Chapter one
introduction

1.1 Introduction:

Heart failure (HF) is a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood. The cardinal manifestations of HF are dyspnea and fatigue, which may limit exercise tolerance, and fluid retention, which may lead to pulmonary and/or splanchnic congestion and/or peripheral edema. Some patients have exercise intolerance but little evidence of fluid retention, whereas others complain primarily of edema, dyspnea, or fatigue. Because some patients present without signs or symptoms of volume overload, there is no single diagnostic test for HF because it is largely a clinical diagnosis based on a careful history and physical examination [1].

Congestive heart failure (CHF) is a common clinical disorder that results in pulmonary vascular congestion and reduced cardiac output. CHF should be considered in the differential diagnosis of any adult patient who presents with dyspnea and/or respiratory failure. The diagnosis of heart failure is often determined by a careful history and physical examination and characteristic chest radiograph findings. The measurements of serum brain natriuretic-peptide and echocardiography have substantially improved the accuracy of diagnosis. Therapy for CHF is directed at restoring normal cardiopulmonary physiology and reducing the hyper-adrenergic state. The cornerstone of treatment is a combination of an angiotensin-convertingenzyme inhibitor and slow titration of a blocker. Patients with CHF are prone to pulmonary complications, including obstructive sleep apnea, pulmonary edema, and pleural effusions. Continuous positive airway pressure and noninvasive positivepressure ventilation benefit patients in CHF exacerbations [2].

Heart failure (HF) resulting in the accumulation of fluid in the lungs and other body tissues. It is related mainly to salt and water retention in the tissues rather than directly to reduced blood flow. Blood pools in the veins (vascular congestion) because the heart does not pump efficiently enough to allow it to return. It may vary from the most minimal symptoms to sudden pulmonary edema or a rapidly lethal shock like state (shock).

Congestive heart failure (CHF) is the most leading cause of admission to hospitals particularly among the old people. The incidence of the disease increased among the population worldwide.

Chronic heart failure (CHF) develops as a result of left ventricular (LV) systolic and/or diastolic dysfunction. It is the only major cardiovascular disease whose prevalence and incidence are thought to be increasing, and it has been predicted that the occurrence of CHF may soon reach epidemic proportions.

A study of heart failure (HF) in the Scottish population showed that hospital discharge rates for CHF increased by almost 60% between 1980 and 1990.

HF is largely a disease of old age and represents the leading hospital diagnosis in older adults. Both the incidence and the prevalence of CHF increase sharply with increasing age such that patients aged >75 face a much greater risk of developing this condition. Estimates calculated within the last decade suggest a prevalence of ∼1–2% in the overall population and >10% in the elderly population.

It has been estimated that there are currently 6.5 million CHF patients in Europe and 5 million in the USA, and these numbers are increasing because of the ageing of the global population and the ability of increasing numbers of individuals to survive to an age when CHF is likely to become a problem [3].

In the past few decades, heart failure has emerged as a major public health problem in developed countries, imposing an escalating burden on their health care systems. In some European countries and the U.S., as much as 1% of the health budget is spent on the management of heart failure. In the U.S., inpatient treatment of heart failure accounts for about U.S. $13 billion per year, and the amount spent on outpatient care is at least 4 times higher. Regretfully, most of the published data on heart failure is based on work in Caucasian populations within the developed world, and data on its incidence, prevalence, etiology, treatment, and outcome in Africa are lacking. As a result of improvements in the control of communicable diseases and malnutrition on the one hand and the migration to the cities with a complete change in the living habits on the other hand, cardiovascular diseases such as hypertension and stroke are increasingly recognized as significant causes of morbidity and mortality in most African countries . Nowadays, cardiovascular diseases account for 7% to 10% of all medical admissions to hospital, with heart failure contributing to 3% to 7%. In this paper we summarize the current state of knowledge on the etiology, treatment, and

outcome of heart failure in Africa. Furthermore, we propose to establish a large prospective multicenter multinational register to define the causes, contemporary treatment, and outcome of patients with heart failure in sub-Saharan Africa. These data will assist in developing a strategy for the early detection, treatment, and prevention of heart failure on the continent [4], therefore the extrapolation statistics yearly of the (CHF) in Sudan is 69,849 [5].

Early diagnosis of congestive heart failure (CHF) is the goal because optimal treatment can prevent or slow (CHF) progression .diagnosis is based on clinical features chest x-ray, electrocardiography (ECG) and echocardiogram. Everyone with suspected (CHF) should undergo an ECG and Chest x-ray even physical signs are normal.

1.2 Problem of the study:

The increasing incidence of congestive heart failure among the native in addition, the variable choices of diagnostic models for the CHF that accompanied with different results could be consider as main problem of the study.

1.3 Objectives of the study:

To measure the heart size for CHF Patients

To determine the common site of enlargement due to CHF

To correlated between chest x-ray and ECG for cardiac enlargement

To determine the incidence percent based on gender, age, and stage

To correlate between the clinical history and heart size

1.4 Thesis outlines:

The following thesis will be laid out in five chapters, chapter one will highlight the introduction, problem of the study, objectives and thesis outlines. Chapter two will show the literature review. Chapter three will deals with methodology. Chapter foursection one will presents the results and section two will show the discussion and analysis. Chapter five will deals with conclusion, recommendation, references and appendices.

Chapter Tow
Literature Review

Heart failure is a condition in which the heart is no longer able to pump out enough oxygen - rich blood. This causes symptoms to occur throughout the body.

Causes and types of heart failure:

Heart failure is often a long-term (chronic) condition, but it may come on suddenly. It can be caused by many different heart problems.

The condition may affect only the right side or only the left side of the heart. More often, both sides of the heart are involved.

The latter classification which is the most commonly used because of its implication the treatment and long term outcome of heart failure patients.

2.1 Types of Heart Failure:

Systolic dysfunction is characterized by a decrease in myocardial contractility. A reduction in the left ventricular ejection fraction (LVEF) results when myocardial contractility is decreased throughout the left ventricle. Cardiac output is maintained in two ways: left ventricular enlargement results in a higher stroke volume, and the Frank-Starling relationship (an increase in contractility in response to an increase in stretch). However, these compensatory mechanisms are eventually exceeded and cardiac output decreases, resulting in the physiologic manifestations of heart failure. The left heart cannot pump with enough force to push a sufficient amount of blood into the systemic circulation. This leads to fluid backing up into the lungs and pulmonary congestion. Systolic dysfunction is a characteristic of dilated cardiomyopathy (DCM). It is also seen in some patients with hypertrophic cardiomyopathy (HCM) who develop progressive left ventricular dilatation and a decrease in LVEF. In general terms, systolic dysfunction is defined as an LVEF less than 40%. The Diastolic dysfunction refers to cardiac dysfunction in which left ventricular filling is abnormal and is accompanied by elevated filling pressures. The diastolic phase of cardiac function includes two components. Left ventricular relaxation is a process that takes place during isovolemic relaxation (the period between aortic valve closure and the mitral valve opening) and then during early rapid filling of the ventricle. Later in diastole, after relaxation is complete, further left ventricular filling is a passive process that depends on the compliance, or distensibility

of the myocardium. The ventricles are unable to relax, and subsequent muscle hypertrophy could and further would lead to inadequate filling. Diastolic dysfunction may lead to fluid accumulation, especially in the feet, ankles, and legs, and some patients may also have pulmonary congestion. For patients with heart failure but without systolic dysfunction, diastolic dysfunction is the presumed cause. Diastolic dysfunction is characteristic of both HCM and restrictive cardiomyopathy (RCM). However, some component of diastolic dysfunction is also common in patients with DCM. In general terms, diastolic dysfunction is defined as an LVEF of greater than 40%.

Diastolic dysfunction is more difficult to identify with echocardiograph scanning than systolic dysfunction, and it may be missed or underestimated in many cases. Doppler scan assessment of transmitral flow is the standard approach to detect diastolic dysfunction, although a variety of other measurements can be used. It is important to understand that some of the symptoms of systolic and diastolic heart failure are similar [6]. The most common causes of heart failure are:

- Coronary artery disease (CAD), a narrowing of the small blood vessels that supply blood and oxygen to the heart. This can weaken the heart muscle over time or suddenly.
- High blood pressure that is not well controlled, leading to problems with stiffness, or eventually leading to muscle weakening.

Other heart problems that may cause heart failure are:

- Congenital heart disease
- Heart attack
- Heart valves that are leaky or narrowed
- Infection that weakens the heart muscle
- Some types of abnormal heart rhythms (arrhythmias)

Other diseases that can cause or contribute to heart failure including over active thyroid, underactive thyroid, emphysema, sarcoidosis, severe anemia and too much iron in the body [7].

2.2 Risk factors of CHF

Heart failure is the final common pathway of many cardiovascular risk factors and of most forms of heart disease. Myocardial ischaemia and reperfusion with evolving oxidative stress, either as repetitive episodes during increased demand or as a cause of

myocardial infarction leading to scar formation and ventricular remodelling, is the most common form of this syndrome. Furthermore, different forms of myocardial disease either due to genetic causes or acquired in response to risk factors such as hypertension and/or diabetes are also of importance. Finally, congenital heart disease, either in the young or in adults, often leads to pump failure and death [8]. Congestive heart failure may be exacerbated by the following lifestyle habits: Unhealthy habits, such as smoking and excessive use of alcohol, Obesity and lack of exercise (May contribute to congestive heart failure, either directly or indirectly through accompanying high blood pressure, diabetes, and coronary artery disease, high salt intake which may cause more fluid retention, Noncompliance with medications and other therapies.

2.2.1 Lifestyle Changes

Several studies have reported reduced risk for heart failure with healthy lifestyle. Healthy weight, avoiding smoking, engaging in exercise, and a healthy diet have been shown to reduce heart failure risk factors including coronary disease, diabetes mellitus, and hypertension. Recently, the physicians' health study investigators reported that healthy lifestyle habits, that is, normal body weight, not smoking, regular exercise, moderate alcohol intake, consumption of breakfast cereals, and consumption of fruits and vegetables were associated with a lower risk of heart failure, with the highest risk of 21.3% in men adhering to none of these habits and the lowest risk of 10.1% in men adhering to 4 or more of them.

2.2.2 Overweight and Obesity

Body mass index is associated with heart failure in a positive and linear fashion in both sexes. Although body mass index in the obese range ($\geq 30\,kg/m^2$) is clearly associated with an increased risk for heart failure, there is controversy regarding body mass index in the overweight range (25 to $29.9\,kg/m^2$). Recent data, however, support that overweight is also associated with heart failure. Abdominal obesity may be a stronger predictor for heart failure than total obesity, even in the absence of coronary heart disease. Several mechanisms by which elevated body mass index increases the risk of heart failure have been proposed including (a) alterations in cardiac loading, (b) changes in cardiac structure and function , (c) activation of neurohumoral and inflammatory pathways , (d) promotion of atherogenic conditions , (e) predisposition to sleep-disordered breathing , and (f) chronic kidney disease . The principal approach

of risk reduction in obese patients should include weight control and physical activity, and control of the associated risk factors such as hypertension, diabetes mellitus, sleeps disorders, and components of the metabolic syndrome. Myocardial changes with non-surgical or surgical weight loss are feasible and minor weight loss is efficacious; a 10% weight reduction ameliorates systolic dysfunction, and weight loss of 8 to 10 kg produces a significant decrease in left ventricular dimensions and mass index and improves diastolic function. Substantial weight loss reduces left ventricular wall thickness and volume, filling pressures and improved diastolic measures and left ventricular systolic function. The role of metabolic and neurohumoral modification may take precedence over the hemodynamic effects as left ventricular mass or functional improvement occurs independently of loading alterations.

2.2.3 Sedentary Exercise Habits

Physical inactivity is an important risk factor for cardiovascular diseases including heart failure. Regular physical activity has important and wide-ranging benefits like reduction in risk of cardiovascular diseases, hypertension, and diabetes. Physical activity is a key determinant of good health and an important component of weight reduction and weight maintenance, improved lipoprotein profile, and reduced risk of hypertension, diabetes mellitus, and coronary artery disease. These favorable influences on cardiovascular risk profile in turn reduce the likelihood of heart failure. Physical activity could also reduce left ventricular hypertrophy and improve endothelial function. Chronic physical activity reduces cytokine production by adipose tissue, skeletal muscles, and endothelial and blood mononuclear cells and up regulates antioxidant enzymes. These modifying effects on heart failure risk factors or intermediate pathways leading to heart failure can reduce incident heart failure. The integration of physical activity into the daily lives of the population has proved to be challenging. Currently, the recommendations of the American College of Sports Medicine and the American Heart Association for regular physical activity in healthy adults 18–65 years include the following. (a) Aerobic Activity Moderate-intensity aerobic physical activity for a minimum of 30 min on five days each week or vigorous-intensity aerobic activity for a minimum of 20 min on three days each week. (b) Muscle-Strengthening Activity. It is recommended that 8–10 exercises should be performed on two or more nonconsecutive days each week using the major muscle

groups. To maximize strength development, a resistance (weight) should be used that allows 8–12 repetitions of each exercise resulting in volitional fatigue.

(c) Activity Dose. Vigorous-intensity activities may have greater benefit than moderate-intensity physical activity.

Walking has been reported as beneficial regarding primary prevention, this should be adopted by individuals who do not adhere to the current recommendations.

2.2.4 Alcohol Consumption

Excessive alcohol consumption is associated with alcoholic cardiomyopathy. Interestingly, other data are consistent with possible benefits of moderate alcohol consumption on the risk of heart failure. The New Haven Epidemiologic Study of the Elderly program and the Cardiovascular Health Study reported a 47% and a 34% lower heart failure risk, respectively. The Framingham heart study reported a 59% lower risk among men who consumed 8 to 14 drinks per week compared with abstainers and only a modest and non significant association in women. Moreover, it has been reported that light-to-moderate alcohol consumption is associated with 40% to 50% lower risk of heart failure with previous myocardial infarction, whereas in the same study the risk of heart failure without antecedent myocardial infarction among heavy drinkers was 1.7-fold higher than in abstainers. Similar findings were reported in the physicians' health study. Beneficial effects of alcohol have also been reported on risk for hypertension, myocardial infarction, and diabetes mellitus, whereas alcohol seems to raise high-density lipoprotein cholesterol, improve insulin sensitivity, lower plasma levels of inflammatory markers and coagulation factors, and raise plasma levels of adiponectin.

2.2.5 Dietary Habits

In the Dietary Approaches to Stop Hypertension (DASH) diet, individuals are encouraged to consume more (i) fruits and vegetables, (ii) grains and grain products, (iii) lean meats, fish, poultry, (iv) low fat or nonfat dairy foods, and (v) nuts, seeds, and legumes, and reduce the consumption of red meat, fat, and sugar while maintaining a low sodium intake. Initially, this was promoted for hypertension; however, recent evidence supports reduction of heart failure risk with an observed 37% lower rate in women who adhere to the DASH diet. The DASH diet may contribute to heart failure prevention by reduction in blood pressure and coronary heart disease. Significantly, the DASH diet reduces low-density lipoprotein

cholesterol levels and oxidative stress and exerts beneficial physiologic effects like estrogenic effects of phytochemicals. Daily consumption of whole-grain breakfast cereals was associated with a 30% lower rate of heart failure, consumption of eggs more than twice per day was associated with a 64% higher rate, consumption of fish was associated with a 20–31% lower heart failure rate depending on consumption, and consumption of 100 mmol or more of sodium was associated with a 26% higher rate; only nut consumption was not associated with heart failure. When animals were fed the high-fructose diet they demonstrated more cardiac remodeling and worse survival. Whole grain cereals could protect against heart failure risk through effects on weight, hypertension, myocardial infarction, and diabetes mellitus. Nutrients contained in whole grain cereals, for example, potassium, may lower blood pressure, phytoestrogens may improve lipid levels and insulin sensitivity, and other constituents exert beneficial effects on lipid and homocysteine levels.

Fish consumption exerts beneficial effects on heart failure risk with about a 20% lower risk associated with an intake of 1 to 2 times per week and about a 30% lower risk with intake ≥3 times per week, compared with intake less than 1 time per month. Estimated intake of marine n-3 fatty acids was associated with 37% lower heart failure risk in the highest quintile of intake compared with the lowest. Short-term trials of fish oil supplementation of 3–5 grams per day may reduce risk whereas dietary doses of about 0.5 grams per day may result in more modest effects that over long term may reduce heart failure risk. It has been reported that broiled or baked fish consumption is inversely associated with systolic blood pressure, C-reactive protein levels, and carotid intimal medial thickness, whereas fried fish intake is positively associated with them, indicating that the type of cooking could impact the effects.

Historically, human ancestors consumed less than 0.25 grams of salt per day. The recent change to the high salt intake of 10–12 g per day presents a challenge to the physiological systems to excrete these large amounts of salt resulting in a rise in blood pressure and increase in the risk for cardiovascular and renal disease. Currently, the Department of Health and Human Services and Department of Agriculture recommends that adults should consume no more than 2300 mg per day of sodium, but specific groups, that is, all persons with hypertension, all middle-aged and older adults, and all blacks, should consume no more than 1500 mg per day of sodium. Overall, 69.2% of the United States adults meet the criteria for the risk groups. There

is overwhelming evidence for a causal relationship between salt intake and blood pressure.

A reduction in salt intake may have other beneficial effects on the cardiovascular system, independent of and additive to its effect on blood pressure and include regression of left ventricular hypertrophy, delay in deterioration of renal function, and reduction in protein uria.

2.2.6 Smoking

Smoking is a strong predictor of heart failure in both men and women, with 45% and 88% increased risk. The deleterious effect of tobacco seems to be independent of the form of use; increased risk for cardiovascular diseases is reported in nonsmoking use of tobacco. There is no "safe" level of smoking; single cigarette may stiffen the left ventricle, and as few as 1 to 4 cigarettes a day double the risk of myocardial infarction. Mechanisms leading to heart failure in smokers include (i) indirect effects, that is by causing or aggravating co-morbidities that are related with heart failure, and (ii) direct effects on the myocardium. In animal models, nicotine induces interstitial fibrosis in the ventricles. Besides nicotine, carbon monoxide is also a significant component of tobacco smoke and causes over expression of growth-related proteins such as calmodulin , calcineurin, and vascular endothelial growth factor. Smoking is associated with higher left ventricular mass, lower stroke volume, ejection fraction, and impaired ventricular diastolic function.

All smokers should be counseled to quit. Patients should be referred to formal cessation programs, and pharmacological therapy should be offered to increase the success rate. Current recommended strategies include the following:

(a)   Medications. Several medications are available for tobacco dependence. Seven first-line medications reliably increase long-term smoking abstinence rates including bupropion SR, nicotine gum or inhaler or lozenge or nasal spray or patch, and varenicline.

(b)   Counseling and Psychosocial Support. Individual, group, telephone practical counseling, and social support are effective, and their effectiveness increases with treatment intensity.

(c)   Combination. The combination of counseling and medication, however, is more effective than either alone, therefore, clinicians should encourage all individuals making a quit attempt to use both counseling and medication.

In the studies of left ventricular dysfunction trials, the risk for heart failure hospitalizations and myocardial infarctions was reduced after quitting. Women's risk of heart disease is reduced by one third within 2 years of quitting and about two thirds within 5 years [9].

2.3 Symptoms of CHF

Symptoms typical of heart failure (breathlessness at rest or on exercise, fatigue, tiredness, ankle swelling) and the signs typical of heart failure (tachycardia, tachypnea, pleural effusion, raised jugular venous pressure, peripheral oedema, hepatomegaly) and such presentation could be shown in table (1) [10].

Table 2.1: shows the common CHF presentations (signs & symptoms) [10]

Dominant clinical feature	Symptoms	Signs
Peripheral oedema/congestion	Breathlessness Tiredness, fatigue Anorexia	Peripheral oedema Raised jugular venous pressure Pulmonary oedema Hepatomegaly, ascites Fluid overload (congestion) Cachexia
Pulmonary oedema	Severe breathlessness at rest	Crackles or rales over lungs, effusion Tachycardia, tachypnea
Cardiogenic shock (low output syndromes)	Confusion Weakness Cold periphery	Poor peripheral perfusion SBP <90 mmHg Anuria or oliguria
High blood pressure (hypertensive heart failure)	Breathlessness	Usually raised BP, LV hypertrophy, and preserved EF
Right heart failure	Breathlessness Fatigue	Evidence of RV dysfunction Raised JVP, peripheral oedema, hepatomegaly, gut congestion

Basic early Investigations are necessary to differentiate heart failure from other conditions and to provide prognostic information. Urinalysis, serum urea and creatinine tests may help to determine if there is kidney failure, since symptoms of kidney disease are similar to those of CHF. Chest X-ray may indicate signs of CHF such as cardiomegaly, pulmonary congestion or pleural effusion and also non-cardiac indications such as lung tumors which account for breathlessness.

Patients suspected of chronic heart failure should receive a range of basic tests. The investigations will vary depending on the presentation but should usually include a full blood count, fasting blood glucose, serum urea and electrolytes, urinalysis, thyroid function and chest X-rays .

2.4 Enlarged heart (cardiomegaly)

An enlarged heart (cardiomegaly) can have various causes. But it's usually caused by high blood pressure (hypertension) or coronary artery disease.

An enlarged heart may not pump blood effectively, resulting in congestive heart failure. Cardiomegaly may improve over time. But most people with an enlarged heart need lifelong treatment with medications.

2.4.1 Types of Enlarged Heart

The heart enlarges in response to damage to the heart muscle, up to a point, enlargement permits the heart to continue to pump blood normally as enlargement progresses, though, the heart's pumping ability declines.

Dilated cardiomyopathy is the main type of cardiomegaly. In dilated cardiomyopathy, the walls of both the left and right side of the heart (ventricles) become thin and stretched; the result is an enlarged heart.

In the other types of enlarged heart, the heart's muscular left ventricle becomes abnormally thick:

Left ventricular enlargement (hypertrophy) is usually caused by high blood pressure.

Hypertrophic cardiomyopathy is an inherited (genetic) condition.

Generally the speaking, the heart's pumping ability is better preserved when the enlarged heart is "thick" rather than "thin".

Most common causes of an enlarged heart are blockages in the heart's blood supply (coronary artery disease) and high blood pressure. An enlarged heart can have many other causes, including: Frequently, no cause for an enlarged heart is identified. This is known as idiopathic dilated cardiomyopathy.

2.4.2 Symptoms of an Enlarged Heart

Most often, an enlarged heart causes no symptoms. If an enlarged heart becomes unable to pump blood effectively, symptoms of congestive heart failure can develop:

Shortness of breath (especially with exertion or when lying flat)

Leg swelling

Increased abdominal girth

Weight gain

Fatigue
Palpitations or skipped heartbeats

Symptoms vary widely in people with an enlarged heart. Some may never have symptoms. Others may have mild symptoms that remain unchanged for years. And some may experience steadily worsening shortness of breath.

2.4.3 Diagnosis of an Enlarged Heart

An enlarged heart may be discovered after a person discusses symptoms of congestive heart failure. Other times, it is discovered in someone without symptom who gets a test for other reasons.

Echocardiography (ultrasound of the heart) is the preferred test to diagnose an enlarged heart. There is no pain or risk from an echocardiogram.

It can accurately measure the heart's Size, muscle thickness and pumping function.

In some cases, echocardiography can suggest potential causes for an enlarged heart.

There may be various other tests to help diagnose an enlarged heart, such as: History: Shortness of breath or other symptoms of congestive heart failure may provide initial clues to the presence of an enlarged heart.

Physical exam: Swelling may be present, and an enlarged heart may produce abnormal sounds when listens with a stethoscope.

Chest X-ray: The type of enlarged heart called dilated cardiomyopathy produces an abnormally large shadow on a chest X-ray film.

Cardiac catheterization, during this procedure to detect blockages in the coronary arteries, the heart's size and pumping function can also be checked.

Blood tests: These may be done to check for causes of enlarged heart, such as: congestive heart failure, thyroid disease, HIV or other viral infection.

CT scans and MRIs: These may help diagnose an enlarged heart in certain situations.

Biopsy: Very rarely, a doctor may recommend taking a small tissue sample from inside the heart to determine the cause of an enlarged heart [11].

2.5 Stages of CHF

The New York heart Association (NYHA) functional classification has proved to be clinically useful and it is employed routinely in most randomized clinical trials. The other describes HF in stages based on structural changes and symptoms. All patients with overt HF are in stages C and D.

Table 2.2: Classification of heart failure by structural abnormality based on American heart association (ACC/AHA), or depending on symptoms relating to functional capacity (NYHA) [12].

ACC/AHA stages of heart failure		NYHA functional classification	
Stage of heart failure based on structure and damage to heart muscle		Severity based on symptoms and physical activity	
Stage A	At high risk for developing heart failure. No identified structural or functional abnormality; no signs or symptoms.	**Class I**	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnoea.
Stage B	Developed structural heart disease that is strongly associated with the development of heart failure, but without signs or symptoms.	**Class II**	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnoea.
Stage C	Symptomatic heart failure associated with underlying structural heart disease.	**Class III**	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in fatigue, palpitation, or dyspnoea.
Stage D	Advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy.	**Class IV**	Unable to carry on any physical activity without discomfort. Symptoms at rest. If any physical activity is undertaken, discomfort is increased.

2.6 Diagnosis of CHF

Chest x-ray, ECG, and an objective test of cardiac function typically echocardiogram should be done. Blood tests, except for BNP levels, are not used for diagnosis but are useful for identifying cause and systemic effects.

2.6.1 Electrocardiography (ECG)

The ECG is an important screening tool that offers practitioners a wealth of information that can be used alongside the history and clinical finding. An ECG provides a measurement of the rate and rhythm of the heart. It also provides information about the health of the electrical system, the size of the heart chambers, and the supply of blood to the heart muscle. ECGs are pivotal in the diagnosis of cardiac ischaemia and infarction, provide the evidence for pacemaker implantation, and detect inherited abnormalities such as cardiomyopathy and long-QT syndrome. ECGs are also useful in detecting non-cardiac pathology, for example, pulmonary emboli and electrolyte disorders [13].

2.6.2 CHEST X-RAY

Congestive heart failure (CHF) is the result of insufficient output because of cardiac failure, high resistance in the circulation or fluid overload. Left ventricle (LV) failure is the most common and results in decreased cardiac output and increased pulmonary venous pressure. In the lungs LV failure will lead to dilatation of pulmonary vessels, leakage of fluid into the interstitium and the pleural space and finally into the alveoli resulting in pulmonary edema.

Right ventricle (RV) failure is usually the result of long standing LV failure or pulmonary disease and causes increased systemic venous pressure resulting in edema in dependent tissues and abdominal viscera.

Increased pulmonary venous pressure is related to the pulmonary capillary wedge pressure (PCWP) and can be graded into stages, each with its own radiographic features on the chest film. This grading system provides a logical sequence of signs in congestive heart failure. In daily clinical practice however some of these features are not seen in this sequence and sometimes may not be present at all. This can seen in patients with chronic heart failure, mitral valve disease and in chronic obstructive lung disease.

2.6.2.1 Stage I – Redistribution:

The normal chest film with the patient standing erect, the pulmonary vessels supplying the upper lung fields are smaller and fewer in number than those supplying the lung bases.

The pulmonary vascular bed has a significant reserve capacity and recruitment may open previously non-perfused vessels and causes distension of already perfused vessel, this results in *redistribution* of pulmonary blood flow. First there is equalization of blood flow and subsequently redistribution of flow from the lower to the upper lobes. The term redistribution applies to chest x-rays taken in full inspiration in the erect position. In daily clinical practice many chest films are taken in a supine or semi-erect position and the gravitational difference between the apex and the lung bases will be less. In the supine position, there will be equalization of blood flow, which may give the false impression of redistribution. In these cases comparison with old films can be helpful.

2.6.2.2 Stage II - Interstitial edema:

Stage II of CHF is characterized by fluid leakage into the interlobular and peribronchial inter-stitium as a result of the increased pressure in the capillaries. When fluid leaks into the peripheral interlobular septa it is seen as Kerley B or septal lines. Kerley-B lines are seen as peripheral short 1-2 cm horizontal lines near the costophrenic angles. These lines run perpendicular to the pleura. And when the fluid leaks into the peribronchovascular inter-stitium, it appears as thickening of the bronchial walls (peribronchial cuffing) and as loss of definition of these vessels (perihilar haze).

2.6.2.3 Stage III - Alveolar edema:

This stage is characterized by continued fluid leakage into the interstitium, which cannot be compensated by lymphatic drainage. This eventually leads to fluid leakage in the alveoli (alveolar edema) and to leakage into the pleural space (pleural effusion). The distribution of the alveolar edema can be influenced by:

- Gravity: supine or erect position and right or left decubitus position
- Obstructive lung disease, i.e. fluid leakage into the less severe diseased areas of the lung.

After treatment we can still see an enlarged cardiac silhouette, pleural fluid and redistribution of the pulmonary blood flow, but the edema has resolved. Pleural effusion is bilateral in 70% of cases of CHF. When unilateral, it is slightly more often on the right side than on the left side. There has to be at least 175 ml of pleural fluid, before it will be visible on a PA image as a meniscus in the costophrenic angle. On a lateral image effusion of > 75 ml can be visible. If pleural effusion is seen on a supine chest film, it means that there is at least 500 ml present.

RV failure is most commonly caused by longstanding LV failure, which increases the pulmonary venous pressure and leads to pulmonary arterial hypertension, thus overloading the RV.

2.6.3 Radiographic signs of RV failure:

- Increased VPW(vascular pedicle width) due to dilatation of the superior vena cava
- Dilatation of azygos vein
- Dilatation of the right atrium [14].

Zahidullah Khan et al, [15] carried out study to determine frequency of various etiological factors responsible for causing CHF at Khyber Teaching Hospital (Beshawar) the study include 100 patients of CHF (60 male and 40 female) with mean age 54 years, after history and clinical examinations. The diagnosis of CHF by using ECG, Echocardiography, chest x-ray and serial blood tests, showed that: ischemic heart disease (IHD) was present in 35% of the patients, hypertension in 27%, rheumatic heart disease in13%, cardiomyopathy in 11% and congenital heart disease in 6%. Five patients initial clinical status was classified according to NYHA functional class. Most of the pt (45%) presented to the unit were in NYHA class III heart failure. 65 patients had LV systolic dysfunction with mean ejection fraction of 41.4% while 35 patients had diastolic V/dysfunction with mean ejection fraction of 66.8% on echocardiography. Three patients had thrombus in left atrium. Four patients had mitral stenosis and two patients had mitral regurgitation, while one patient had aortic stenosis. None of the patients had normal ECG. Left ventricular hypertrophy criteria on ECG were identified in 24% of patients.

Margaret et al, [16], made study to determine the incidence increasing of CHF among male and female in (united state), according to the data from the national hospital discharge survey 2000-2010. There were 1 million hospitalizations for CHF in 2000 and in 2010. Most of CHF hospitalization were for those aged 65 and over but the proportion under 65 increased significantly from 2000-2010 (35.5 compared with 32.8), but the trends were different for those under and over age 65. From 2000-2010 the rate of CHF and hospitalizations for males under age 65 increased significantly while the rate for females aged 65 and over decreased significantly. In both years 2000 and in 2010; a great share of inpatient under 65 compared with those aged 65 and over were discharged to their homes. In 2000 and in 2010 there were an estimated I million Hospitalization for CHF, while most of this hospitalization were for those aged 65 and over and the share of CHF hospitalization for those under age 65 increase from 23% to 29% over this time period. Such distribution has been shown in Figure (2.1).
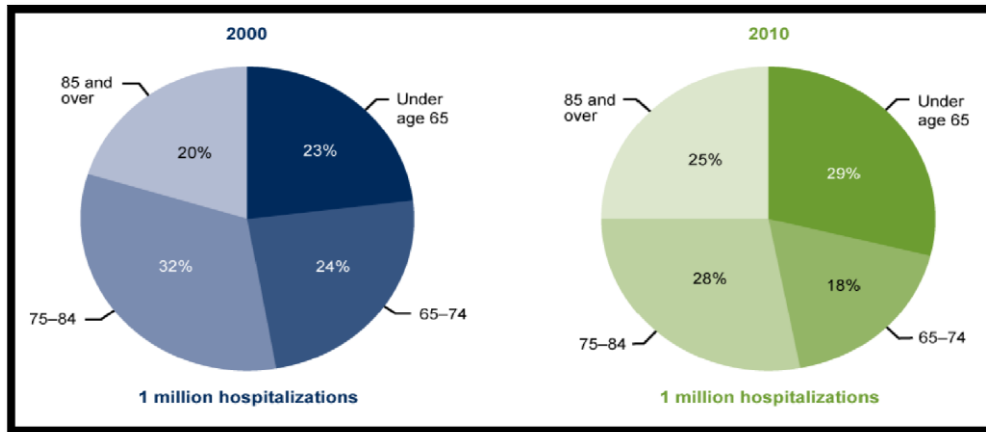
Figure 2.1: Percent distribution of hospitalization for CHF by age in years in USA during 2000 and 2010 (CDC/NCHS, National Hospital Discharge Survey, 2000–2010).

Djoussé et al, [17], found that according to study reported in Journal of American Medicine Association in (2009) that: unhealthy habits such as smoking and excessive use of alcohol, obesity and lack of exercise may contribute to CHF, either directly or indirectly through accompanying high blood pressure, diabetes and CAD.

Satish Kenchaiah et al, [18] made study to investigate the relation between the bodymass index and the incidence of heart failure among 5881 participants in the Framingham heart study. the mean age 55 years, 54 percent women, with the use of Cox proportional-hazards models, the body-mass index was evaluated both as a continuous variable and as a categorical variable (normal,18.5 to 24.9; overweight, 25.0 to 29.9; and obese, 30.0 or more). Their study of 14 years follow up revealed that: heart failure developed in 496 subjects (258 women and 238 men), there was an increase in the risk of heart failure of 5 percent for men and 7 percent for women for each increment of 1 in body-mass index. As compared with subjects with a normal body-mass index, obese subjects had a doubling of the risk of heart failure. For women, the hazard ratio was 2.12 (95 percent confidence interval, 1.51 to 2.97); for men, the hazard ratio was 1.90 (95 percent confidence interval, 1.30 to 2.79). A graded increase in the risk of heart failure was observed across categories of body mass index. The hazard ratios per increase in category were 1.46 in women (95% confidence interval, 1.23 to 1.72) and 1.37 in men (95 percent confidence interval, 1.13 to 1.67) [18].

Pollehn et al, [19] carried out study to show the importance of the electrocardiographic differential diagnosis of ST segment depression (STD) in patients presenting with acute chest pain. They found that: STD is associated with

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acute coronary syndromes (ACS), both acute ischemia and acute infarction, this electrocardiographic pattern, however, may also be found in patients with nonischemic events, such as left bundle branch block (LBBB), left ventricular hypertrophy (LVH), and those with therapeutic digitalis levels. The following cases Figure (2.2) illustrate the use of ECG in patients presenting with chest pain and electrocardiographic STD attributable to ACS, LVH, LBBB or digitalis.

Figure (2.2): shows the various causes of electrocardiographic ST segment depression. (A) ST segment depression related to non-infarction ischemia, horizontal in morphology. (B) Reciprocal ST segment depression in lead III in a patient with acute anterior wall AMI. (C) Lead V2 STD attributable to posterior wall AMI. (D) Digoxin effect (E) Left ventricular hypertrophy. (F) Left bundle branch block.

In the following examples of ECG, some cases studies have been carried out and diagnosed by Eric et al, [20]: Figure (2.3-), shows the following ECG presentations, Atrial fibrillation, Normal QRS duration, Left axis deviation (LAD) noted and Paced atrial rhythm.
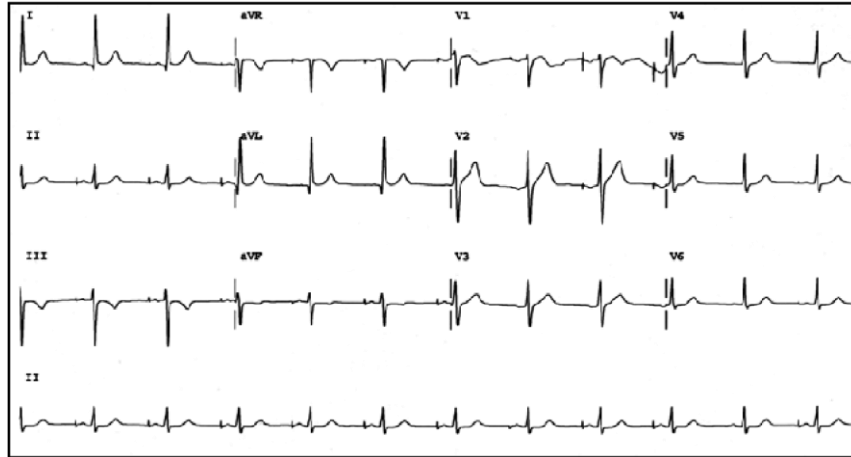
Figure 2.3: shows atrial fibrillation, Normal QRS duration, Left axis deviation (LAD) noted and Paced atrial rhythm
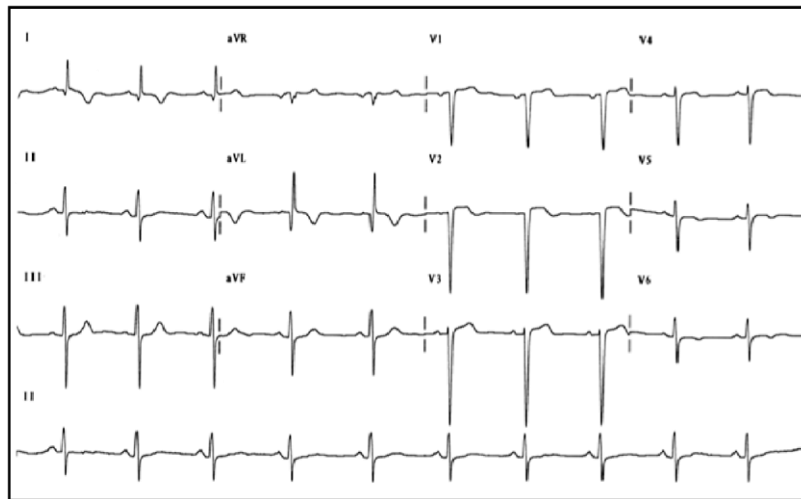
Figure 2.4: shows an ECG with obvious Normal cardiac axis, Normal QRS duration, and Lateral wall infarction.

On other hand the detection of CHF by using chest x-ray also has considerable rule, in this realm here are some cases presented by some scholars.

Figure 2.5: shows 5CXR findings: cardiomegaly, Edema and haziness of vascular margins.

Gromadziński et al, [21] carried out study for CHF patients images to example the findings of CHF in chest x-ray and reveals the CHF phases.
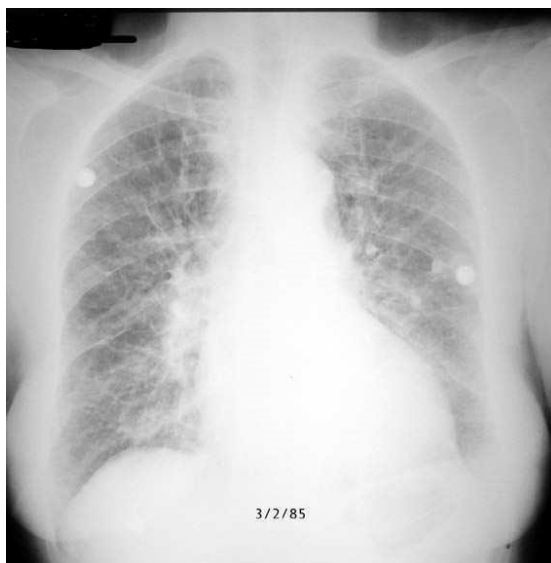
Figure 2.6: CXR finding: cephalization on vessels in upper lobes of the lungs which is more prominent as manifestation on pulmonary venous hypertension (vascular phase).
.
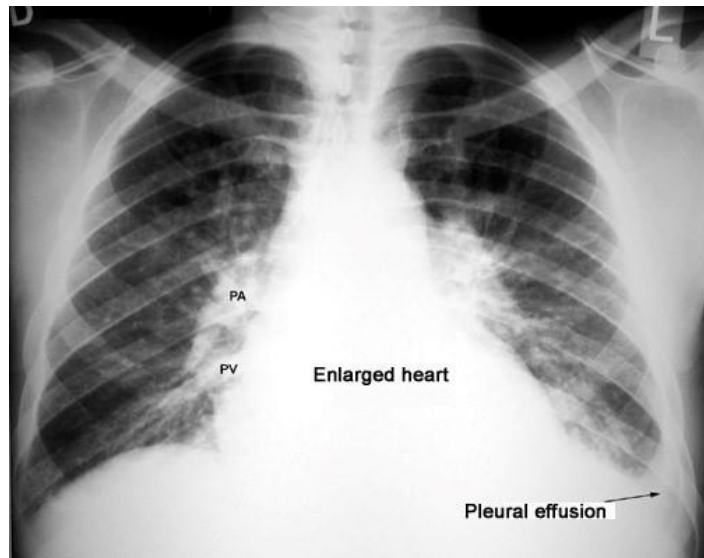
Figure 2.7: CXR finding: Hilar fullness with haziness enlarged pulmonary veins with perivascular fluid collection leads to full hazy hilar and vessels

Figure 2.8: CXR shows bilateral diffuse soft fluffy alveolar infiltrates with each other in abutterfly distribution (alveolar phase).
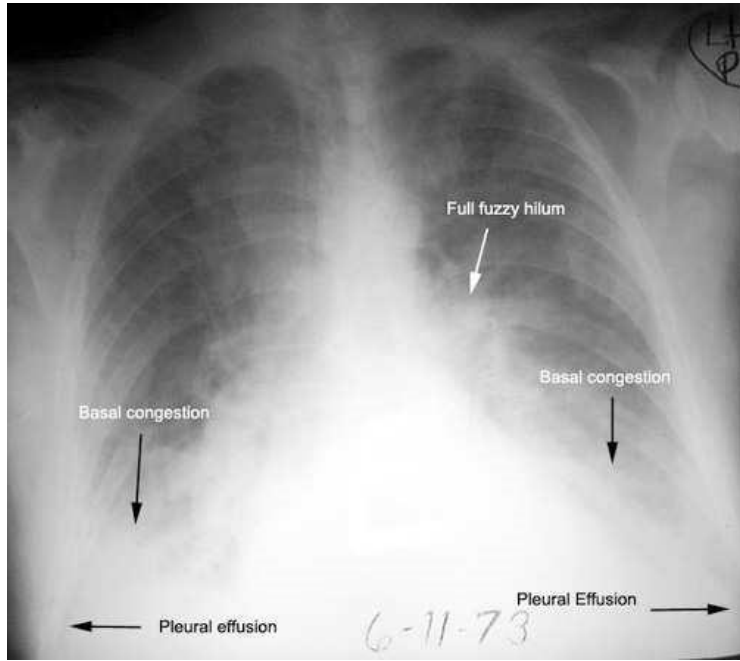
Figure 2.9: CXR shows cardiomegaly, bilateral hilar congestion, fuzzy full hilum and bilateral pleural effusion.

Chapter Three
Methodology of Study

The following is retrospective comparative study of chest x-ray and electrocardiogram ECG findings in congestive heart failure (CHF). The data was collected for 120 patients from Khartoum and Omdurman hospitals and the variables were: the age, gender, and clinical history, etiology, habits, BMI, heart/chest ratio, enlarged chamber and the relative deviated wave in ECG.

3.1 Tools & Equipments

- x-ray system
- ECG system

3.2 Method (Technique):

- X-RAY CHEST

The patient standing or sitting facing the cassette in posterior anterior projection to avoid magnification of the heart size, the chin extended and centered to the middle of the top of the cassette which support at stand buckey, the median sagittal plane is adjusted at right-angles to the middle of the cassette. The hands put at the trunk with the palm of the hands facing the tube.

The direction of the central ray is horizontal at right-angles at the level of fifth thoracic vertebra; exposure is made in full normal arrested inspiration, as a poor inspiration will make the heart look larger. The focal film distance is 72 inches to avoid distortion of the heart size. Then the heart/chest ratios have been calculated depending on maximum transverse diameter of the heart multiplied by the maximum width of the thorax

- ECG

When making a recording of ECG, The patient must lie down and relax. The six standard leads, which are recorded from the electrodes attached to the limbs, can be thought of as looking at the heart in vertical plane (that's from the sides or the feet). Calibration of the record with the 1 MV signal, then record the six standard leads. Leads 1, II and VL look at the left lateral surface of the heart, III and VF attached to the inferior surface, and VR looks at the RT atrium.

The V lead is attached to the chest wall by means of a suction electrode, and recordings are made from six positions, overlying the 4th and 5th rib spaces. The six

numbered V leads look at the heart in horizontal plane, from the front and the left side. Thus leads V1 and V2 look at the right ventricle, V3 and V4 look at the septum between the ventricles and the anterior wall of the left ventricle, and V5 and V6 look at the anterior and lateral walls of the left ventricle . As with the limb leads, the chest leads each show a different ECG pattern. In each lead the pattern is characteristics, being similar in different individuals who have normal hearts.

3.3 The shape of the ECG:

The muscle of the atria is small, and the electrical changes accompanying the contraction of the atria are there for small. Contraction of the atria is associated of the ECG wave called P.

The ventricular mass is large, and so there is a large deflection of when the ECG the ventricles are depolarized. This is called the QRS complex.

The T wave of the ECG is associated with the return of the ventricular mass to its resting electrical state (re-polarization).

3.4 Calibration:

A limited amount of information is provided by the height of the P waves, QRS complexes and T waves, provided the machine is properly calibrated. A Standard signal of 1Mv should move the stylus vertically 1 cm (two large squares), and this calibration should be included with every record.

Chapter Four Section-One
Results of the Study

The following chapter will highlight the results related to the data collected from the CHF patients at Khartoum, Omdurman, Aljazeera, and Kosti teaching Hospitals.
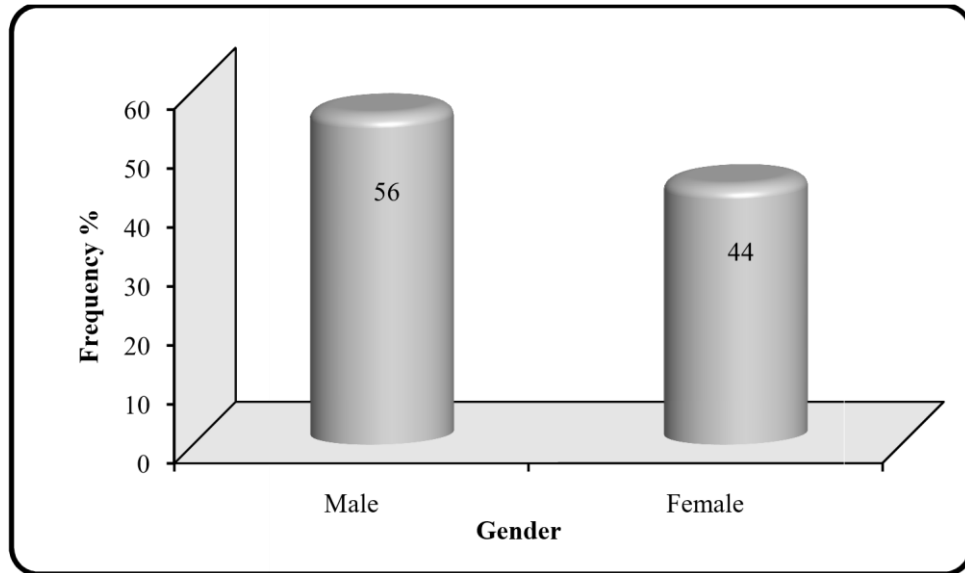

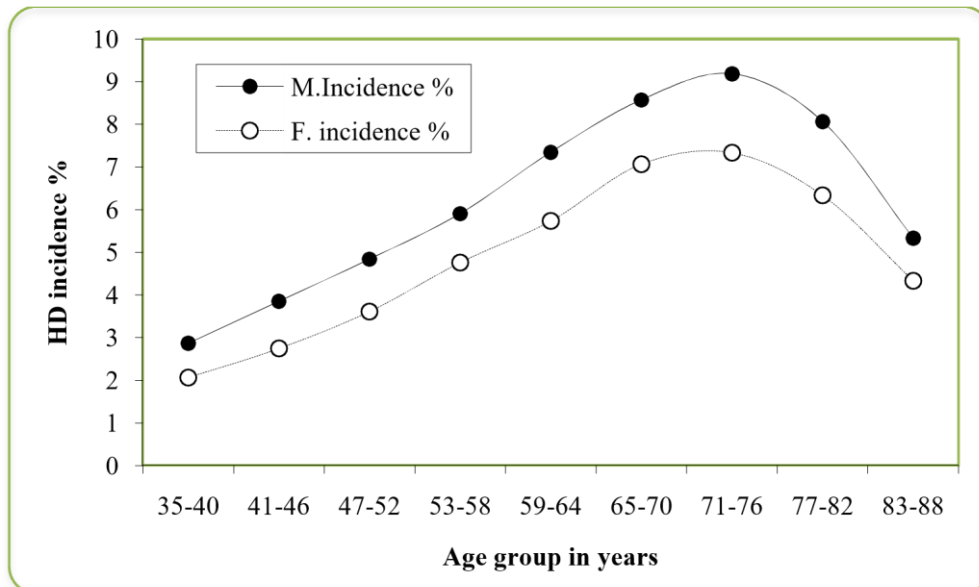Figure 4.1: shows the incidence percent of Heart Diseases (CHF) based on gender in Sudan 2015.

Figure 4.2: shows the Heart Diseases (CHF) distribution in Sudan based on age group during June 2014 – June 2015.
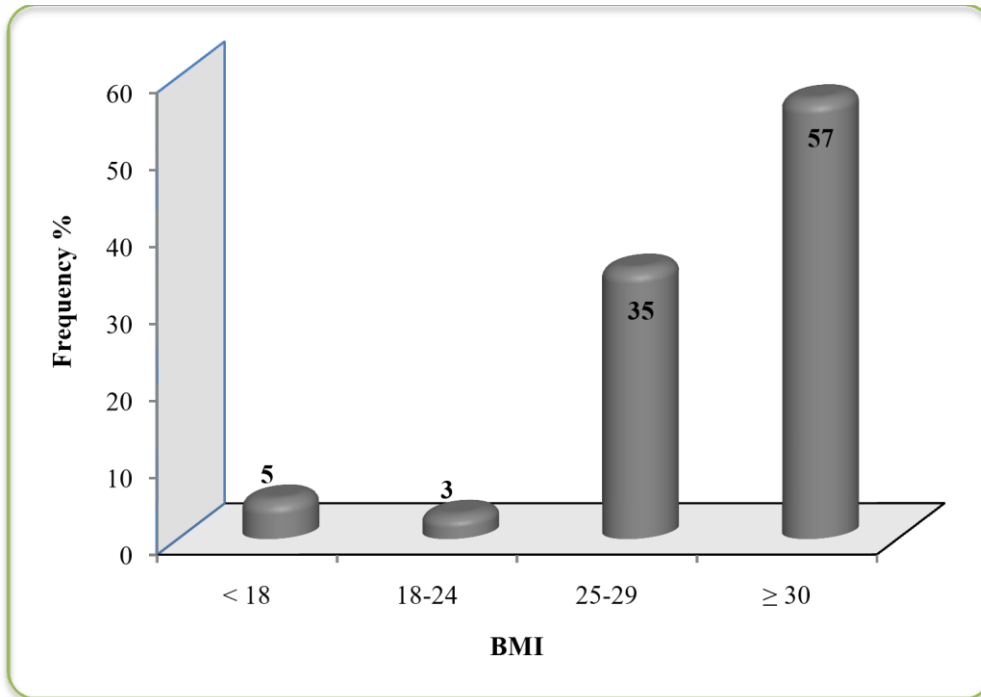
Figure 4.3: shows the body mass index BMI and the relevant frequency% among population during June 2014 – June 2015.
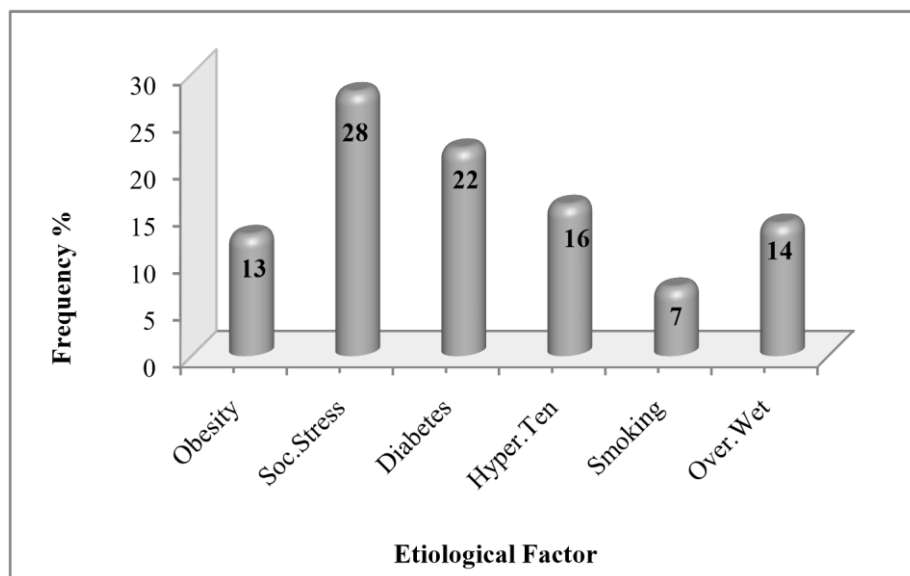
Figure 4.4: shows the common cofactor for cardiovascular diseases among Sudanese population during June 2014-June 2015.
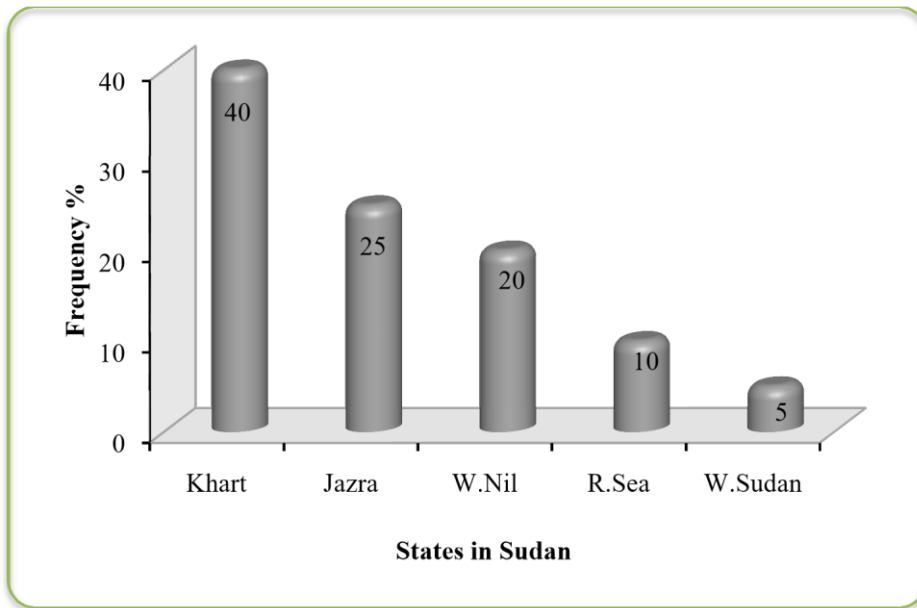
Figure 4.5 shows the incidence percent of Heart Diseases (CHF) in the states of Sudan during June 2015 up to June 2015.
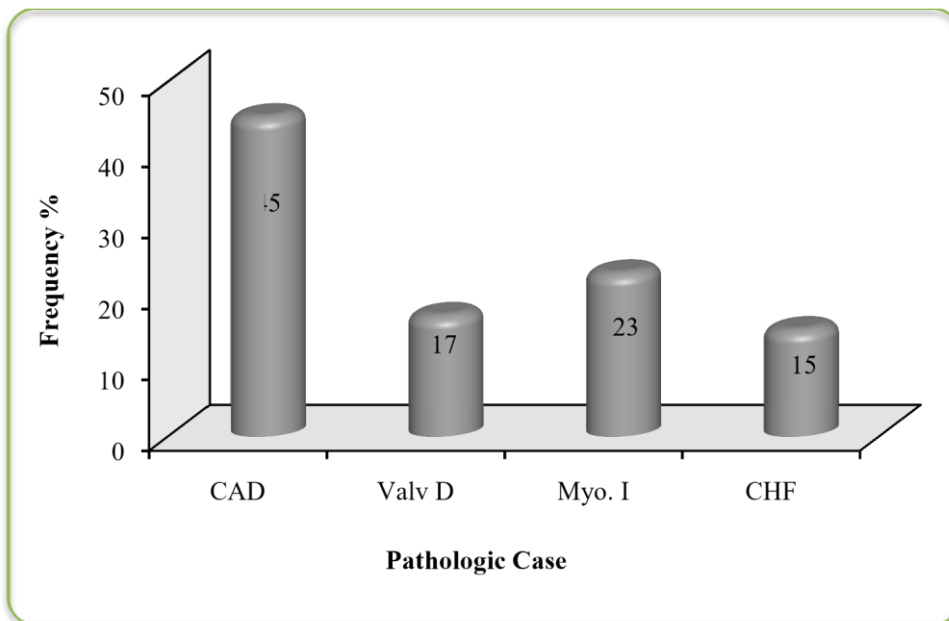

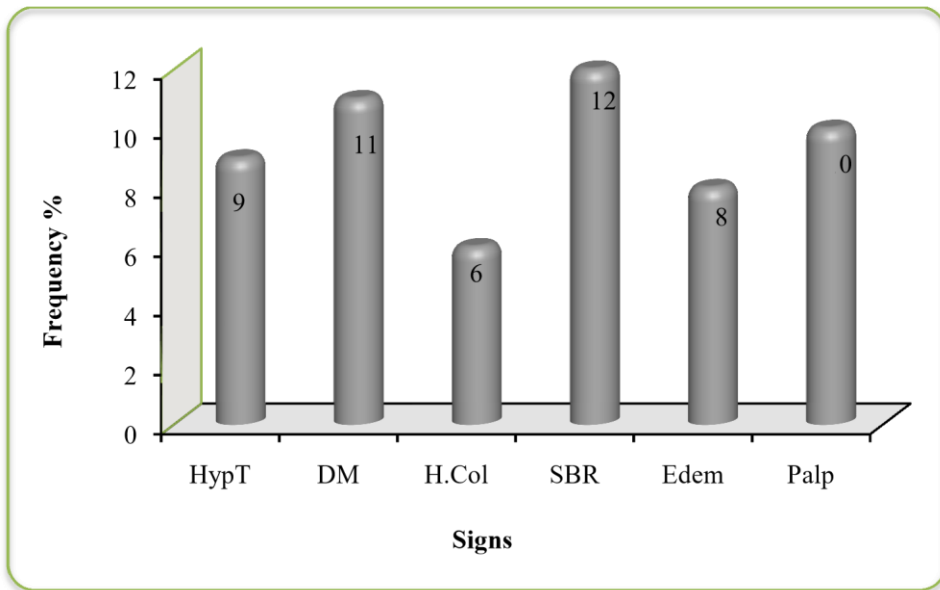Figure 4.6: shows the common types of Heart Diseases in Sudan during June 2015 up to June 2015.

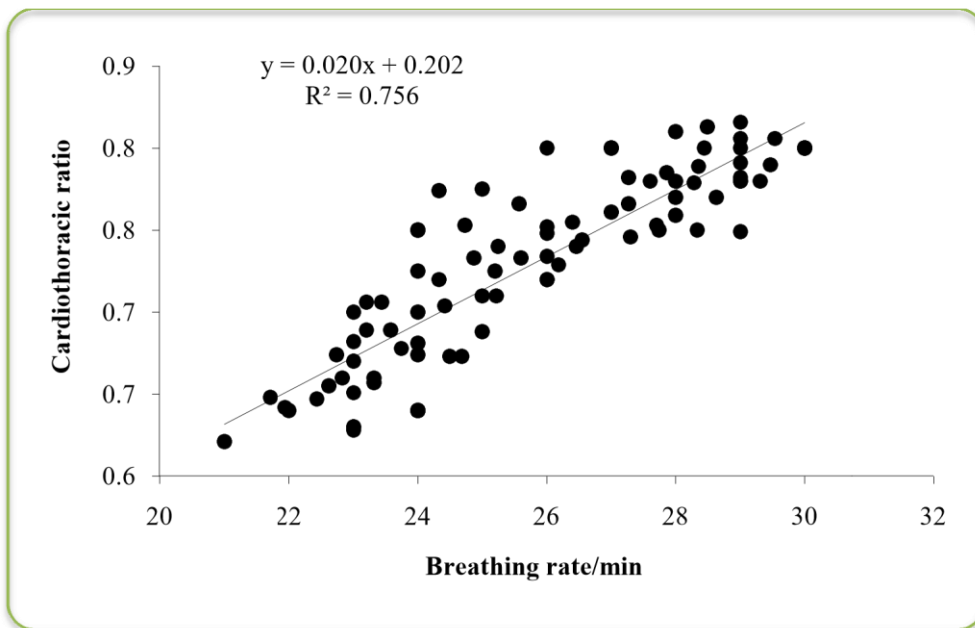Figure 4.7 shows the common symptoms of patient's Heart Diseases in Sudan during June 2014 – June 2015.

Figure 4.8: shows the correlation between the cardiothoracic ratio and the breathing rate
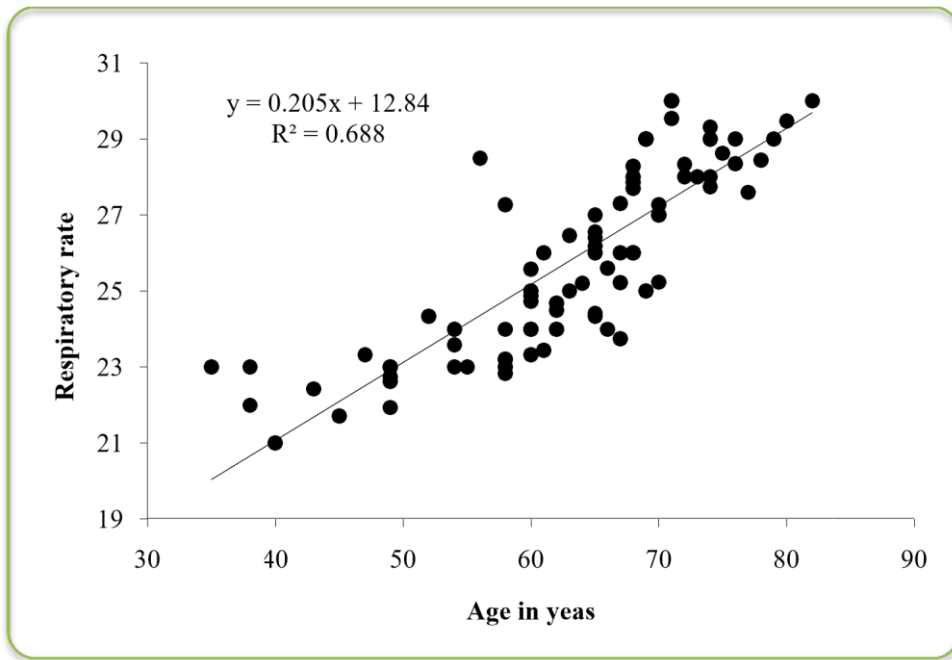
Figure 4.9 shows the correlation between the respiratory rate/min and the age in years
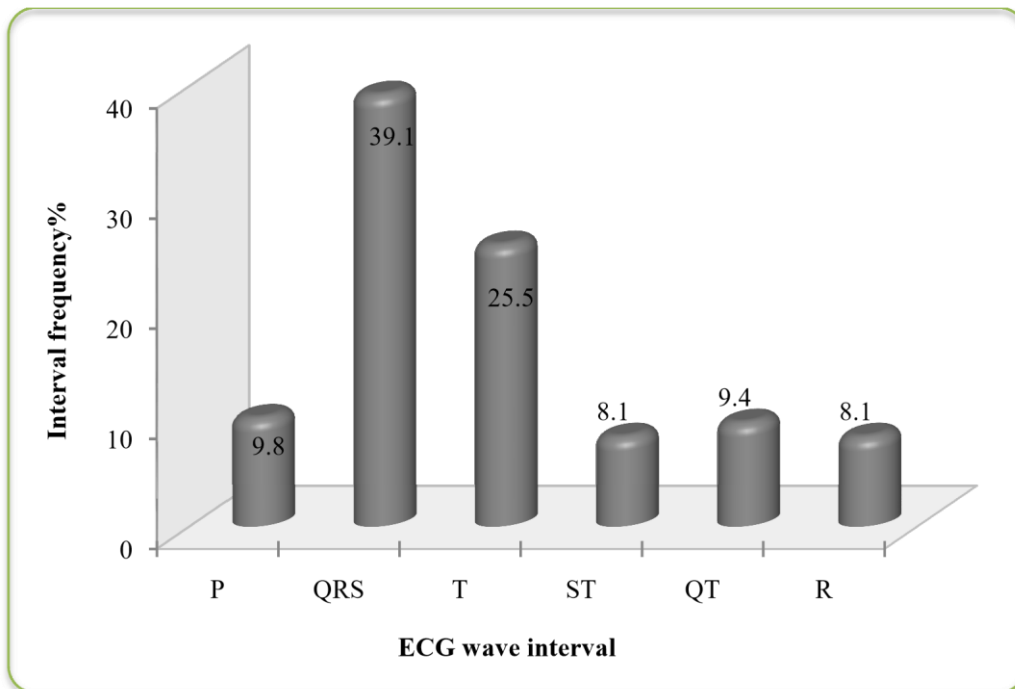

Figure 4.10 shows the deviated wave of ECG for CHF patients distributed based on frequency%
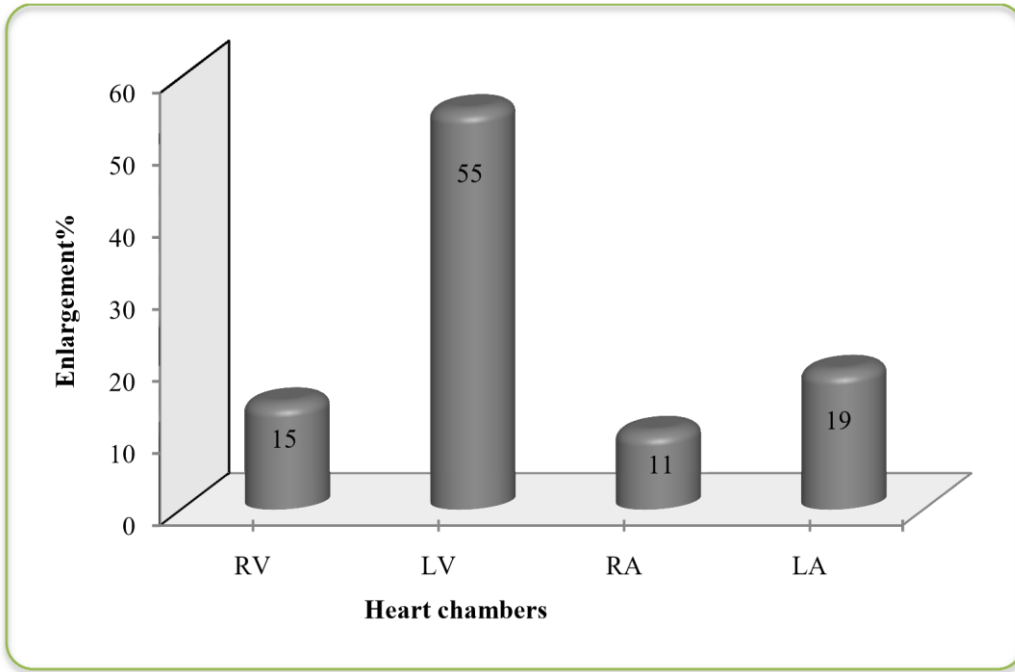
Figure 4.11: shows the heart chambers enlargement frequency % for CHF patients

Figure 4.12: shows the stages of CHF distributed based on frequency%

The chart shows a scatter plot with:
- Y-axis: The Peak of R wave (mm), ranging from 0 to 40
- X-axis: Cardiothoracic Ratio (cm), ranging from 0 to 1
- Trend line equation: $y = 29.724x + 9.9295$
- $R^2 = 0.8809$
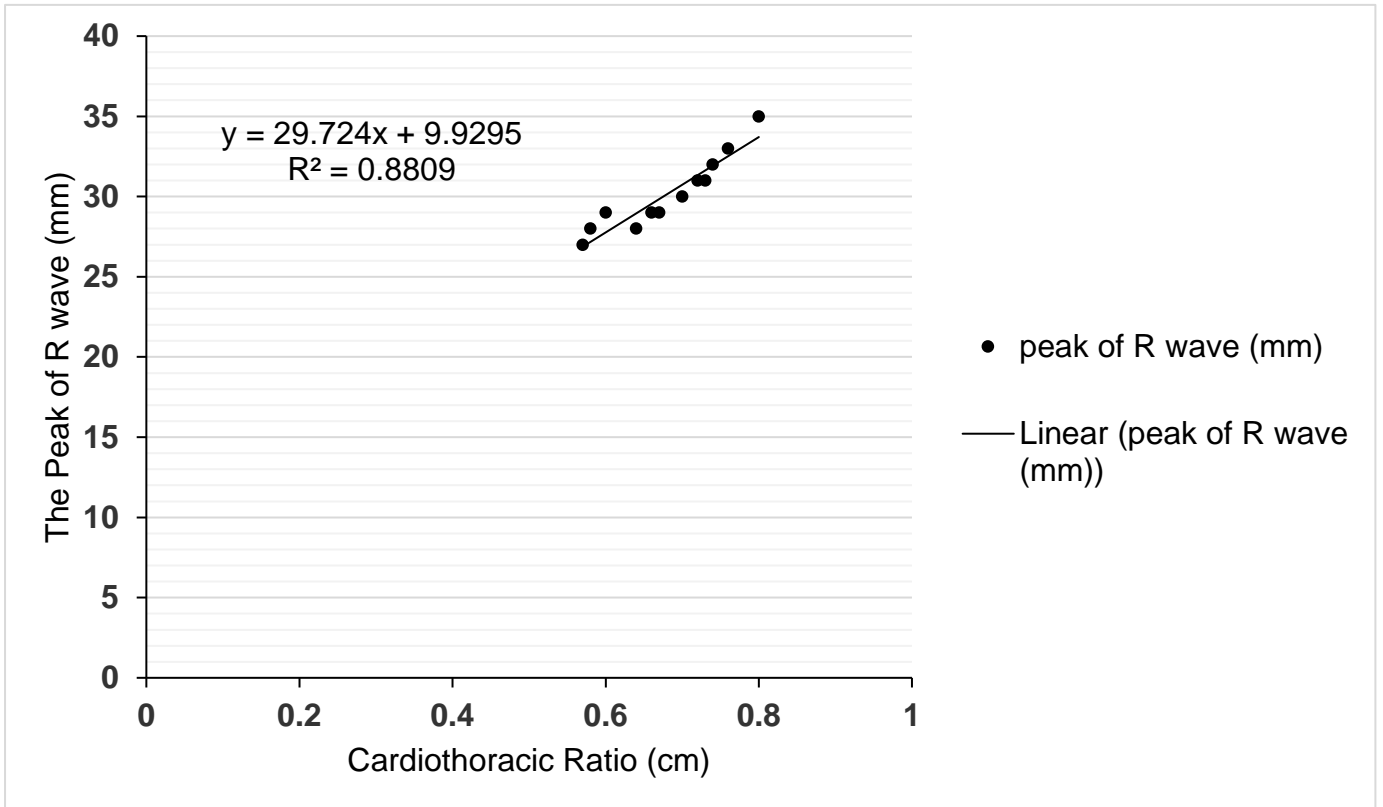- Legend: peak of R wave (mm); Linear (peak of R wave (mm))

Figure 4-13: shows the correlation between cardiothoracic ratio and the beak of R wave.

Chapter Four-Section Two
Discussion & Analysis

Figure 4.1: shows the incidence percent of Heart Diseases (CHF) based on gender in Sudan 2015. The analysis reveals that: the CHF is predominant among male with a percent of 56 relative to 44% among female. In comparison with the study carried out by Jackson et al, [22], which is agreed that the heart diseases is more common among male, such high incidence of heart diseases among male could be ascribed to different factors such as: social stress, bare economic status relative to loss or missing of the basic life needs among Sudanese male communities, and poverty; in addition to other general expected factors such as utilization of chemical fertilization in farms, fast food consumption and personal habits, other induction factor for heart disease is the obesity and over weight as stated by Adil et al, [23] in which they stated that: overweight and obesity is associated with the morbidity and mortality of many health conditions, such as coronary heart diseases CHD, Type 2 diabetes, gall bladder disease, ischaemic stroke, osteoporosis, sleep apnoea and some types of cancers; hence such facts ascertaining the high incidence of HD among Sudanese population as well as the increment of incidence in future.

Figure 4.2: shows the Heart Diseases (CHF) distribution in Sudan based on age group. The analysis reveals that: the CHF have been increased following the age increment and the high incidence of heart disease have occurred among age group of 65-77 years old in both gender, however the incidence of heart diseases obviously appeared with higher incidence percent along the entire age groups among male; which could be ascribed to exposure to many induction factors; as the age itself in addition to other factors mentioned in introduction and as well Mai, [24] highlighted some early signs or precursors of CVD in youngsters interact, such as teasing behavioral (diet, exercise, sleep, smoking, screen time), environmental (neighborhood, access to parks, crowding, toxin exposure), and psychological (stress, coping, social support), however the common associated factors among the study sample were the obesity, smoking and social stress.

Figure 4.3 shows the body mass index BMI and the relevant frequency% in the selected sample of the study, in which the majority of the sample were either obese representing 57% or over weight representing 35%, based on the obesity conventional

definition as having a body mass index (BMI) of 25 kg/m^2 and 30 kg/m^2, respectively [25]. In view of correlating such fact with CVD, Bahaaedin et al, [26] showed that: there is significant relationship between left ventricle ejection fraction and BMI (P < 0.001), as well Hubert et al, [27]; after 26 years of follow-up they concluded that obesity, was a significant independent predictor of CVD, including CHD, coronary death and congestive heart failure in both men and women; and stroke in women after adjustment for risk factors. Same study done by Wallis et al, [28], in which they showed that: CVD risk (including angina, myocardial infarction, CHD or stroke) was higher among overweight men (*Relative risk and confidence interval CI*) (RR 1.24; 95% CI: 1.07–1.44), and obese men (RR 1.38; 95% CI: 1.12–1.69) and obese women (RR 1.38; 95% CI: 1.14–1.68) after adjustment for age, smoking, high blood pressure, high cholesterol and diabetes. Therefore such results strengthen the probability of CVD among the Sudanese population. And the best idea to prevent CVD and achieve an optimal health is by keeping the BMI in the median range 21–23 kg/m^2 for adult populations, while the goal for individuals have to maintain a BMI in the range 18.5–24.9 kg/m^2 [29]. Also this finding is agreed with Flegal et al, [30] in which they stated that: overweight and obesity are in increasing metabolic disorder in both gender and were representing 56% to 65% and 23% to 31% respectively. Such obtained fact is agreed with some studies that confirm the obesities have association with many disorders such as: (*Obesity is associated with numerous comorbidities, such as dyslipidemia, hypertension (HTN), reduced insulin sensitivity, diabetes mellitus, left ventricular (LV) hypertrophy, certain cancers, and sleep apnea/sleep-disordered breathing*) [31]. As well Obesity is considered as an independent risk factor for cardiovascular disease (CVD), including HTN, coronary heart disease (CHD), and heart failure (HF) and is associated with an increased risk of morbidity and mortality [32]. The effect of obesity on the heart morphology and physiology could be ascribed to volume and pressure overload on the heart [33].

Figure 4.4 shows the common causatives factors for cardiovascular diseases in Sudan during June 15, 2014-June 15, 2015. The analysis reveals that: the most common cofactors for induction of CHF were the social stress, diabetes, hypertension and overweight which representing 28%, 22%, 16% and 14% respectively, with less impact of obesity and smoking which representing

13% and 7% respectively. Such facts have been agreed with the study findings given by Mendis et al [29] in which they stated that: the diabetes (*which is defined as having a fasting plasma glucose value of 7.0 mmol/l (126 mg/dl) or higher*) increases the events of cardiovascular diseases by two to three folds relative to normal people, hypertension is a leading cause of CVD, smoking inducing the CVD by 10% relative to all CVD and the increment of blood cholesterol level by even 1% can put someone at a 2% higher risk of heart disease [34].

Figure 4.5 shows the incidence percent of Heart Diseases in the states of Sudan during June 2015 up to June 2015. Among these states; Khartoum state was the most endemic with CHF showing a percent of 40% of the total sample, Aljazeera state showed 25%, White Nile showed 20%, Red Sea showed 10% and West of Sudan showed 5%. The high incidence in Khartoum and Aljazeera states could be ascribed to high population, life style changes and stress, feeling of hopeless, low economic status and poverty, in addition to the confirmed factors such as obesity and overweight, however the incidence of HD also being increasing among the youth recently in Sudan.

Figure 4.6 shows the common types of Heart Diseases in Sudan during June 2015 up to June 2015. It is so obvious that: the common type of heart diseases was the coronary arteries diseases representing 45% of the total sample, Valves diseases represent 17%, Myocardial infarctions represent 23% and congestive heart failure was 15%. The high incidence of CAD could be due to susceptibility of coronary arteries to many pathogenic factors, for instance: progressive narrowing due to atherosclerosis, high blood pressure, high cholesterol and/or triglycerides in blood, diabetes, smoking, excessive weight, and lack of a regular exercise program [35].

Figure 4.7 shows the common signs of patient's Heart Diseases in Sudan during June 2014 – June 2015, which were Hypertension, Diabetes mellitus, High Cholesterol, breathing rate, edema, palpitation (*sudden pounding, fluttering, or racing feeling in the heart*) with relative frequencies 9%, 11%, 6%, 12%, 8% and 10% respectively. Such relative signs have been mentioned by WHO, [36] and the high incidence of short breathing rate, diabetes mellitus and palpitation could be ascribed to compensating breathing mechanism for having sufficient oxygen and for metabolic syndrome e.g. uric acid metabolic [37].

Figure 4.8 shows the correlation between the cardiothoracic ratio (CTR) and the breathing rate (BR), in which there is obvious significant relationship ($R^2 = 0.8$) that: as the cardiothoracic ration increases; the breathing rate/min increases and the correlation could be fitted in the following equation: y = 0.020x + 0.202, where *y* refers to cardiothoracic ratio and *x* refers to breathing rate/min. the analysis revealed that all the patients have CTR exceeding the normal range (0.5) which in turn has direct impact on the breathing rate increment beyond the normal rang 12-20 bpm [38, 39]. And as has been mentioned by George et al, [40] that Abnormal patterns of breathing are frequently caused by injury to respiratory centers in pons and medulla, use of narcotic medications, metabolic derangements, and respiratory muscle weakness; here the factors of metabolic and respiratory muscles weakness are playing major rules.

Figure 4.9 shows the correlation between the respiratory rate/min and the age in years. It reveals that: there is increasing proportional relationship between the age and respiratory rate/min; such relation fits the following equation: y = 0.205x + 12.84, where y refers to respiratory rate/min and x refers to age in years which is significant as $R^2 = 0.7$. Although the normal respiratory rate are 12-20, 15-20, 18-25, 20-30, 2540 and 30-60 breath per minutes (bpm) for adults, 10 years old, 6 years old, 3 years old, 6 months old and from birth to 6 weeks respectively; the HD patients showed breathing rate greater than normal relative to average resting respiratory rates [38, 39]. Figure 4.10: shows the deviated wave of ECG for CHF patients distributed based on frequency%. The analysis reveals that: the common deviated ECG waves were QRS and T with frequencies of 39.1% and 25.5% respectively, with consideration that: the QRS wave represents the state of left ventricle (*depolarization of ventricles*) while T wave state the re-polarization of the ventricles, such result indicates that: the common heart enlargement occurs in the left ventricle and could be ascribed to excessive exercise imparted on, which is in turn assigned for certain pathology somewhere ells in the body like (*abnormal heartbeat (arrhythmia), High blood pressure, Heart valve disease, Disease of the heart muscle (cardiomyopathy), High blood pressure in the artery connecting your heart and lungs (pulmonary hypertension), Fluid around your heart (pericardial effusion) Fluid around your heart (pericardial effusion), Low red*

blood cell count (anemia), Thyroid disorders, and Excessive iron in the body (hemochromatosis)).

Figure 4.11: shows the heart chambers enlargement frequency% for CHF patients derived from chest x-ray. Obviously, it reveals that: the enlarged chamber in the heart was the left ventricle that representing 55% relative to other chambers as 19%, 15%, and 11% for the left atrium, right ventricle and right atrium respectively, which is corresponding with the ECG result. The presence of certain subtleties in the cardiac shape may point to a particular pathology and thus help narrow the differential diagnosis. Enlargement or hypoplasia of a particular component of the heart will alter the normal shape of the cardiac silhouette. Therefore, each aspect of the heart border should be examined to assess for abnormalities [41].

Figure 4.12: shows the stages of CHF distributed based on frequency%. As the classes of CHF are I, II, III, and IV with certain symptoms like: Paroxysmal Nocturnal Dyspnea (PND) (short breathing when laying supine), experience dyspnea with moderate exertion, difficult to carry out activities of daily life/respiratory distress, continual distress/un able to do normal activities respectively. The study analysis revealed that: the most common stage of CHF was stage III that showing 55% among the sample relative to stage I (2%), II (30%) and IV (13%). The high incidence of stage III could be ascribed to severe symptoms and signs that lead the patients to go for medication with special consideration that: Sudanese population have not the culture of going to medical doctor for mild symptoms, however in developed countries the common presented stage is stage (I), which is due to medical health caring, availability of medication, in expensive as mentioned by Khawaja et al, [42].

Figure 4.13: shows the correlation between the Cardiothothoracic ratio in the Chest x-ray and the peak of the R wave in the ECG of the same patient .reveals that when the cardiothoracic ratio increased the peak of R wave also increased.

Figure 4.13: show the correlation between cardiothoracic ratio (CTR) and peak of R wave , in which there is obvious significant relationship (R^2 =0.9) that :as the CTR increases the peak of R wave increases and the correlation could be fitted in the following equation y=29.724x+9.9295 where y refer to peak of R wave and x refer to CTR :the analysis revealed that all the patient have CTR exceeding the normal range (0.5) which in turns has direct impact on the peak of R wave increament.

Chapter Five
Conclusion and Recommendation

5.1 Conclusion:

Based on the analyzed data of this study and the obtained results, the conclusion could be summarized in the following paragraph:

CHF is predominant among male with a percent of 56 relative to 44% among female and as the age increases with high incidence occurred among age group of 65-77 years and the majority of the sample was either obese representing 57% or overweight representing 35%. The most common cofactors for induction of CHF were the social stress, diabetes, hypertension and overweight which representing 28%, 22%, 16% and 14% respectively, with less impact of obesity and smoking which representing 13% and 7% respectively. Within the states of Sudan, Khartoum was the most endemic with CHF showing a percent of 40%, Aljazeera showed 25%, White Nile showed 20%, Red Sea showed 10% and West of Sudan showed 5%. And the common type of heart diseases was the coronary arteries diseases representing 45% of the total sample, Valves diseases represent 17%, Myocardial infarctions represent 23% and congestive heart failure was 15% with the common signs of as: Hypertension, Diabetes mellitus, High Cholesterol, increased breathing rate, edema, palpitation with relative frequencies 9%, 11%, 6%, 12%, 8% and 10% respectively. The cardiothoracic ration increases and hence increase the breathing rate/min. and the correlation could be fitted in the following equation: $y = 0.020x + 0.202$, where y refers to cardiothoracic ratio and x refers to breathing rate/min. The cardiothoracic ration increases and hence increase the peak of the R wave. And the correlation could be fitted in the following equation : $y=29.724x+9.9295$.where y refers to peak of R wave and x refer to the cardiothoracic ratio and all the patients have CTR exceeding the normal range (0.5) which in turn has direct impact on the breathing rate increment beyond the normal rang 12-20 bpm. Also there is increasing proportional relationship between the age and respiratory rate/min; such relation fits the following equation: $y = 0.205x + 12.84$, where y refers to respiratory rate/min and x refers to age in years which is significant as ($R^2 = 0.7$), with relative common deviated ECG waves as: QRS and T with frequencies of 39.1% and 25.5% respectively and the enlarged chamber in the heart was the left ventricle that representing 55%, and 19%, 15%, and 11% for the left atrium, right ventricle and right atrium respectively, with the most common stage of

CHF was stage III that showing 55% among the sample relative to stage I (2%), II (30%) and IV (13%).

5.2 Recommendation:

After successful achievement of the current research objectives; the worth to be recommended could be highlighted in the following points:

- Employing of social specialists in and should have influence in decision made by constitutional court in the country.

- Dispense the culture of low fat and cholesterol diets and recommend of sports and exercise.

- Encourage more researches in such related realm i.e. heart diseases and epidemiological factors.

- Initiate the public screening program to assess and detect the early heart diseases stimulants.

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Appendices
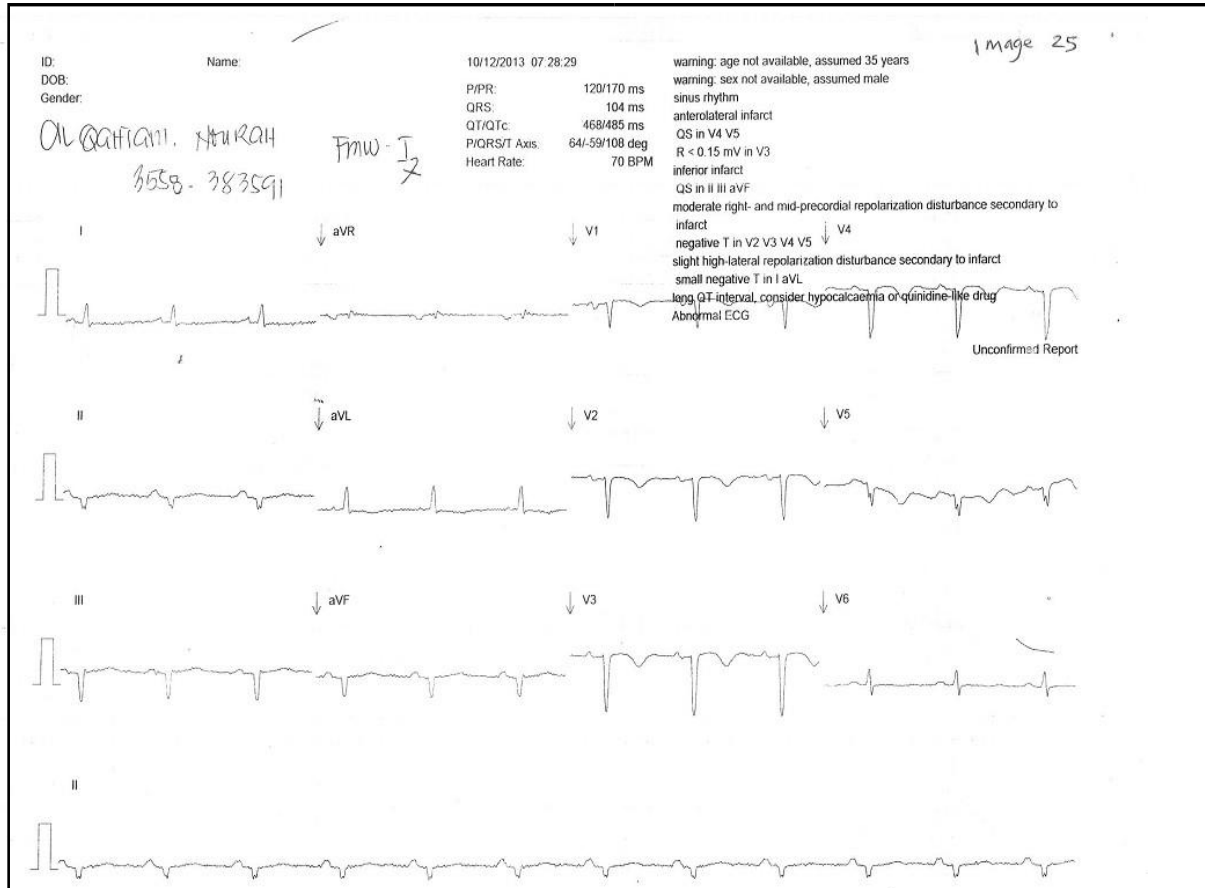
Vent. rate	79 bpm	Normal sinus rhythm
PR interval	146 ms	Rightward axis
QRS duration	96 ms	Anterior infarct, age undetermined
QT/QTc	446/511 ms	T wave abnormality, consider lateral ischemia
P-R-T axes	64 105 122	Prolonged QT
		Abnormal ECG

Technician:
Test ind:

Referred by: Unconfirmed

I   aVR   V1   V4

II   aVL   V2   V5

III   aVF   V3   V6

V1

II

V5
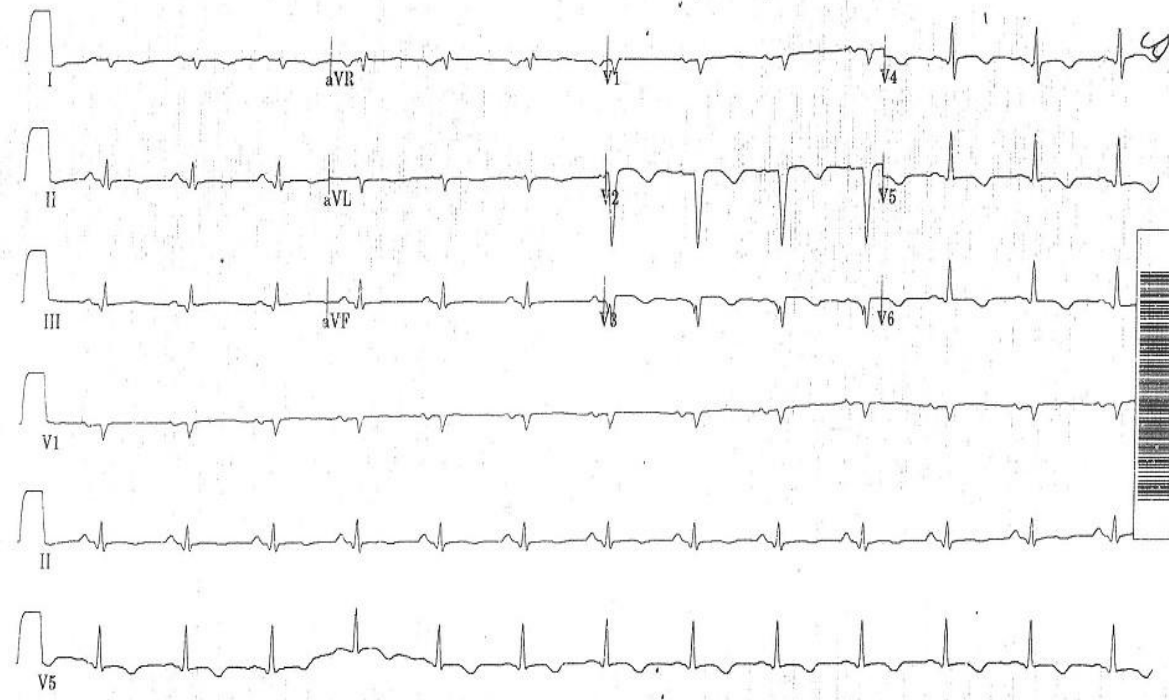
20 Hz   25.0 mm/s   10.0 mm/mV   4 by 2.5s + 3 rhythm lds   MAC55 009A   12SL™ v237

ECG shows :
Normal sinus rhythm
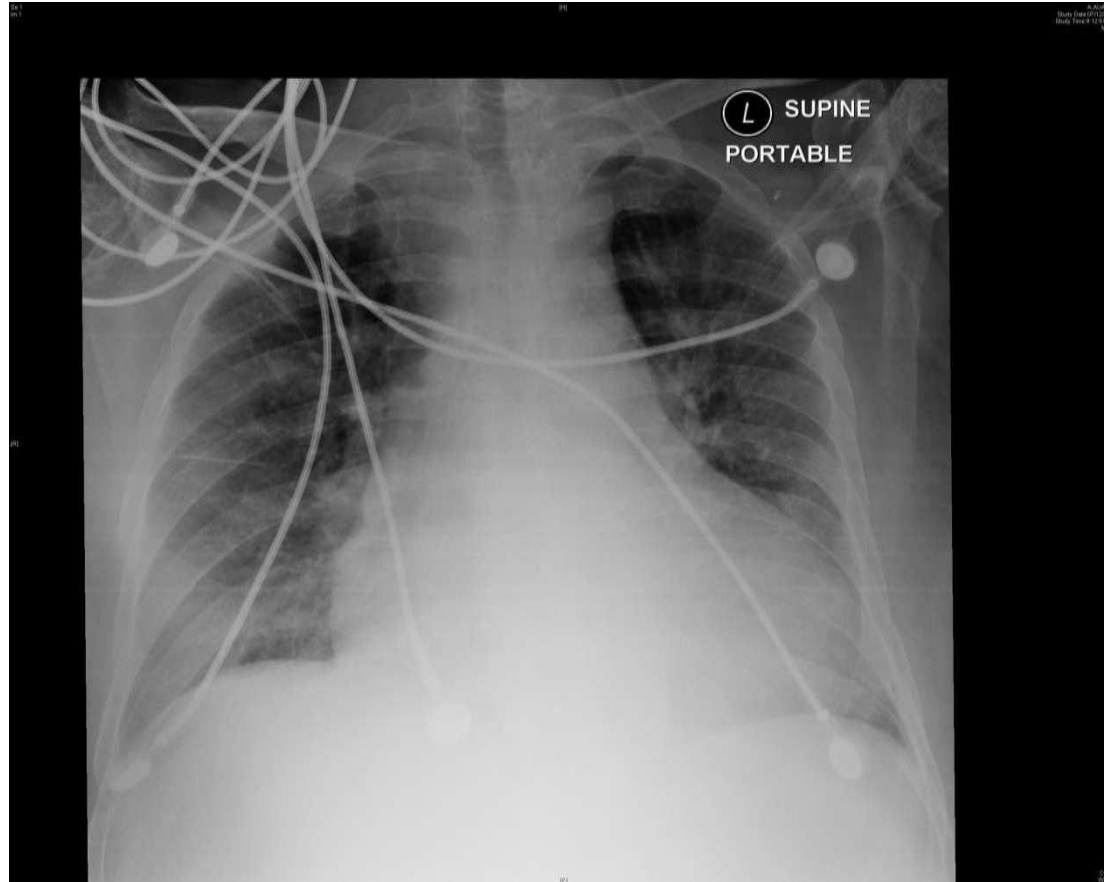T wave negative (inverted) in V2 V3 V4 V5 indicate infarction .
Long QT interval indicate hypocalcaemia .
Normal duration of QRS complex .
The QRS is upward in lead I and downward in II and III indicate left axis deviation (LAD), consider upward deviation .
Chest x-ray shows :
Cardiomegaly (Cardiothoracic ratio (CTR) = 0.7 cm .

ECG shows :
Normal sinus rhythm.
RT axis deviation (the QRS in lead I down ward).
Inverted T wave indicate ischemia .
Chest x-ray shows : Cardiomegaly , CTR=0.7 cm Image

48
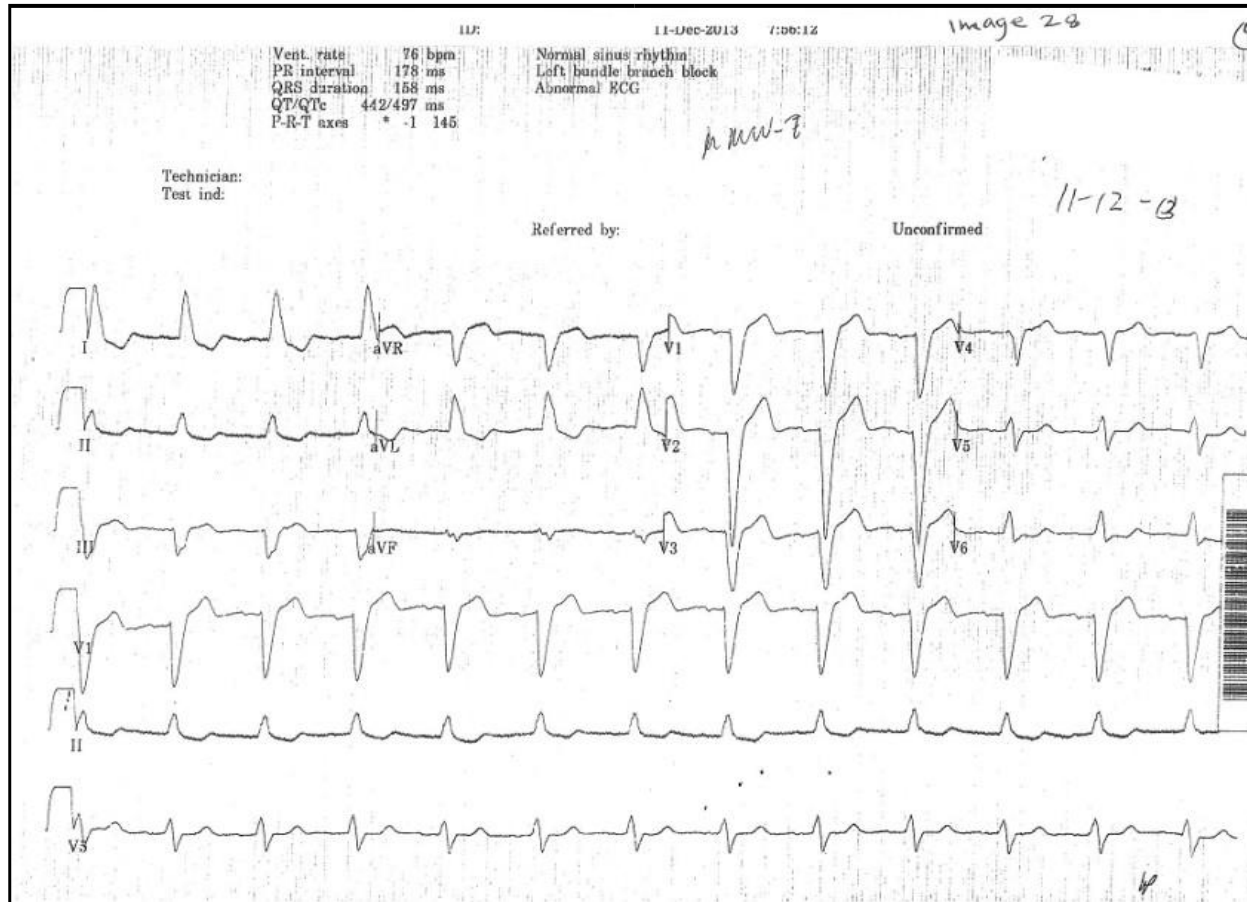
ECG shows :
Normal sinus rhythm .
Widened QRS duration consider LBBB .
LAD considered .
Chest x-ray shows :
Cardiomegaly. CTR=0.6 CM

Image 33   3/9/2013   8:10:00 PM

Rate 105   . Age not entered, assumed to be 50 years old for purpose of ECG interpretation
             . Sinus tachycardia...rate> 99
PR   175   . Left atrial enlargement.................................P, P'>60mS, <-0.15mV V1
QRSD   86   . Borderline T abnormalities, anterior leads.................T flat or neg, V2-V4
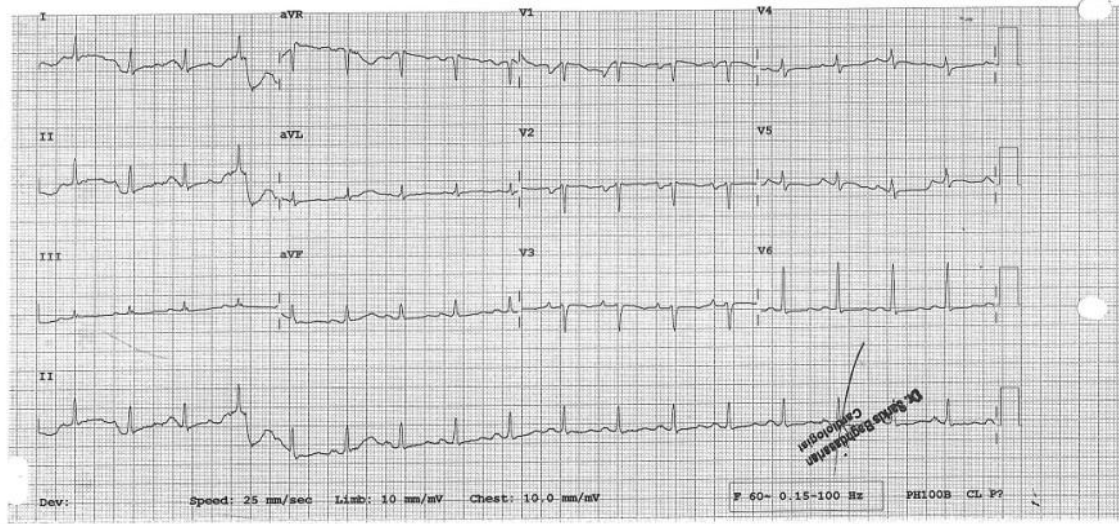QT   403   . Prolonged QT interval..QTc >500mS
QTc   533

--AXIS--
P   35
QRS   35   . - ABNORMAL ECG -
T   28

Name: QAETHAH ALI YEHYA A
Gender: FEMALE
DOB: 08/03/1983
Nationality: Saudi
Medical File No. 1433204687

Unconfirmed Diagnosis

27/4/34 ⌀8.05

I   aVR   V1   V4

II   aVL   V2   V5

III   aVF   V3   V6

II

Dev: Speed: 25 mm/sec   Limb: 10 mm/mV   Chest: 10.0 mm/mV   F 60~ 0.15-100 Hz   PH100B CL F?

50
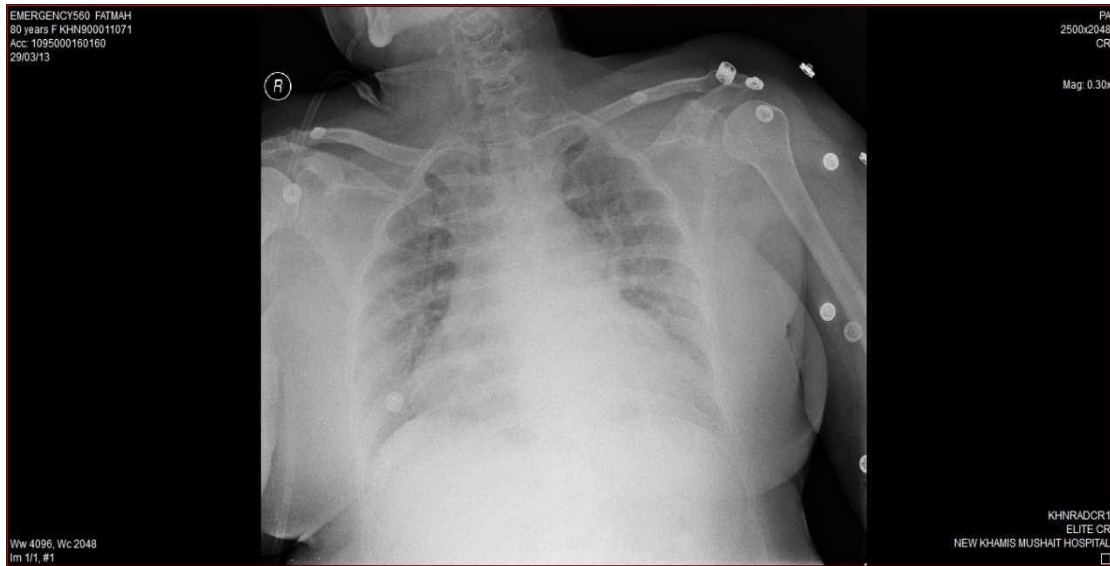
ECG shows:
Sinus tachycaredia.
Normal QRS duration.
Normal cardiac axis .
Inverted T wave in V4 indicated ischemia .
P wave is broad in lead I,II and III consider left a trial enlargement .
Chest x-ray shows :
Cardiomegaly , CTR =0.7 cm