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Assessmentof Heart in Diabetic Patients using Echocardiography

تخطيط القلب لمرضى السكري باستخدام تخطيط صدى القلب

Athesis submitted for partial fulfillment of M.Sc. degree in medical diagnostic ultrasound

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الآية

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قال تعالي:

اقْرَأْ بِاسْمِ رَبِّكَ الَّذِي خَلَقَ (1) خَلَقَ الْإِنْسَانَ مِنْ عَلَقٍ (2) اقْرَأْ وَرَبُّكَ الْأَكْرَمُ (3) الَّذِي عَلَّمَ بِالْقَلَمِ (4) عَلَّمَ الْإِنْسَانَ مَا لَمْ يَعْلَمْ (5)

صدق الله العظيم

سورة العلق الآيات (1-5)

Dedication

To;

My parents...

The soul of my late brother…

My husband & kids…

And my brothers…

Acknowledgment

First of all, I thank Allah the Almighty for helping me complete this project. I thank Dr. Mohamed Elfadil Mohamed my supervisor, for his help and guidance.

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I am greatly indebted to my parents, my husband and my daughter Duaa and my son Khaled and my brothers for bearing with me during the past several months.

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Finally I would like to thank everybody who helped me prepare and finish this study.

Abstract

Heart can be affected by many morbidities regarding the diabetic status, this study was aimed to assess the impact of diabetes on the heart by echocardiographyin order to detect the major complications of the heart early.This study was concerned with study and assessment of heart changes in diabetic patient using echocardiography in order to characterize these lesions or changes prior to the development of symptoms. So this can lead to early clinical detection as well as providence of clinical base for these changes for future scope.A total of 50 diabetic patients were studied by using echocardiography this study has been done in the echo departments in National Ribat University Hospital and the Military Hospital – Sudan in the period from Aug – Sep 2015, aged between 25-85 years old. Echocardiographic machines with motion mode(M –mode) and Doppler capabilities was used. The probe is of a convex, or better to be a sector type. The transducer is a phased - array 3.5-7.5 MHZ, ultrasound gel is applied to the transducer to prevent any attenuation or artifacts. A questionnaire is used to collect the data and to number the patients.The main study results were, 16% were diagnosed with normal results and 84% with significant morbidities, the percentage of diabetes in females is higher than males, the most affected age group is 60-69 years old, the highest morbid result found is the ischemic heart disease with diastolic dysfunction 20% of the total research population, the mean ± SD for the LV EF% was 61.26±11.634, LV EF% correlated inversely with LVESD and Lt Atrium Diameter; whereas correlated proportionally with LVPW, cardiac valves and blood flow mostly were normal. We concluded from this study that the use of the echocardiography in characterizing these lesions or changes prior to the development of symptomatic congestive heart failure sub-clinical left ventricular dysfunction (systolic or diastolic) exists for sometimes. So this can lead to early clinical detection as well as providence of clinical base for these changes for future scope.

المستخلص

يمكن أن يتأثر القلببالعديد من الحالات المرضية فيما يتعلق بمرض السكري، تهدف هذه الدراسة إلى تقييم تأثير مرض السكري على القلب عن طريق السونار من أجل الكشف عن التعقيدات الكبيرة في القلب في وقت مبكر. وتهتم هذه الدراسة بمعرفة وتقييم التغيرات في القلب في مريض السكري باستخدام تخطيطسونار القلب من أجل تميز هذه التغييرات الأولية في تطور الأعراض. ولذلك فإن هذا يمكن أن يؤدي إلى الكشف السريري المبكر وكذلك العناية الكاملة مستقبلا. تمت دراسة مجموعه 50 مريض باستخدام تخطيطسونار القلب و تم إجراء هذه الدراسة في مستشفى جامعة الرباط الوطني والمستشفى العسكري - السودان في الفترة من أغسطس.سبتمبر. 2015، تتراوح أعمارهم بين 25-85 عاما. تم استخدام آلات تخطيطسونار القلب مع وضع الحركة (M -mode) وقدرات دوبلر. وعلى مراحل محول - مجموعة 3،5-7،5MHZ ، يتم تطبيق هلام الموجات فوق الصوتية لمحول لمنع أي تخفيف أو الأعمال الفنية. يتم استخدام الاستبيان لجمع البيانات وعددهم المرضى. وقد تم تشخيص النتائج الرئيسية للدراسة 16٪ وكانت النتائج طبيعية و 84٪ مع مرضية هامة، نسبة السكري في الإناث أعلى من الذكور، والفئة العمرية الأكثر تضررا هي 60-69 سنة، وهي أعلى نتيجة مرضية وجدت هي مرض نقص التروية القلبية مع اختلال وظيفي الانبساطي 20٪ من مجموع السكان البحوث، المتوسط ± الانحراف المعياريلنسبة معدل ضخ البطين الأيسر وكان 61.26 ± 11.634،نسبة معدل ضخ البطين الأيسر يرتبط عكسيا مع LVESD والملازم اتريوم القطر. في حين ترتبط بشكل متناسب معLVPW ، كانت الصمامات القلبية وتدفق الدم الطبيعي في الغالب. خلصنا من هذه الدراسة أن استخدام تخطيطسونار القلب في تشخيص هذه التغييرات قبل وضع من أعراض قصور القلب الاحتقاني دون السريرية اختلال البطين الأيسر (الانقباضي أو الانبساطي) موجود لفي بعض الأحيان. ولذلك فإن هذا يمكن أن يؤدي إلى الكشف السريري المبكر من قاعدة السريرية لهذه التغييرات عن نطاق المستقبل.

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Chapter one

1.1 Introduction

The heart is the most important vital organ in the body located within the chest in the left side posterior to the sternum in the middle mediastinum, relatively small conical approximately the size of a person's clenched fist (Sinnatamby, 2004). It weighs about 250 to 350 grams, rotated such that its right side or border (Right atrium and ventricle) located more anteriorly, while its left side or border (Left atrium and ventricle) is located more posteriorly. It pumps the deoxygenated blood to the lungs through the pulmonary trunk via the pulmonary valve to be oxygenated. This right ventricular systole is done by the myocardium including the inter-ventricular septum. On the other hand the left atrial filling (diastole) is with oxygenated blood which is send by both passive and active flow to the left ventricle via the mitral valve {atrial systole},however, this is also the left ventricular diastole . Its systole is the ejection of blood to the aorta via the aortic valve by the strong and large myocardium of this chamber. This cardiac cycle is controlled by the conduction system of the heart and the autonomic nervous system (Sweenand Wholly, 1998).

These alternate cycles of the wall contraction and relaxation develop the so called blood pressure. Minimum blood pressure is essential to push the blood through the blood vessels to the body tissues for nutrient supply and waste exchange.Heart disease is a complication that may affect people with diabetes if their condition develops.Coronary heart disease is recognized to be the cause of death for 80% of people with diabetes; however, the NHS states that heart attacks are largely preventable.(NHS 2009).

Diabetes mellitus (DM) also known as simply diabetes, is a group of metabolic diseases in which there are high blood sugar levels over a prolonged period.(WHO2014).

This high blood sugar produces the symptoms of frequent urination, increased thirst, and increased hunger. Untreated, diabetes can cause many complications. (WHO2014). Acute complications include diabetic ketoacidosis and non-ketotic hyperosmolar coma. (Kitabchi et al 2009).

Serious long-term complications include heart disease, stroke, kidney failure, foot ulcers and damage to the eyes.Diabetes is due to either the pancreas not producing enough insulin, or the cells of the body not responding properly to the insulin produced.[5] There are three main types of diabetes mellitus:Type 1 DM results from the

body's failure to produce enough insulin. This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes". The cause is unknown, Type 2 DM begins with insulin resistance, a condition in which cells fail to respond to insulin properly.[3] As the disease progresses a lack of insulin may also develop.[6] This form was previously referred to as "non-insulin-dependent diabetes mellitus" (NIDDM) or "adult-onset diabetes". The primary cause is excessive body weight and not enough exercise and Gestational diabetes, is the third main form and occurs when pregnant women without a previous history of diabetes develop a high blood glucose level.(WHO 2014).

Echocardiogram, often referred to as a cardiac echo or simply an echo, is a sonogram of the heart. (It is not abbreviated as ECG, which in medicine usually refers to an electrocardiogram.) Echocardiography uses standard two-dimensional, three-dimensional, and Doppler ultrasound to create images of the heart.Echocardiography has become routinely used in the diagnosis, management, and follow-up of patients with any suspected or known heart diseases. It is one of the most widely used diagnostic tests in cardiology. It can provide a wealth of helpful information, including the size and shape of the heart (internal chamber size quantification), pumping capacity, and the location and extent of any tissue damage. An Echocardiogram can also give physicians other estimates of heart function such as a calculation of the cardiac output, ejection fraction, and diastolic function (how well the heart relaxes).Echocardiography can help detect cardiomyopathies, such as hypertrophic cardiomyopathy, dilated cardiomyopathy, and many others.The biggest advantage to echocardiography is that it is non-invasive and has no known risks or side effects.Not only can an echocardiogram create ultrasound images of heart structures, but it can also produce accurate assessment of the blood flowing through the heart, using pulsed or continuous wave Doppler ultrasound. This allows assessment of both normal and abnormal blood flow through the heart. Colour Doppler as well as spectral Doppler is used to visualize any abnormal communications between the left and right side of the heart, any leaking of blood through the valves (valvular regurgitation), and to estimate how well the valves open (or do not open in the case of valvular stenosis).Echocardiography was also the first ultrasound subspecialty to use intravenous contrast.

1.2. The problem of the study

Myocardial involvement in diabetics may occur relatively early in the course of the disease, initially impairing early diastolic relaxation and when more extensive, it causes decreased myocardial contraction. Prior to the development of symptomatic congestive heart failure sub-clinical left ventricular dysfunction (systolic or diastolic) exists for sometimes. However, frequency of progression from pre-clinical to clinically evident myocardial dysfunction is not established. Therefore the introduction of cardiac imaging using ultrasound may give better advantage in early detection of cardiac morbidity.

1.3. Thestudy objectives:

1.3.1. The general objectives

The general aim of this study was to assess the impact of diabetes on the heart by echocardiographyin order to detect the major complications of the heart early.

1.3.2. The specific objectives

- To assess the left ventricular ejection fraction in diabetic patients.
- To measure the left ventricular wall thickness and ventricular diameter in both systolic and diastolic functions.
- To identify the hypertrophy in diabetic patient heart.
- To assess and characterize the valvular lesions that result from diabetes
- To identify the motion activity of the myocardium and the diastolic as well as systolic dysfunction if ever in diabetes.
- To assess the blood flow in diabetes using Doppler capability
- To assess the impact of age, sex, body mass index, duration of the diabetes, the medication on the echocardiogram results.

1.4. Significance of the study:

This study was concerned with study and assessment of heart changes in diabetic patient using echocardiography in order to characterize these lesions or changes Prior to the development of symptomatic congestive heart failure sub-clinical left ventricular dysfunction (systolic or diastolic) exists for sometimes. So this can lead to early clinical detection as well as providence of clinical base for these changes for future scope.

1.5. Overview of the study:

This study was consist of five chapters, chapter one was an introduction introduce briefly this thesis and contained; general introduction, general and specific objectives, significant of the study and overview of the study. Chapter two was literature review about general theoretical background which contains anatomy, physiology and pathology, in addition to the previous study. Chapter three was describe the methodology (material, method) used in this study. Chapter four was included result of presentation of final finding of study; chapter five included discussion, conclusion and recommendation for future scope in addition to references and appendices.

Chapter Two

2.1. Anatomy

The heart is a muscular pump that ejects blood into the vascular tree with sufficient pressure to maintain optimal circulation. Average weight of the heart in an adult male is 300-350 gm while that of an adult female is 250-300 gm. Heart is divided into four chambers: a right and a left atrium both lying superiorly, and a right and a left ventricle both lying inferiorly and are larger. The atria are separated by a thin interatrial partition called interatrial septum, while the ventricles are separated by thick muscular partition called interventricular septum. The thickness of the right ventricular wall is 0.3 to 0.5 cm while that of the left ventricular wall is 1.3 to 1.5 cm. The blood in the heart chambers moves in a carefully prescribed pathway:

venous blood from systemic circulation → right atrium → right ventricle → pulmonary arteries → lungs → pulmonary veins → left atrium → left ventricle → aorta → systemic arterial supply. Wall of the heart consists mainly of the myocardium which is covered externally by thin membrane, the epicardium or visceral pericardium, and lined internally by another thin layer, the endocardium.The transport of blood is regulated by cardiac valves: two loose flap-like atrioventricular valves, tricuspid on the right and mitral (bicuspid) on the left; and two semilunar valves with three leaflets each, the pulmonary and aortic valves, guarding the outflow tracts. The normal circumference of the valvular openings measures about 12 cm in tricuspid, 8.5 cm in pulmonary, 10 cm in mitral and 7.5 cm in aortic valve.

The myocardium is the muscle tissue of the heart composed of syncytium of branching and anastomosing, transversely striated muscle fibres arranged in parallel fashion. The space between myocardial fibres contains a rich capillary network and loose connective tissue. The myocardial fibres are connected to each other by irregular joints called as intercalated discs. They represent apposed cell membranes of individual cells which act as tight junctions for free transport of ions and action potentials. The cardiac myocyte is very rich in mitochondria which is the source of large amount of ATP required for cardiac contraction. The cardiac muscle fibre has abundant sarcoplasmic reticulum corresponding to endoplasmic reticulum of other cells. Transverse lines divide each fibre into sarcomeres which act as structural and functional subunits. Each sarcomere consists of prominent central dark A-band attributed to thick myosin filaments and flanked on either side by light I-bands consisting of thin actin filament. The actin bands are in the form of twisted rods overlying protein molecules called tropomyosin. These protein molecules are of 3 types: troponin-I, troponin- T, and troponin-C.

Troponin molecules respond to calcium ions in cyclical contraction-relaxation of myocardial fibres. Myocardial fibres are terminally differentiated cells and do not regenerate but there is recent evidence that new cardiac myocytes can be formed from stem cells recruited from the circulation. The conduction system of the heart located in the myocardium is responsible for regulating rate and rhythm of the heart. It is composed of specialized Purkinje fibres which contain some contractile myofilaments and conduct action potentials rapidly. The conduction system consists of 4 major components: ((Stuon et.al (1990), fouad et.al (2000)).

The sinoatrial (SA) node is located in the posterior wall of the right atrium adjacent to the point at which the superior vena cava enters the heart. It is also called cardiac pacemaker since it is responsible for determining the rate of contraction for all cardiac muscle. The atrioventricular (AV) bundle conducts the impulse from the SA node to the AV node, the atrioventricular (AV) node is located on the top of the interventricular septum and receives impulses from the SA node via AV bundle and transmits them to the bundle of His and the bundle of His extends through the interventricular septum and divides into right and left bundle branches which arborise in the respective ventricular walls. These fibres transmit impulses from the AV node to the ventricular walls.The pericardium consists of a closely apposed layer, visceral pericardium or epicardium, and an outer fibrous sac, the parietal pericardium. The two layers enclose a narrow pericardial cavity which is lined by mesothelial cells and normally contains 10-30 ml of clear, watery serous fluid. This fluid functions as lubricant and shock absorbant to the heart. The endocardium is the smooth shiny inner lining of the myocardium that covers all the cardiac chambers, the cardiac valves, the chordae tendineae and the papillary muscles. It is lined by endothelium with connective tissue and elastic fibres in its deeper part. The valve cusps and semilunar leaflets are delicate and translucent structures. The valves are strengthened by collagen and elastic tissue and covered by a layer of endothelium (valvular endocardium).

2.1.1. Myocardial blood supply:

The cardiac muscle, in order to function properly, must receive adequate supply of oxygen and nutrients. Blood is transported to myocardial cells by the coronary arteries which originate immediately above the aortic semilunar valve. Most of blood flow to the myocardium occurs during diastole.The major coronary trunks, which supplying blood to specific segments of the heart: The anterior descending branch of the left coronary artery supplies most of the apex of the heart, the anterior surface of the left ventricle, the adjacent third of the anterior wall of the right ventricle, and the anterior two-third of the

interventricular septum. The circumflex branch of the left coronary artery supplies the left atrium and a small portion of the lateral aspect of the left ventricle). The right coronary artery supplies the right atrium, the remainder of the anterior surface of the right ventricle, the adjacent half of the posterior wall of the left ventricle and the posterior third of the interventricular septum. There are 3 anatomic patterns of distribution of the coronary blood supply, depending upon which of the coronary arteries crosses the crux. Crux is the region on the posterior surface of the heart where all the four cardiac chambers and the interatrial and interventricular septa meet. These patterns are as under:

Right coronary artery preponderance is the most common pattern. In this, right coronary artery supplies blood to the whole of right ventricle, the posterior half of the interventricular septum and a part of the posterior wall of the left ventricle by crossing the crux, balanced cardiac circulation is the next most frequent pattern. In this, the right and left ventricles receive blood supply entirely from right and left coronary arteries respectively. The posterior part of the interventricular septum is supplied by a branch of the right coronary while the anterior part is supplied by a branch of the left coronary artery, Left coronary preponderance is the least frequent pattern. In this, the left coronary artery supplies blood to the entire left ventricle, whole of interventricular septum and also supplies blood to a part of the posterior wall of the right ventricle by crossing the crux. Coronary veins run parallel to the major coronary arteries to collect blood after the cellular needs of the heart are met. Subsequently, these veins drain into the coronary sinus.

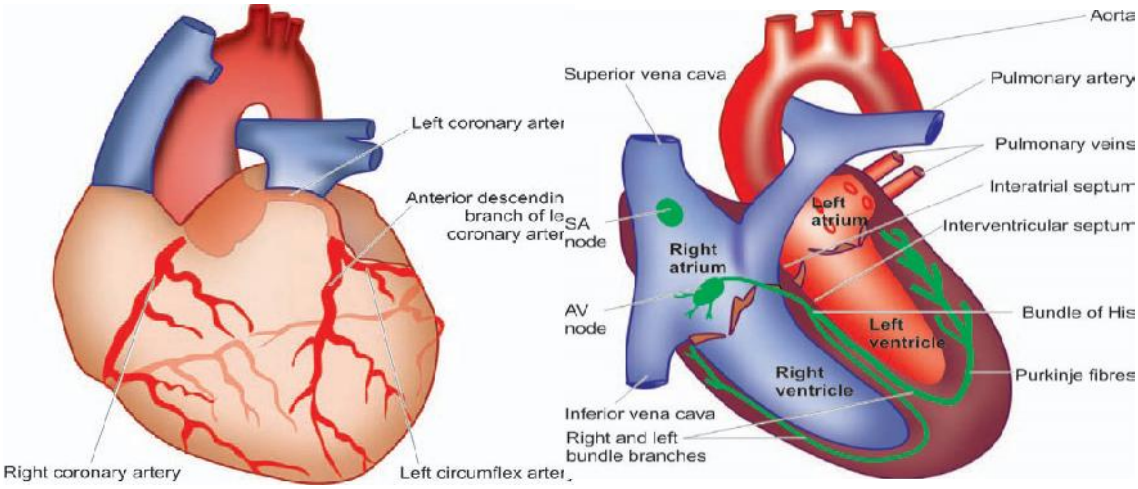

Figure 2.1. Show the heart blood vessels and general anatomy (L. Drake et.al 2014)

2.2. Pathology of the heart:

For the purpose of pathologic discussion of heart diseases, they are categorized on the basis of anatomic region involved and the functional impairment. Accordingly, topics on heart diseases are discussed in this chapter under the following headings:

Heart failure, Congenital heart diseases, Ischemic heart disease, Hypertensive heart disease, Corpulmonale, Rheumatic fever and rheumatic heart disease, Non-rheumatic endocarditis, Valvular diseases and deformities, Myocardial disease, Pericardial disease, Tumours of the heart and Pathology of cardiovascular interventions. It may be mentioned here that pattern of heart diseases in developing and developed countries is distinct due to difference in living standards. In children, valvular diseases are common all over the world, but in developing countries including India, infections, particularly rheumatic valvular disease, is the dominant cause compared to congenital etiology in affluent countries. On the other hand, ischemic heart disease and hypertensive cardiomyopathy are the major heart diseases in adults in western populations. Chopra P et.al (2003)

2.2.1. Heart failure:

Heart failure is defined as the pathophysiologic state in which impaired cardiac function is unable to maintain an adequate circulation for the metabolic needs of the tissues of the body. It may be acute or chronic. The term congestive heart failure (CHF) is used for the chronic form of heart failure in which the patient has evidence of congestion of peripheral circulation and of lungs. CHF is the end-result of various forms of serious heart diseases.

2.2.1.1. Etiology:

Heart failure may be caused by one of the following factors, either singly or in combination: Intrinsic pump failure; the most common and most important cause of heart failure is weakening of the ventricular muscle due to disease so that the heart fails to act as an efficient pump. The various diseases which may culminate in pump failure by this mechanisms are as under: Ischemic heart disease, Myocarditis, Cardiomyopathies, Metabolic disorders e.g. beriberi and Disorders of the rhythm e.g. atrial fibrillation and flutter. Increased workload on the heart: Increased mechanical load on the heart results in increased myocardial demand resulting in myocardial failure. Increased load on the heart may be in the form of pressure load or volume load. Increased pressure load may occur in the following states: Systemic and pulmonary arterial hypertension, Valvular disease e.g. mitral stenosis, aortic stenosis, pulmonary stenosis and chronic lung diseases.Increased volume load occurs when a ventricle

is required to eject more than normal volume of the blood resulting in cardiac failure. This is seen in the following conditions: Valvular insufficiency, severe anemia, Thyrotoxicosis, Arteriovenous shunts, Hypoxia due to lung diseases. Impaired filling of cardiac chambers: Decreased cardiac output and cardiac failure may result from extra-cardiac causes or defect in filling of the heart:Cardiac tamponade e.g. haemopericardium, hydropericardium and Constrictive pericarditis.

2.2.1.2. Types of Heart Failure:

Heart failure may be acute or chronic, right-sided or left sided, and forward or backward failure. Depending upon whether the heart failure develops rapidly or slowly, it may be acute or chronic: Acute heart failure. Sudden and rapid development of heart failure occurs in the following conditions: Larger myocardial infarction, Valve rupture, Cardiac tamponade, Massive pulmonary embolism, acute viral myocarditis and acute bacterial toxemia.

In acute heart failure, there is sudden reduction in cardiac output resulting in systemic hypotension but oedema does not occur. Instead, a state of cardiogenic shock and cerebral hypoxia develops.Chronic heart failure. More often, heart failure develops slowly as observed in the following states: Myocardial ischemia from atherosclerotic coronary artery disease, Multi-valvular heart disease, and Systemic arterial hypertension, chronic lung diseases resulting in hypoxia and pulmonary arterial hypertension and Progression of acute into chronic failure. In chronic heart failure, compensatory mechanisms like tachycardia, cardiac dilatation and cardiac hypertrophy try to make adjustments so as to maintain adequate cardiac output. This often results in well-maintained arterial pressure and there is accumulation of oedema.

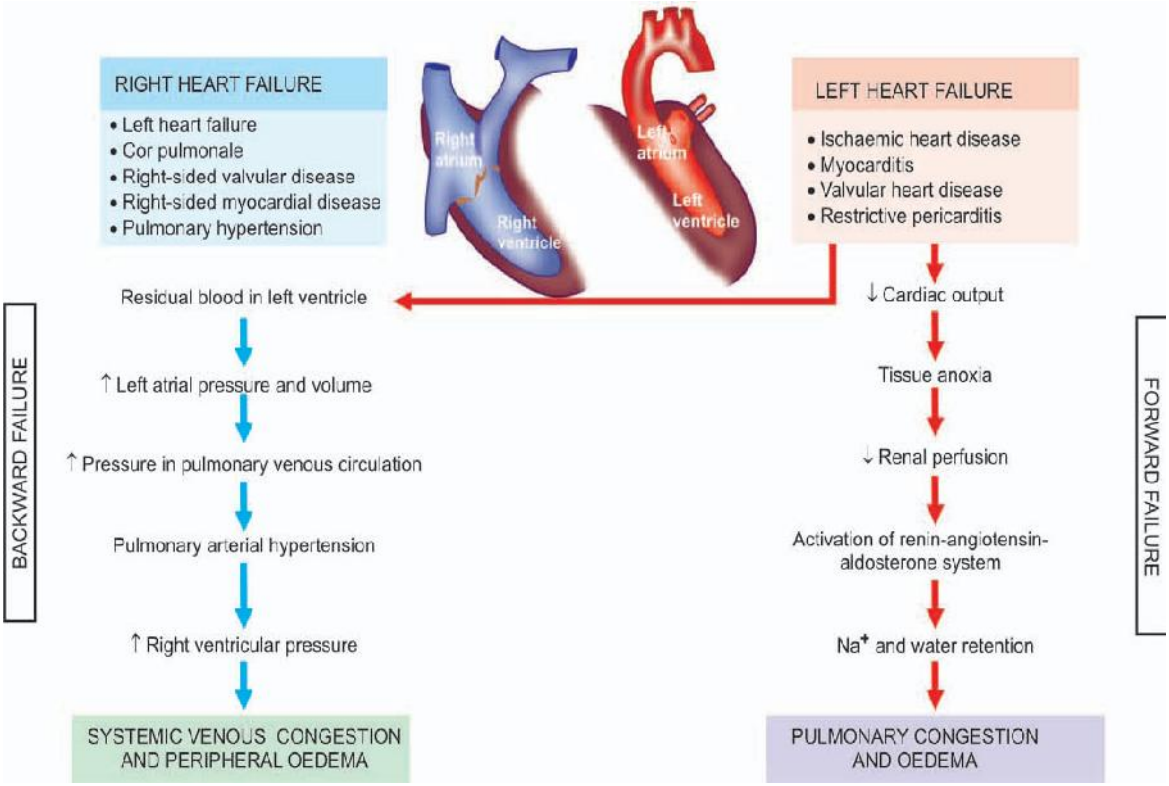

Figure 2.2. Show the type and mechanism of heart failure.

2.2.2. Ischemic Heart Disease (IHD):

Ischemic heart disease IHD is defined as acute or chronic form of cardiac disability arising from imbalance between the myocardial supply and demand for oxygenated blood. Since narrowing or obstruction of the coronary arterial system is the most common cause of myocardial anoxia, the alternate term 'coronary artery disease CAD' is used synonymously with IHD. IHD or CAD is the leading cause of death in most developed countries (about one-third of all deaths) and somewhat low incidence is observed in the developing countries. Men develop IHD earlier than women and death rates are also slightly higher for men than for women until the menopause. As per rising trends of IHD worldwide, it is estimated that by the year 2020 it would become the most common cause of death throughout world.

2.2.2.1. Etiopathogenesis:

IHD is invariably caused by disease affecting the coronary arteries, the most prevalent being atherosclerosis accounting for more than 90% cases, while other causes are responsible for less than 10% cases of IHD. Therefore, it is convenient to consider the etiology of IHD under three broad headings: Coronary atherosclerosis; Superadded changes in coronary atherosclerosis; and Non-atherosclerotic causes.

2.2.2.1.1. Coronary Atherosclerosis:

Coronary atherosclerosis resulting in 'fixed' obstruction is the major cause of IHD in more than 90% cases. The general aspects of atherosclerosis as regards its etiology, pathogenesis and the morphologic features of atherosclerotic lesions have already been dealt with at length this study. Here, a brief account of the specific features in pathology of lesions in atherosclerotic coronary artery disease in particular are presented. Its distribution: Atherosclerotic lesions in coronary arteries are distributed in one or more of the three major coronary arterial trunks, the highest incidence being in the anterior descending branch of the left coronary, followed in decreasing frequency, by the right coronary artery and still less in circumflex branch of the left coronary. About one third of cases have single-vessel disease, most often left anterior descending arterial involvement; another one-third have two vessel disease, and the remainder have three major vessel disease. Location: Almost all adults show atherosclerotic plaques scattered throughout the coronary arterial system. However, significant stenotic lesions that may produce chronic myocardial ischemia show more than 75% (three-fourth) reduction in the cross-sectional area of a coronary artery or its branch. The area of severest involvement is about 3 to 4 cm from the coronary ostia, more often at or near the bifurcation of the arteries, suggesting the role of hemodynamic forces in atherogenesis. Fixed atherosclerotic plaques: The atherosclerotic plaques in the coronaries are more often eccentrically located bulging into the lumen from one side. Occasionally there may be concentric thickening of the wall of the artery. Atherosclerosis produces gradual luminal narrowing that may eventually lead to 'fixed' coronary obstruction. The general features of atheroma's of coronary arteries are similar to those affecting elsewhere in the body and may develop similar complications like calcification, coronary thrombosis, ulceration, hemorrhage, rupture and aneurysm formation. Corri R et.al (2003)

2.2.2.1.2. Superadded Changes in Coronary Atherosclerosis:

The attacks of acute coronary syndromes, which include acute myocardial infarction, unstable angina and sudden ischaemic death, are precipitated by certain changes superimposed on a pre-existing fixed coronary atheromatous plaque. These changes are as under: Acute changes in chronic atheromatous plaque. Though chronic fixed obstructions are the most frequent cause of IHD, acute coronary episodes are often precipitated by sudden changes in chronic plaques such as plaque Haemorrhage, fissuring, or ulceration that results in thrombosis and embolization of athermanous debris. Acute plaque changes are brought about by factors such as sudden coronary artery spasm, tachycardia, intraplaqueHaemorrhage and hypercholesterolemia, Coronary artery thrombosis. Transmural acute myocardial

infarction is often precipitated by partial or complete coronary thrombosis. The initiation of thrombus occurs due to surface ulceration of fixed chronic athermanous plaque, ultimately causing complete luminal occlusion. The lipid core of plaque, in particular, is highly chromogenic. Small fragments of thrombotic material are then dislodged which are embolized to terminal coronary branches and cause micro infarcts of the myocardium. Corri R et.al (2003).

Local platelet aggregation and coronary artery spasm: Some cases of acute coronary episodes are caused by local aggregates of platelets on the atheromatous plaque, short of forming a thrombus. The aggregated platelets release vasospasmic mediators such as thromboxane A2 which may probably be responsible for coronary vasospasm in the already atherosclerotic vessel. Based on progressive pathological changes and clinical correlation, American Heart Association (1995) has classified human coronary atherosclerosis into 6 sequential types in ascending order of grades of lesions

2.2.2.1.2. Non-atherosclerotic Causes:

Several other coronary lesions may cause IHD in less than 10% of cases. These are as under: Vasospasm. It has been possible to document vasospasm of one of the major coronary arterial trunks in patients with no significant atherosclerotic coronary narrowing which may cause angina or myocardial infarction, Stenosis of coronary ostia. Coronary ostial narrowing may result from extension of syphilitic aortitis or from aortic atherosclerotic plaques encroaching on the opening, Arteritis.Various types of inflammatory involvements of coronary arteries or small branches like in rheumatic arteritis, polyarteritisnodosa, thrombo-angiitisobliterans (Buerger's disease), Takayasu's disease, Kawasaki's disease, tuberculosis and other bacterial infections may contribute to myocardial damage, Embolism. Rarely, emboli originating from elsewhere in the body may occlude the left coronary artery and its branches and produce IHD. The emboli may originate from bland thrombi, or from vegetations of bacterial endocarditis; rarely fat embolism and air embolism of coronary circulation, Thrombotic diseases. Another infrequent cause of coronary occlusion is from hypercoagulability of the blood such as in shock, polycythaemiavera, sickle cell anaemia and thrombotic thrombocytopenic purpura, Trauma: Contusion of a coronary artery from penetrating injuries may produce thrombotic occlusion, Aneurysms.Extension of dissecting aneurysm of the aorta into the coronary artery may produce thrombotic coronary occlusion. Rarely, congenital, mycotic and syphilitic aneurysms may occur in coronary arteries and produce similar occlusive effects and Compression.Compression of a coronary from outside by a primary or secondary tumour of the heart may result in coronary occlusion.

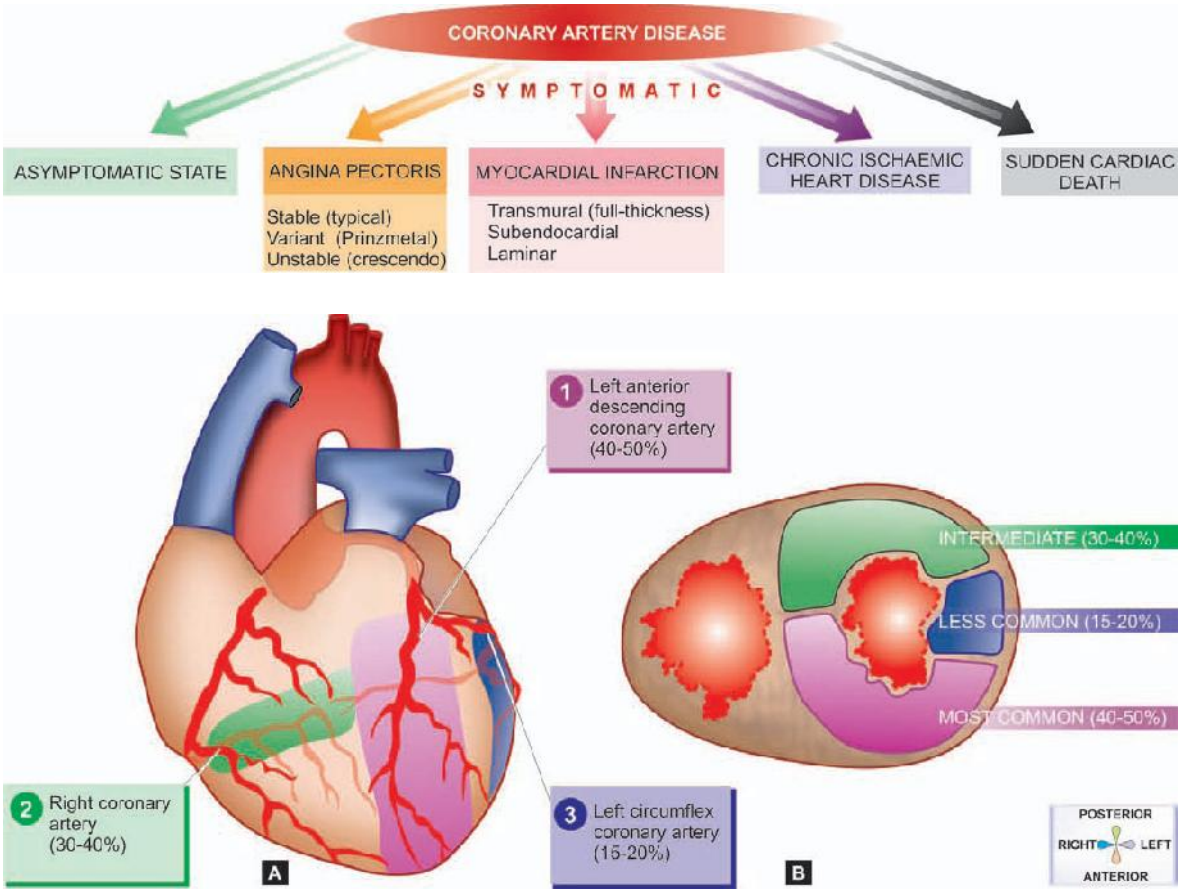

Figure 2.3. Common locations and the regions of involvement in myocardial infarction, the figure shows region of myocardium affected by stenosis of three respective coronary trunks in descending order shown as: 1) left anterior descending coronary, 2) right coronary and 3) left circumflex coronary artery. A, as viewed from anterior surface. B, as viewed on transverse section at the apex of the heart.

2.3. Previous studies

Devereux et al., 1999 stated that whether diabetes mellitus (DM) adversely affects left ventricular (LV) structure and function independently of increases in body mass index (BMI) and blood pressure is controversial. Echocardiography was used in the Strong Heart Study, a study of cardiovascular disease in American Indians, to compare LV measurements between 1810 participants with DM and 944 with normal glucose tolerance. Participants with DM were older (mean age, 60 versus 59 years), had higher BMI (32.4 versus 28.9 kg/m^2) and systolic blood pressure (133 versus 124 mm Hg), and were more likely to be female, to be on antihypertensive treatment, and to live in Arizona (all P<0.001). In analyses adjusted for covariates, women and men with DM had higher LV mass and wall thicknesses and lower

LV fractional shortening, mid-wall shortening, and stress-corrected mid-wall shortening (all $P<0.002$). Pulse pressure/stroke volume, a measure of arterial stiffness, was higher in participants with DM ($P<0.001$ independent of confounders). And he conclude that; non–insulin-dependent DM has independent adverse cardiac effects, including increased LV mass and wall thicknesses, reduced LV systolic chamber and myocardial function, and increased arterial stiffness. These findings identify adverse cardiovascular effects of DM, independent of associated increases in BMI and arterial pressure that may contribute to cardiovascular events in diabetic individuals.

Yetkin et al,. 2015 aimed to investigate the relation of cardiovascular risk factors and hematological parameters with collateral development in patients with severely stenotic (≥95 %) and totally occluded coronary artery disease including at least one major coronary artery.The study population was selected from the patients who underwent coronary angiography between January 2008 and March 2009. Five hundred and two patients who had at least one coronary artery stenosis ≥95 % (368 men; mean age 59 ± 10 years) comprised the study population. Of the 502 patients, 228 had total occlusion in at least one major epicardial coronary artery. Collateral artery grading was performed by using Cohen–Rentrop method to the vessel with coronary artery stenosis of ≥95 % and patients with chronic total occlusions (CTO). Patients with grade 0–1 collateral development were regarded as the poor collateral group, and patients with grade 2–3 collateral development were regarded as the good collateral group. The study results showed: Two hundred and fifty-eight (51 %) of 502 patients had poor collateral development, and 244 (49 %) had good collateral development. Logistic regression analysis revealed that DM was independently associated with poor CVD in patients with ≥95 % stenosis ($p < 0.001$). Additionally, female gender and DM were found to be independently associated with poor CVD in patients with CTO ($p = 0.005$ and $p < 0.001$, respectively). Monocyte count was found to be independent of CVD neither in patients with ≥95 % stenosis nor in patients with CTO. Which concluded that DM is an

independent factor for poor coronary CVD both in patients with severe coronary artery stenosis and in patients with CTO. Female gender or being in post-menopausal period is another negative risk factor for poor CVD in addition to DM in patients with CTO.

In adults, overweight and obesity are linked to increased risk of heart disease, type 2 diabetes (high blood sugar), high blood pressure, certain cancers, and other chronic conditions. Research has shown that obese children are more likely to be overweight or obese as adults.

Sutton, et.al 2002, was compare the Effects of Rosiglitazone and Glyburide on Cardiovascular Function and Glycemic Control in Patients With Type 2 Diabetes, and he stated that this open-label, active-controlled study investigated the cardiac safety and antihyperglycemic effect of rosiglitazone (RSG) in patients with type 2 diabetes.The 203 patients randomly assigned to RSG (4 mg b.i.d.) or glyburide (GLB) (titrated to achieve optimal glycemic control for the first 8 weeks only to limit the risk of hypoglycemia; mean 10.5 mg/day), 118 had an echocardiogram performed at week 52. Left ventricular (LV) mass index, ejection fraction, and left ventricular end-diastolic volume were assessed by M-mode echocardiography at baseline and weeks 12, 28, and 52; 24-h ambulatory blood pressure was assessed at baseline and at weeks 28 and 52. Glycemic control was assessed by measuring fasting plasma glucose (FPG) and HbA. his main result was; either treatment produced an increase in LV mass index that exceeded 1 SD. Ejection fraction did not change in either group. Both groups had clinically insignificant increases in LV end-diastolic volume. RSG, but not GLB, caused a statistically significant reduction in ambulatory diastolic blood pressure. Both treatments reduced HbA_{1c} and FPG.A total of 52 weeks of therapy with RSG (4 mg b.i.d.) did not adversely affect cardiac structure or function in patients with type 2 diabetes and produced significant and sustained reductions in hyperglycemia. Decreases in ambulatory diastolic blood pressure with RSG were superior to those with GLB.

Diabetes mellitus-related cardiomyopathy (DMCMP) was originally described as a dilated phenotype with eccentric left ventricular (LV) remodelling and systolic LV dysfunction. Recently however, clinical studies on DMCMP mainly describe a restrictive phenotype with concentric LV remodelling and diastolic LV dysfunction. Both phenotypes are not successive

stages of DMCMP but evolve independently to respectively heart failure with preserved left ventricular ejection fraction (HFPEF) or reduced left ventricular ejection fraction (HFREF). Phenotype-specific pathophysiological mechanisms were recently proposed for LV remodelling and dysfunction in HFPEF and HFREF consisting of coronary microvascular endothelial dysfunction in HFPEF and cardiomyocyte cell death in HFREF. A similar preferential involvement of endothelial or cardiomyocyte cell compartments explains DMCMP development into distinct restrictive/HFPEF or dilated/HFREF phenotypes. Diabetes mellitus (DM)-related metabolic derangements such as hyperglycaemia, lipotoxicity, and hyperinsulinaemiafavour development of DMCMP with restrictive/HFPEF phenotype, which is more prevalent in obese type 2 DM patients. In contrast, autoimmunity predisposes to a dilated/HFREF phenotype, which manifests itself more in autoimmune-prone type 1 DM patients. Finally, coronary microvascular rarefaction and advanced glycation end-products deposition are relevant to both phenotypes. Diagnosis of DMCMP requires impaired glucose metabolism and exclusion of coronary, valvular, hypertensive, or congenital heart disease and of viral, toxic, familial, or infiltrative cardiomyopathy. In addition, diagnosis of DMCMP with restrictive/HFPEF phenotype requires normal systolic LV function and diastolic LV dysfunction, whereas diagnosis of DMCMP with dilated/HFREF phenotype requires systolic LV dysfunction. Treatment of DMCMP with restrictive/HFPEF phenotype is limited to diuretics and lifestyle modification, whereas DMCMP with dilated/HFREF phenotype is treated in accordance to HF guidelines. (Petar M. Seferović, Walter J. Paulus, 2015).

Chapter Three

Methodology

3.1. Materials

Echocardiographic machines with motion mode(M –mode) and Doppler capabilities was used. The probe is of a convex, or better to be a sector type.The transducer is a phased - array 3.5-7.5 MHZ, ultrasound gel is applied to the transducer to prevent any attenuation or artefacts. A questionnaire is used to collect the data and to number the patients.

3.2. Methods:

From the ultrasound technique, in which there are five windows the so called: (Right &Left parasternal view, apical view, sub costal view, and suprasternal view); that use two positions either left lateral decubitus or supine, from it the following results were collected: LVPW size or thickness (using M –mode), IVS size (using M –mode), LVESD (using M-mode), LVEDD (using M-mode), Valvular sizes (using M-mode), Blood flow (using Doppler) and Ejection fraction. Also Name, sex, age, height and weight with the body index, duration of the disease, type of the medications, the follow up visits, and the last three readings of Diabetes which will indicate the control of the disease.

3.2.1. The studyarea

The study conducted in Khartoum city at Ribat University Hospital and The Military Hospital

3.2.2. Duration of the Study

FromAugust to September 2014; all attending diabetic patients.

3.2.3. The study population

In a random way a total of fifty patients were the sample unit in this study where EIGHT of them are control.

3.2.4. The inclusion criterion

Any diabetic patient attending the hospital in that period mentioned.

3.2.5. The exclusion criteria

Any child (less than 18 years) or a pregnant women with diabetes and the patients with cardiovascular diseases that related to other than diabetes, pulmonary diseases, neurological diabetes, smokers, and renal diseases which are not related to diabetes.

3.2.6. Data collection variables:

LVPW size or thickness (using M –mode), IVS size (using M –mode), LVESD (using M-mode), LVEDD (using M-mode), Valvular sizes (using M-mode).Blood flow (using Doppler), and Ejection fraction.

3.2.7. Method of data analysis:

Finally these data tabulated, described, represented and analysed using SPSS version 21.0,putting in mind that the p value is 0.05 using the chi square test to know the significance . The results of this analysis put in a scientific frames and facts from which a medical decision or recommendation was created.

Chapter Four

Results

Table (4.1) showed the frequency distribution of gender within the collected data

Gender	Frequency	Percent
Male	22	44.0
Female	28	56.0
Total	50	100.0

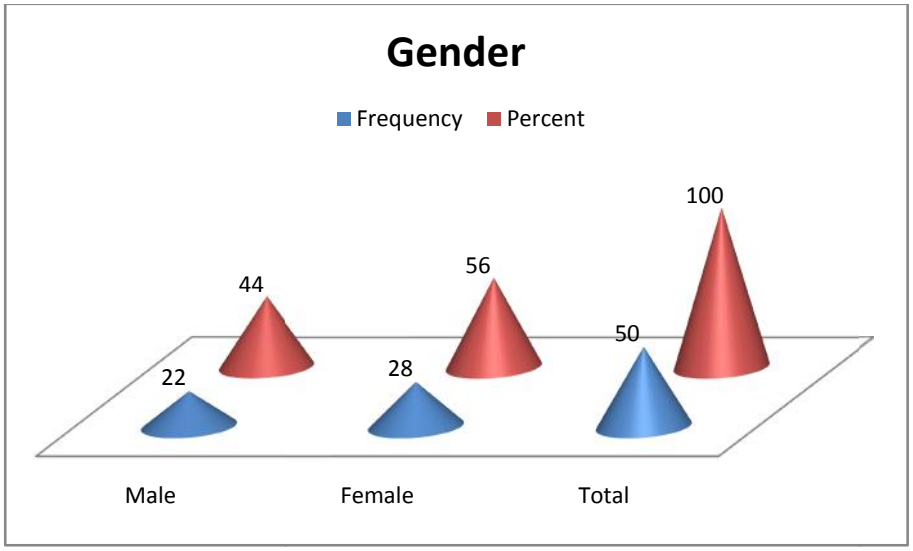

Figure (4.1): showed the frequency distribution of the gender

Table (4.2) showed the frequency distribution within the collected data of follow up

Follow Up	Frequency	Percent
Yes	48	96.0
No	2	4.0
Total	50	100.0

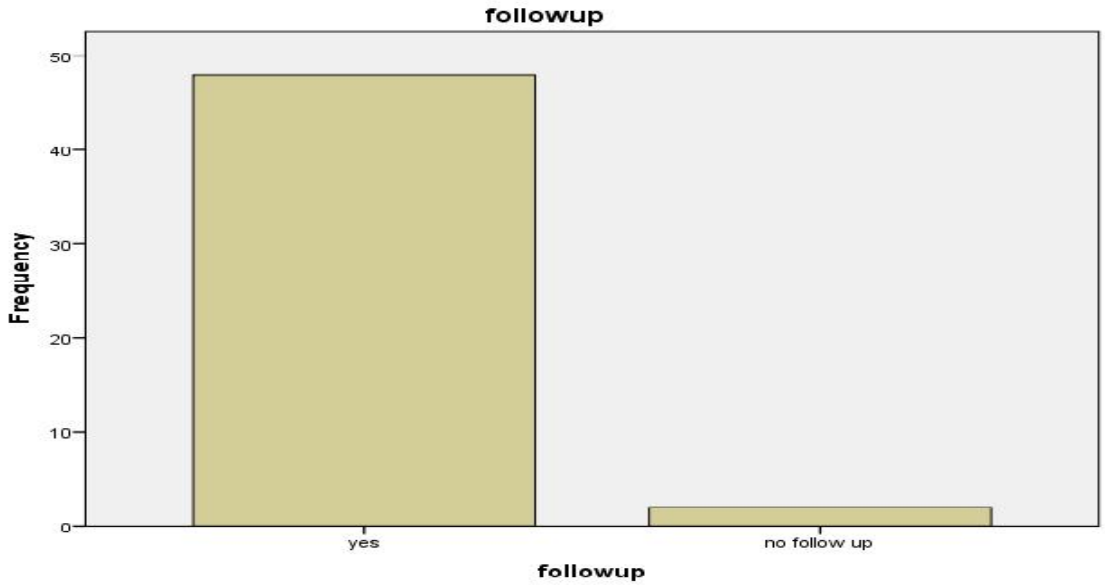

Figure (4.2): showed the frequency distribution of the follow up

Table (4.3) showed the frequency distribution within the collected data of gender

medication		Frequency	Percent
Valid	yes	47	94.0
	no medications	3	6.0
	Total	50	100.0

Figure (4.3): showed the frequency distribution within the collected data of medication

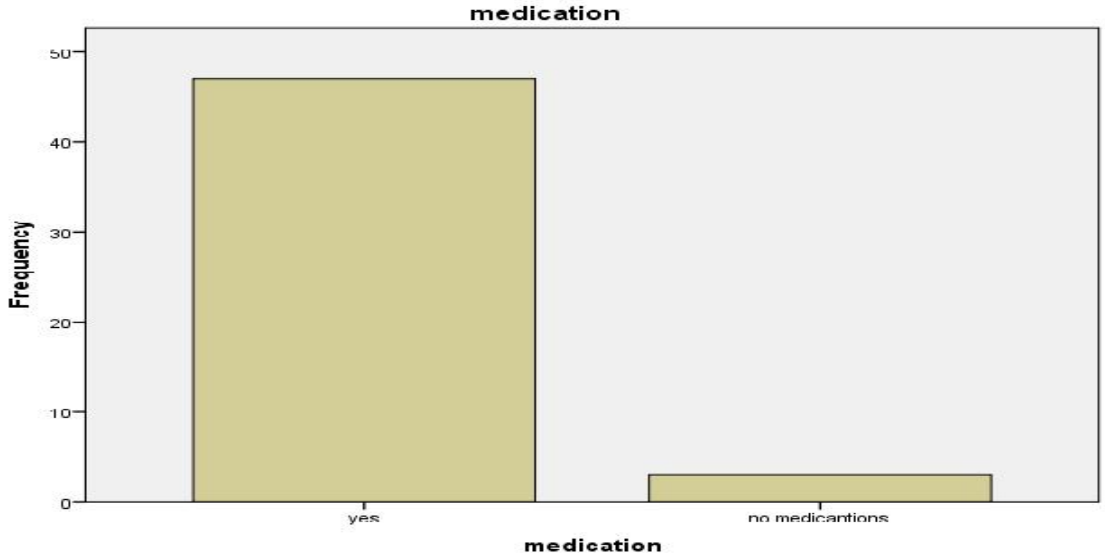

Table (4.4)) showed the frequency distribution within the collected data of age group

Age group	Frequency	Percent
20-29	2	4.0
30-39	1	2.0
40-49	8	16.0
50-59	13	26.0
60-69	14	28.0
70-79	9	18.0
80-89	3	6.0
Total	50	100.0

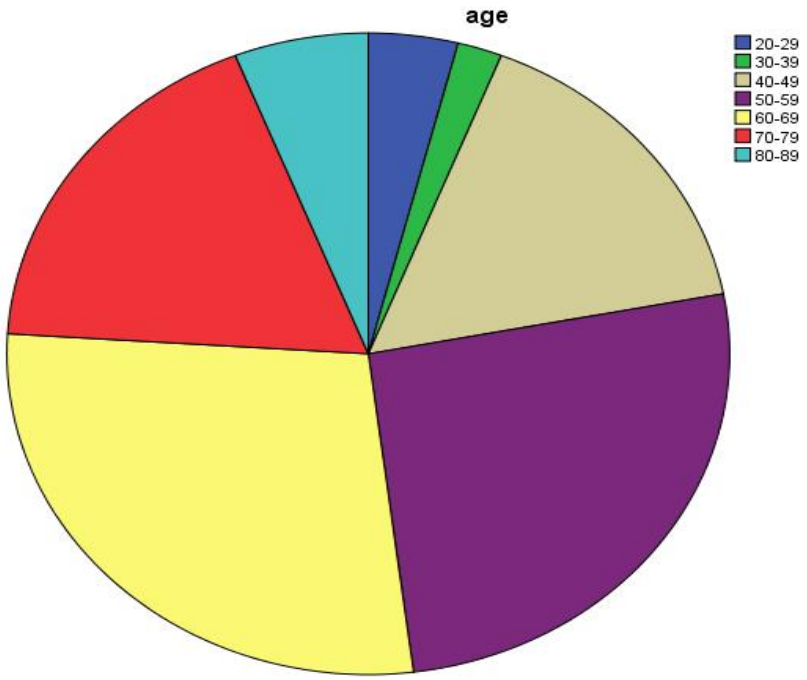

Figure (4.4): showed the frequency distribution within the collected data of age group

Table (4.5) showed the frequency distribution within the collected data ofwhether fasting blood sugar or random tests

Fasting or Random Blood Sugar	Frequency	Percent
Fasting	38	76.0
Random	12	24.0
Total	50	100.0

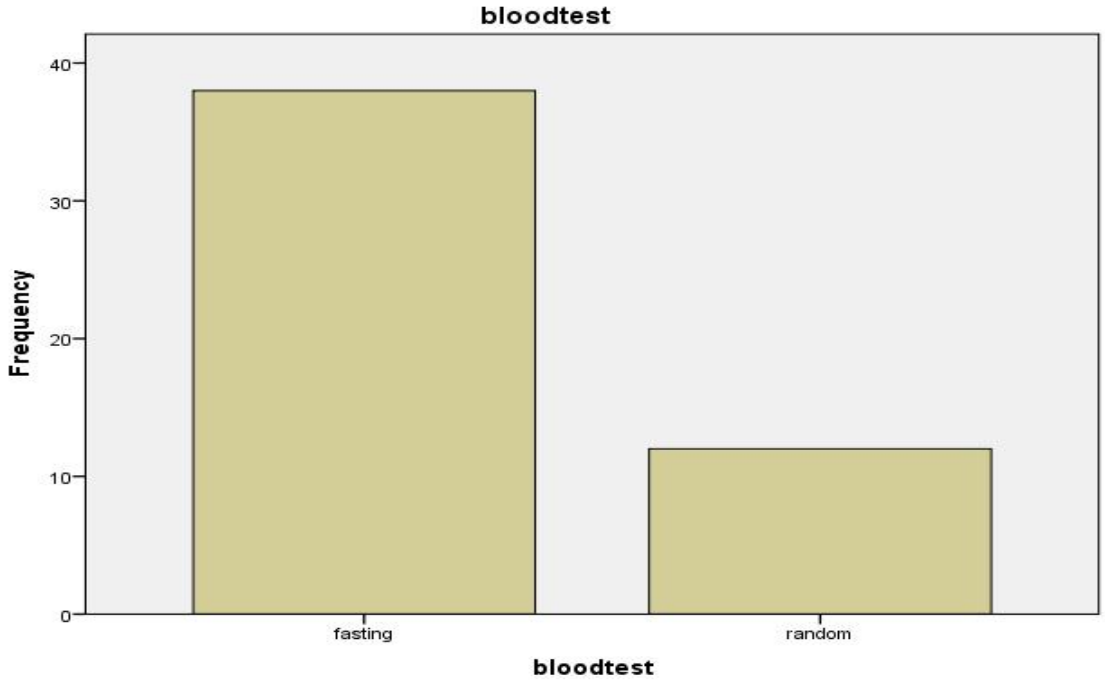

Figure (4.5): showed the frequency distribution within the collected data of blood test

Table (4.6) showed the frequency distribution within the collected data blood test result

Blood test result		Frequency	Percent
Valid	normal	27	54.0
	abnormal	23	46.0
	Total	50	100.0

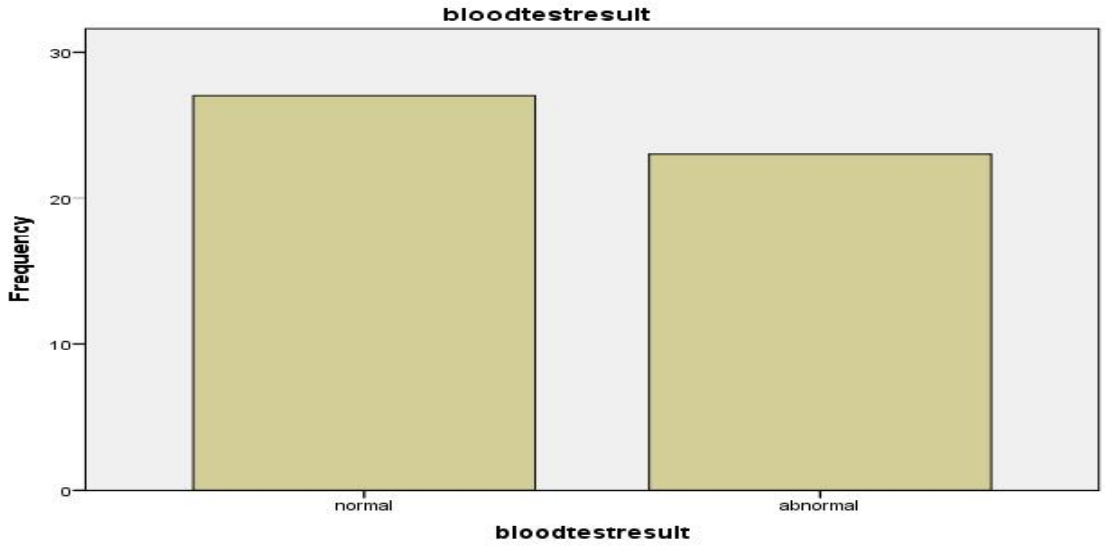

Figure (4.6): showed the frequency distribution within the collected data of blood test result

Table (4.7) showed the frequency distribution within the collected data of medical history

Medical History	Frequency	Percent
Diabetes Mellitus (DM)	10	20.0
Diabetes Mellitus + Hypertension (DM + HTN)	29	58.0
Diabetes Mellitus + Hypertension + Other (DM + HTN + Other)	10	20.0
Diabetes Mellitus + Other (DM + Other)	1	2
Total	50	100

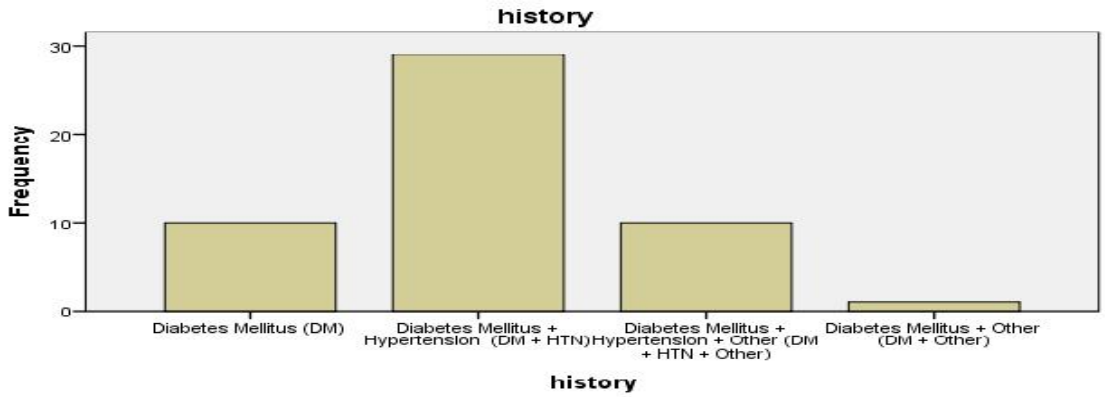

Figure (4.7): showed the frequency distribution within the collected data of medical history

Table (4.8) showed the frequency distribution within the collected data of use of medication among the age group

		Medication		Total
		yes	no medications	
age	20-29	1	1	2
	30-39	1	0	1
	40-49	7	1	8
	50-59	13	0	13
	60-69	14	0	14
	70-79	8	1	9
	80-89	3	0	3
Total		47	3	50

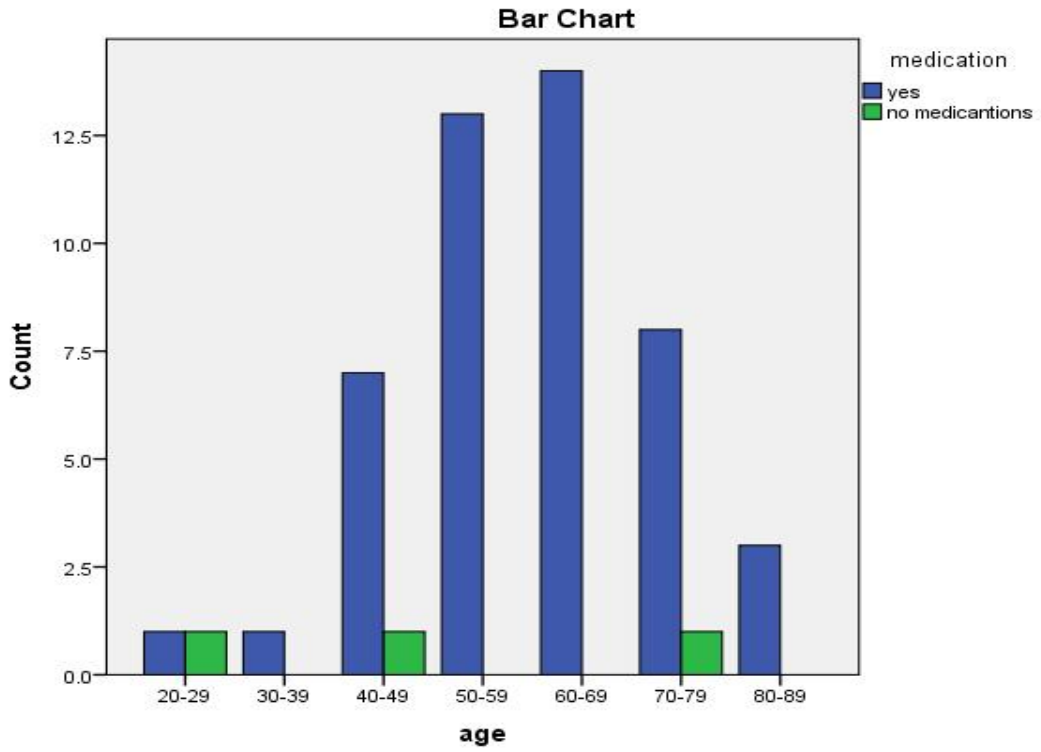

Figure (4.8): showed the frequency distribution within the collected data of use of medication among the age group

Table (4.9) showed the frequency distribution within the collected data of blood test results among the age group

		Blood test result		Total
		normal	abnormal	
age	20-29	1	1	2
	30-39	0	1	1
	40-49	2	6	8
	50-59	8	5	13
	60-69	10	4	14
	70-79	4	5	9
	80-89	2	1	3
Total		27	23	50

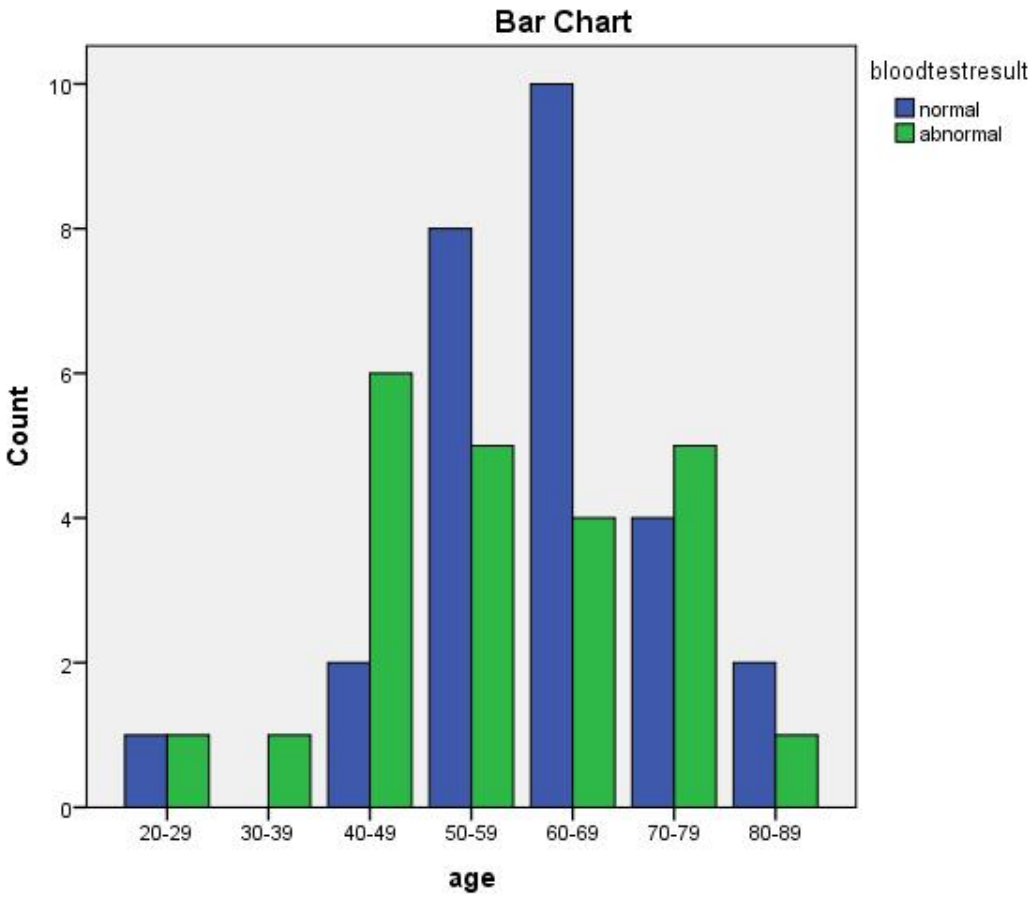

Figure (4.9) showed the frequency distribution within the collected data of blood test results among the age group

Table (4.10)showed the frequency distribution within the collected data of A/E ratio among the age group

		E/A ratio				Total
		normal	grade one	grade two	grade three	
age	20-29	1	1	0	0	2
	30-39	1	0	0	0	1
	40-49	2	3	1	0	6
	50-59	4	5	3	1	13
	60-69	4	4	5	1	14
	70-79	0	2	7	0	9
	80-89	0	1	2	0	3
Total		12	16	18	2	48

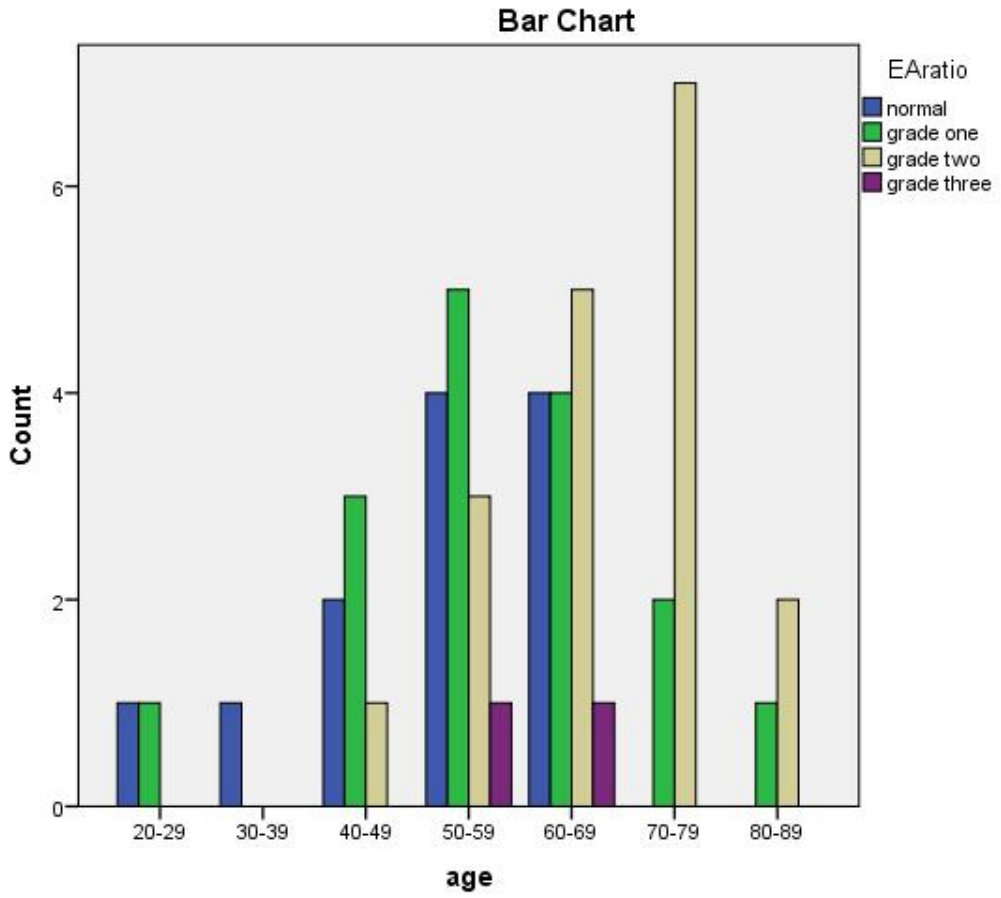

Figure (4.10)showed the frequency distribution within the collected data of A/E ratio among the age group

Table (4.11) showed the frequency distribution within the collected dataof status as control group who had normal echo finding and the abnormal findings group

Status	Frequency	Percent
Normal	8	16.0
Abnormal	42	84.0
Total	50	100.0

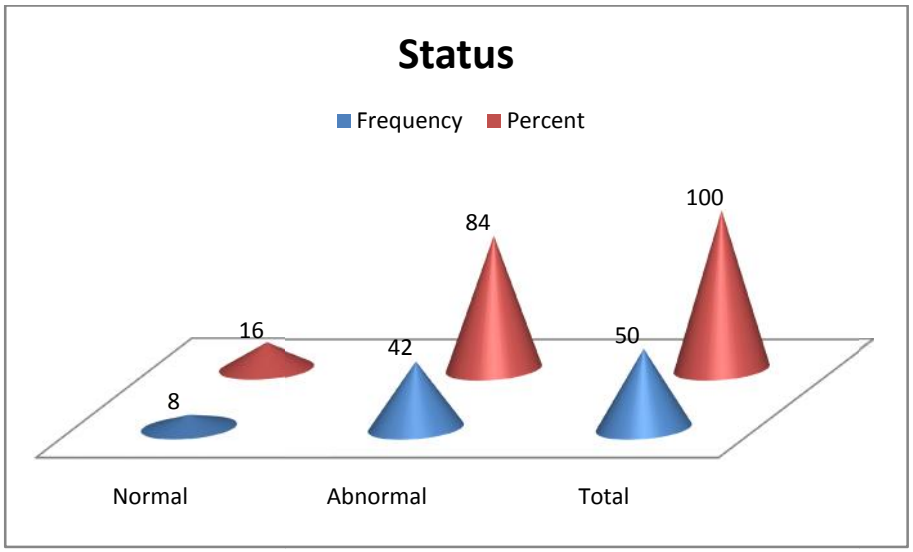

Figure (4.11) showed the frequency distribution within the collected dataof status as control group who had normal echo finding and the abnormal findings group

Table (4.12) showed the frequency distribution within the collected dataof valves status

Valves	Frequency	Percent
Normal	44	88
Degenerative	2	4.0
Sclerotic	4	8.0
Total	50	100.0

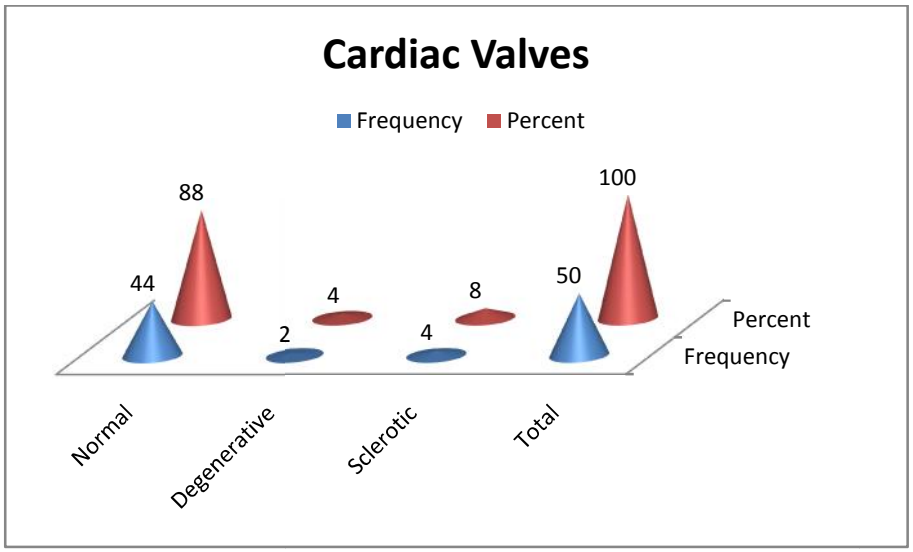

Figure (4.12) showed the frequency distribution within the collected dataof valves status

Table (4.13) showed the frequency distributionwithin the collected data of blood flow

Blood flow		Frequency	Percent
Valid	normal	40	80.0
	Regurgitation	9	18.0
	stenosis	1	2.0
	Total	50	100.0

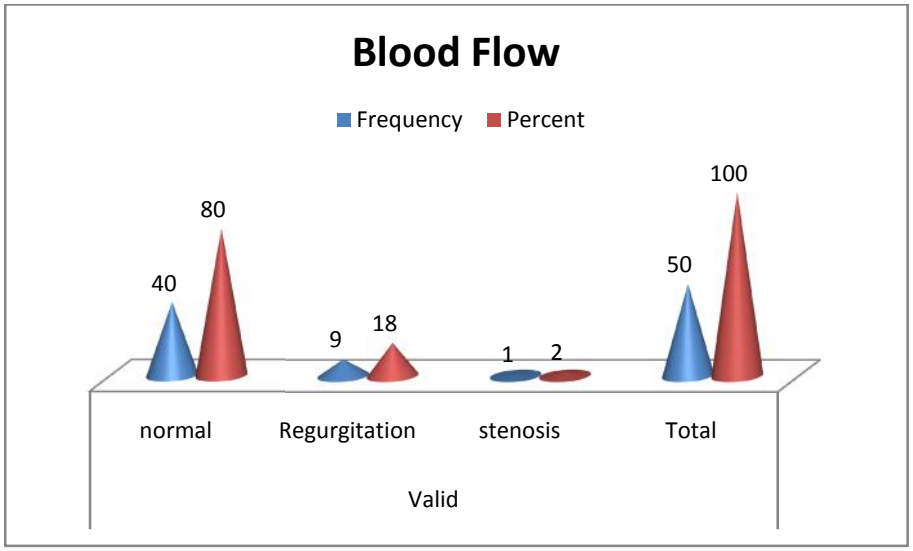

Figure (4.13) showed the frequency distribution within the collected data of blood flow

Table (4.14) showed the frequency distribution within the collected data of Echo Results

Echo Results	Frequency	Percent
Normal echo study	8	16.0
Normal LV systolic function and diastolic dysfunction	9	18.0
LVH with good LV function	9	18.0
LVH with diastolic dysfunction & good systolic function	7	14
IHD with good LV systolic function and diastolic dysfunction	10	20
IHD with good LV systolic function and diastolic dysfunction and others	7	14
Total	50	100.0

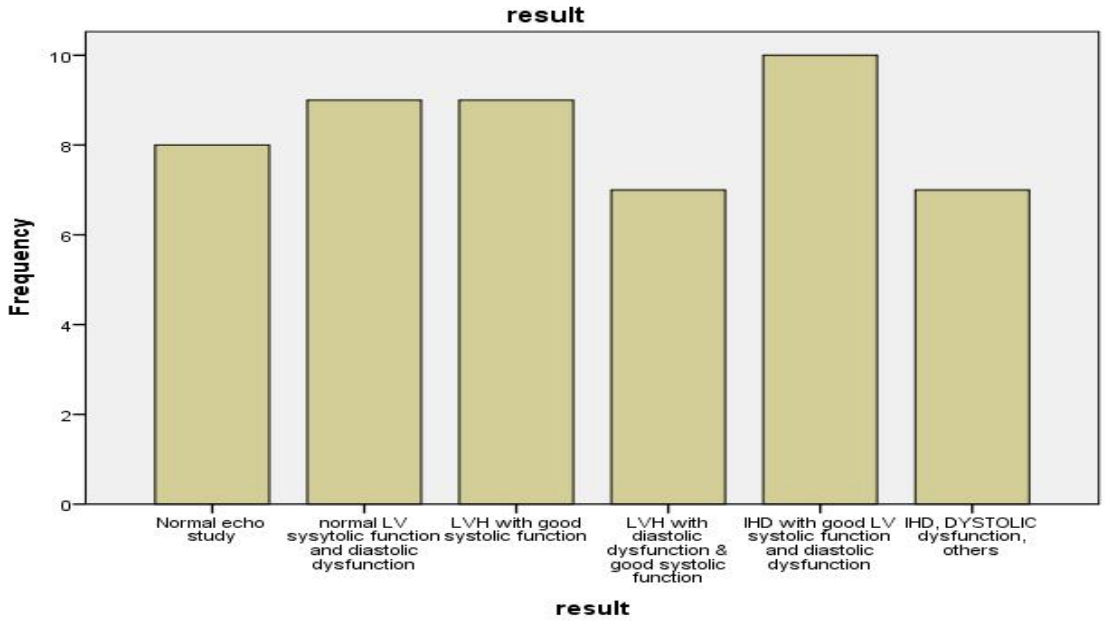

Figure (4.14) showed the frequency distribution within the collected data of Echo Results

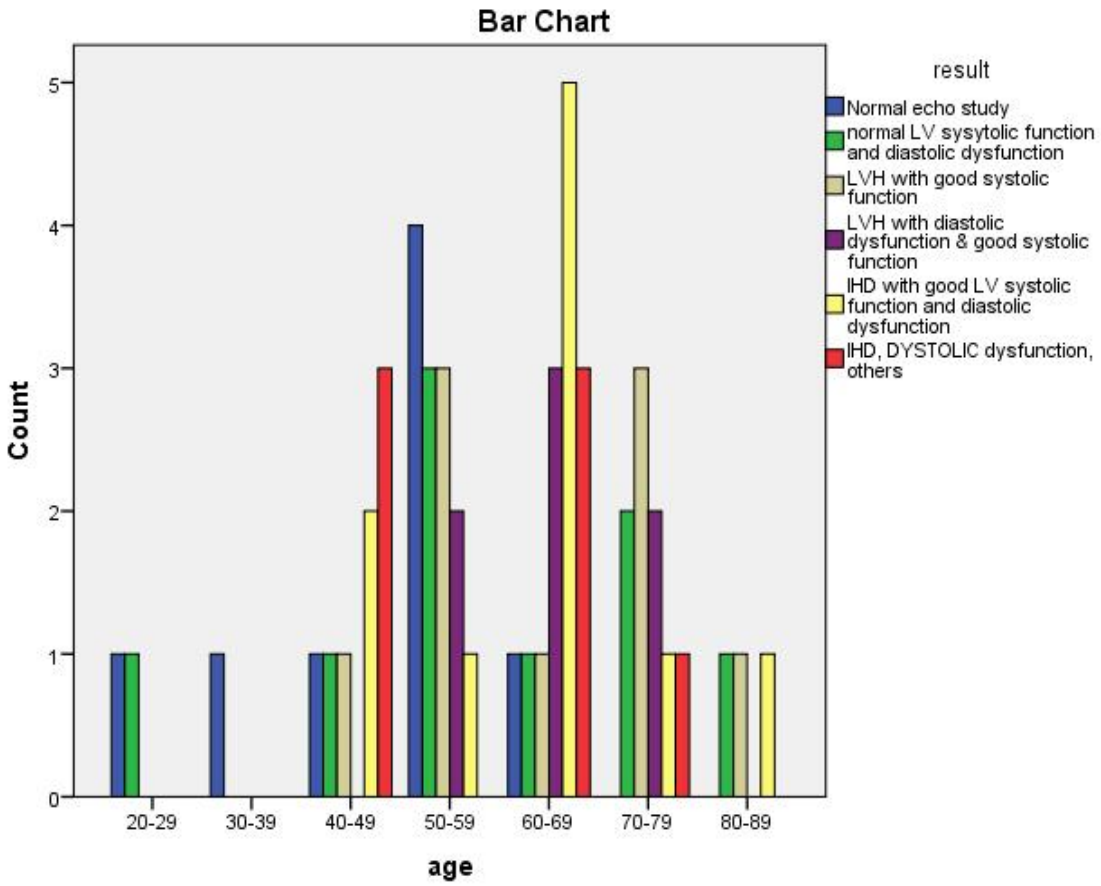

Figure (4.15) showed the frequency distribution within the collected dataof echo results among the age group

Table (4.15) showed The statistics (mean ± SD) of the different variables within the collected data

Variables	Mean±SD
Age	58.84±13.642
Height	162.46±9.451
Weight	72.44±13.197
BMI	27.632±5.8940
Duration	8.07158±6.814764
Control	171.68±85.311
LVEF percent	61.26±11.634
LVPW	11.158±2.5025
IVS	12.092±3.0118
LVESD	30.554±7.7404
LVEDD	47.254±5.9244
E/A Ratio	1.208±0.8742
Aortic Root	29.624±3.1523

Table (4.16) showed the significance Coefficients[a] within the collected data

Coefficients[a]				
Model		Unstandardized Coefficients	t	Sig.
		B		
3	(Constant)	104.679	11.080	.000
	LVESD	-1.113	-9.079	.000
	LVPW	1.408	3.654	.001
	LtAtrium	-.697	-2.990	.005

a. Dependent Variable: LVEF percent

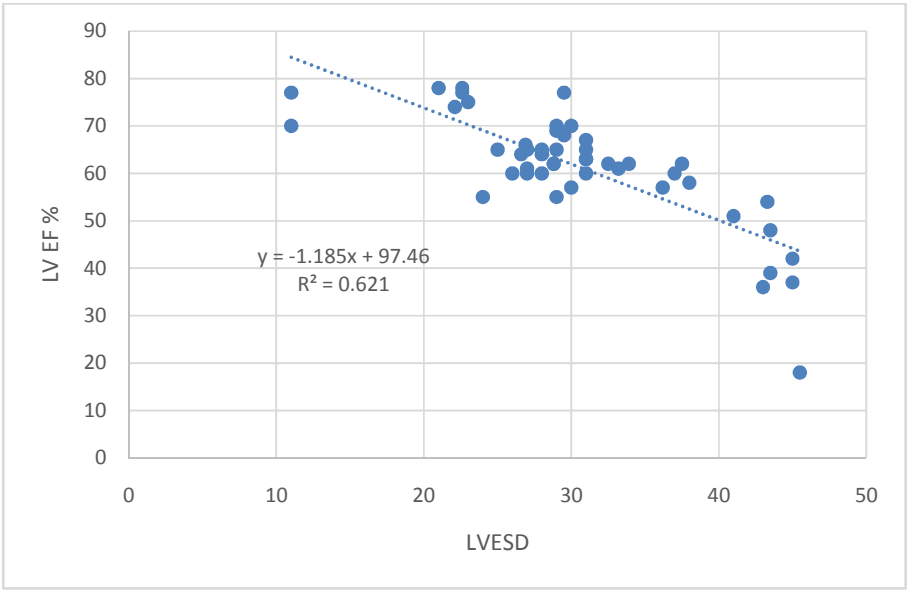

Figure (4.16) scatter plot showed the correlation between the LVEF% and LVESD (mm)

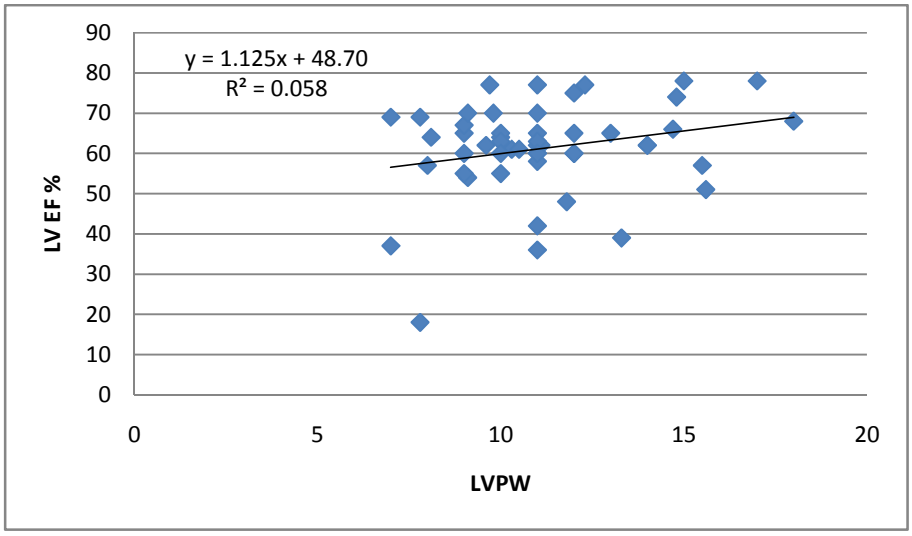

Figure (4.17) scatter plot showed the correlation between the LVEF% and LVPW (mm)

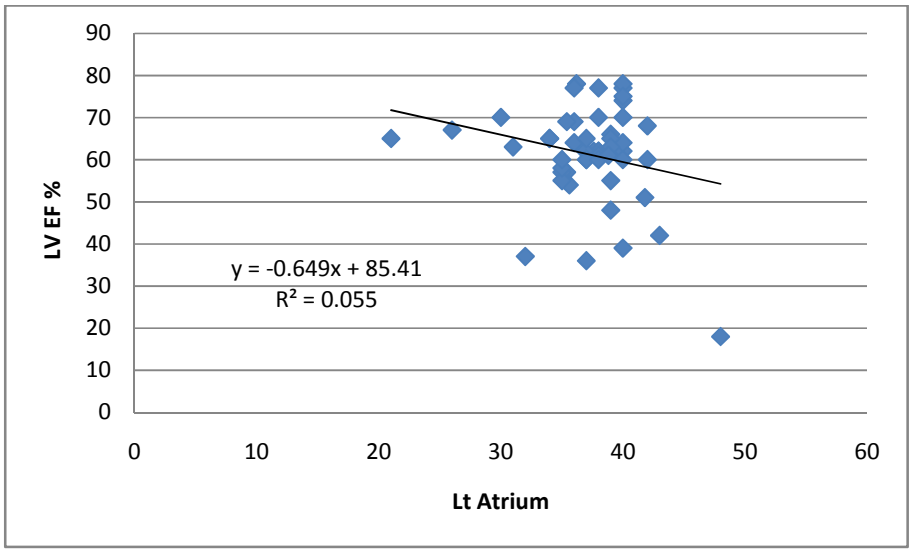

Figure (4.18) scatter plot showed the correlation between the LVEF% and Lt. Atrium Diameter (mm)

Table (4.17) showed the Medical History * Conclusion Cross-tabulation within the collected data

Medical History * echo results Cross-tabulation								
Count								
		Echo Results						Total
		Normal echo study	Normal LV systolic function and diastolic dysfunction	LVH with good LV function	LVH with diastolic dysfunction & good systolic function	IHD with good LV systolic function and diastolic dysfunction	IHD with good LV systolic function and diastolic dysfunction and others	
Medical History	Diabetes Mellitus (DM)	6	3	0	0	1	0	10
	Diabetes Mellitus + Hypertension (DM + HTN)	2	3	7	5	6	6	29

	Diabetes Mellitus + Hypertension + Other (DM + HTN + Other)	0	2	2	2	3	1	10
	Diabetes Mellitus + Other (DM + Other)	0	1	0	0	0	0	1
Total		8	9	9	7	10	7	50

Table (4.18) showed Blood Flow * Valves Cross-tabulation within the collected data

Blood Flow * Valves Cross-tabulation					
Count					
		Valves			Total
		Normal	Degenerative	Sclerotic	
Blood Flow	Normal	37	0	3	40
	Abnormal	7	2	1	10
Total		44	2	4	50

Chapter Five

Discussion, conclusion and recommendations

5.1. Discussion

This study has been done in the echo departments in National Ribat University Hospital and the Military Hospital – Sudan which aim to assess the effect of diabetes on heart by echocardiography and it was done on 50 diabetic patients 44% Male and 56% Female; in age between 25-85 years old. 96% of the patients are following up with their doctors for the diabetes and the other illnesses, and 94% on medication for diabetes.

The study variables measured were age, height, weight, BMI, Duration of diabetes, control of diabetes by blood sugar test, LVEF %, LVPW diameter (mm), IVS diameter (mm), LVESD (mm), LVEDD (mm), E/A ratio, Aortic root Diameter (mm), which having mean±SD 58.84±13.642, 162.46±9.451, 72.44±13.197, 27.632±5.8940, 8.07158±6.814764, 171.68±85.311, 61.26±11.634, 11.158±2.5025,12.092±3.0118, 30.554±7.7404, 47.254±5.9244, 1.208±0.8742, 29.624±3.1523, respectively.

The percentage of males in this study is 44% and Females is 56% which reflect the higher incidence of diabetes mellitus among the female which agreed with Devereux et al., 1999 that were more likely to be female. Another statement from Yetkin et al., 2015 that Female gender or being in post-menopausal period is another negative risk factor for poor Coronary collateral vessel development (CVD) in addition to DM in patients with chronic total occlusions (CTO).

The highest incidence of diabetes mellitus was among the age group 60-69 years old with frequency of 14 and percentage of 28%

The group of patients who had follow up represent 96% of the collected data; and those who were on medication represent 94% and this reflected in the blood test result which done in

76% of the research population with fasting blood glucose tests and 24% random blood glucose tests; among which the normal results are 54% that reflected the control of diabetes and 46% of abnormal results which indicated the uncontrolled blood glucose levels; the age group 60-69 years old had the highest frequencies 14 and 10 on usage of medication and normal blood glucose result respectively.

The cross-tabulated data of the blood flow and the valves within the collected data; the normal blood flow seen valves mainly through the normal valves in 37 cases and also in 3 cases through the sclerotic valves; the abnormal blood flow seen in seven cases through normal valves, two degenerative valves, and one sclerotic valve. SeeTable (4.18).

The study showed the predominanceof Diabetes in addition to Hypertension in the clinical history within the collected data with frequency of 29 and percentage of 58%.

The control group who had normal echo findingsin this study represent 16% of the collected data and84% had abnormal echo results. 88% of the patients had normal valves and the rest had 8% sclerotic changes and 4% degenerative changes, the normal blood flow had frequency of 40 and 80% of the collected data.

The highest incident of echo results abnormalities was among the age groups of 60-69 and 50-59years old. Those result concluded in Ischemic heart disease with diastolic dysfunction; followed by both left ventricular hypertrophy with good left ventricular function, and normal systolic function with diastolic dysfunction; then thenormal echo study, and the least for both groups with IHD with good LV systolic function and diastolic dysfunction with other abnormalities like dilated heart, clots or smoke in the heart chambers and high SPAP, and the other group of LVH with diastolic dysfunction and good systolic function; represented as 20%,18%,16% and14%respectively.

The relationship between the LV EF% and LVESD was investigated, the strong correlation showed an inverse relationship where the LV EF% decrease by 1.1851% for every one millimeter (mm) from LVESD because the increase of ESD can lead to decrease cardiac contractility where its affect the volume of blood ejected from the ventricles due to reduction of systolic pressure, ($y=-1.1851x + 97.468$) $R^2 = 0.6217$.

A significant correlation where noted between LVEF% and Lt Atrium diameter (mm) where its decreased by 0.6493 for every one millimeter (mm) increase in atrial diameter possibly due to reduced stroke volume for a given end-diastolic pressure according to a reduction in end-diastolic volume. ($y=-0.6493x +85.411$) $R^2 = 0.0553$.

The relationship between the LV EF% and Left Ventricular Posterior Wall(LVPW) was also investigated, the strong correlation showed the proportional relationship where the LV EF% increased by 1.1251 for every one millimeter of LVPW because its affecting the volume

ejected from the ventricle due to the increase of the systolic pressure.($y=1.1251x + 48.706$) $R^2 = 0.0586$.

The cross-tabulated data of the medical history and the echo results showed the highest incident of the left ventricular hypertrophy with good ventricular function among the group of population who had diabetes and hypertension; followed by ischemic heart disease IHD with good LV systolic function and diastolic dysfunction result and ischemic heart disease IHD with good LV systolic function and diastolic dysfunction with other abnormalities such as valvular abnormities, clots and smoke inside the heart chambers and cardiac dilatation.With the frequencies of 29, 10, 10, and 1 respectively.

5.2Conclusion

Heart can be affected by many morbidities regarding the diabetic status and this study was conducted to characterize and assess these morbidities in diabetic patients using echocardiography,

A total of 50 diabetic patients were studied by using echocardiography.This study has been done in the echo departments in National Ribat University Hospital and the Military Hospital – Sudan in the period from Aug – Sep 2015, aged between 25-85 years old; where 16% were diagnosed with normal results and 84% with significant morbidities.

The main study results were, the percentage of diabetes in females is higher than males, the most affected age group is 60-69 years old, the highest morbid result found is the ischemic heart disease with diastolic dysfunction 20% of the total research population, the mean ± SD for the LV EF% was 61.26±11.634, LV EF% correlated inversely with LVESD and Lt Atrium Diameter; whereas correlated proportionally with LVPW, cardiac valves and blood flow mostly were normal.

5.3Recommendation

- Further studies to be conducted with more detailed investigations for better patient care,
- To establish policies and procedure guidelines in the management of the heath care facilities.
- To catch the train of the state of art technology which will improve the outcome in the patient prognosis.

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