Sudan University of Sciences and Technology
College of Graduate Studies

Evaluation of Heart in Hypertensive Patients using Echocardiography

تقييم القلب في مرضى ارتفاع ضغط الدم باستخدام الموجات فوق الصوتية لتصوير القلب

A Thesis Submitted for Partial Fulfillment of Requirements
Of MSC Degree in Medical Diagnostic Ultrasound

By:

Sawad Mohammed Mousa Gampose

Supervisor:

Dr.Asmaa Ibrahim Ahmed Elamin

2016

Dedication

To my lovely parents & my brother Khalid

To my husband Amaar

To all my friends

Acknowledgement

Firist full of thanks to allah. And a lot of thanks and great fullness to my supervisor Dr Asmaa Ibrahim Ahmed Elamin for her help and guidance.

I owe my most sincere gratitude to Dr Esam Eldeen Eltayeb who give me the apportunity to work with him in the department of echocardiography in Atbra medical complex during the process of collection of data.

Abstract

This descriptive study carried out in order to assess the heart in hypertensive patients. The study was done in Atbara Medical Complex, Atbara city from the September to November 2016.

There were 50 patients, 30 female, 20 male all of them suffering from hypertension selected randomly their ages between 25-80years with mean age 57.5 years. Echocardiography using 2.5-6.5 MHZ sector transducer was performed.

The study found that the majority 78% of patients had a duration of hypertension from 1-5 years. The study found out that 82% of patients had a diastolic dysfunction , 46%of patients had valvular lesions, 32% of patients had left ventricular hypertrophy and 23% of patients had left atrium dilatation.

The study concluded that the hypertension can affect the heart structure and function and use of echocardiography was very important to evaluate this.

Finally the study recommended that any hypertensive patients must be examined by echocardiography as routine exam to follow up and to avoid the complications.

ملخص البحث

هذه دراسه وصفيه اجرييت لتقييم القلب فى المرضى المصابين بارتفاع ضغط الدم. اقيمت هذه الدراسه بمجمع عطبره الطبى في مدينة عطبره فى الفتره من سبتمبر الى نوفمبر 2016م .

حيث اخذ عدد 50 مريض 30 من الاناث و 20 من الذكور اعمارهم تتراوح مابين 25-80 سنه وكلهم يعانون من ارتفاع ضغط الدم وتم فحصهم باستخدام الموجات فوق الصوتيه للقلب.

وجدت الدراسه ان معظم المرضى بنسبة 78% كان لديهم ارتفاع ضغط الدم من مدة سنه الى خمسه سنوات. وايضا وجدت الدراسه ان 82% من المرضى كان لديهم خلل فى وظيفة القلب الانبساطيه, 46% من المرضى لديهم اصابات فيى صمامات القلب ,32% من المرضى لديهم تضخم فى بطين القلب الايسر و 23% من المرضى لديهم توسع فى اذين القلب الايسر.

وخلصت الدراسه ال ان ارتفاع ضغط الدم يؤثر فى بنية ووظيفة القلب واستخدام الموجات فوق الصوتيه لتصوير القلب مهم جدا لتقييم هذا.

واخيرا اوصت الدراسه بان اى مريض لديه ارتفاع فى ضغط الدم يجب ان يفحص بواسطة الموجات فوق الصوتيه لتصوير القلب كفحص روتينى للمتابعه وتجنب المضاعفات.

List of Contents

List of abbreviations

U S	Ultrasound
3D	Three dimension
2D	Tow dimension
LV	Left ventricle
LA	Left atrium
RV	Right ventricle
RA	Right atrium
IVS	Inter ventricular septum
SVC	Superior vena capa
EF	Ejection fraction
LVDD	Left ventricular diastolic function
LVSD	Left ventricular diastolic function
IVC	Inferior vena capa
LVPW	Left ventricular posterior wall
LVH	Left ventricular hypertrophy
ECHO	Echo cardio graphy
MHZ	Mega hertz
LAX	Long axis
SAX	Short axis
AO	Aorta
AV	Aortic valve
MV	Mitral valve
TV	Tricuspid valve
AR	Aortic rigurgetation
MR	Mitral rigurgetation
HTN	hyprertension
TTE	Trans thoracic echo
KAD	Coronary artery diseases
BP	Blood pressure

Chapter One

Introduction

1-1 Introduction:

The heart is most important vital organ in the body located within the chest in left side posterior to the sternum in the middle mediastinum ,relatively small conical approximately the size of person's clenched fist. It weight about 250 to 350 gram. It has four chambers the two superior receiving chambers are the atria, and the two inferior pumping chambers are the ventricles ,rotated such that its right side or border (right atrium and ventricle) is located more anteriorly while its left side or border (left atrium and ventricle) is located more posteriorly (shummys2004).

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 myocardium including the interventricular septum, on the other hand the left atrial filling with oxygenated blood which is send by both passive and active flow to the left ventricle via mitral valve (atrial systole) , however this is also left ventricular diastole, its systole is the ejection of blood to the aorta via aortic valve by strong and large myocardium of this chamber , this cardiac cycle controlled by conduction system of the heart and the autonomic nervous system (Roderick NM mac Sween & Keith Wholly,1998).

These alternate cycles of wall contraction and relaxation develop the so called blood pressure. Minimum blood pressure is essential to push the blood through blood vessels to the body tissues for nutrient supply and waste exchange. High blood pressure more than (140/90) is considered in some type of classification as hypertension. Hypertenstion (HTN) or high blood pressure ,sometime called arterial hypertension, is chronic medical condition in which the blood pressure in the arteries is elevated (ChobanianAV, Bakris GL, Black HR et al 2003).

This requires the heart work harder than normal to circulate blood through the blood vessels. Blood pressure is summarized by two measurements, systolic and diastolic, which depend on whether heart muscle is contracting (systole) or relaxed between beats (diastole) and equate to maximum and minimum pressure respectively. Normal blood pressure at rest is with the range of 100-140mmHg (systolic top reading) and 60-90mmHg (diastolic bottom reading). Hypertension is classified as either primary or secondary hypertension, about 90-95% of cases are categorized as primary hypertension which means high blood pressure with no obvious underlying medical cause, the remaining 5-10% of cases are secondary hypertension which caused by other condition that affect the kidneys, arteries, heart or endocrine system. Uncontrolled and prolonged elevation of BP can lead to a variety of changes in the myocardial structure, coronary vasculature, and conduction system of the heart. These changes in turn can lead to the development of left ventricular hypertrophy (LVH), coronary artery disease (KAD), various conduction system diseases, and systolic and diastolic dysfunction of the myocardium, (carreterooa, 2002).

Sound is a disturbance propagating in a material – air, water, body tissue or a solid substance. Each sound is characterized by its frequency and its intensity. Frequency is measured in hertz (Hz). Sound of frequency higher than 20 kHz cannot be perceived by the human ear and is called ultrasound. Use of ultrasound to examine the heart is called echocardiography (echo) is a safe, powerful, non-invasive and painless technique (Sam Kaddoura, 2002).

1-2 Statement of the problem:

Uncontrolled and prolonged elevation of BP can lead to a variety of changes in the myocardial structure, coronary vasculature, and conduction system of the heart. These changes in turn can lead to the development of left ventricular hypertrophy (LVH), coronary artery disease (CAD), various

conduction system diseases, and systolic and diastolic dysfunction of the myocardium. The problem here is that weather there is really cardiac complications or not and to what extent.

1-3 Research objectives:

1-3-1 General objectives:

To study the heart in hypertensive patients.

Specific objectives:

- To determine the duration of hypertension among patients.
- To evaluate the heart chambers dimensions.
- To evaluate the left ventricular walls thickness (left ventricular posterior wall-LVPW, interventricular septum-IVS).
- To characterize the valvular lesions that result from HTN (aortic, mitral, tricuspid and pulmonary stenosis and regurgitation).
- To identify the motion activity of the myocardium and the diastolic as well as the systolic dysfunction if ever in HTN.

1-4 Overview of the study:

Chapter one is an introduction, statement of the problem and study objectives. While Chapter two will include a highlight the literature reviews. Chapter three deals with the methodology, where it provides an outline of material and methods. While chapter four include the results, and finally Chapter five include discussion of results, conclusion and recommendation, references and appendices.

Chapter Tow

Literature review & previous studies

2-1 Anatomy of the heart:

The heart is relatively small, roughly the same size as your closed fist. It is about 12 cm long, 9 cm wide at its broadest point, and 6 cm thick, with an average mass of 250 g in adult females and 300 g in adult males. You can visualize the heart as a cone lying on its side. The pointed apex is formed by the tip of the left ventricle (a lower chamber of the heart) and rests on the diaphragm. It is directed anteriorly, inferiorly, and to the left. The base of the heart is its posterior surface. It is formed by the atria (upper chambers) of the heart, mostly the left atrium (Tortora & Derrickson, 2008).

The heart is divided by vertical septa into four chambers: the right and left atria and the right and left ventricles. The right atrium lies anterior to the left atrium, and the right ventricle lies anterior to the left ventricle.The walls of the heart are composed of cardiac muscle, the myocardium, covered externally with serous pericardium, the epicardium, and lined internally with a layer of endothelium, the endocardium (Snell, 2012).

2-1-1The heart valves:

There are four valves that regulate the blood flow through the heart,the tricuspid valve regulate the blood flow between the right atrium and right ventricle, the pulmonary valve controls blood flow from right ventricle into the pulmonary arteries, which carry blood to the lungs to pick up oxygen, the mitral valve lets oxygen-rich blood from lungs pass from the left atrium into the left ventricle ,the aortic valve opens the way for oxygen-rich blood to pass from left ventricle into the aorta (Michael Ronaldo 1976).

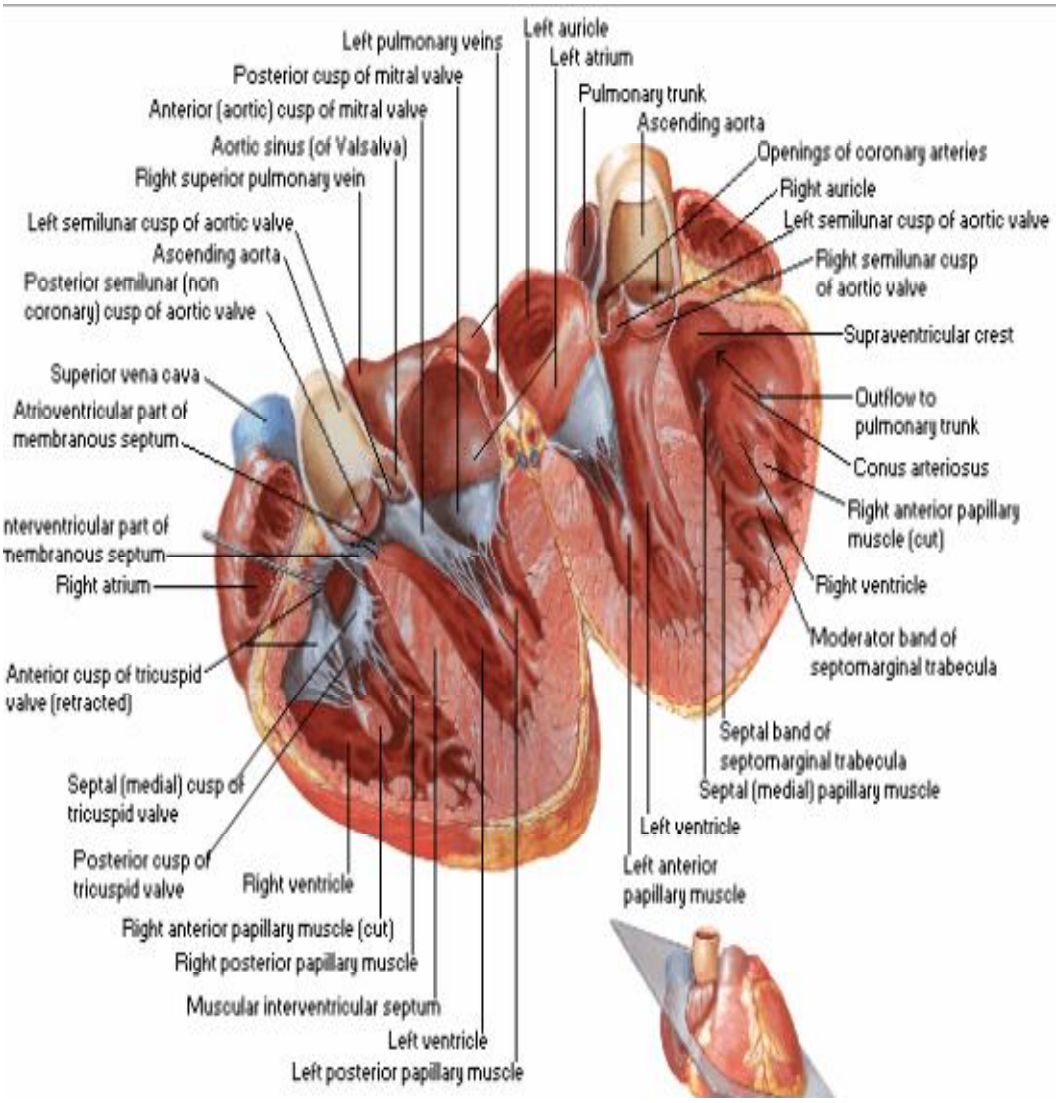

Fig (2-1) cross sectional image showing the anatomy of heart.(Sobota, 2006)

2-1-2 Blood supply to the heart:

Nutrients are not able to diffuse quickly enough from blood in the chambers of the heart to supply all the layers of cells that make up the heart wall. For this reason, the myocardium has its own network of blood vessels, the coronary or cardiac circulation. The coronary arteries branch from the ascending aorta and encircle the heart like a crown encircles the head. While the heart is contracting, little blood flows in the coronary arteries because they are squeezed shut.When the heart relaxes, however, the high pressure of blood in the aorta propels blood through the coronary arteries, into capillaries, and then into coronary veins (Tortora & Derickson, 2008) .

2-2 Physiology of the heart:

The heart is a muscular pump. The series of changes that take place within it as it fills with blood and empties is referred to as the **cardiac cycle.** The normal heart beats70 to 90times per minute in the resting adult and 130 to150 times per minute in the newborn child. Blood is continuously returning to the heart; during ventricular systole (contraction), when the atrioventricular valves are closed, the blood is temporarily accommodated in the large veins and atria. Once ventricular diastole (relaxation) occurs, the atrioventricular valves open, and blood passively flows from the atria to the ventricles. When the ventricles are nearly full, atrial systole occurs and forces the remainder of the blood in the atria into the ventricles. The sinuatrial node initiates the wave of contraction in the atria, which commences around the openings of the large veins and milks the blood toward the ventricles. By this means, blood does not reflux into the veins. The cardiac impulse, having reached the atrioventricular node, is conducted to the papillary muscles by the atrioventricular bundle and its branches. The papillary muscles then begin to contract and take up the slack of the chordate tendineae. Mean while, the ventricles start contracting and the atrioventricular valves close. The spread of the cardiac impulse along the

atrioventricular bundle and its terminal branches, including the Purkinje fibers, ensures that myocardial contraction occurs at almost the same time throughout the ventricles. Once the intraventricular blood pressure exceeds that present in the large arteries (aorta and pulmonary trunk), the semilunar valve cusps are pushed aside, and the blood is ejected from the heart. At the conclusion of ventricular systole, blood begins to move back toward the ventricles and immediately fills the pockets of the semilunar valves. The cusps float into apposition and completely close the aortic and pulmonary orifices (Snell, 2012).

2-3 Pathology of the heart:

2-3-1 Hypertensive heart diseases:

Elevated BP leads to adverse changes in cardiac structure and function in 2 ways: directly, by increased afterload, and indirectly, by associated neurohormonal and vascular changes .Elevated 24-hour ambulatory BP and nocturnal BP have been demonstrated to be more closely related to various cardiac pathologies, especially in black persons. The pathophysiologies of the various cardiac effects of hypertension differ and are described in this section.

2-3-1-1 Left ventricular hypertrophy (LVH):

LVH, defined as an increase in the mass of the left ventricle, is caused by the response of myocytes to various stimuli accompanying elevated BP. Myocyte hypertrophy can occur as a compensatory response to increased afterload. Mechanical and neurohormonal stimuli accompanying hypertension can lead to activation of myocardial cell growth, gene expression (of which some occurs primarily in fetal cardio myocytes), and, thus, to LVH. In addition, activation of the renin-angiotensin system, through the action of angiotensin II onangiotensin I receptors, leads to growth of interstitium and cell matrix components. In summary, the development of LVH is characterized by myocyte hypertrophy and by an

imbalance between the myocytes and the interstitium of the myocardial skeletal structure. Various patterns of LVH have been described, including concentric remodeling, concentricLVH, and eccentric LVH. Concentric LVH is an increase in LV thickness and LV mass with increased LV diastolic pressure and volume, commonly observed in persons with hypertension, this is a marker of poor prognosis in these patients. Compare concentric LVH with eccentric LVH, in which LV thickness is increased not uniformly but at certain sites, such as the septum. Although the development of LVH initially plays a protective role in response to increased wall stress to maintain adequate cardiac output, it later leads to the development of diastolic and, ultimately, systolic myocardial dysfunction (Douglas, 2014).

2-3-1-2 Left atrial abnormalities:

Frequently under appreciated, structural and functional changes of the left atrium are very common in patients with hypertension. The increased afterload imposed on the LA by the elevated LV end-diastolic pressure secondary to increased BP leads to impairment of the left atrium and left atrial (LA) appendage function, plus increased LA size and thickness. Increased LA size accompanying hypertension in the absence of valvular heart disease or systolic dysfunction usually implies chronicity of hypertension and may correlate with the severity of LV diastolic dysfunction. In addition to LA structural changes, these patients are predisposed to atrial fibrillation. Atrial fibrillation, with loss of atrial contribution in the presence of diastolic dysfunction, may precipitate overt heart failure (Karman, 2014).

2-3-1-3 Valvular disease:

Although valvular disease does not cause hypertensive heart disease, chronic and severe hypertension can cause aortic root dilatation, leading to significant aortic insufficiency. Some degree of hemodynamically insignificant aortic insufficiency is often found in patients with uncontrolled

hypertension. An acute rise in BP may accentuate the degree of aortic insufficiency, with return to baseline when the BP is better controlled. In addition to causing aortic regurgitation, hypertension is also thought to accelerate the process of aortic sclerosis and cause mitral regurgitation (Karman, 2014).

2-3-1-4 Heart failure:

Heart failure is a common complication of chronically elevated BP. Patients with hypertension fall into 1 of the following categories:

Asymptomatic but at risk of developing of heart failure - Stage A or B, per the American College of Cardiology (ACC) American Heart Association (AHA) classification, depending on whether or not they have developed structural heart disease as a consequence of hypertension Suffering from symptomatic heart failure - Stage C or D, per the ACC/AHA classification Hypertension as a cause of CHF is frequently under recognized, partly because at the time heart failure develops, the dysfunctioning left ventricle is unable to generate the high BP, thus obscuring the heart failure's etiology. The prevalence of asymptomatic diastolic dysfunction in patients with hypertension and without LVH may be as high as 33%.Chronically elevated afterload and the resulting LVH can adversely affect the active early relaxation phase and the late compliance phase of ventricular diastole (Douglas, 2014).

2-3-1-5 Diastolic dysfunction:

Diastolic dysfunction is common in persons with hypertension. It is often, but not invariably, accompanied by LVH. In addition to elevated afterload, other factors that may contribute to the development of diastolic dysfunction include coexistent coronary artery disease, aging, systolic dysfunction, and structural abnormalities such as fibrosis and LVH. Asymptomatic systolic dysfunction usually follows. Early LV diastolic dyssynchrony may be associated with LV remodeling and contribute to LVdiastolic dysfunction in

patients with hypertension. The level of diastolic dysfunction appears to correlate with increasing severity of hypertension, and peak myocardial systolic strain rate may be an independent factor in the extent of LV remodeling and diastolic function (Simon, 2013).

2-3-1-6 Systolic dysfunction:

Later in the course of disease, the LVH fails to compensate by increasing cardiac output in the face of elevated BP, and the LV cavity begins to dilate to maintain cardiac output. As the disease enters the end stage, LV systolic function decreases further. This leads to further increases in activation of the neurohormonal and renin-angiotensin systems, leading to increases in salt and water retention and increased peripheral vasoconstriction. Eventually, the already compromised LV is overwhelmed, and the patient progresses to the stage of symptomatic systolic dysfunction (Simon, 2013).

2-3-1-7 Myocardial ischemia:

Patients with angina have a high prevalence of hypertension. Hypertension is an established risk factor for the development of coronary artery disease, almost doubling the risk. The development of ischemia in patients with hypertension is multi factorial. Importantly, in patients with hypertension, angina can occur in the absence of epicardial coronary artery disease. The reason for this is 2-fold. Increased afterload secondary to hypertension leads to an increase in LV wall tension and transmural pressure, compromising coronary blood flow during diastole. In addition, the microvasculature beyond the epicardial coronary arteries has been shown to be dysfunctional in patients with hypertension, and it may be unable to compensate for increased metabolic and oxygen demand. The development and progression of arteriosclerosis, the hallmark of coronary artery disease, is exacerbated in arteries subjected to chronically elevated BP. Shear stress associated with hypertension and the resulting endothelial dysfunction cause impairment in the synthesis and release of the potent vasodilator nitric oxide. A decreased

nitric oxide level promotes the development and acceleration of arteriosclerosis and plaque formation. Morphologic features of the plaque are identical to those observed in patients without hypertension (Karman, 2014).

2-3-1-8 Cardiac arrhythmias:

Cardiac arrhythmias commonly observed in patients with hypertension include atrial fibrillation, premature ventricular contractions (PVCs), and ventricular tachycardia (VT). The risk of sudden cardiac death is increased. Various mechanisms thought to play a part in the pathogenesis of arrhythmias include altered cellular structure and metabolism, in homogeneity of the myocardium, poor perfusion, myocardial fibrosis, and fluctuation in afterload. All of these may lead to an increased risk of ventricular tachyarrhythmias. Atrial fibrillation (paroxysmal, chronic recurrent, or chronic persistent) is observed frequently in patients with hypertension. In fact, elevated BP is the most common cause of atrial fibrillation in the Western hemisphere. In one study, nearly 50% of patients with atrial fibrillation had hypertension. Although the exact etiology is not known, LA structural abnormalities, associated coronary artery disease, and LVH have been suggested as possible contributing factors. The development of atrial fibrillation can cause decompensation of systolic and, more importantly, diastolic dysfunction, owing to loss of atrial kick, and it also increases the risk of thromboembolic complications (Karman, 2014).

2-4 Ultrasound Imaging:

2-4-1 physics:

Sound is a disturbance propagating in a material – air, water, body tissue or a solid substance. Each sound is characterized by its frequency and its intensity. Frequency is measured in hertz (Hz). Sound of frequency higher than 20 kHz cannot be perceived by the human ear and is called ultrasound. Ultrasound results from the property of certain crystals to transform electrical oscillations (varying voltages) into mechanical oscillations

(sound). This is called the piezoelectric effect . The same crystals can also act as ultrasound receivers since they can effect the transformation in the opposite direction (mechanical to electrical). At the core of any echo machine is this piezoelectric crystal transducer. When varying voltages are applied to the crystal, it vibrates and transmits ultrasound. When the crystal is in receiving mode, if it is struck by ultrasound waves, it is distorted. This generates an electrical signal which is analysed by the echo machine. The signals that return to the transducer therefore give evidence of depth and intensity of reflection. These are transformed electronically into grey scale images on a TV screen or printed on paper. Echocardiography (echo) the use of ultrasound to examine the heart is a safe, powerful, non-invasive and painless technique. Echo studies are carried out using specialized ultrasound machines. Ultrasound of different frequencies (in adults usually 2–4 MHz) is transmitted from a transducer (probe) which is placed on the subject's anterior chest wall. This is transthoracic echo (TTE). The transducer usually has a line or dot to help rotate it into the correct position to give different echo views (Sam Kaddoura, 2002).

2-4-2 echo technique:

Three echo methods are in common clinical usage:

2-4-2-1 Tow dimensional echocardiography:

Two-dimensional (2D) images form the basis of the echocardiographic study, providing structural and functional information as well as guiding the use of M-mode and Doppler techniques. Ultrasound waves generated from the ultrasound transducer travel to the heart and are then reflected back to the transducer are analyzed and displayed on a TV screen to give real-time imaging of the heart chambers, valves and blood vessels (Solomon, 2003).

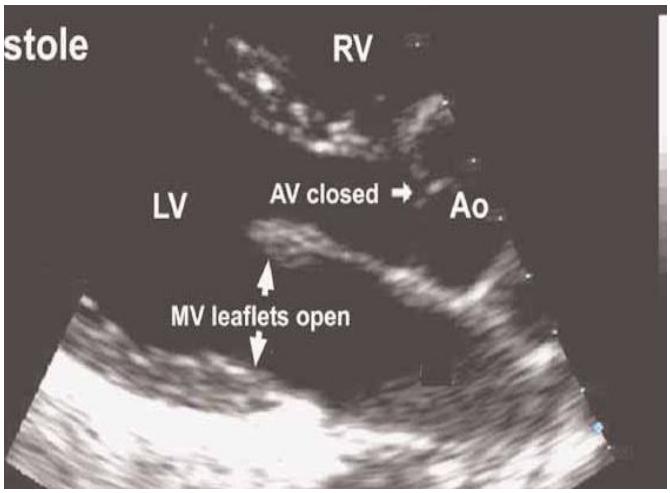

Fig 2-2 2D Image demonstrating the following cardiac structures: RV, right ventricle; LV, left ventricle; AV, aortic valve, Ao, aorta; MV, mitral valve. (Solmon, 2003)

2-4-2-2 M mode echocardiography:

M-mode images (M stands for "motion") can be thought of as a one dimensional, or "ice-pick" image, recorded over time. The high resolution of M-mode images, and the ability to correlate them with a simultaneously recorded electrocardiogram, makes M-mode the image of choice for many measurements (Solomon, 2003).

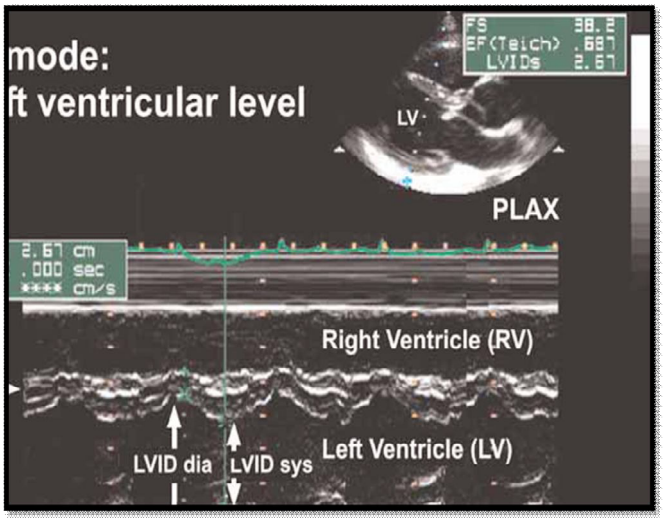

Fig 2-3 M-mode through the left ventricle. From this view, measurements of left ventricular wall thickness, and end-diastolic and end systolic diameter can be made. (Solmon, 2003)

2-4-2-3 Doppler echocardiography:

Doppler echocardiography is used to measure blood flow; it can assess flow velocity, direction, and turbulence. It can be used to measure the severity of valvular narrowing (stenosis), to detect valvular leakage (regurgitation) and can show intracardiac shunts such as ventricular septal defects (VSDs) and atrial septal defects (ASDs) (Solomon, 2003).

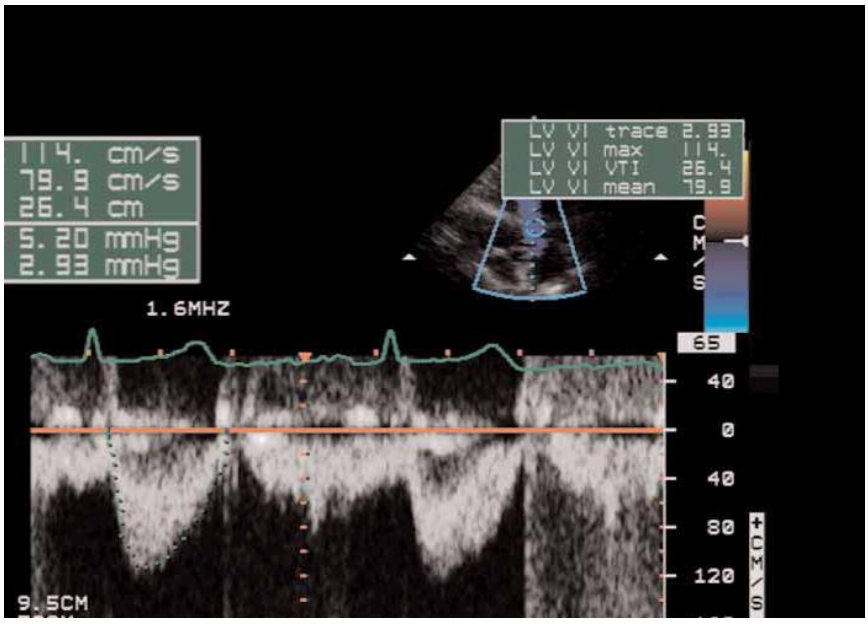

Fig 2-4 Pulsed Doppler through the left ventricular outflow tract.

The waveform demonstrates the velocity of blood (*y*-axis), with time on the *x*-axis. (Solmon, 2003)

2-4-3 Normal sonographic appearance of heart:

Echo studies are carried out using specialized ultrasound machines. Ultrasound of different frequencies (in adults usually 2–4 MHz) is transmitted from a transducer (probe) which is placed on the subject's anterior chest wall. This is transthoracic echo (TTE). The transducer usually has a line or dot to help rotate it into the correct position to give different echo views. The subject usually lies in the left lateral position and ultrasound jelly is placed on the transducer to ensure good images. An echo examination usually takes 15–20minit. The standard echocardiographic images are acquired by transducer maneuvers within four standard anatomical positions—left parasternal (or simply parasternal), left apical, subcostal, and suprasternal (Solomon, 2003).

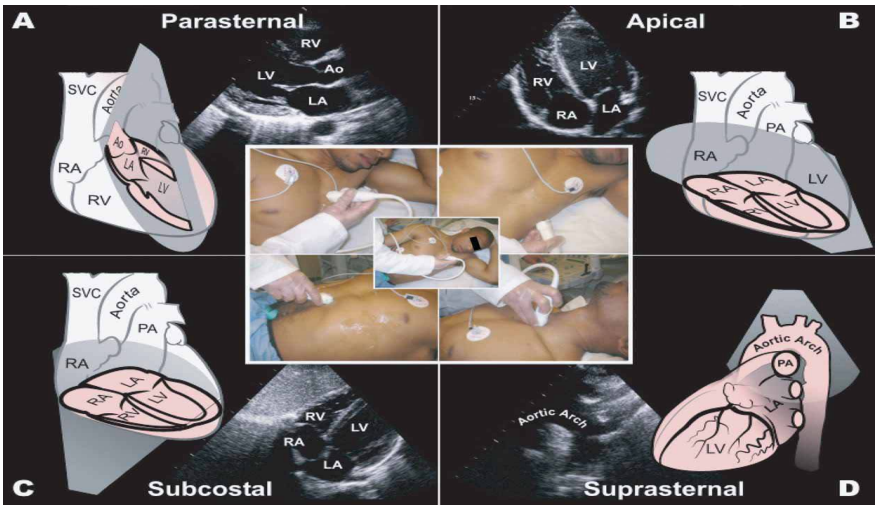

Fig 2-5 showing transducer placement for each of the major echocardiographic views: **(A)** parasternal location for parasternal long and short-axis; **(B)** apical location for apical four-, two-chamber and long-axis views; **(C)** subcostal location for subcostal views; **(D)** suprasternal location for suprasternal notch view.(Solmon,2003)

2-5 previous studies:

Study (1):

D Savage, etal 1979 their study include 234 subject with mild to moderate systemic hypertension examined by echocardiography. After adjusting the echocardiographic values for age and body surface area, they found abnormally increased ventricular septal thickness in 61% of patients, increased left atrial and left ventricular internal dimension in 5.7% and decreased left ventricular ejection fraction in 15% of subjects.

Study (2):

KanitkarSA, etal 2013 they are studied hypertensive change by 2D echocardiography in elderly patients with isolated systemic hypertension. Their study conclude 76 patient more than 60 years of age, 45 were males and 31 were females ,48 patients (63.1%) had diastolic dysfunction ,46 patients (60.5%) had LVH and 36 patients (47.4%) had systolic dysfunction.

Study (3):

A Akintunde, etal 2008 their study to determine the prevalence diastolic abnormalities in 100 hypertensive patients theirs result include diastolic dysfunction was detected 85% of 100 hypertensive patients. 76% had early diastolic dysfunction while 9% shows late diastolic dysfunction, only 15% of these patients are likely to have normal diastolic function.

Study (4):

Maria A Martinez, etal 2003 their study conclude 250 patients recently diagnosed with mild hypertension underwent clinical evaluation including electrocardiography (ECG), microalbuminuria measurement , 24h blood monitoring and ECHO. Level of cardiovascular risk was sratified, initially using routine procedures including ECG to assess target organ damage and then again after detection of LVH by ECHO. The result shows the frequency of echocardiographic LVH was 32%, substaintially higher than that detected by ECG (9%). Initial cardiovascular risk stratification yielded the following

result: 30% low risk, 49% medium risk, 16% high risk and 5% very high risk subjects. The detection of LVH by ECHO provoked a significant change in the risk stata distribution, particularly in those patients initially classified as being at medium risk. In this group, 40% of subjects were reclassified as high risk subjects according to ECHO information. The new classification was as follow:23% low risk, 30% medium risk, 42% high risk and 5% very high risk subjects. Their conclusion show substantial proportion of mildly hypertensive patients presenting in primary care have LVH detected by ECHO.

Study (5):

Mohammed, 2015 he study the role of echocardiography in heart diseases especially left ventricle of patients with hypertension in 50 subjects (mean age 56-70 year 46%), gender(27% mles,54% females. The study conclude that left ventricle hypertrophy found in many stages, moderate (38%) and severe (14%).

Chapter Three

Material and methods

3-1 Materials:

3-1-1 Machine:

An echocardiography machine **(MY LAB 30GOLD CARDIOVASCULAR)** ,with Doppler and M-mode capability is used , the probe of sector type (2.5-6.5 MHZ) .

3-1-2 Sampling:

50 hypertensive patients selected randomly.

3-1-2-1 inclusion criteria:

Hypertensive patients.

3-1-2-2 exclusion criteria:

All cardiac patients not hypertensive.

3-2 Methods:

The study was conducted in **Atbara medical complex** in Atbara city from the first of September to the thirty of November 2016.

3-2-1 technique:

3-2-1-1 preparation:

Patient for echocardiography not need preparation.

3-2-1-2 position of patient:

The patient lie supine and rotated slightly to the left with left arm supporting the head.

3-2-3-3 scanning technique:

Start with long axis plane (LAX) this plane runs from the right shoulder to the left hip and cuts the left side of the heart along its long axis, the LA , LV & AO are imaged with MV & AV. Then scan the short axis plane (SAX) this is perpendicular to LAX. It runs from the left shoulder to the right hip a cuts the heart in cross section, cross sectional images from apex to the base of the heart can be imaged by serial tilt of the transducer and short axis views are obtained at different levels, from the aorta to the LV apex. Continue scan by shifting the transducer to the apex of the heart to scan (Apex 4- chambers view). This plane runs parallel to the sternum and chest wall toward the

right shoulder and cuts all the 4-chambers (LA, LV, RA, RV) & AV, MV and TV of the heart.

3-2-2 Image interpretation:

Both normal and abnormal should be recorded. Variation from normal size should be accompanied by measurements. image should be labelled with patient identification , examination date , side (right & left) of anatomic position and it printed on thermal paper ,CD or DVD.

3-2-3 data analysis:

All data collected from patient were entered in a data sheet which is prepared for study contain patient age, sex and sonographic finding were analyzed by using SPSS under windows, where frequency distribution, cross-tabulation and line graph were used.

Chapter Four

Results

This study was carried out on 50 hypertensive patients were examined by echocardiography with theirs following result according to age, gender duration of disease and ultrasound finding.

Table 4.1 show statistical parameters for age and systolic function of heart for all patients:

	Age	Systolic %
Mean	57.50	70.36
Median	55.50	70.00
Std. Deviation	11.817	8.822
Minimum	25	30
Maximum	80	84

Table 4.2 show frequency distribution for Gender:

Gender	Frequency	Percentage
Female	30	60 %
Male	20	40 %

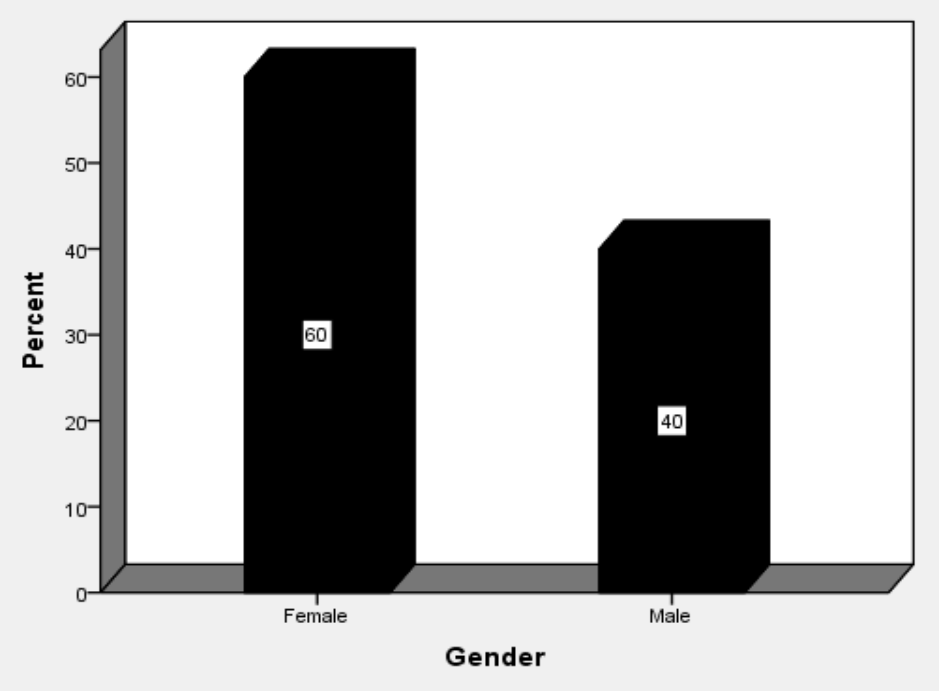

Figure 4.1 show frequency distribution for Gender.

Table 4.3 show frequency distribution for duration of disease:

Duration of Disease	Frequency	Percentage
1	12	24 %
2	2	4 %
3	11	22%
4	1	2%
5	13	26%
6	4	8%
8	1	2%
10	2	4%
15	1	2%
20	2	4%
35	1	2%

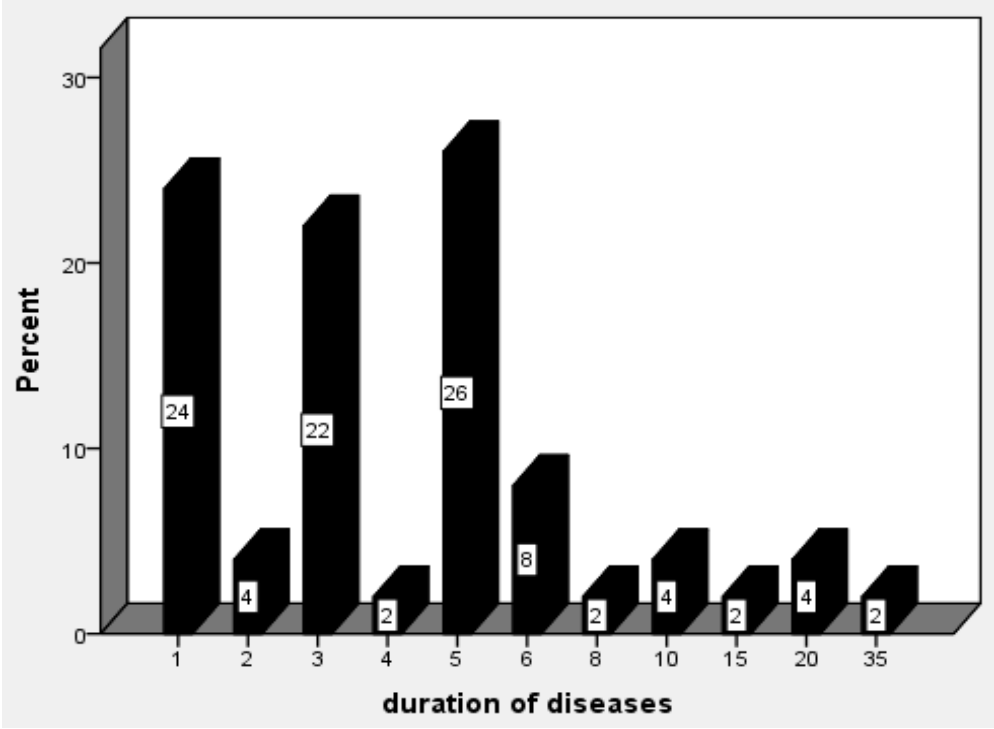

Figure 4.2 show frequency distribution for duration of disease.

Table 4.4 show frequency distribution for Chambers Dimension:

Chambers Dimension	Frequency	Percentage
Normal	38	76 %
Dilated LA	11	22 %
All Chambers	1	2 %

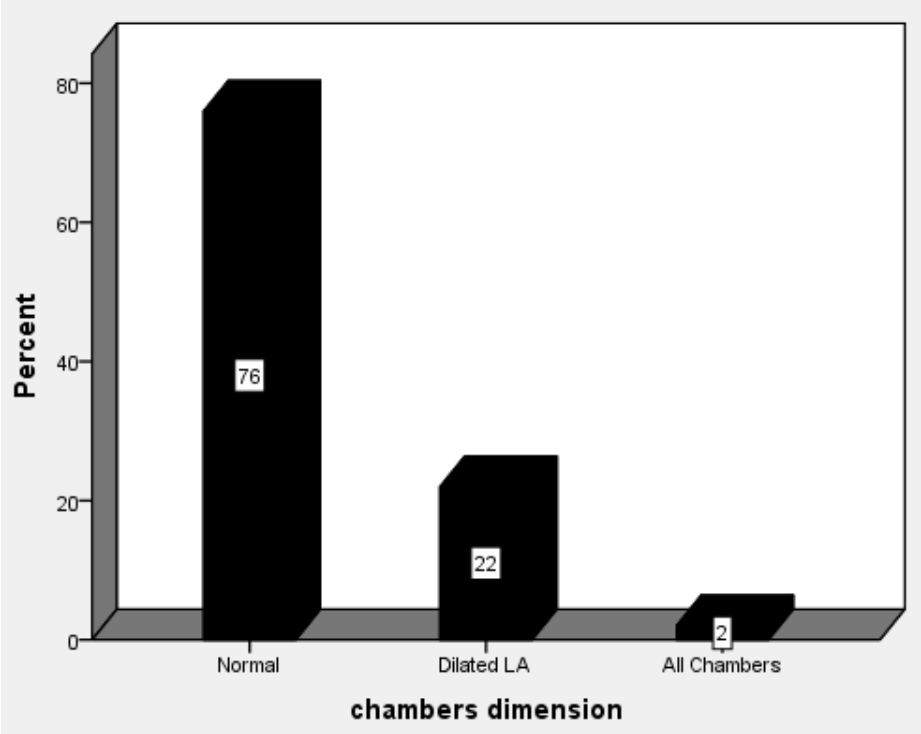

Figure 4.3 show frequency distribution of chambers dimension.

Table 4.5 show frequency distribution of LV walls Thickness:

LV walls Thickness	Frequency	Percentage
Normal	32	64 %
Mild LVH	16	32 %
Moderate LVH	2	4 %

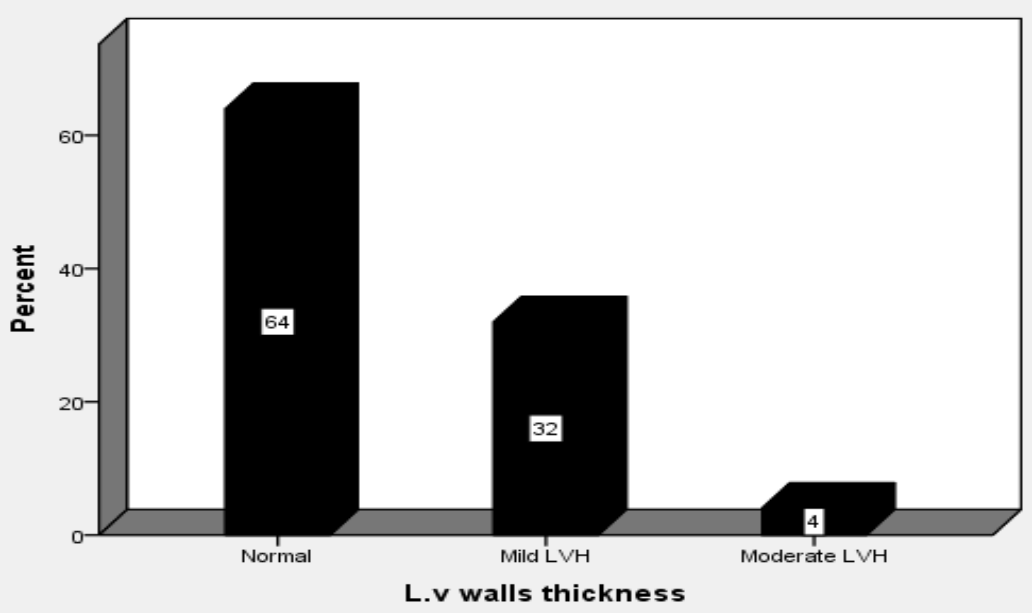

Figure 4.4 show frequency distribution of LV walls Thickness.

Table 4.6 show frequency distribution of Valvular Lesions:

Valvular Lesion	Frequency	Percentage
Normal	27	54 %
Thick MV	7	14 %
Thick AV	9	18 %
Mild AR	4	8 %
Mild MR	2	4 %
Moderate MR	1	2 %

Table 4.7 show frequency distribution of Regional wall motion:

Regional wall motion	Frequency	Percentage
NO	49	98 %
Global hypokinsea	1	2 %

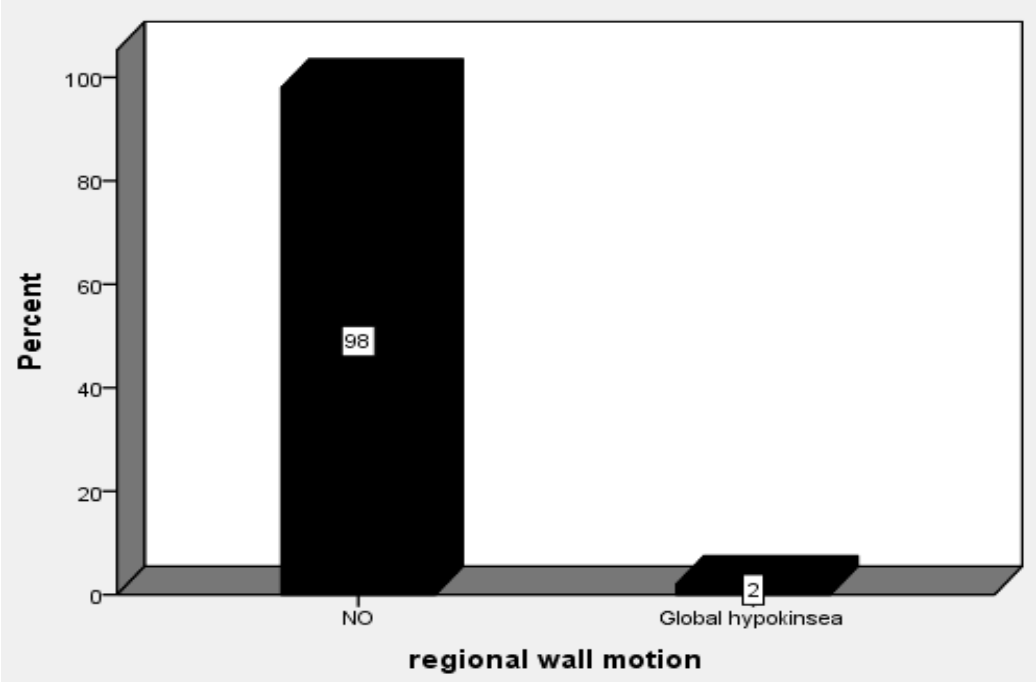

Figure 4.5 show frequency distribution of Regional wall motion.

Table 4.6 show frequency distribution of Diastolic Function:

Diastolic Function	Frequency	Percentage
Advance diastolic dysfunction	3	6 %
Grade1 diastolic dysfunction	38	76 %
Normal	9	18 %

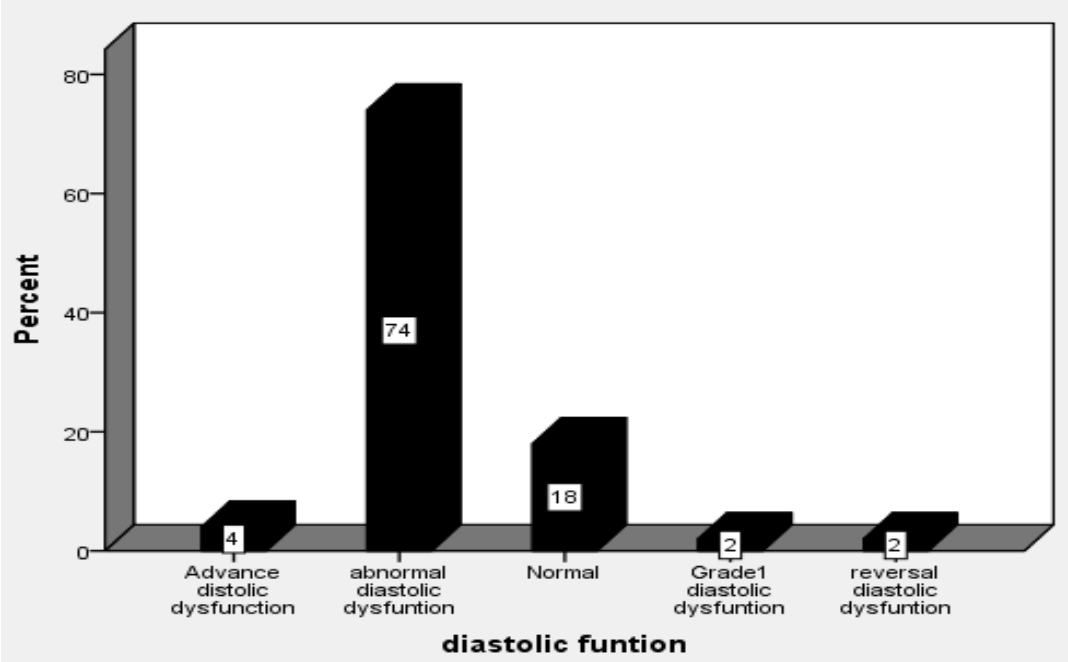

Figure 4.6 show frequency distribution of Diastolic Function.

Table 4.9 show correlation between of Diastolic Function with gender:

diastolic function * Gender Cross tabulation

diastolic function	Gender		Total
	Female	Male	
Advance diastolic dysfunction	1	2	3
Normal	6	3	9
Grade1 diastolic dysfunction	23	15	38
reversal diastolic dysfunction	0	1	1
Total	30	20	50

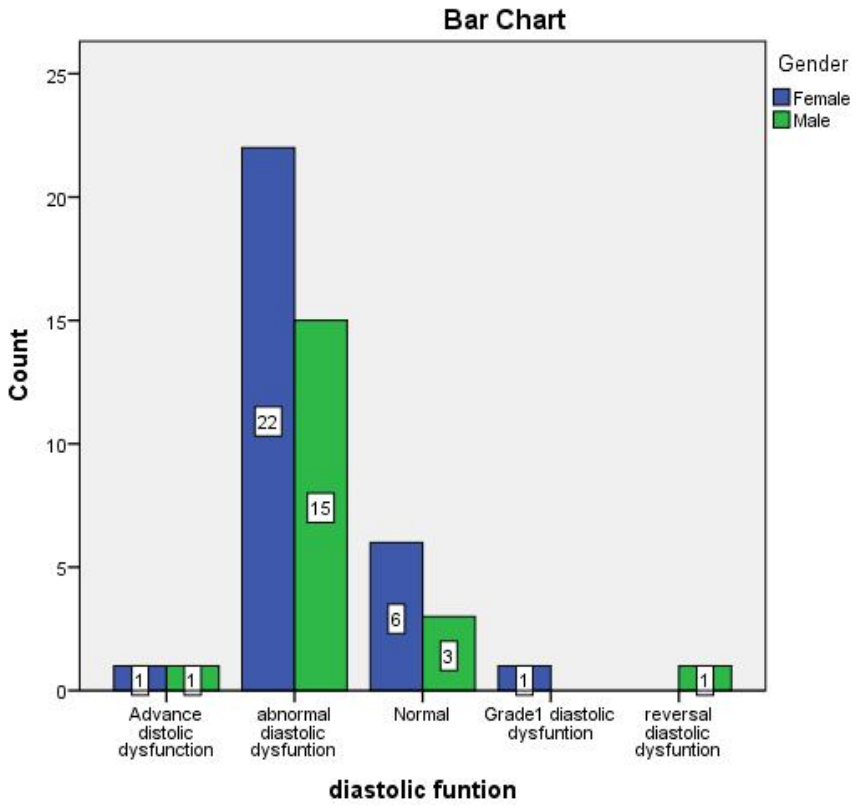

Figure 4.7 show correlation between of Diastolic Function with gender.

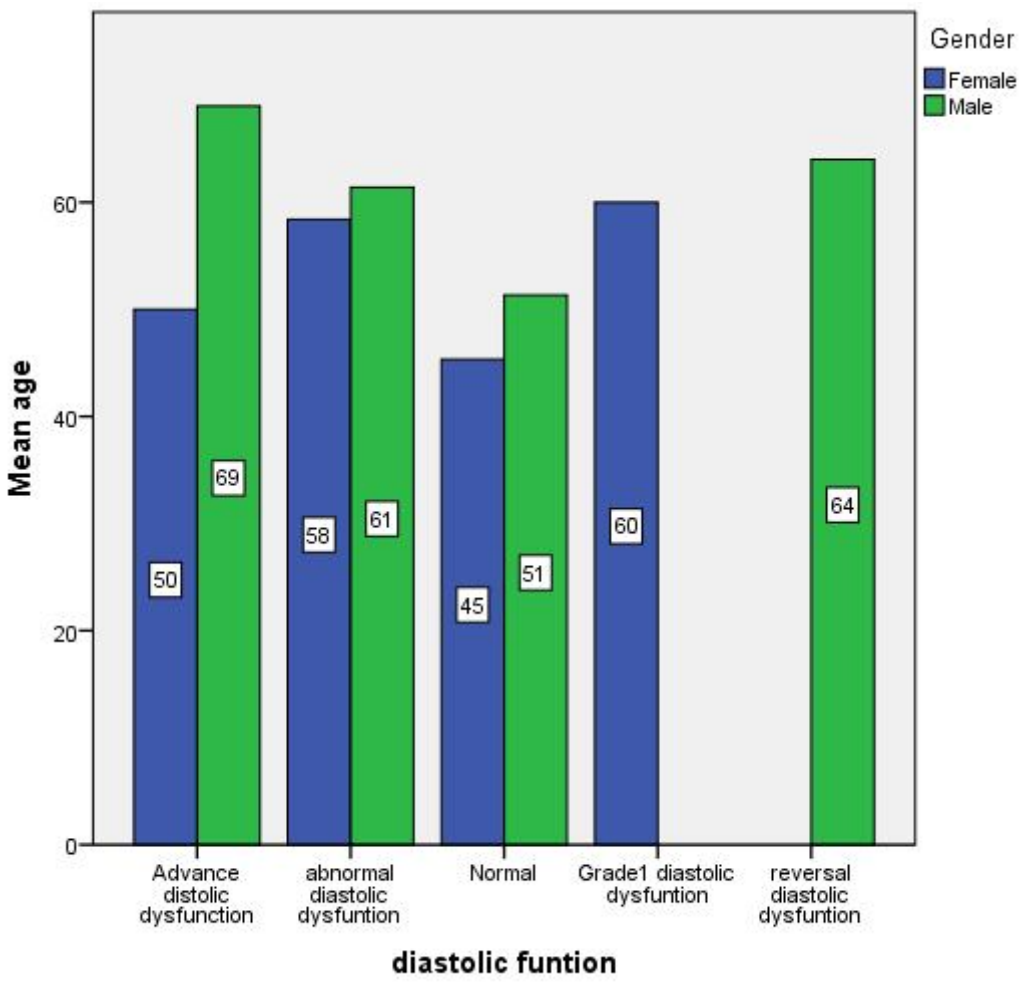

Figure 4.8 show correlation between of Diastolic Function with gender and Age.

CHAPTER FIVE

5-1discussion:

This study has been done in echo department of Atbara medical complex ,Atbara city to study the heart of hypertensive patients. The data of this study was collected from 50 hypertensive patients.

The result of this study reveal that the age of the patients ranged between 25-80 years with mean age 57.5yars, table (4-1).

Table (4-2) shows the distribution of gender, and it indicate that 60% (30) of patient was female and 40% (20) was male.

Table (4-3) shows the distribution of duration of disease and it indicate that the majority of patients 78% had a duration of hypertension from 1 to 5years.

Table (4-4) shows the distribution of chambers dimension and it indicate that 76% (38 of selected patients) had normal chambers dimension but 22% (11 of patients) had dilated left atrium and 2% (1 of selected patients) had dilated all chambers.

Table (4-5) shows the distribution of left ventricular wall thickness and it indicate that 32% (16 of patients) had mild left ventricular hypertrophy, this agree with previous study of (Maria,A, etal, 2003) and 4% (2 of patients) had moderate left ventricular hypertrophy.

Table (4-6) shows the valvular lesions that affect the heart of hypertensive patients of this study and it reveal that 46% (23 of patients) had abnormal valves represented as :(18%(9) of patients had thick aortic valve, 8%(4) of patients had mild aortic regurgitation, 14%(7) of patients had thick mitral valve, 4% (2) of patients had mild mitral regurgitation and 2% (1) of patients had moderate mitral regurgitation).

Table (4-7) shows the distribution of regional wall motion and it reveal that 98% (49) of patient had no regional wall motion but 2% (1) only had global hypokinsea.

Table (4-8) shows the distribution of diastolic function and it indicate that 82% (41) of patients had abnormal diastolic function, 76% (38) of patients had early (grade 1) diastolic dysfunction and 6% (3) of patients had advance diastolic dysfunction. This agree with previous study of (A Akintunde, etal, 2008).

The correlation between diastolic function * gender and age demonstrate that 9 patients 18% of total number of sample 6 female and 3 males with mean age 45, 51 years respectively had normal diastolic function, 38 patients 76% of total number of sample 23 female, 15 male with mean age 58, 61years respectively had grade 1 diastolic dysfunction and 3 patients 6% of total number of sample 1 female, 2 male with mean age 50, 59 years respectively had advance diastolic dysfunction as show in table (4-9) and figure (4-8).

From the statistical study and analysis that done, the study found that the overall incidence of hypertension among the patient is high in women than men. Also the study found that the hypertension was affect the heart function mainly diastolic function as well as it was affect the heart morphology mainly the left ventricular walls thickness (LVH) and left atrium size.

5-2Conclusions:

The study conclude that the hypertension was affect on heart morphology and function mainly diastolic function ,left ventricular walls thickness and left atrium size and this can be detected and evaluated by echocardiography which is available, relatively low cost and absence of ionizing radiation. Early diagnosis of hypertensive heart diseases and institution of treatment will be reduce the morbidity and improve the outcomes by preventing future development of heart failure.

5-3Recommendation:

1. Further studies with more sample.
2. In all emergency departments must be modern echocardiography machine with qualified trained echocardiographer.
3. Any hypertensive patient must do echocardiography as routine follow up.
4. It is important to educate patients about the nature of their disease and the risks associated with untreated hypertension.

References:

- A Akintund, P Akinwusi, O Opaadijo, R Adebayo, S Ogunyemi, 2008 prevalence of echocardiophic indices of diastolic dysfunction in patients with hypertension at Teriary Health Faccility in Negeria. The Internal Journal of Cardiology Volume6 Number2.
- Amico AF, Lichtenberg GS, Reisner SA, Stone CK, Schwartz RG,Meltzer RS.1989 Superiority of visual versus computerized echocardiographic estimation of radionuclide left ventricular ejectionfraction. Am Heart J ;118:1259–1265.
- Becher H, Burns PN. 2000 Handbook of Contrast Echocardiography: LV function and Myocardial Perfusion. Frankfurt: Springer-Verlag, 2.
- Cho GY, Park WJ, Han SW, Choi SH, Doo YC, Oh DJ, Lee Y.2004 Myocardial systolic synchrony measured by Doppler tissue imaging as a role of predictor of left ventricular ejection fraction improvement in severe congestive heart failure. J Am SocEchocardiogr;17:1245–1250.
- Chummy.Sinnatamby,2004 last's anatomy regional &Applied 10th edition, Churchill Livingstone,China,
- Cohen GI, Pietrolungo JF, Thomas JD, Klein AL,1996 A practical guide to assessment of ventricular diastolic function using Doppler echocardiography. J Am CollCardiol;27:1753–1760.
- Devereux RB, Alonso DR, Lutas EM, et al,1986 Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol;57:450–458.
- Douglas L. Man MD ,Douglas P. Zipes MD. Peter LibyMD.Robert O. Bono MD MS, 2014 Braunwalds Heart Disease, 10th edition.
- Kanitkar SA, Kalyan M Gaikwad AN, Singh N, Bhate AS, Midhum M, 2013 echocardiographic assessment of hypertensive change in

elderly patients with isolated systolic hypertension and it is correlation with pulse pressure.Med j DY patilUniv (cited 2016 Dec 17)”6:75-8.

- Michael Ronald Eleson Dean, 1976 Basic Anatomy and Physiologyfor Radiographers.
- Roderick NM Mac Sween& Keith Wholly, mur’s,1998 text book of pathology, 13th edition, Arnold international edition New York .
- Sam Kaddoura ,2002echo made easy, first edition,Churchill Livingstone, China.
- Simon Suvarna ,2003 cardiac pathology.
- Snell R.S.,2012 clinical anatomy for medical students ,9th edition ,philladellpia ,Lippincott Willams and Wilkins.
- Solomon SD. Principles of echocardiography. In: Braunwald E, Goldman L., eds. Primary Cardiology, 2nd ed. Philadelphia: Saunders-Elsevier, 2003.
- Tortora J.G &Derrickson 13, 2011 principle of anatomy and physiology 13th edition, Newgersy, John Wily &Sons, inc.

Appendixes

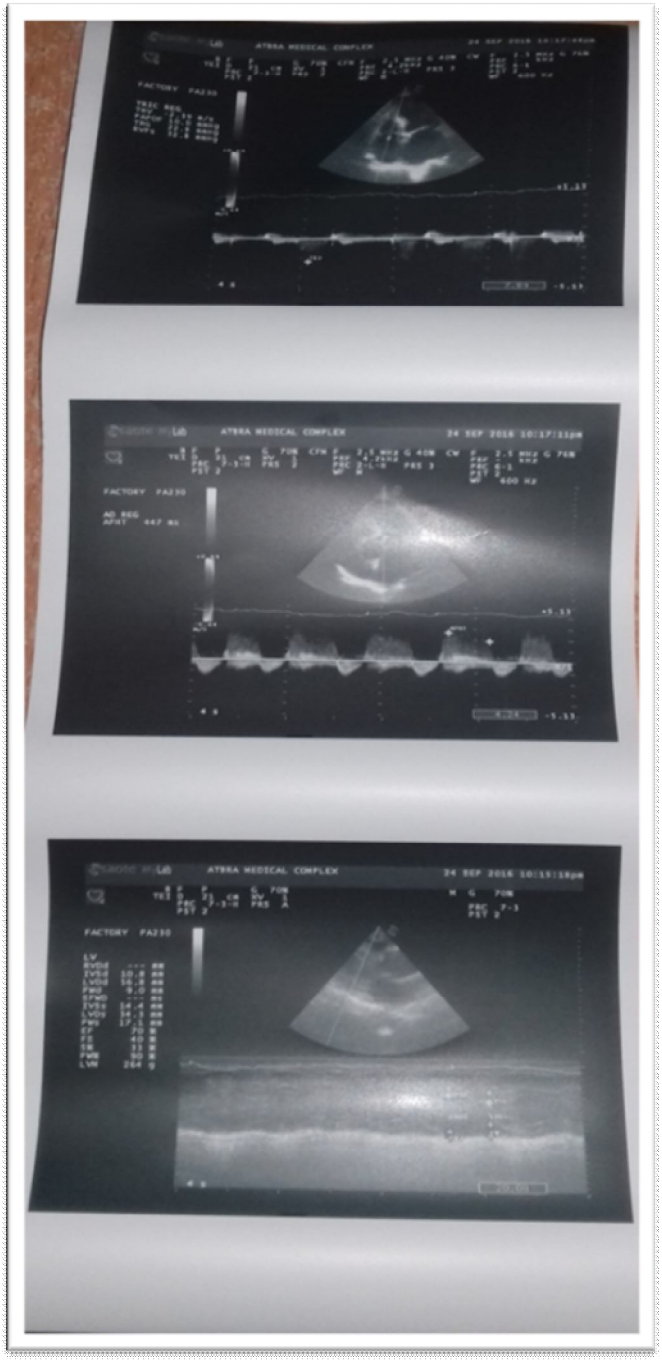

Image NO (1) 70 years old male, LAX view showing thickening of aortic valve with moderate aortic regurgitation.

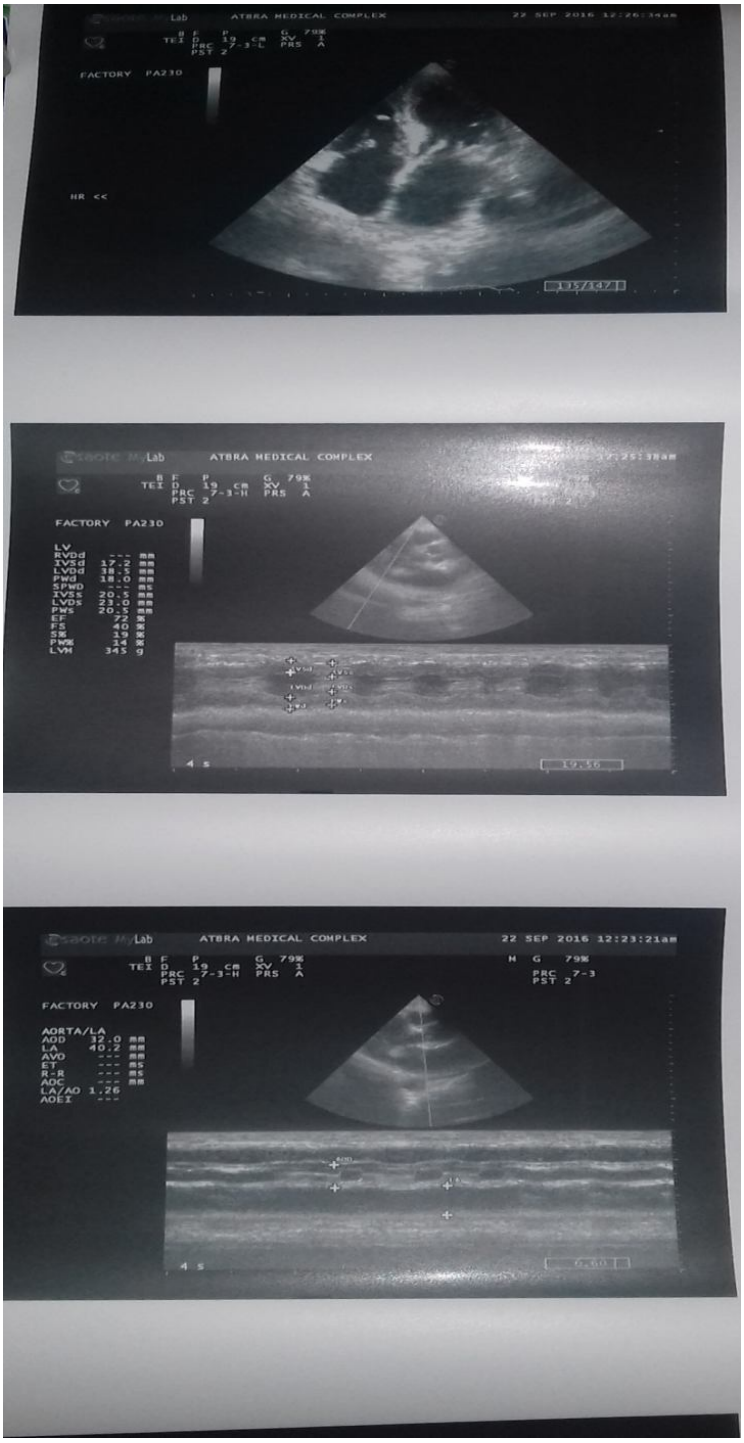

Image NO (2) 50 years old female, LAX view showing moderate LVH.

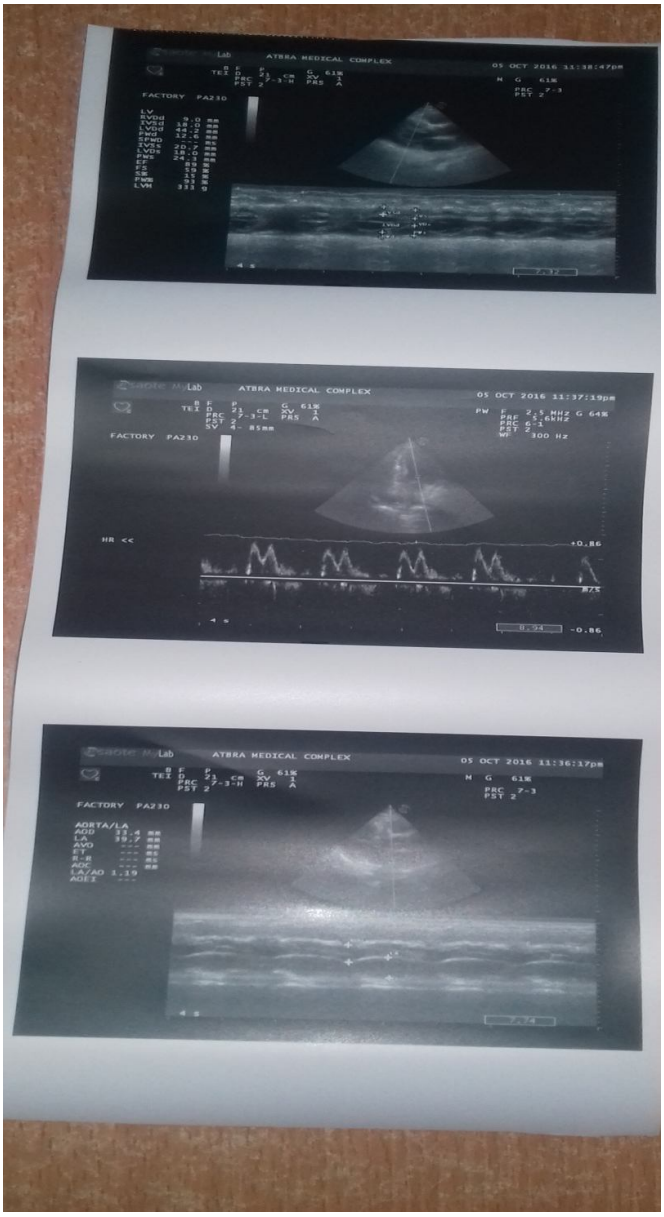

Image NO(3) 53 years old female, LAX, and apical 4chamber view showing LVH and diastolic dysfunction.

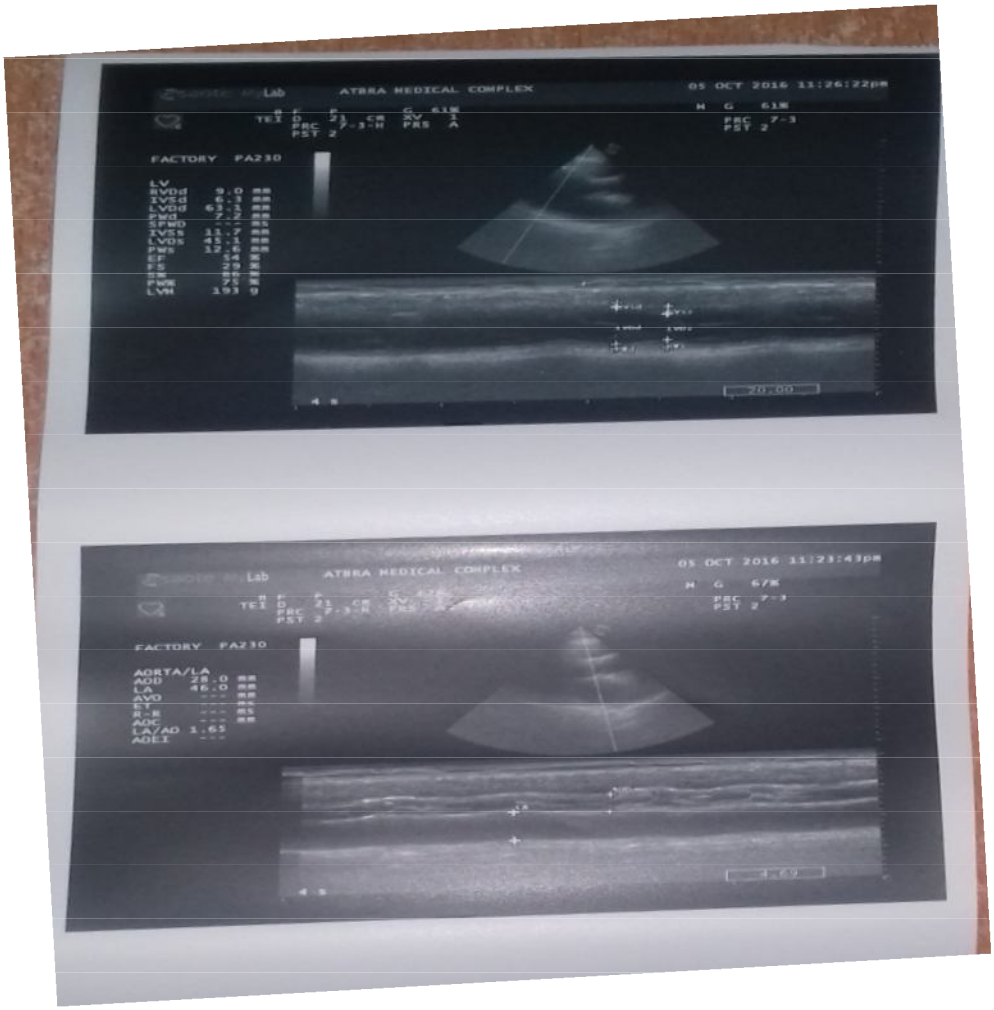

Image NO(4) 80 years old male, LAXview showing dilated LA.

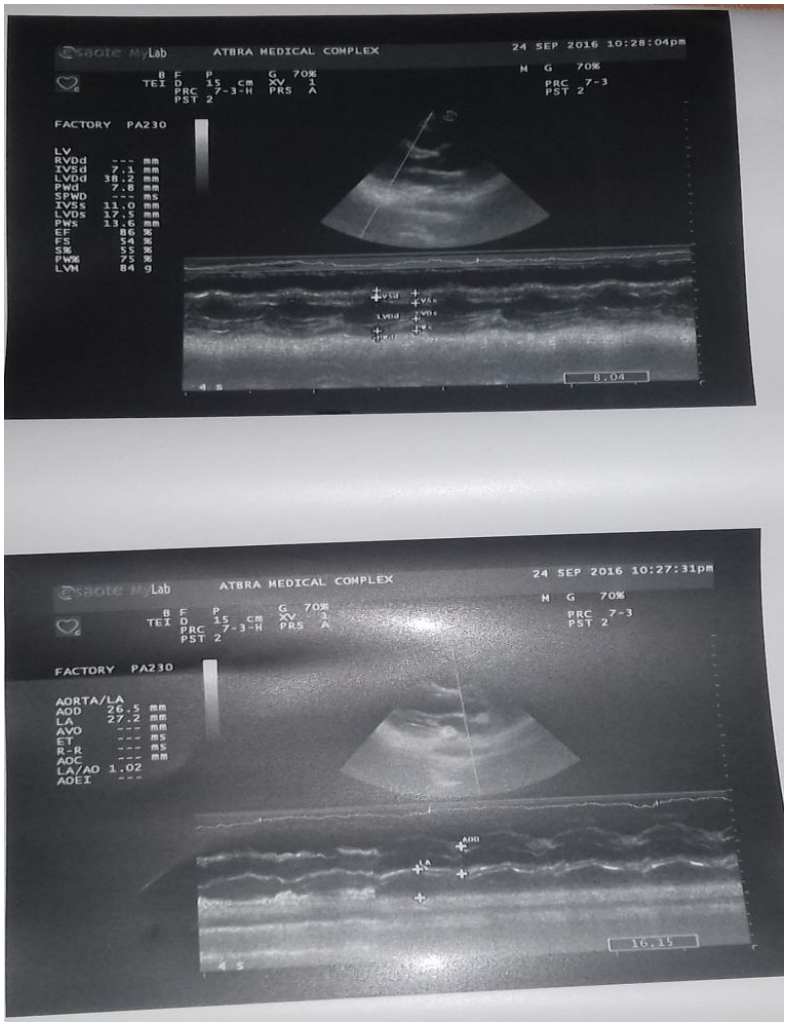

Image NO(5) female 40 years old, LAXview showing thickening of mitral valve.

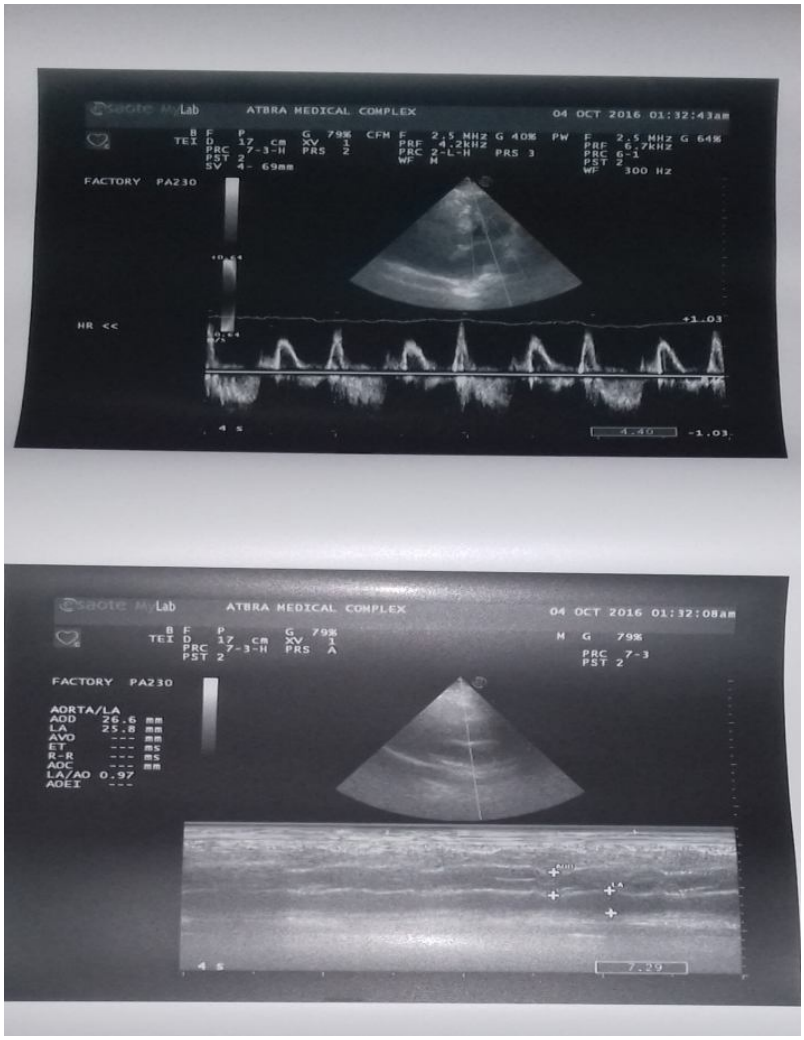

Image NO(6) 47 years old female LAXview showing mild LVH and diastolic dysfunction.

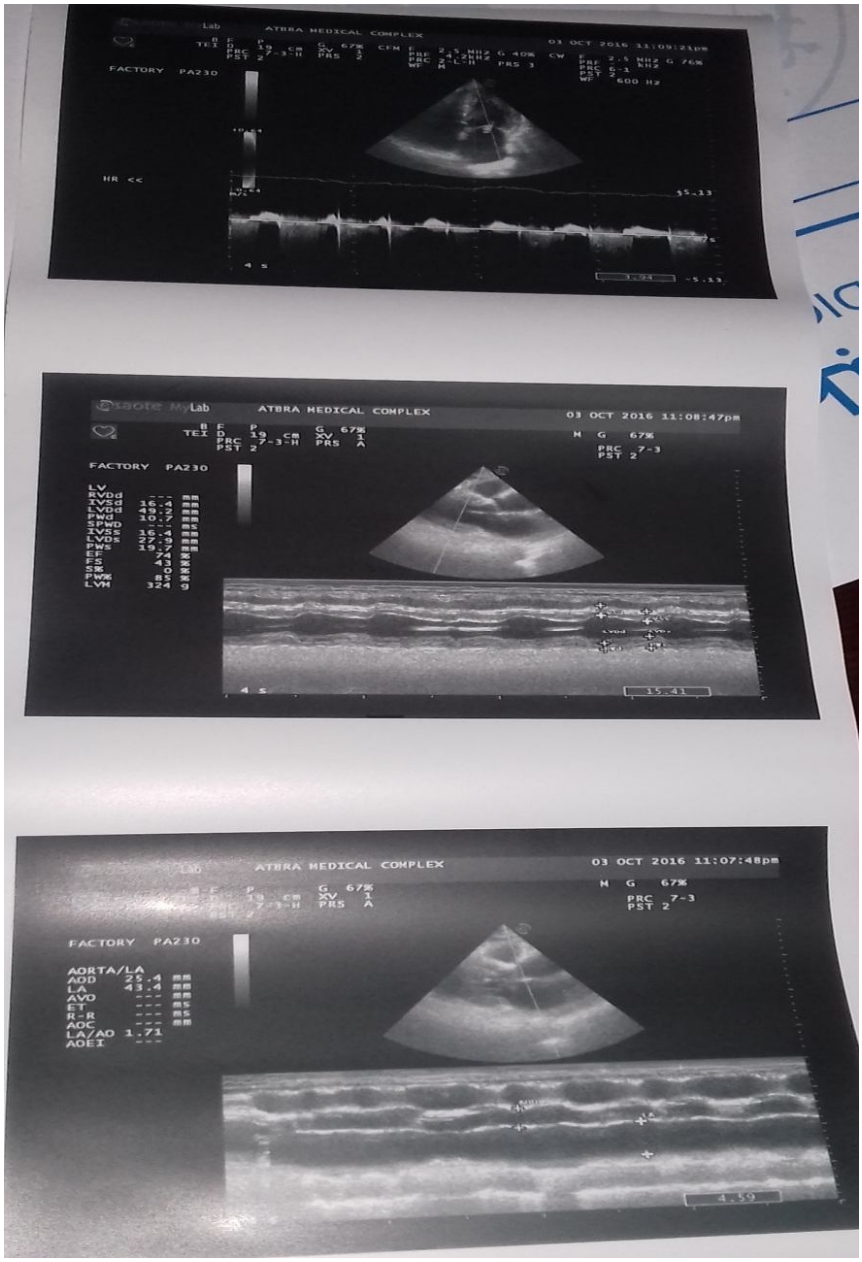

Image NO (7) 74 years old male LAX and apical 4 chambers view showing dilated LA and mild MR.

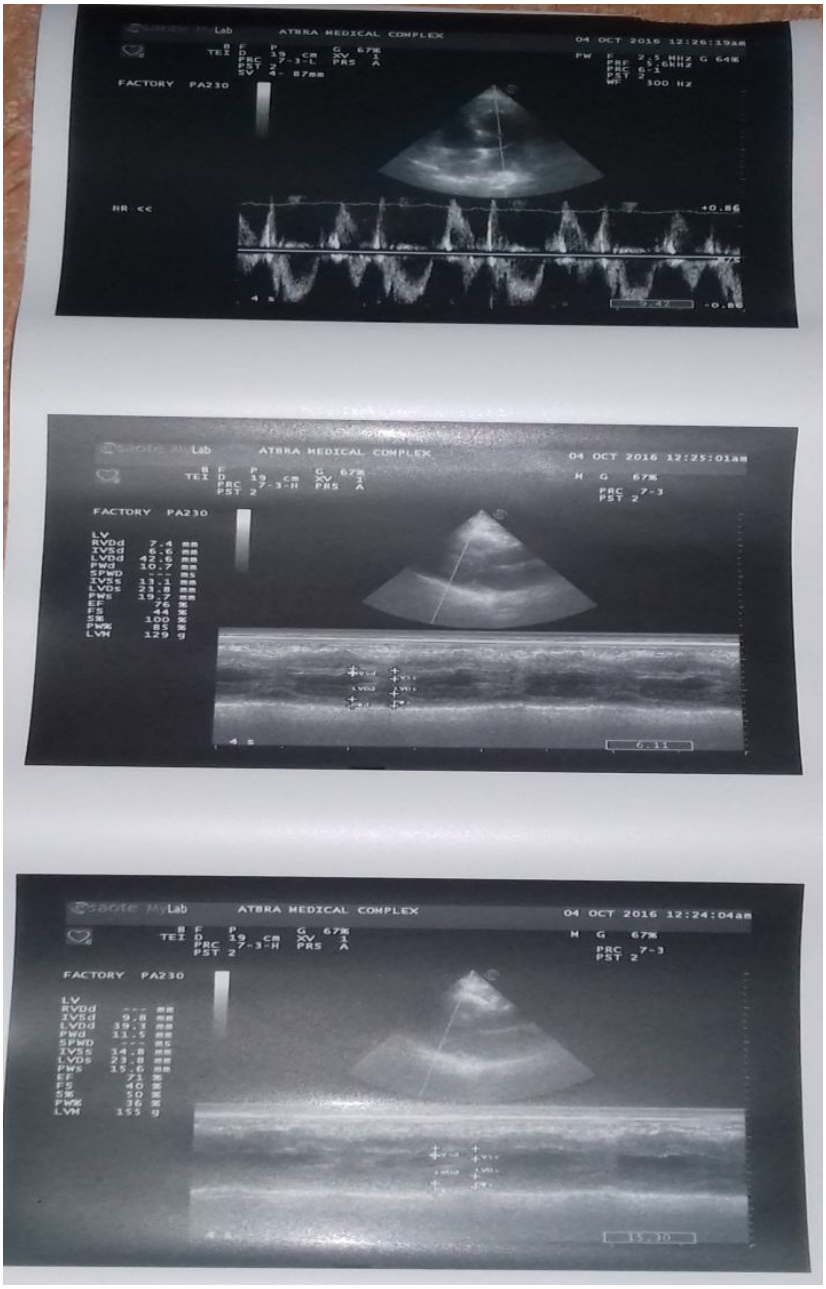

Image NO (8) 65 years old male 4 chambers view showing abnormal diastolic function.

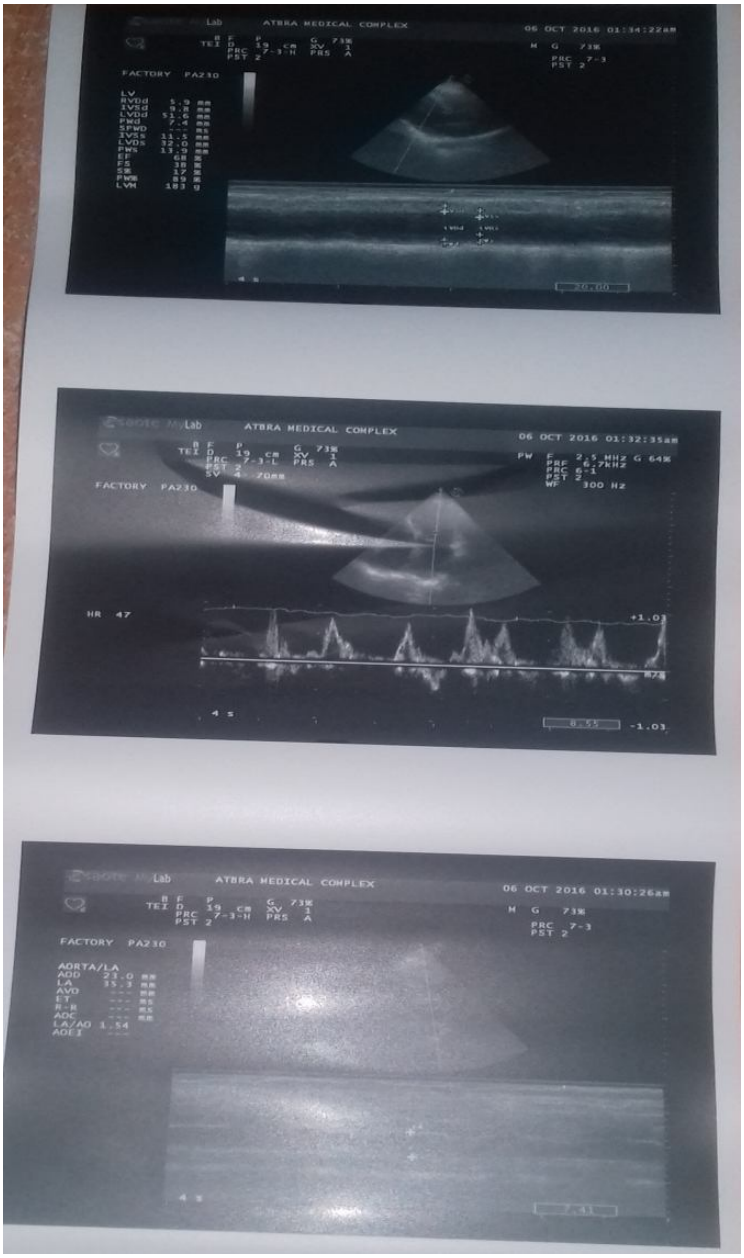

Image NO(9) 56 years old female LAXview showing LVH and ab normal diastolic function.

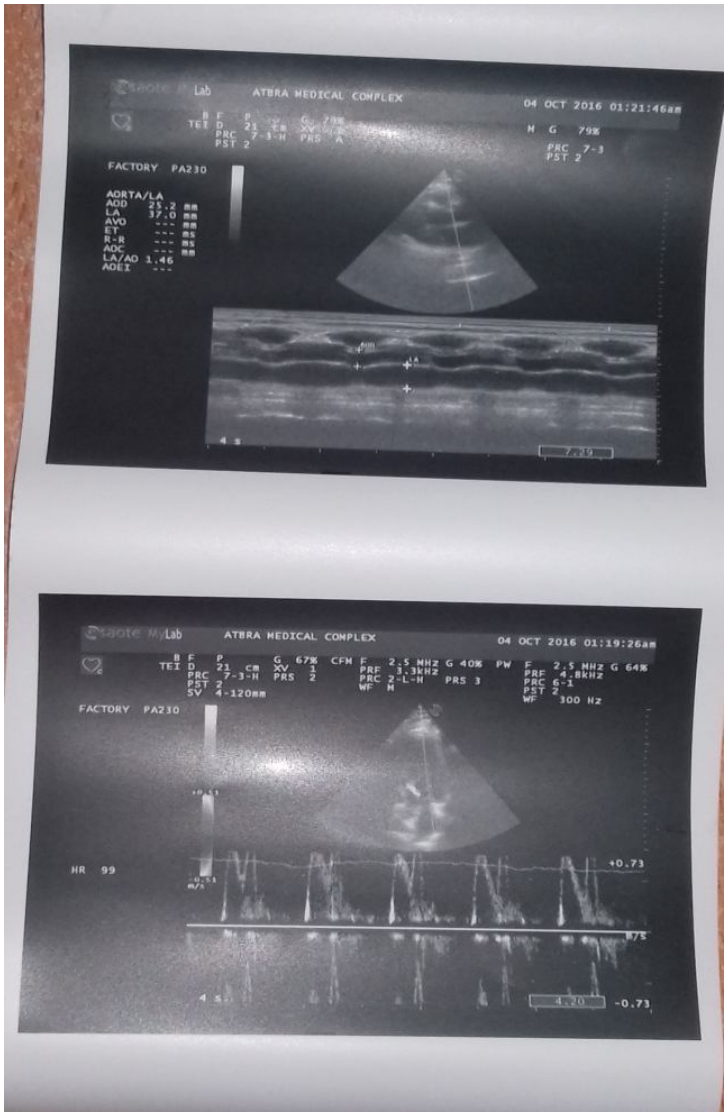

Image NO(10) 50years old female apical 4 chambers view showing abnormal diastolic function.