	145	48	102	33	1.05	0.64	108	IRIR	L	-	+	-	+	-	1.10	3.6	5.7	4.4	45	0.5	2.7	2.6	CALC	THK	N	N
35	S1 VARIABLE	-	-	8.9	7400	90	4	3	240	120	38	110	40	0.9	1.9	120	IRIR	N	-	+	LVH	-	-	1.00	4.1	5.2	3.4	50	0.5	2.6	2	N	THK	N	N
36	S1 S2 MUFFLED	-	-	10.7	9600	68	5	0	210	140	42	160	94	1.46	0.47	75	IRIR	L	-	+	LVH	-	-	1.10	3.6	5.7	4.4	60	0.5	2.7	2.6	CALC	THK	N	N
37	S1 VARIABLE	-	-	12.5	9700	88	2	0	146	88	34	94	88	2.39	2.26	90	IRIR	N	-	-	-	-	-	1.20	4	3.9	2.6	60	0.5	2.3	1.9	CALC	THK	N	N
38	S1 VARIABLE	-	-	11.5	10600	78	2	6	208	117	33	290	38	1.38	0.79	160	IRIR	N	-	+	-	-	-	1.10	4.6	5.5	4.6	45	0.6	3	2.3	CALC	THK	N	N
39	S1 VARIABLE	PSM	-	12.1	10400	48	7	35	204	160	35	109	38	1.34	2.4	100	IRIR	N	-	-	-	-	-	0.90	5.4	4.5	3	60	0.5	2.8	2.1	N	CALC, THK	N	N
40	S1 VARIABLE	ESM	-	5.4	11100	84	6	1	176	110	46	130	18	0.8	1.9	130	IRIR	N	-	-	-	-	-	1.00	4.2	5.2	4.4	60	0.5	2.7	2.2	N	CALC, THK	N	N
41	S1 VARIABLE	MDM	-	6.4	9600	90	2	2	220	180	40	96	30	1.8	4.2	139	IRIR	N	-	+	LVH	-	-	0.90	4.9	4.3	2.78	50	0.5	2.8	3	N	N	N	N
42	S1 VARIABLE	PSM	-	8.6	5600	78	2	2	230	140	36	180	97	2.7	1.4	80	IRIR	N	-	-	LVH	-	-	1.00	5.5	5.4	3.9	60	0.5	2.8	3	THK	THK	N	N
43	S1 VARIABLE	ESM	-	7.4	7000	85	2	4	190	106	44	324	15	0.8	0.9	132	IRIR	L	-	-	-	-	-	1.00	5.5	6.2	4.9	25	0.8	2.1	2.8	THK	THK	N	N
44	S1 VARIABLE	ESM	-	10.6	9100	76	4	2	200	140	40	98	27	0.7	0.2	100	IRIR	N	-	+	LVH	-	-	1.20	5.5	6.2	4.4	25	0.8	2.1	2.8	THK	THK	N	N
45	S1 VARIABLE	-	-	14.5	13000	79	5	2	280	180	40	96	30	1.67	0.6	110	IRIR	N	-	-	-	-	-	0.90	4.2	4	7.8	60	0.6	2.1	2.7	N	N	N	N
46	S1 VARIABLE	ESM	-	13.6	12600	57	7	1	115	79	27	48	34	0.9	2.7	60	IRIR	L	-	-	LVH	-	-	1.10	3.6	5.7	4.4	60	0.7	2.7	2.6	CALC	THK	N	N
47	S1 VARIABLE	-	-	13.2	10100	71	7	18	300	220	32	140	74	2.28	1.917	140	IRIR	N	-	-	-	-	-	0.90	4.1	4.8	3.1	50	0.6	2.1	2	N	N	N	N
48	S1 VARIABLE	-	-	10.8	15400	84	6	8	124	106	38	90	20	1.37	0.9	100	IRIR	R	-	-	-	-	-	1.20	4	3.9	2.6	60	0.9	2.4	2.8	CALC	THK	N	N
49	S1 VARIABLE	-	-	12.6	7400	51	1	2	232	130	40	210	29	0.92	0.74	100	IRIR	L	LBBB	-	-	-	-	0.90	5.4	5.6	4.3	30	0.8	2.6	2.5	N	N	N	N
50	S1 VARIABLE C3 GALLOP	-	-	10.2	8400	84	8	2	215	145	48	102	33	1	0.64	110	IRIR	N	-	-	-	+	-	1.10	5.6	5.7	4.4	45	0.5	2.7	2.6	CALC	THK	N	N

ANNEXURE III - MASTER CHART

Serial number	Investigations														
	Echocardiography							Doppler study				CHA2DS2 VASc Score	CHADS2 Score	Creatnine clearance (mL/min)
	Chambers				Septae	Great artery								
	Ventricle		Atrium											
	Left	Right	Left	Right		Aorta	Pulmonary artery	Mitral valve	Aortic valve	Tricuspid valve	Pulmonary valve (ACT)			
28	DILATED	N	DILATED	N	N	N	N	0.6	2.2	N	80	2	1	58.54
29	LVH	N	N	N	N	N	N	1.2	0.6	0.8	30	5	3	28.78
30	LVH	N	DILATED	DILATED	N	N	MILD PAH	0.6	2.2	1.8	80	4	2	36.80
31	DILATED	N	DILATED	DILATED	N	N	N	0.7	1.13	0.6	80	5	3	53.89
32	DILATED	DILATED	DILATED	DILATED	N	N	MOD PAH	2.1	2	2.1	45	3	2	15.87
33	N	N	N	N	N	N	N	2.1	0.8	N	28	4	2	51.39
34	N	N	DILATED	N	N	N	DILATED	0.6	3.8	0.6	40	3	1	35.42
35	N	N	DILATED	DILATED	N	N	N	1.1	0.8	0.8	65	1	0	73.23
36	DILATED	N	DILATED	N	N	N	DILATED	0.6	3.8	0.9	40	4	2	38.47
37	LVH	N	DILATED	DILATED	N	N	N	0.6	2.2	1.4	80	4	3	22.29
38	DILATED	N	DILATED	N	N	N	N	1.2	1.1	2.1	81	1	0	42.75
39	LVH	N	DILATED	DILATED	N	N	N	0.8	1.1	N	65	1	0	46.36
40	N	N	DILATED	N	N	N	N	0.8	3.8	3	60	1	0	59.90
41	N	N	DILATED	N	N	N	N	1.3	0.8	1.4	80	3	2	38.33
42	DILATED	N	DILATED	DILATED	N	N	DILATED	1.5	1.3	3.1	80	3	2	20.45
43	DILATED	DILATED	DILATED	DILATED	N	N	DILATED	0.7	1.6	N	86	3	2	82.64
44	DILATED	DILATED	DILATED	DILATED	N	N	DILATED	0.7	1.6	N	85	4	3	60.24
45	N	N	DILATED	N	N	N	N	0.7	0.9	N	80	2	1	33.53
46	DILATED LVH	N	DILATED	N	N	N	DILATED	0.6	3.8	2	40	5	4	55.56
47	N	N	DILATED	N	N	N	N	0.8	1	N	65	5	4	33.78
48	LVH	N	DILATED	DILATED	N	N	MILD PAH	0.6	2.2	1.1	50	3	1	43.43
49	DILATED	N	DILATED	DILATED	N	N	MILD PAH	0.7	1.18	0.6	80	4	3	62.56
50	N	N	DILATED	N	N	N	DILATED	0.6	3.8	1.3	40	1	0	48.61