Sudan University of Science and Technology

College of Graduate Studies and Scientific Research

Evaluation of the left ventricle in hypertensive patients By Using echocardiograph

تقويم البطين الايسر لدى المرضى المصابين بارتفاع ضغط الدم باستخدام الموجات فوق الصوتية للقلب

A thesis submitted in partial fulfillment for the requirements of Master degree in Medical Ultrasound

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بسم الله الرحمن الرحيم

قال تعالى

أَفَلَمْ يَسِيرُوا فِي الأَرْضِ فَتَكُونَ لَهُمْ قُلُوبٌ يَعْقِلُونَ بِهَا

أَوْ آذَانٌ يَسْمَعُونَ بِهَا فَإِنَّهَا لاَ تَعْمَى الأَبْصَارُ وَلَكِن تَعْمَى القُلُوبُ

الَتِي فِي الصُّدُور

صدق الله العظيم ،،،

[الحج: 46]

Dedication

To My Parents,

To My Brothers,

To My Friends

And Finally,

To My Family

Acknowledgements

I thank God for enabling me to complete this thesis.

I sincerely thank **Dr.Alsafi Ahmed Abdalla**, the supervisor of my thesis for his continuous help, supervision and guidance.

I greatly thank all those who supported and helped me to complete this thesis.

Specially thanks for my colleagues at military hospital.

Very much thanks to **Dr.Abdelalkarim Ahmed.**

Abstract

The objective of this study was to to describe the echocardiographic features of Left ventricle among hypertensive patients using echocardiography .

Retrospectively this study carried out in 50 patient referred to Omdurman military hospital in echocardiography department from march 2014 to June 2014. Their ages ranged between 22 and 83 (mean: 55.9; SD: 15.9) years.consecutive patients referred for echocardiogram.The demographic parameters including age, sex, systolic were taken. Patients were categorized into groups of males and females. Subjects underwent M-mode and 2D echocardiogram with color Doppler study. Reference values were derived for end-diastolic and end-systolic left ventricular internal dimensions, left ventricular wall thickness.

Sonographic appearance showed abnormalities in patients; LV dimension was 34% and LV hypertrophy was 84%. It also shows the effect of followup and medication.

Using echo cardiography the effects of hypertension on cardiac character and ejection fraction (EF) during systole and diastole was studied and the relation was found to be significant

الملخص

الهدفت من هذه الدراسة وصف ملامح تخطيط صدى القلب للبطين الأيسر لدى مرضى ارتفاع ضغط الدم باستخدام تخطيط صدى القلب .

بأثر رجعي أجريت هذه الدراسة في مستشفي السلاح الطبى بامدرمان في الفترة من فى الفترة من مارس 2014 وحتى يونيو 2014 .شملت الدراسة عدد خمسون(50) مريض وتراوحت أعمارهم بين 22 و 83 (متوسط : 55.9 ، الانحراف المعيارى 15.9) تم تحويلهم إلى المستشفى العسكري أم درمان الى قسم تخطيط صدى القلب ودلك باستخدام تقنية الموجات فوق الصوتية والدوبلر وقياس مؤشراته المختلفة.

أوضحت الدراسة المعلومات الخاصة بالمرضى كالسن ، والجنس ، وتم تصنيف المرضى إلى مجموعات من الذكور والإناث. حيث اجريت الدراسة باستخدام مخطط صدى القلب مع دراسة لون دوبلر. وقد استمدت القيم المرجعية للبطين الايسر شملت الأبعاد الداخلية فى وضع نهاية الانبساط ونهاية الانقباض ، سمك الجدار البطيني .

أظهرت الدراسة ان التضخم فى عضلة القلب يمثل 84٪ من المرضى. واظهرت أيضا تأثير المتابعة و الاستخدام المنتظم للدواء. خلصت الدراسة ان تصوير صدي القلب له دور فعال في تقويم امراض الشرايين التاجيه و امراض القلب الخلقيه وضخ عضله القلب عند الانقباض و الانبساط.

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Table of Abbreviations

Abbreviation	Meaning
ECG	Electrocardiograph
ECHO	Echocardiography
EF	Ejection faction
HCM	Hypertrophic cardiomyopathy
LA	Left atrium
CW	Continuous wave
E-wave	Early wave of mitral flow
IAS	Interatrial septum
IVS	Interventricular septum
LV	Left Ventricle
LVEDD	Left ventricular end–diastolic diameter
LVESD	Left ventricular end–systolic diameter
LVH	Left ventricular hypertrophy
M-mode	Motion mode
PHT	Pulmonary hypertension
PW	Pulsed wave
PAP	Pulmonary artery pressure
RA	Right atrium
RV	Right ventricle

CHAPTER ONE

Introduction

Hypertensionis a condition in which the arteries have persistently elevated blood pressure. Every time the human heart beats, it pumps blood to the whole body through the arteries .Blood pressure is the force of blood pushing up against the blood vessel walls. The higher the pressure the harder the heart has to pump .Hypertension can lead to damaged organs, as well as several illnesses, such as renal failure (kidney failure), aneurysm, heart failure, stroke, or heart attack. Researchers from UC Davis reported in the Journal of the American Academy of Neurology that high blood pressure during middle age may raise the risk of cognitive decline later in life(Cannel and Cobb, 1992)

The normal level for blood pressure is below 120/80, where 120 represent the systolic measurement (peak pressure in the arteries) and 80 represents the diastolic measurement (minimum pressure in the arteries). Blood pressure between 120/80 and 139/89 is called prehypertension (to denote increased risk of hypertension), and a blood pressure of 140/90 or above is considered hypertension. Hypertension may be classified as essential or secondary. Essential hypertension is the term for high blood pressure with unknown cause. It accounts for about 95% of cases. Secondary hypertension is the term for high blood pressure with a known direct cause, such as kidney disease, tumors, or birth control pills, (Haider,1998).

1.1. Problem of the study:

Cardiac problem encountered every day during normal examination especially on those with high risk factor and it might goes without being notice till the onset of a sever complication. Various type of test has been conducted in different hospital and clinical centers to assess the cardiac problem including echocardiography. Most of the applied methods showed a major deficiency either in safety issue or the sufficient information regarding the heart condition which lead to wrong decision in management; which in turn lead to complication; in this essence a long standing hypertensive might affect the

heart and it will lead to heart failure, therefore it important to use echocardiography modality to assess the disease and hence prevent further complication.

1.2. Objectives

The main purposes of this study was to study the Lt ventricle of patients with hypertension using echocardiography in order to classify the septum size and Lt ventricle dimension for early prediction of heart problems.

1.2.1. Specific objectives:

- To correlate the result of echocardiogram with the left ventricle (of hypertensive patient) finding and echocardiography.
- To find the accuracy of echocardiography in assessing LV ventricle hypertrophy in hypertensive patient.
- To find the characteristics that associated LV in a hypertensive patient.

1.3. Significance of the study:

This study will highlight the issue of early diagnosis of possible problem in the left ventricles of hypertensive patients where it will help in early detection of abnormalities so complication can be avoided.

1.4. Overview of the study:

This study consisted of five chapters with chapter one which includes introduction, objectives (general and specific) , problem and Anatomy , physiology , effect of hypertension in the heart, Chapter tow previous study. Chapter three Method and methodology, data collection ,analysis .Chapter four deals with result .Chapter five discussion, recommendation and references.

Theoretical background and literature review

2.1. Anatomy of the Heart

The human heart is a four-chambered muscular organ, shaped and sized roughly like a man's closed fist with two-thirds of the mass to the left of midline.

The heart is enclosed in a pericardial sac that is lined with the parietal layers of a serious membrane. The visceral layer of the serous membrane forms the epicedium.

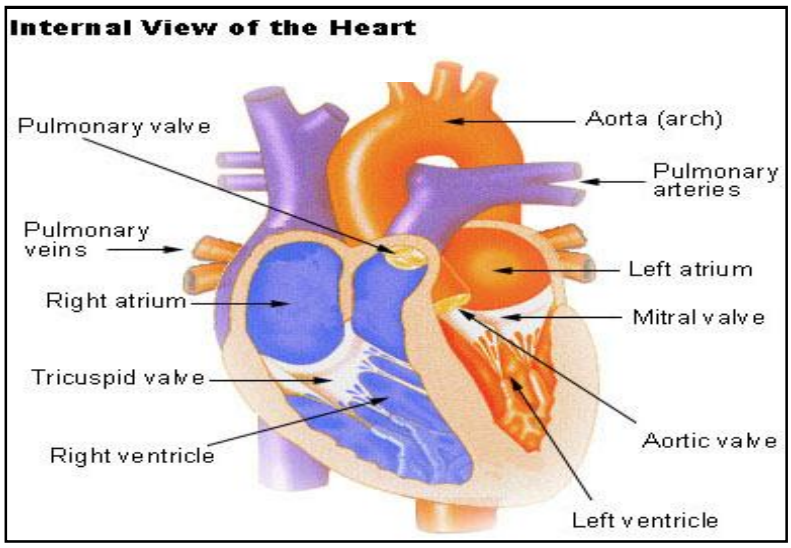


Figure (2.1):Internal View of the Heart (Haider A,1998)

2.1.1. Layers of the Heart Wall

Three layers of tissue form the heart wall. The outer layer of the heart wall is the epicardium, the middle layer is the myocardium, and the inner layer is the endocardium.

2.1.2. Chambers of the Heart

The internal cavity of the heart is divided into four chambers, right atrium, right ventricle, left atrium, left ventricle the two atria are thin-walled chambers that receive blood from the veins. The two ventricles are thick-walled chambers that forcefully pump blood out of the heart. Differences in thickness of the heart chamber walls are due to variations in the amount of myocardium present, which reflects the amount of force each

chamber is required to generate. The right atrium receives deoxygenated blood from systemic veins; the left atrium receives oxygenated blood from the pulmonary veins.

2.1.3. Valves of the Heart

Pumps need a set of valves to keep the fluid flowing in one direction and the heart is no exception. The heart has two types of valves that keep the blood flowing in the correct direction. The valves between the atria and ventricles are called atrioventricular valves (also called cuspid valves), while those at the bases of the large vessels leaving the ventricles are called semilunar valves.

The right atrioventricular valve is the tricuspid valve. The left atrioventricular valve is the bicuspid, or mitral, valve. The valve between the right ventricle and pulmonary trunk is the pulmonary semilunar valve. The valve between the left ventricle and the aorta is the aortic semilunar valve.

When the ventricles contract, atrioventricular valves close to prevent blood from flowing back into the atria. When the ventricles relax, semilunar valves close to prevent blood from flowing back into the ventricles.

2.1.4. Pathway of Blood through the Heart

While it is convenient to describe the flow of blood through the right side of the heart and then through the left side, it is important to realize that both atria and ventricles contract at the same time. The heart works as two pumps, one on the right and one on the left, working simultaneously. Blood flows from the right atrium to the right ventricle, and then is pumped to the lungs to receive oxygen. From the lungs, the blood flows to the left atrium, then to the left ventricle. From there it is pumped to the systemic circulation.

2.1.5. Blood Supply to the Myocardium

The myocardium of the heart wall is a working muscle that needs a continuous supply of oxygen and nutrients to function efficiently. For this reason, cardiac muscle has an

extensive network of blood vessels to bring oxygen to the contracting cells and to remove waste products.

The right and left coronary arteries, branches of the ascending aorta, supply blood to the walls of the myocardium. After blood passes through the capillaries in the myocardium, it enters a system of cardiac (coronary) veins. Most of the cardiac veins drain into the coronary sinus, which opens into the right atrium.

2.2. Physiology of the Heart

The conduction system includes several components. The first part of the conduction system is the sinoatrialnode. Without any neural stimulation, the sinoatrial node rhythmically initiates impulses 70 to 80 times per minute. Because it establishes the basic rhythm of the heartbeat, it is called the pacemaker of the heart. Other parts of the conduction system include the atrioventricular node, atrioventricular bundle, bundle branches, and conduction myofibers. All of these components coordinate the contraction and relaxation of the heart chambers.

2.2.1. Cardiac Cycle

The cardiac cycle refers to the alternating contraction and relaxation of the myocardium in the walls of the heart chambers, coordinated by the conduction system, during one heartbeat. Systole is the contraction phase of the cardiac cycle, and diastole is the relaxation phase. At a normal heart rate, one cardiac cycle lasts for 0.8 second.

2.2.2. Heart Sounds

The sounds associated with the heartbeat are due to vibrations in the tissues and blood caused by closure of the valves. Abnormal heart sounds are called murmurs.

2.2.3. Heart Rate

The sinoatrial node, acting alone, produces a constant rhythmic heart rate. Regulating factors are reliant on the atrioventricular node to increase or decrease the heart rate to adjust cardiac output to meet the changing needs of the body. Most changes in the heart

rate are mediated through the cardiac center in the medulla oblongata of the brain. The center has both sympathetic and parasympathetic components that adjust the heart rate to meet the changing needs of the body.

Peripheral factors such as emotions, ion concentrations, and body temperature may affect heart rate. These are usually mediated through the cardiac center .

2.2.4. Blood

Blood is the fluid of life, transporting oxygen from the lungs to body tissue and carbon dioxide from body tissue to the lungs. Blood is the fluid of growth, transporting nourishment from digestion and hormones from glands throughout the body. Blood is the fluid of health, transporting disease-fighting substances to the tissue and waste to the kidneys. Because it contains living cells, blood is alive. Red blood cells and white blood cells are responsible for nourishing and cleansing the body.

Without blood, the human body would stop working.

2.2.5. Effects Of Hypertension in The Heart

The cause of hypertensive heart disease is chronically elevated blood pressure (BP); however, the causes of elevated BP are diverse. Essential hypertension accounts for 90% of cases of hypertension in adults. Secondary causes of hypertension account for the remaining 10% of cases of chronically elevated BP.

According to the Framingham Study, hypertension accounts for about one quarter of heart failure cases.[1] In the elderly population, as many as 68% of heart failure cases are attributed to hypertension .Community-based studies have demonstrated that hypertension may contribute to the development of heart failure in as many as 50-60% of patients. In patients with hypertension, the risk of heart failure is increased by 2-fold in men and by 3-fold in women.

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, complications that manifest clinically as angina or myocardial infarction, cardiac arrhythmias (especially atrial fibrillation), and congestive heart failure (CHF).

Thus, hypertensive heart disease is a term applied generally to heart diseases, such as LVH (seen in the images below), coronary artery disease, cardiac arrhythmias, and CHF, that are caused by the direct or indirect effects of elevated BP. Although these diseases generally develop in response to chronically elevated BP, marked and acute elevation of BP can lead to accentuation of an underlying predisposition to any of the symptoms traditionally associated with chronic hypertension.

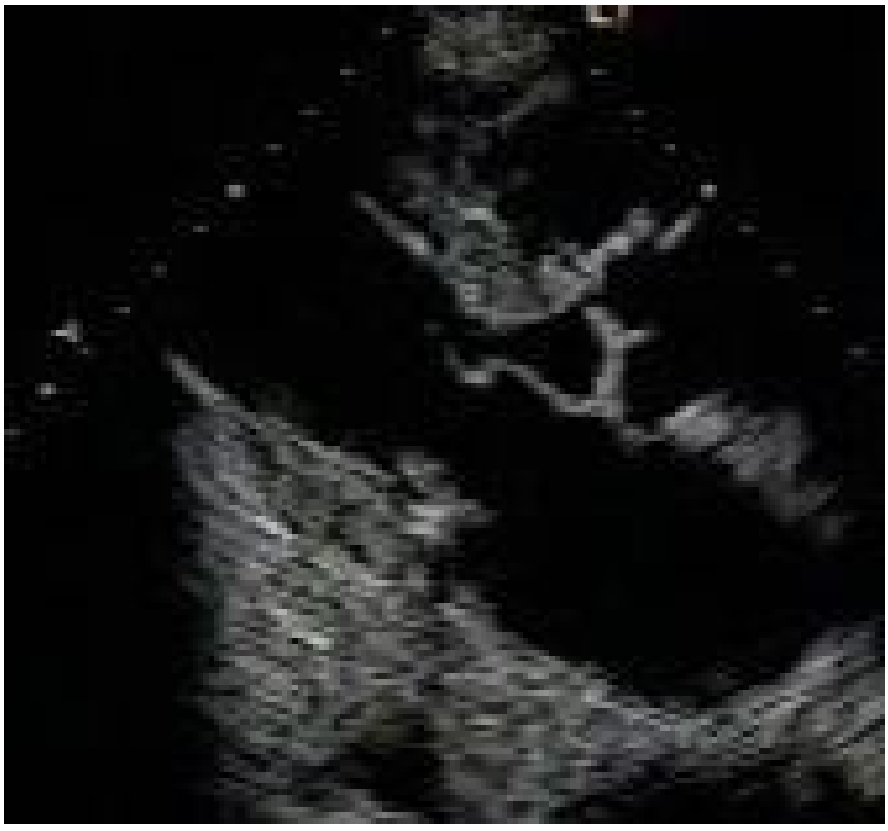


Figure (2.2): Two-dimensional echocardiogram (parasternal long axis view)

Two-dimensional echocardiogram (parasternal long axis view) from a 70-year-old woman showing concentric left ventricular hypertrophy and left atrial enlargement,(Kannel W, Cobb J 199).

Figure (2.3): Gross specimen of the heart with concentric left ventricular hypertrophy, (Kannel W, Cobb J 199).

2.2.6. Differentials

The conditions should also be considered when evaluating hypertensive heart disease there is, coronary artery atherosclerosis, hypertrophic Cardiomyopathy. athlete's heart (with LVH) , congestive heart failure due to other etiologies,atrial fibrillation due to other etiologies, diastolic dysfunction due to other etiologies and a sleep apnea,(Kannel W, Cobb J 1992).

2.2.7. Patient education

It is important to educate patients about the nature of their disease and the risks associated with untreated hypertension. In addition, dietary modifications and the importance of regular exercise, taking medications regularly, weight loss, and avoiding medications and foods that can potentially elevate blood pressure should be emphasized.

Table (2.1): reading of blood pressure in diastole and systole

Category	Systolic BP, mm Hg	Diastolic BP, mm Hg
Optimal	< 120	< 80
Prehypertension	120-139	80-89
Stage I	140-159	90-99
Stage II	>160	>100

2.2.8. Etiology

The etiology of hypertensive heart disease is a complex interplay of various hemodynamic, structural, neuroendocrine, cellular, and molecular factors. These factors play integral roles in the development of hypertension and its complications; however, elevated BP itself can modulate these factors.

Obesity has been linked to hypertension and LVH in various epidemiologic studies, with as many as 50% of obese patients having some degree of hypertension and as many as 60-70% of patients with hypertension being obese.

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.

2.3. Pathology of the heart

2.3.1. Left ventricular hypertrophy

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 cardiomyocytes), and, thus, to LVH. In addition, activation of the renin-angiotensin system, through the action of angiotensin II on angiotensin 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,(Avdić S, et al 2007).

Various patterns of LVH have been described, including concentric remodeling, concentric LVH, 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.

Interestingly, findings from a prospective study (The Multiethnic Study of Atherosclerosis [MESA] trial) also indicate a higher risk of developing systemic hypertension among patients in the higher quartiles of the LV mass at baseline, (Verdecchia P 1990).

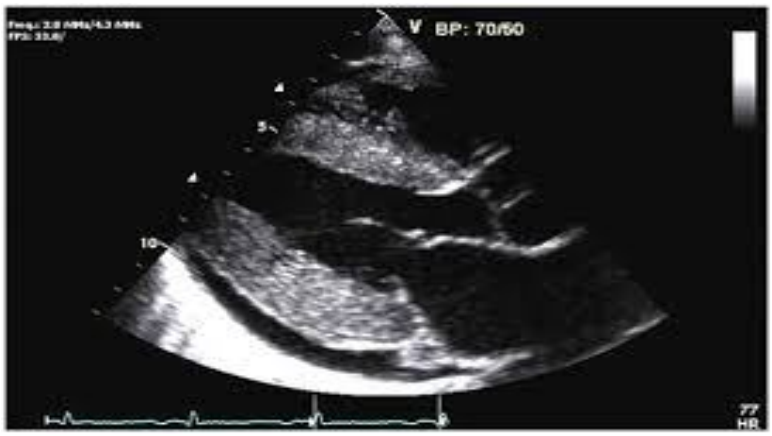


Figure (2.4): Concentric LVH (Verdecchia P 1990).

2.3.2. Left atrial abnormalities

Frequently underappreciated, 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, (Avdić S, et al 2007).

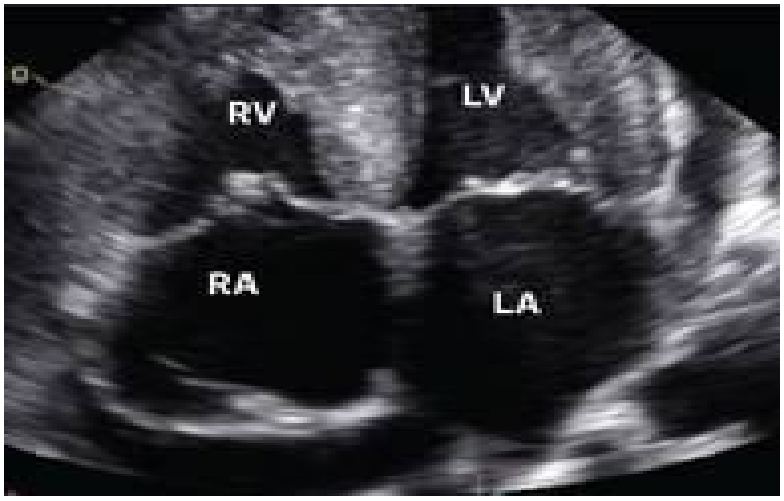


Figure (2.5): Dilated Right, left atrium.

2.3.3. 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, (Kannel W, Cobb J.1992).

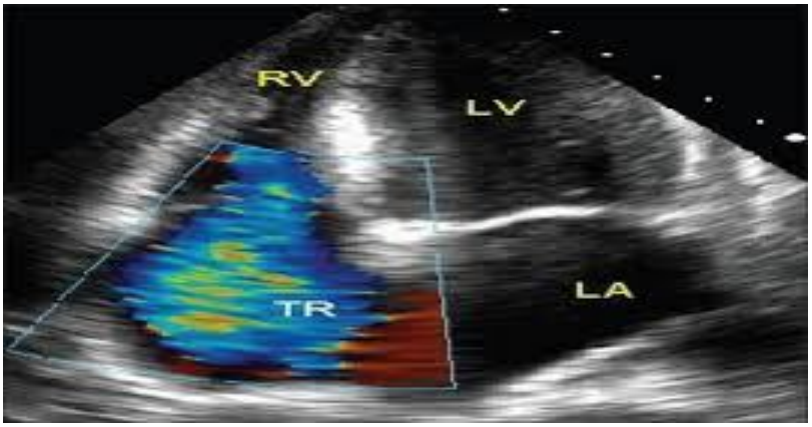


Figure (2.6): Tricuspid regurgitation (Kannel W, Cobb J.1992).

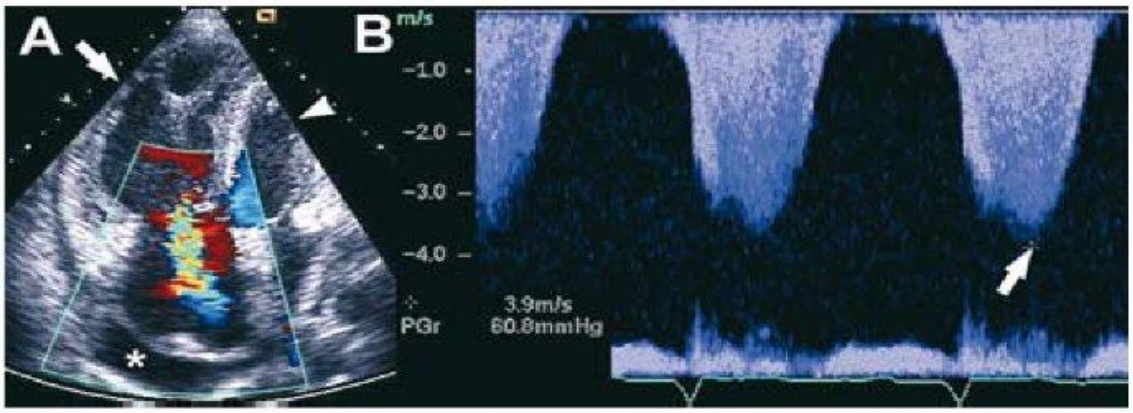


Figure (2.7): Mitral regurgitation, (Verdecchia P 1990).

2.3.4. Heart failure

Heart failure is a common complication of chronically elevated BP. Patients with hypertension fall into one of these categories like, 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, and 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 after load and the resulting LVH can adversely affect the active early relaxation phase and the late compliance phase of ventricular diastole, (Verdecchia P 1990).

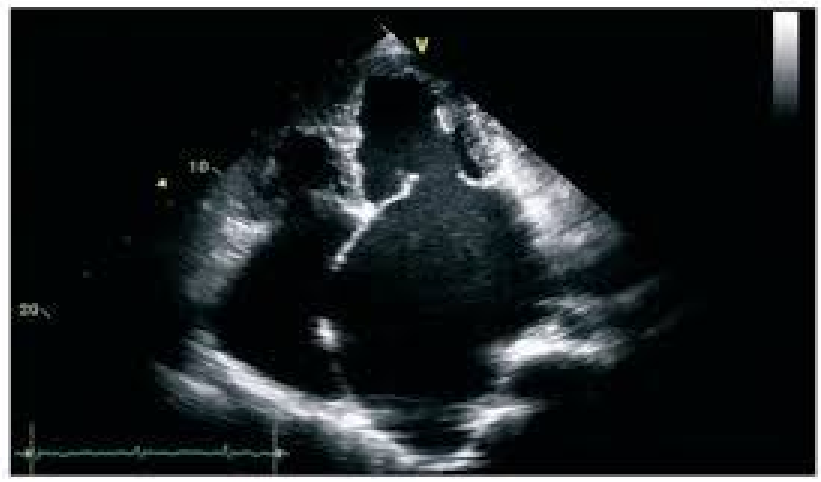


Figure (2.8): Left heart failure, (Dorr M, 2005).

2.3.5. Diastolic dysfunction

Diastolic dysfunction is common in persons with hypertension. It is often, but not invariably, accompanied by LVH. In addition to elevated after load, 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, (Dorr M, et al 2005).

2.3.6. 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, (Dorr M, et al 2005).

2.3.7. Decompensation

Apoptosis, or programmed cell death, stimulated by myocyte hypertrophy and the imbalance between its stimulants and inhibitors, is considered to play an important part in the transition from compensated to decompensated stage. The patient may become

symptomatic during the asymptomatic stages of the LV systolic or diastolic dysfunction, owing to changes in afterload conditions or to the presence of other insults to the myocardium (e.g. ischemia, infarction). A sudden increase in BP can lead to acute pulmonary edema without necessarily changing the LV ejection fraction.

Generally, development of asymptomatic or symptomatic LV dilatation or dysfunction heralds rapid deterioration in clinical status and a markedly increased risk of death. In addition to LV dysfunction, right ventricular (RV) thickening and diastolic dysfunction also develop as results of septal thickening and LV dysfunction, (Dorr M, et al 2005).

2.3.8. 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 multifactorial.

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, (Avdić S, et al 2007).

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.

2.3.9. 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, inhomogeneity of the myocardium, poor perfusion, myocardial fibrosis, and fluctuation in Afterload,(Avdić S, et al 2007).

All of these may lead to an increased risk of ventricular tachyarrhythmias.

Atrial fibrillation (paroxysmal, chronic recurrent, 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, most notably stroke. Premature ventricular contractions, ventricular arrhythmias, and sudden cardiac death are observed more often in patients with LVH than in those without LVH.

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, (Avdić S, et al 2007).

2.4. Pervious Study

Radaideh et al (2013) studied the pattern of left ventricular hypertrophy caused by hypertension and to compare it with idiopathic hypertrophic Cardiomyopathy.

The retrospective study was conducted at the echocardiography lab of Rashid Hospital, Dubai, from January 2009 to January 2010. Cases of 11 patients with significant left ventricular hypertrophy (septum >15mm), due to underlying hypertension, were analyzed and compared with 11 cases of idiopathic hypertrophic.

Cardiography (septum >15mm) to assess the two groups with similar baseline echocardiographic features.

Although the pattern of hypertrophy in hypertensive patients was more concentric (n=5; 45%), there was also asymmetrical septal hypertrophy in 4 (36%) cases, particularly the elderly with sigmoid shape septum. Therewas evidence of resting mid-cavity gradient due to reduced left ventricular end-systolic diameter in 4 (36%) cases.

Although the equation between hypertension and left ventricular hypertrophy is more concentric, it can be associated with left ventricular outflow tract obstruction and significant mid-cavity gradients similar to thatseen in idiopathic hypertrophic cardiomyopathy,(Johan Sundström, et al). The increased risk associated with left ventricular hypertrophy (LVH) diagnosed echocardiographically (Echo-LVH) or electrocardiographically (ECG-LVH) is well known, but the clinically relevant question of how much additional prognostic information would be provided by echocardiographically assessing LVH if a subject's ECG-LVH and hypertension status are known has not been addressed.

We investigated whether Echo-LVH and ECG-LVH predicted total and cardiovascular mortality and morbidity independently of each other and of other cardiovascular risk factors by using a population-based sample of 475 men investigated at age 70 with a median follow-up time of 5.2 years. Echocardiographic left ventricular mass index (LVMI) predicted total mortality (hazards ratio [HR] 1.44, 95% CI 1.09 to 1.92, for a 1-SD increase in LVMI) and cardiovascular mortality (HR 2.38, 95% CI 1.52 to 3.73)

independently of ECG-LVH and other cardiovascular risk factors. ECG-LVH, defined as Cornell product >244μV/s, predicted total mortality (HR 2.89, 95% CI 1.41 to 5.96) independently of LVMI and other cardiovascular risk factors. Thus, Echo-LVH and ECG-LVH provided complementary prognostic information, especially in hypertensive subjects.

Echo-LVH and ECG-LVH predict mortality independently of each other and of other cardiovascular risk factors, implying that Echo-LVH and ECG-LVH in part carry different prognostic information. Therefore, to fully assess the increased risk associated with these conditions, both ECG and echocardiography should be performed.

MorgantiAet al (2008) reviewed recent literature on the prevalence of LVH, as assessed by echocardiography, in order to offer updated information on the magnitude of subclinical alterations in LV structure in contemporary human hypertension. A MEDLINE search using key words 'left ventricular hypertrophy', 'hypertension', 'echocardiography' and 'cardiac organ damage' was performed in order to identify relevant papers. Full articles published in English language in the last decade, (1 January 2000-1 December 2010), reporting studies in adult or elderly individuals, were considered. A total of 30 studies, including 37,700 untreated and treated patients (80.3% Caucasian, 52.4% men, 9.6% diabetics, 2.6% with CV disease) were considered. LVH was defined by 23 criteria; its prevalence ranged from 36% (conservative criteria) to 41% (less conservative criteria) in the pooled population. LVH prevalence was not different between women and men (range 37.9-46.2 versus 36.0-43.5%, respectively). Eccentric LVH was more frequent than concentric hypertrophy (range 20.3-23.0 versus 14.8-15.8, respectively, $P<0.05$); concentric phenotype was found in a consistent fraction (20%) of both genders. Despite the improved management of hypertension in the last two decades, LVH remains a highly frequent biomarker of cardiac damage in the hypertensive population. Our analysis calls for a more aggressive treatment of hypertension and related CV risk factors leading to LVH.

RJ – Brazil et al (2006) studied sixty hypertensive patients, grouped according to the Joint National Committee stages of hypertension. Using the single- and two-dimensional Doppler Echocardiography, we analyzed the left ventricular mass and the geometric patterns through the correlation of left ventricular mass index and relative wall thickness. On ambulatory blood pressure monitoring we assessed the means and pressure loads in the different geometric patterns detected on echocardiography. We identified three left ventricular geometric patterns: 1) concentric hypertrophy, in 25% of the patients; 2) concentric remodeling, in 25%; and 3) normal geometry, in 50%. Casual systolic blood pressure was higher in the group with concentric hypertrophy than in the other groups (p=0.001). Mean systolic pressure in the 24h, daytime and nighttime periods was also higher in patients with concentric hypertrophy, as compared to the other groups (p=0.003, p=0.004 and p=0.007). Daytime systolic load and nighttime diastolic load were higher in patients with concentric hypertrophy (p=0.004 and p=0.01, respectively).

Left ventricular geometric patterns show significant correlation with casual systolic blood pressure, and with means and pressure loads on ambulatory blood pressure monitoring.

The occurrence of ventricular hypertrophy undoubtedly represents an important marker of increased risk for cardiovascular events; therefore, the importance of identifying the patterns of hypertrophy in patients with hypertension is widely justified.

A recent study by Mensah et al reported that measurement of the left ventricular mass on echocardiography proved to be better for predicting the evolutional process of hypertension as compared with other variables, such as systolic blood pressure, diastolic blood pressure, and hypertension staging.

Left ventricular hypertrophy is no longer considered an adaptive process that compensates the pressure imposed on the heart and has been identified as an independent and significant risk factor for sudden death, acute myocardial infarction, and congestive heart failure.

In cardiac hypertrophy, anomalous collagenous proteins and other types of contractile proteins (myosin with different functional properties) are produced. These myosins show a lower activity of the ATPase enzyme and a lower velocity of power generation. Likewise the formation of anomalous collagenous proteins, changes in contractile proteins also occur. Concomitantly, sarcomeres and fibroblasts proliferate and become hyperplastic. These adaptations aim to maintain the cardiac contractile capacity to compensate the pressure load imposed on the heart.

The cardiac adaptive process is not always beneficial for functioning of the heart and causes alteration in myocardial fibers, in the cardiac capacity to respond to adrenergic stimuli, in the left ventricular diastolic function, in the coronary artery flow, and finally in the contractile function.

Left ventricular hypertrophy causes important derangements in coronary artery flow. If hypertrophy is mild, the increase in coronary circulation may proportionally accompany the increase in left ventricular mass through vascular neoformation or use of mechanisms of flow reserve. In the cases of significant increase in ventricular mass, generation of new vessels and capillaries is disproportional to the increase in mass. Concomitantly to these myocardial changes, hypertrophy of the walls of the resistance coronary vessels occurs.

Coexistence of these alterations significantly limits the perfusion of left ventricular muscle mass. The decrease in the coronary flow reserve contributes to the pathogenesis of myocardial ischemia and heart failure in patients with left ventricular hypertrophy. Direct effects on myocardial electrical instability may also occur, contributing to severe arrhythmias and sudden death, which have a high incidence in patients with left ventricular hypertrophy.

According to Devereux et al, the increase in left ventricular mass represents a common final pathway towards which adverse effects on the cardiovascular system converge, resulting in a higher vulnerability to complications.

Studies by Levy et al and Kannel reported that left ventricular hypertrophy is a strong indicator of bad prognosis in hypertensive individuals and in the general population. The risk of congestive heart failure, acute myocardial infarction, and sudden death is 6- to 8-fold higher in patients with left ventricular hypertrophy.

The term "concentric remodeling" has recently been used to explain volumetric or geometric alterations due to injuries to the myocardium, and it probably means that a structural or biochemical alteration of the muscle, vascular, or interstitial compartments is happening.

Koren et al 12 and Casale et al, among others, have shown that even in patients with normal left ventricular mass, definition of the left ventricular geometry, and in particular of concentric remodeling, is an important marker of cardiovascular risk.

The hemodynamic stress is clearly a critical determinant for left ventricular hypertrophy, and elevated blood pressure is its major triggering factor.

Materialand methodology

A total of 50 consecutive hypertensive patients arriving at the echocardiography department of the Military hospital in Omdurman were recruited in this study. The sample consisted of 30 males and 20 females. Their ages ranged from 22 to 83 (mean: 55.9, SD:15.9) years. The study duration was form March 2014to June 2014.

3.1. Materials : ultrasound machine MY LAB 50 SN 03486

(transducer):convex (5MHz)

Inclusion criteria: patient attending to echocardiography department in Omdurman military hospital with diagnostic of hypertensive.

Exclusion criteria: patient with diagnostic of essential hypertensive above 5 years.

Echocardiography:

How echocardiography was preformed:

Echocardiography data consisted of four measurements: left ventricular diastolic dimension (LVDd); left ventricular systolic dimension (LVSd); interventricular sepal thickness (IVS); and posterior wall thickness (PW).The first four variables were used to create only two categorical variables that described the LV status of the patient, namely, LV dimension and LV hypertrophy. In addition, data on follow up and medication were available as indicator variables.

Sites of transducer placement:

Air being a poor conductor of ultrasound, the transducer should be placed at points without lung interference .Such sites are:

1. left parasternal space (standard point)
2. apex of the heart
3. suprasternal notch (to study aorta & major branches)

4. xiphisternum – useful in patients emphysematous lung
5. right parasternal space in dextroposition&dextrocardia
6. oesophagus,using a trans—oesophageal transducer

3.2. Method:

Technique of echocardiography:

Thetrancsducer is placed on the fourth left intercostal space near the sternal edge, various sections may be made.

Three planes commonly used to the heart are:

1. long axis plane (LAX) .
2. short axis plane (SAX) .
3. apex four chamber plane (A 4 Ch) .

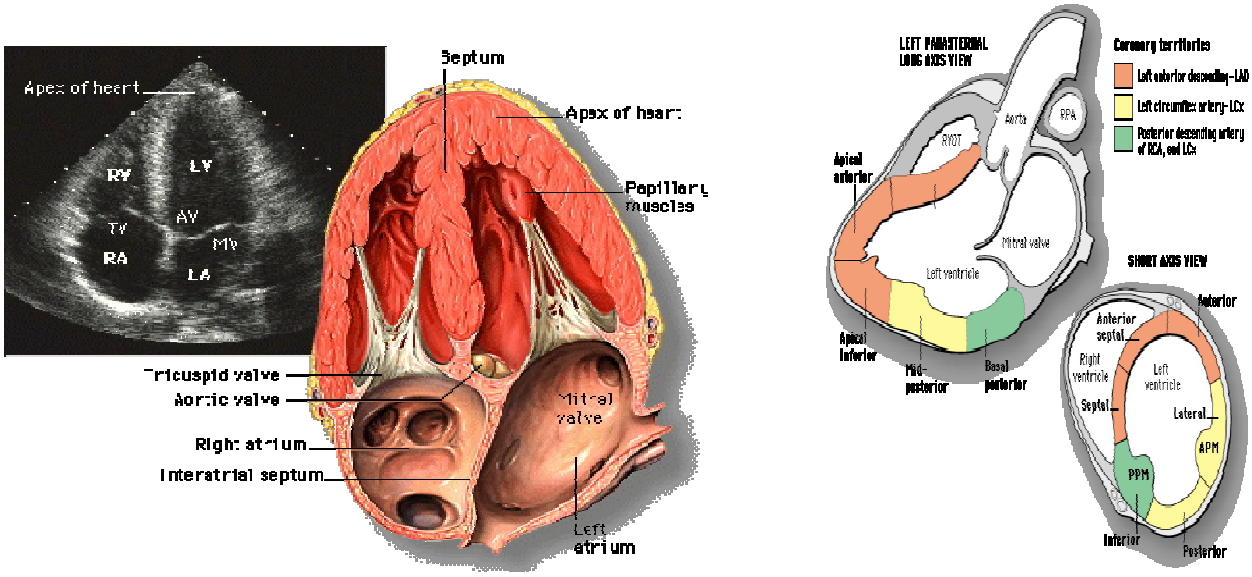


Figure (3.1): Long axis plane (LAX)

- 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.

- The coronary sinus is imaged in cross section at the atrioventricular groove

The pericardial space is normally visible in systole as a capillary space posterior to LV

- The descending thoracic aorta is cut obliquely and is seen posterior to the heart as an oval shaped pulsatile structure.

- After completing the standard LAX imaging, the transducer is tilted to the right until the plane of the scan cuts the tricuspid valve(TV). This view is called the RV – inflow view.

Parasternal Long-Axis View (PLAX):

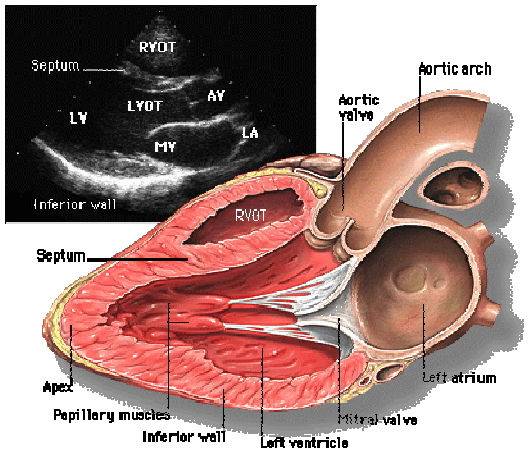
Transducer position: left sternal edge; 2nd – 4th intercostal space

Marker dot direction: points towards right shoulder.

Most echo studies begin with this view

It sets the stage for subsequent echo views.

Many structures seen from this view



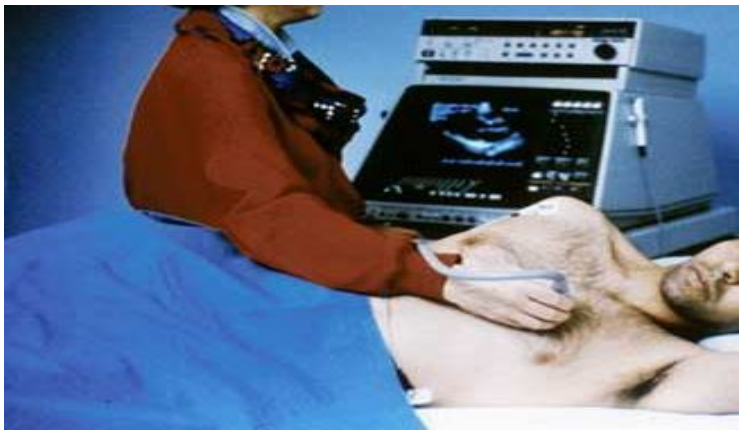


Figure (3.4): show site of transducer in long axis veiw

Short axis plane(SAX):

This is perpendicular to LAX. It runs from the left shoulder to the right hip and cuts the heart in cross section.

Cross sectional images from the apex to the base of the heart can be imaged by serial tilt of the transducer infero- superiorly.

Three sections are important of (SAX).

1- Caudal section at LV apex: the LV is imaged in cross section with two papillary muscles at (3) O Clock & (8) O clock potions respectively .

2- Mid section at LVinflow: the LV body is seen in cross section with two cusps of the mitral (the anterior & posterior leaflets) – which show a fish – mouth like opening and closing motion.

3- Tilted interiorly to obtain aortic valve level which showsmercedes shape like.

Apex 4- chamber view:

This is perpendicular to the other planes.

The transducer is shifted to the apex of heart .the plane runs parallel to the sternum and the chest wall towards the right shoulder. This plane cuts all the 4- chambers (LA, LV, RA, RV)& AV, MV & TV of the heart.

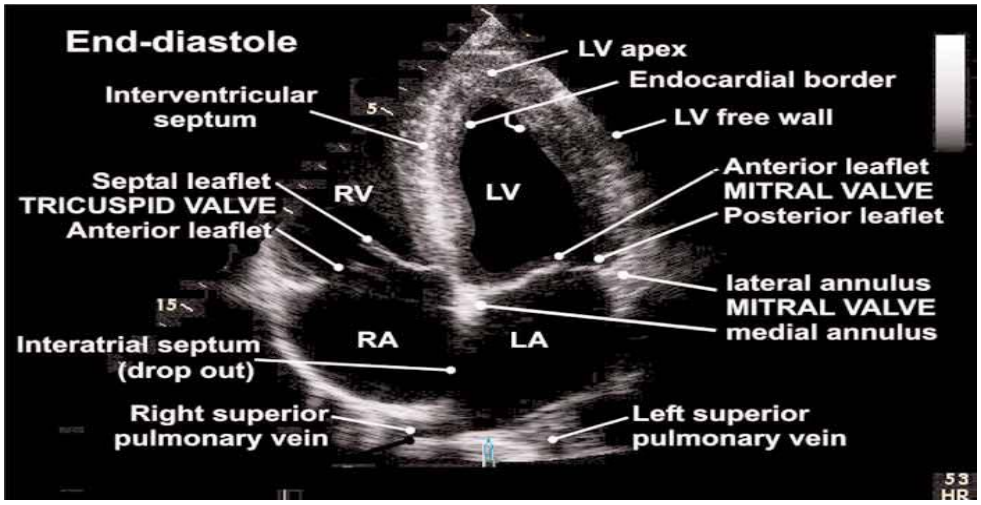


Figure (3.5): Apical four - chamber view

One can image the 2-chamber view of LA and LV by rotating the transducer by 90 degree. This view images the anterior and posterior walls of LV,whereas the 4- chamber view images its medial and lateral walls. Thus these views are complementary to each other.

Apical 2-Chamber View (AP2CH)

Transducer position: apex of the heart

Marker dot direction: points towards left side of neck (45) anticlockwise from AP4CH view)

Good for assessment of

LV anterior wall

LV inferior wall

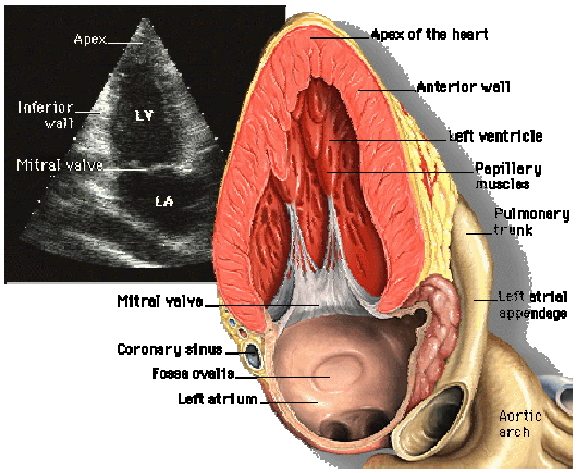


Figure (3.6): apical 2-chamber view

M- Mode Echocardiography

Current technique of M–mode study.

During 2-D study, the cursor line is placed on the valve or the point of heart whose motion has to be recorded for analysis.

The display mode is then switched to M-mode to obtain the desired recording.

All measurements are to be made from leading edge to leading edge.

An M- mode echocardiogram is not a "picture" of the heart, but rather a diagram that shows how the positions of its structures change during the course of the cardiac cycle.

M-mode recordings permit measurement of cardiac dimensions and motion patterns.

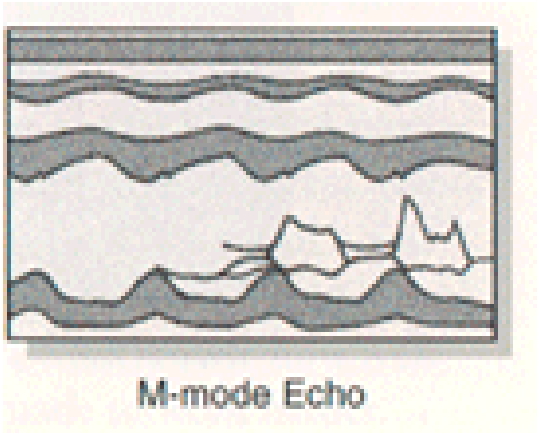
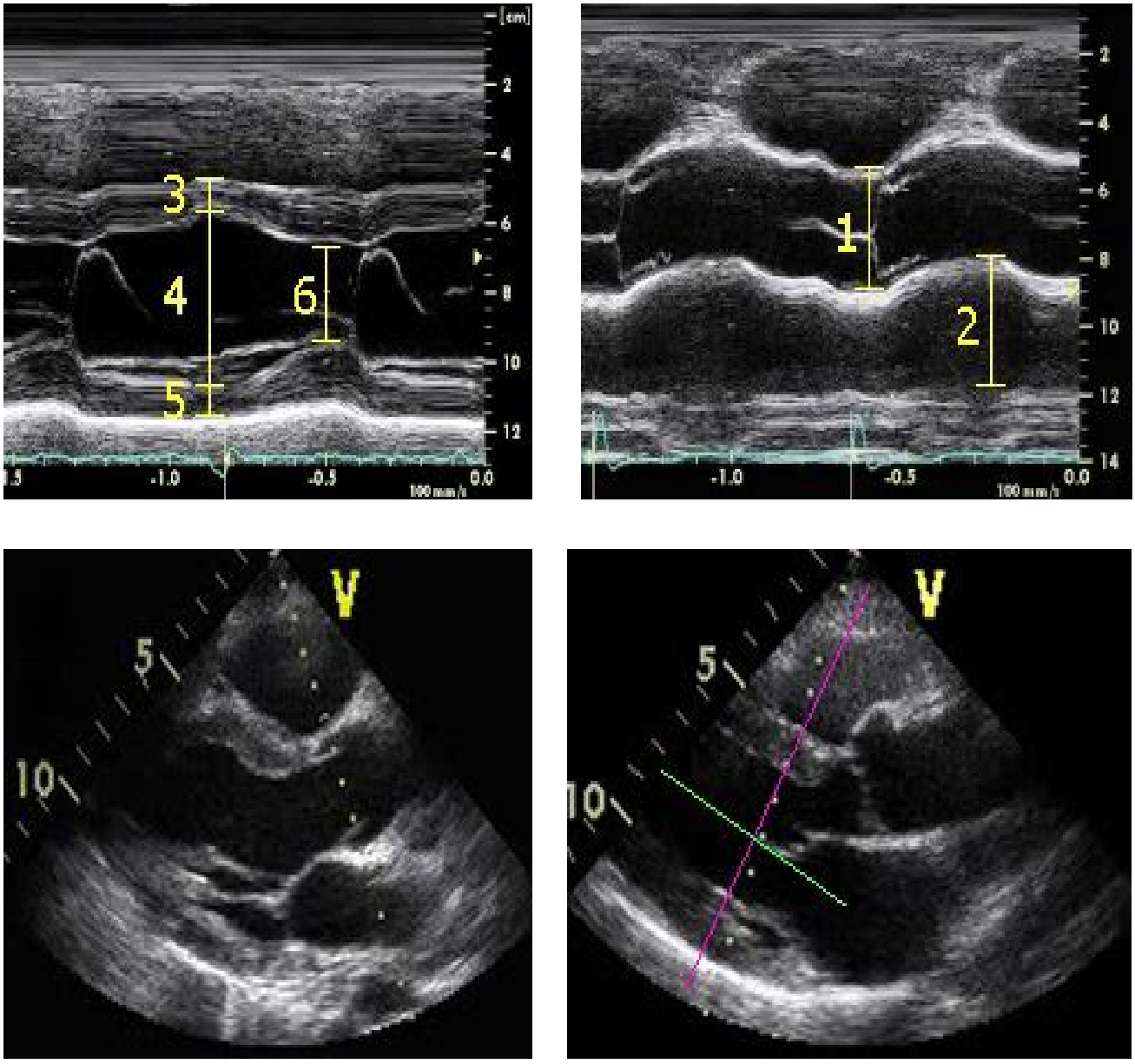


Figure (3.7): M-mode Echo



Left atrial dimension

Figure (3.8): M-mode view

This is measured from the anterior margin of the posterior aortic wall to the leading edge of the left atrial posterior wall at end systole. The aortic valve cusp should be seen in the aortic root so that the direction of the ultrasound beam is standardized.

Normal value = (19— 40 mm).

The ventricles:

1\ RV internal dimension.

This is measured from the left side of the anterior right ventricular wall or (0.5 cm) below the non- moving chest wall echoes (if the anterior right ventricular wall is not visualized clearly) to the right side of the inter ventricular septum over the R wave of the electrocardiogram.

To standardize the beam direction, the measurement should be taken only when the tip of mitral valve or chordae are seen in adults.

In children the measurements are taken when both the anterior and posterior mitral leaflets are seen Normal value: supine 0.7 to 2.3 cm & left lateral = 0.7 to 2.5 cm

2\ Inter ventricular septal thickness, at end diastole over the R wave of ECG & at end systole the maximal interventricularseptal thickness is measured.

IVS motion normally is opposite to that of LV posterior wall. In LV volume overload the septal motion is exaggerated. In RV overload theSeptal motion is paradoxic.

3\ LV posterior wall thickness

At end diastole from anterior edge of the endocardium to the anterior edge of the epicardium at end diastole over the R wave of ECG. Normal value = (6—12 mm)

At end systole is measured similar to the previous one at end diastole.

LVIDd :

This is measured from the anterior edge of the left side of the IVS to the anterior edge of posterior endocardium or myocardium over the R wave of ECG.

The normal value:(3.5 – 5.0 cm).

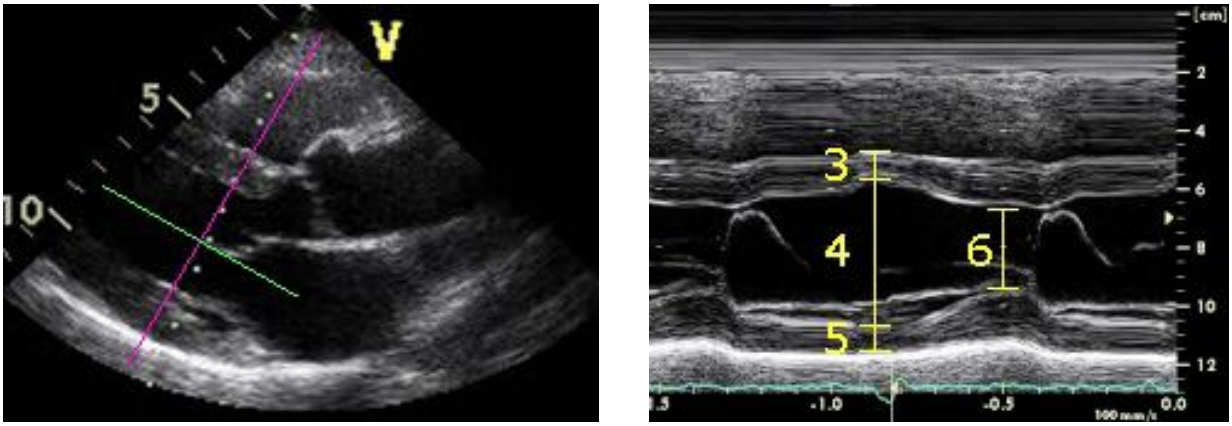


Figure (4.1): show diastole with open valve

LVIDs:

- This is shortest vertical distance between the anterior edge of the IVS endcardium or myocardium to the anterior edge of the LV posterior wall endocardium or myocardium
- Normal value is variable

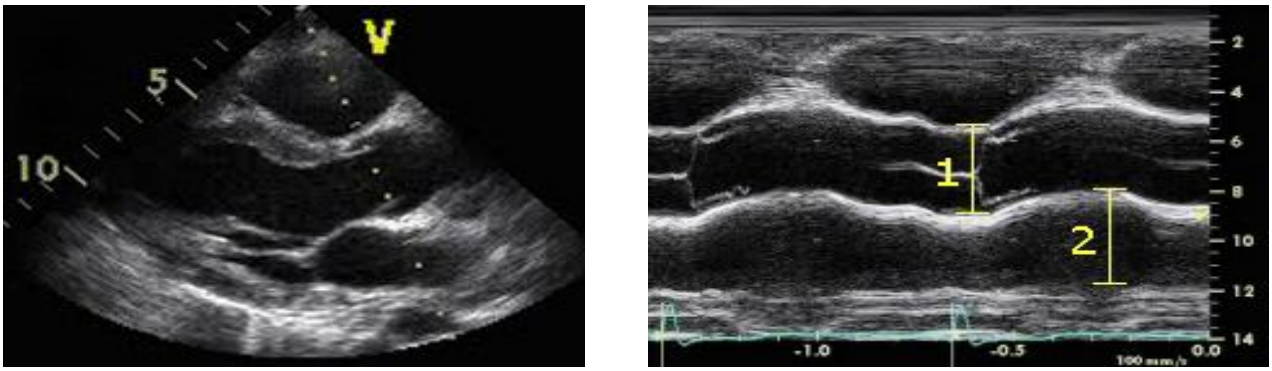


Figure (4.2): show systole with close mitral valve

This is shortest vertical distance between the anterior edge of the IVS endcardium or

myocardium to the anterior edge of the

CHAPTER FOUR

Results

4.1. Summary of echo findings

Table (2.2): Summary of the echo findings of the studied cases

Echo Finding	No.	%
Mild LVH	13	26%
Normal echo study	8	16%
Moderate Concentric LVH	5	10%
Moderate LVH	4	8%
Dilated LV & Mild LVH	3	6%
Dilated LV & Moderate LVH	3	6%
Dilated LV	2	4%
Mild Concentric LVH	2	4%
Concentric Mild LVH	1	2%
Concentric moderate LVH & Small LV	1	2%
Dilated LV & Concentric LVH	1	2%
Mild Dilated LV	1	2%
Mild Dilated LV & Concentric Moderate LVH	1	2%
Mild Dilated LV & mild LVH	1	2%
Mild Dilated LV & Moderate LVH	1	2%
Mild LVH & small LV	1	2%
Moderate Concentric LVH & Small LV	1	2%
Dilated LV & Moderate Concentric LVH	1	2%
Total	50	100%

It is evident from Table (4.1) that only eight cases out of the total (16%) had normal echo result. The rest, however, had varying patterns of adaptation. The most three dominant adaptation patterns were: Mild LVH (26%); Moderate concentric LVH (10%); and moderate asymmetric LVH (8%).

4.2. Effectof follow up

The effects of follow up and of medication on **LV dimension** and **LV hypertrophy** was investigated both graphically and formally assuming no difference between the follow up groups and the medication groups in the studied sample as regards those two variables.

The distribution of patients by Follow up status and LV dimension is depicted in Figure (4.1)

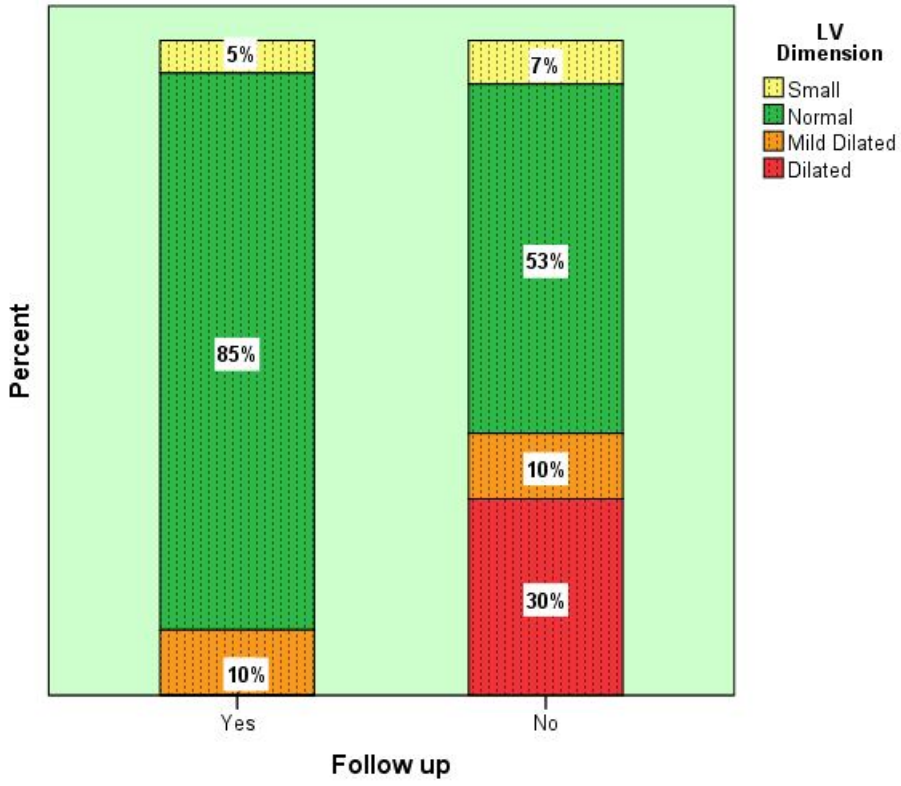


Figure (4.3}: Distribution of patients by Follow up and LV dimension

It is evident from Figure (4.1) above that the patients who had follow up did not suffer from dilated LV, while 30% of those without follow up had dilated LV. Further, the percentage of cases with normal LV dimension was 38% less in the no follow up group compared to the follow up group.

A cross tabulation of data regarding LV dimension and the follow up status of the patients is shown in Table (4.2) below.

Table (2.2): LV dimension by Follow up Cross tabulation

Follow up		LV dimension				Total
		Small	Normal	Mild Dilated	Dilated	
	No	2	16	3	9	30
	Yes	1	17	2	0	20
Total		3	33	5	9	50

Fisher's exact test showed a statistically significant difference, as regards the LV dimension, between those who were on regular follow up and those who were not (p=0.02).

4.2.2. The distribution of patients by Follow up status and LV hypertrophy is shown in figure (4.2).

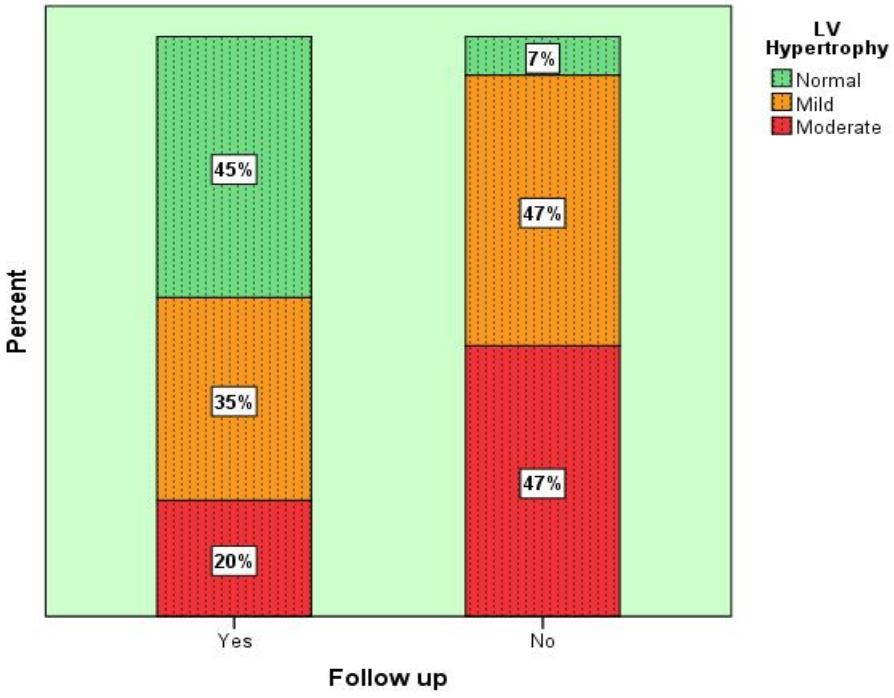


Figure (4.4): Distribution of patients by Follow up and LV hypertrophy

A cross tabulation of data regarding LV hypertrophy and the follow up status of the patients is shown in Table (2.4) below.

Table (2.3): Follow up and LV Hypertrophy Cross tabulation

Follow up	LV Hypertrophy			Total
	Normal	Mild	Moderate	
No	2	14	14	30
Yes	9	7	4	20
Total	11	21	18	50

Fisher's exact test - showed a statistically significant difference, as regards the LV hypertrophy, between those who were on regular follow up and those who were not (p=0.007).

4.3. Effect of medication

4.3.1. Effect of medication on LV dimension

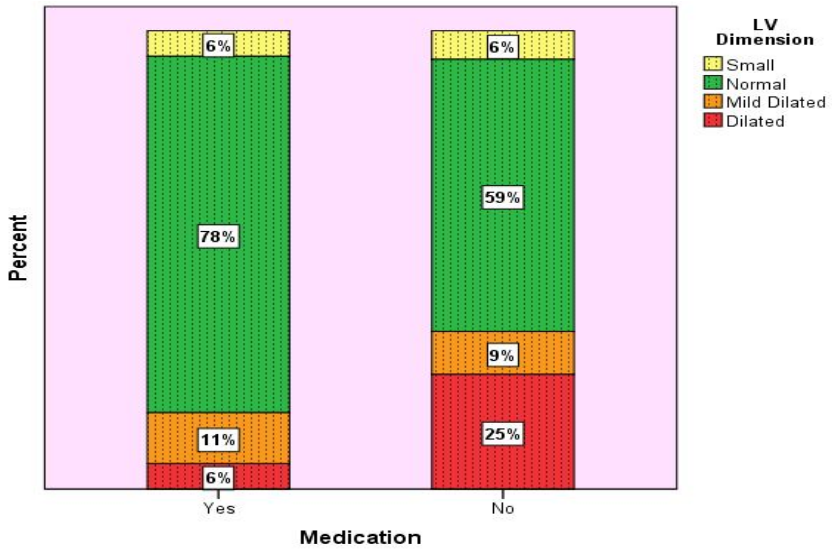


Figure (4.5): Distribution of patients by Medication and LV dimension

Figure (4.3) shows that there were four times as high a percent of patients with dilated LV among those who did not take medication compared to those who did, and about a quarter less a percent of patients with normal LV dimension among those who did not take medication compared to those who did.

A cross tabulation of data regarding LV dimension and whether medication was received by the patients is shown in Table (4.4) below.

Table (2.4): Medication by LV Dimension Cross tabulation

Medication	LV Dimension				Total
	Small	Normal	Mild Dilated	Dilated	
No	2	19	3	8	32
Yes	1	14	2	1	18
Total	3	33	5	9	50

Fisher's exact test - showed that there was not a statistically significant difference between those who were on medication and those who were not as regards the LV dimension (p=0.361).

4.3.2. Effect of Medication on LV hypertrophy

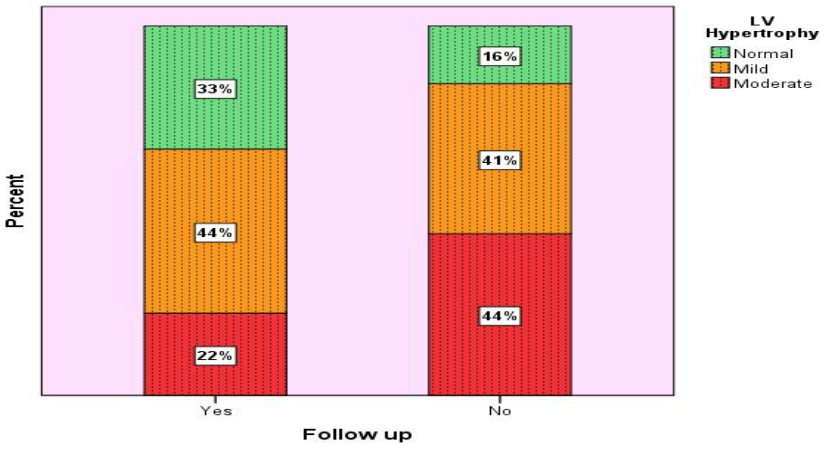


Figure (4.6): Distribution of patients by Medication and LV hypertrophy

Figure (4.4) shows that the percentage of patients who did not take medication and yet had no LV hypertrophy was about half that of patients who did, while the percentage of those with moderate LV hypertrophy was twice as big in patients who did not take

medication compared to those who did. However the percentage of those with mild LV hypertrophy was about the same in both groups

A cross tabulation of data regarding LV hypertrophy and whether medication was taken by the patients is shown in Table (4.5) below.

Table (2.5): Medication by LV hypertrophy Cross tabulation

Medication	LV Hypertrophy			Total
	Normal	Mild	Moderate	
No	5	13	14	32
Yes	6	8	4	18
Total	11	21	18	50

Also using Fisher's exact test – data showed that there was not a statistically significant difference, as regards LV hypertrophy, between those who were on medication and those who were not (p=0.178), i.e. the given data set did not provide sufficient evidence to reject the null hypothesis of equality of percentages across the two follow up groups.

CHAPTER FIVE

5.1. Discussion

Hypertension is the commonest of the cardiovascular risk factors, whose prevalence in Sudan ishigh among rural and urban residents respectively. Uncontrolled hypertension leads to a number of structural changes in the heart which eventually cumulates into interstitial fibrosis, myocardial wall thickness and functional alteration such as diastolic dysfunction.

Noninvasive assessment of diastolic filling by echocardiography provides important information about left ventricular (LV) status in selected subsets of patients.

This study was designed to assess values for two-dimensional (2D) M-mode echocardiographic dimensions in detection of Changes in Echocardiographic Parameters among hypertensive patients

The heart is the pump of the circulatory system, so it is reasonable that the increased increased arterial pressure affects it from the early stages of hypertension and it actually suffers the commonest hypertensionrelated organ damage. Any provoked alterations involving either the anatomy or the functionality of the heart can easily be detected and imaged by echocardiography, which represents a real-time, quick, reproducible, cheap, and widespread method.

This is why echocardiography is one of the very first examinations that a hypertensive patient is recommended to undergo. The echocardiographic assessment of the heart of a hypertensive patient is performed ontwo levels: i) an anatomic approach, which includes measurement of the heart cavities (Table 1), and ii) a functional approach, which includes assessment of indices of function. Overall, to summarize, a global echocardiographic evaluation of a patient with hypertension should include assessment of the following: a) left ventricular hypertrophy, cardiac mass and geometry; b) left ventricular function; c) left atrial volume and function; d)

the thoracic aorta; and e) coronary artery patency, to investigate the possible coexistence of coronary artery disease.

All the Echocardiographic reference parameters that we use to compare during Echocardiographic studies are derived from those defined in the western world. We till date do not have a proper reference range based on studies conducted in sudan or africa itself. It is a well knonwn fact that that the population in sudan has a very much different genetic and physical make up as compared to the population in the west. The difference in the body size in itself brings into question the reference range that we quote as normal values derived from studies conducted in the west. We have thus made an effort to study a hypertensive population in Sudan, for recording changes of echocardiographic parameters.

Despite many technical limitations (interobserver variability, low quality imaging in obesepatients, obstructive lung disease, etc.) echocardiography is more sensitive than electrocardiography in identifying left ventricular hypertrophy and predicting cardiovascular risk, thus assisting in the selection of appropriate therapy. Given the relationship between increased left ventricular mass and cardiovascular risk, in its latest guidelines for the management of arterial hypertension the European Society of Cardiology has included measurement of the dimensions of the left ventricle and further calculation of mass(Table 1). Proper evaluation includes measurement of the interventricular septum, left ventricular posterior wall thickness, and end-diastolic diameter.

Finally, it is well known that the variability of echo measurements is non-trivial.Echocardiography provides a reliable means of assessing left ventricular systolic function. Left ventricular ejection fraction, as well as endocardial and mid-wall fractional shortening, are the most practical systolic indices that have also been proposed as possible additional predictors of cardiovascular events.

The Framingham study showed that the hazard for developing heart failure in hypertensive as compared with normotensive subjects was about twofold in men and

threefold in women,(Shahgaldiet al, 2009),thus documenting the importance of assessing left ventricular function in hypertensive heart disease.

The conventional way of assessing left ventricular function with echocardiography is via the left ventricular ejection fraction, determined by applying Simpson's method of discs.(Shahgaldiet al, 2009) If the left ventricular ejection fraction is initially evaluated to be <50%, there is a nearly tenfold increased risk for hospitalization for congestive heart failure as compared to hypertensive patients with a normal ejection fraction. Despite the widespread clinical use of the left ventricular ejectionfraction, it should be kept in mind that it is a load-dependent systolic index. From this point of view, it is clearly very important to identify the slightest initial impairment of left ventricular function, using additional indices apart from ejection fraction that are not load-dependent. This was the reason for the introduction into clinical practice of mid-wall fractional shortening,a systolic index of left ventricular function that is relatively independent of afterload. Notably, hypertensive patients with left ventricular hypertrophy and a normal ejection fraction have been found to have abnormal mid-wall fractional shortening.(Devereux et al,. 2000)

Data showed that the prevalence of abnormality in patients' LV dimensions was 34% (Exact Clopper-Pearson 95% confidence interval: 21.2% to 48.8%); the prevalence of LV hypertrophy was 78% (Exact Clopper-Pearson 95% confidence interval: 64% to 88.5%); and the prevalence of abnormal LV dimensions and/or LV hypertrophy was 84% (Exact Clopper-Pearson 95% confidence interval: 70.9% to 92.8%). The echocardiogram findings of the studied cases are summarized in Table (1) this agree with Sumbul J et al 2009.

From the results shown above there is a high prevalence of abnormalities in the left ventricular of the studied hypertensive cases. The prevalence of abnormality in patients' LV dimensions was 34% (95% CI: 21.2% to 48.8%); the prevalence of LV hypertrophy was 78% (95% CI: 64% to 88.5%); and the prevalence of abnormal LV dimensions and/or LV hypertrophy was 84% (95% CI: 70.9% to 92.8%).

The top three prevailing adaptation patterns were: Mild LVH (26%); Moderate concentric LVH (10%); and moderate asymmetric LVH (8%).Follow up appeared to have played a significant role in reducing the severity of both LV dilation and LV hypertrophy in hypertensive patients.

Medication, however, did not show a significant reduction in the percentages of the various LV abnormalities in hypertensive patients - possibly due to the anticipated inaccuracy in answering the question about whether the patients took their medication regularly or as prescribed by their physicians.

Essential hypertensive is the term for high blood pressure with unknown cause. It accounts for about 95% of case.Secondary hypertensive is the term for high blood pressure with a known direct cause.

From this result the prevalence of abnormality in patients LV dimensions was 34% and the prevalence of abnormal LV hypertrophy was 84%. The top three prevailing adaptation patterns were mild LVH 26% moderate concentric LVH 10% and moderate asymmetric LVH 8%

Medication however did not show significant reduction in the percentage of the various LV abnormalities in hypertensive patients. Possibly due to the anticipated inaccuracy in answering the question about whether the patients took their medication regularly or as prescribed by their physicians.

5.2-Conclusion

From the results shown above, it can be concluded that there is a high prevalence of abnormalities in the left ventricular of the studied hypertensive cases. Follow up appeared to have played a significant role in reducing the severity of both LV dilation and LV hypertrophy in hypertensive patients.

Medication, however, did not show a significant reduction in the percentages of the various LV abnormalities in hypertensive patients - possibly due to the anticipated inaccuracy in answering the question about whether the patients took their medication regularly or as prescribed by their physicians.

This study attempts to simulate the basic clinical or experimental echocardiography M-mode protocol in hypertensive patients. echocardiography itself could be used for heart disease screening in clinical patients Precise echocardiographic measurements in a study employing experimental rats could result in a precise description of models with zero-pain and good clinical relevance. We have established reliable regression equations for the calculation of basic echocardiographic parameters Despite its technical limitations, echocardiography is really a significant tool for the evaluation of a hypertensive patient. Assessing a hypertensive patient echocardiographically does not simply represent adherence to a routine examination procedure that has limited clinical value. Conventional echocardiography, alongside newer, richer techniques, provides invaluable information about the extent of heart damage related to hypertension and cardiovascular risk, thus helping us to achieve better management and apply better treatment.

This study is small study consisting of randomized populations of people in Khartoum state and the study population cannot be the representation of whole sudanese population. Relatively the sample size was also small.

5-3-Recommendation

- Since echocardiography is not currently endorsed as a routine investigation for Sudanese hypertensive patients, despite its being a non-invasive and non-expensive method of revealing cardiac functional and morphological changes, we recommend its use in follow up and monitoring of hypertensive patients in the Sudan.
- A sufficiently large sample is needed in future studies to assess the prevalence of various patterns of LV adaptation in hypertensive patients.
- Echocardiographic machines must be made available in emergency hospitals/departments together with well trained staff.
- As technological advances allow more detailed examination of these structures, our knowledge of normal anatomy and pathologic conditions will allow for more accurate and detailed examinations.
- Further study needed to show the relation of Echo and other imaging modalities and cardiac function.
- One imaging modality is unable to reliably distinguish various cardiac diseases.
- Ultrasound investigation should be used as routine for any patient complaining of heart disorders.
- All the Echocardiographic reference parameters that we use to compare during Echocardiographic studies are derived from those defined in the western world. We till date do not have a proper reference range based on studies conducted in sudan or africa itself. It is a well knonwn fact that that the population in sudan has a very much different genetic and physical make up as compared to the population in the west. The difference in the body size in itself brings into question the reference range that we quote as normal values derived from studies conducted in the west. We have thus made an effort to study a hypertensive population in Sudan, for recording changes of echocardiographic parameters.

- Despite many technical limitations (interobserver variability, low quality imaging in obese patients, obstructive lung disease, etc.)
- This study is small study consisting of randomized populations of people in Khartoum state and the study population cannot be the representation of whole sudanese population. Relatively the sample size was also small.

- The results of this study strongly indicate the need for a larger scale study to further establish ethnic-specific and gender-specific echocardiographic reference values for Sudanese population.
- This study is small study consisting of randomized populations and the study population cannot be the representation of whole Sudan population.

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Appendix 1

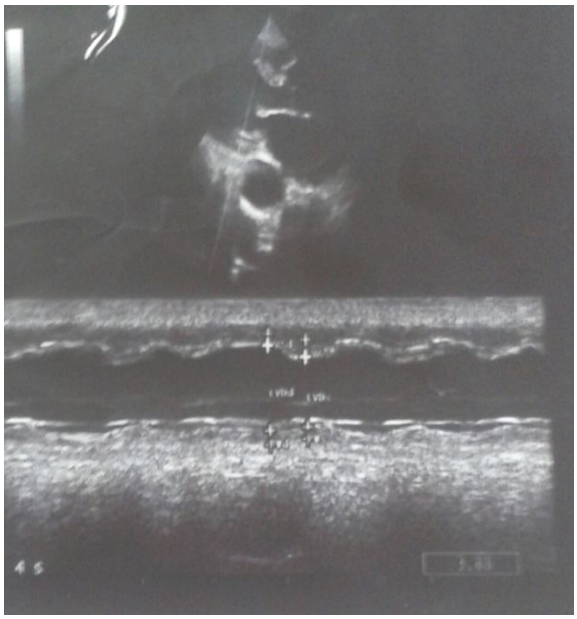


Figure (5.1) Female,70years, EF(59), LVED(4.8), LVES(3.3), IVS(1.5), PW(1.2)

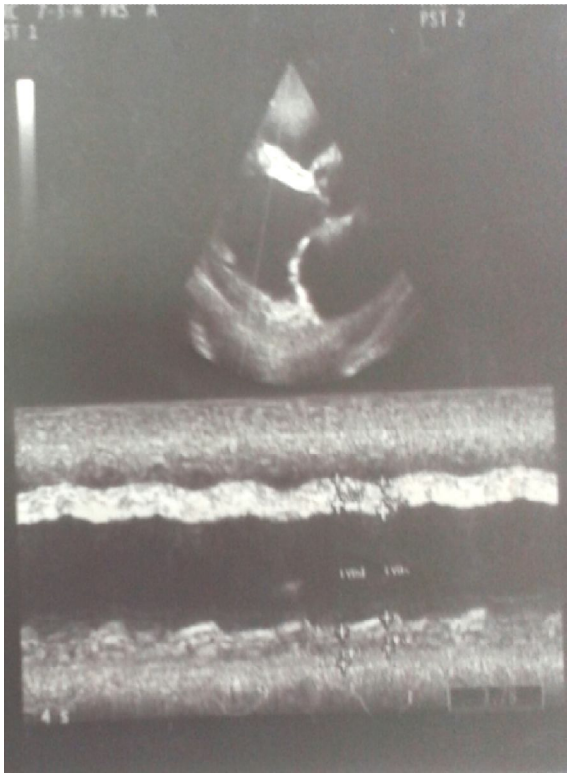
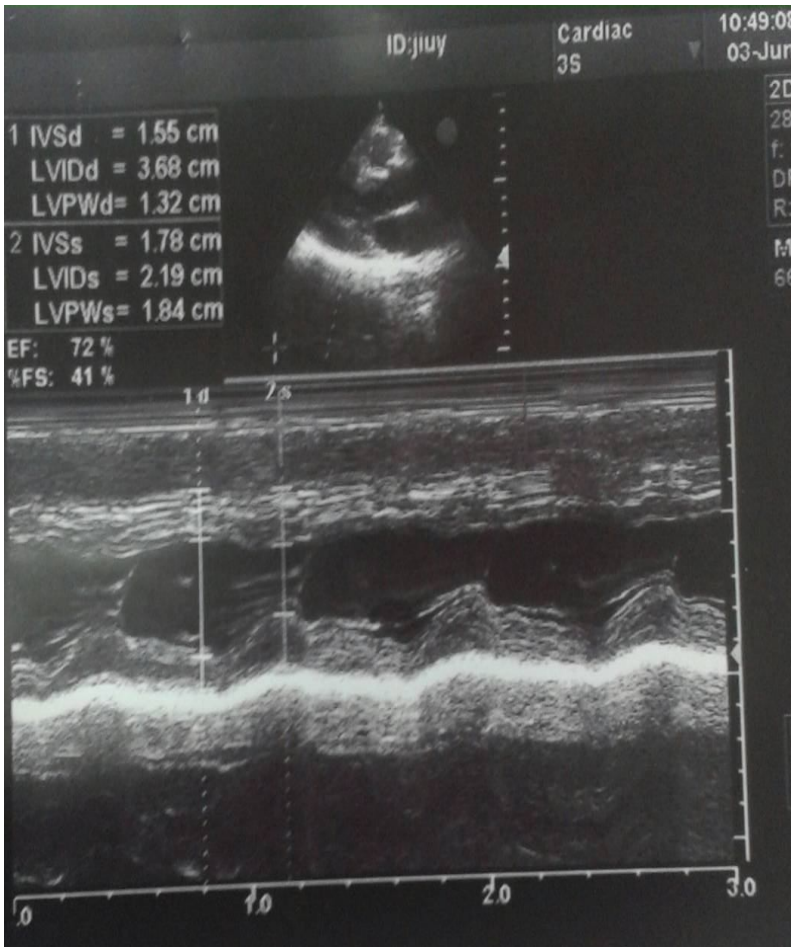


Figure (5.2) male,51years, EF(33), LVED(6.8), LVES(5.3), IVS(1.5), PW(1.2)



Figure(5.3) male,40years, EF(78), LVED(4.7), LVES(2.2), IVS(1.5), PW(1.3)

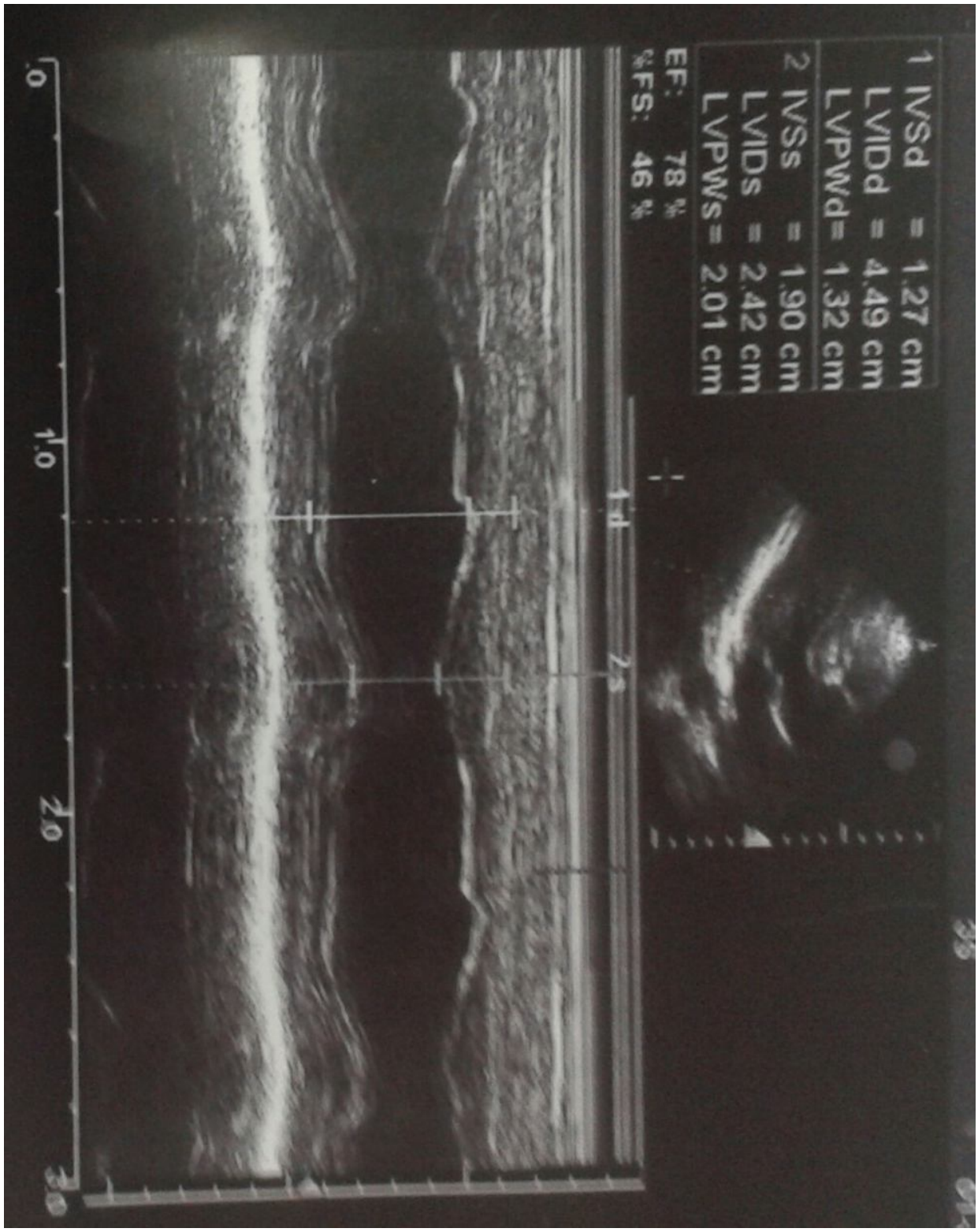


Figure (5.4) male,72years, EF(78), LVED(4.5), LVES(2.4), IVS(1.3), PW(1.3)

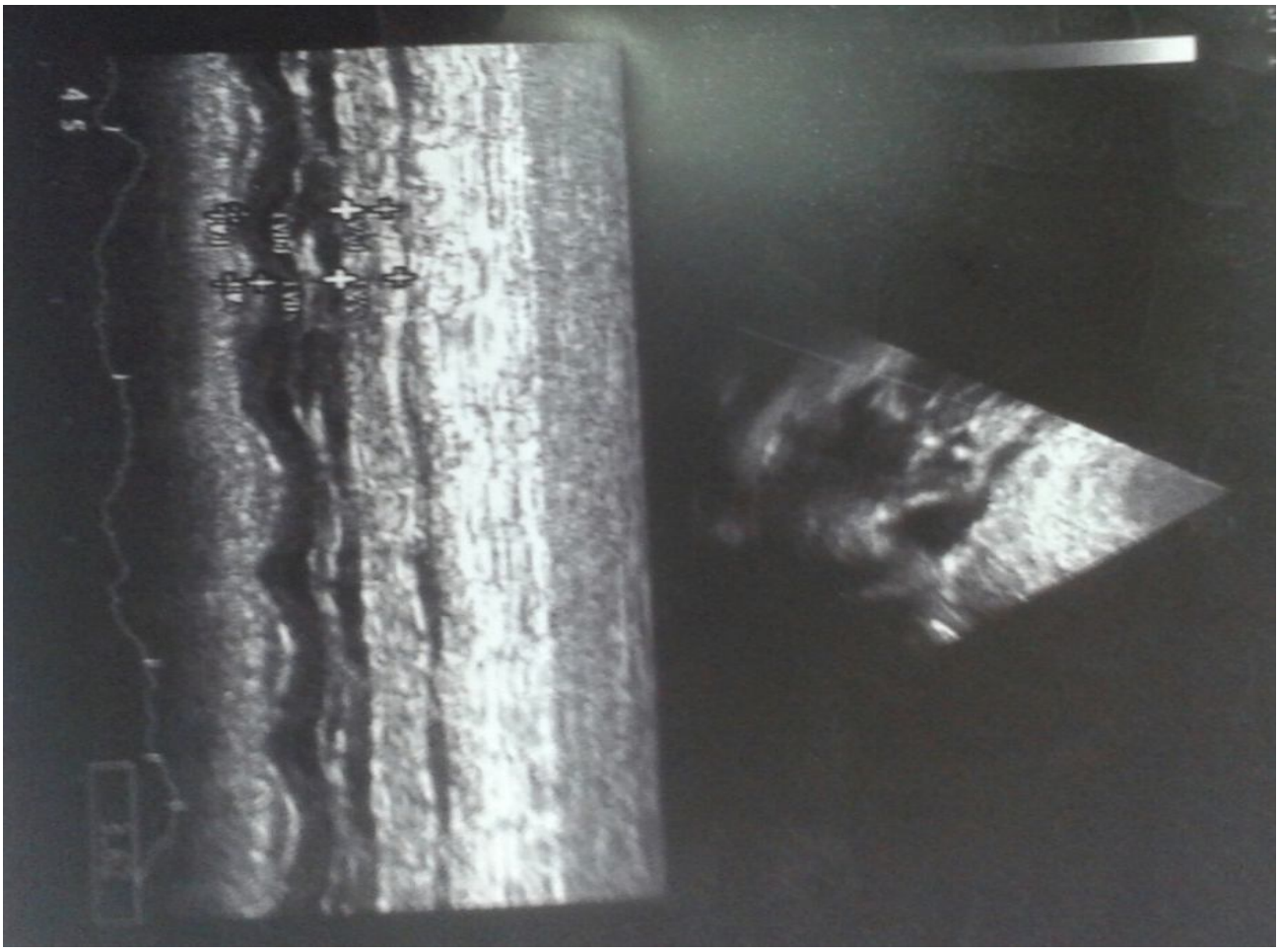
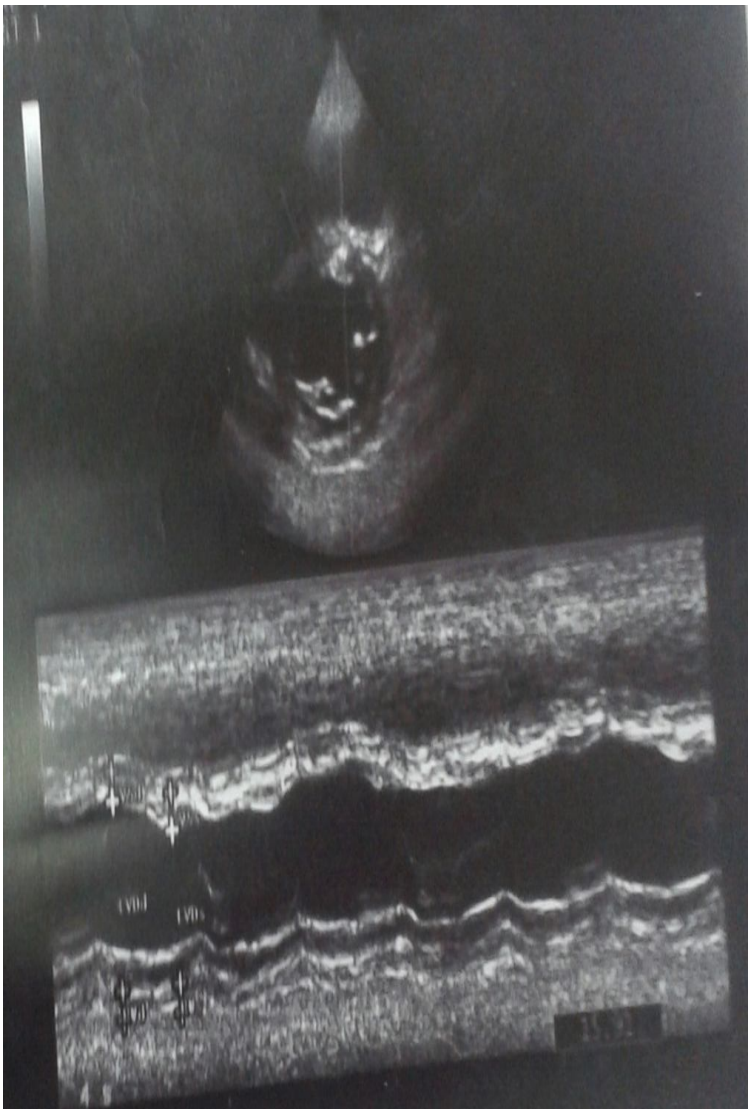


Figure (5.5) Female,75years, EF(55), LVED(4.5), LVES(2.4), IVS(1.6), PW(0.5)



Figure(5.6) Female,68years, EF(39), LVED(5.3), LVES(4.3), IVS(0.8), PW(0.8)

Appendix (B)
Data collection sheet

No	Age	Gender[1]	L V demission		Septum demission		Follow up		Medication	
			LVED	LVSD	IVC	PW	Yes	No	Yes	No
1.	35 years	M	4.4	2.9	1.2	1.1	√		√	
2.	37 years	M	4.2	2.4	1.4	1.2		√	√	
3.	57 years	M	4.6	2.7	1.4	1.0	√			√
4.	31 years	F	4.4	2.9	1.1	0.7	√		√	
5.	74 years	M	4.9	2.3	1.5	1.0		√		√
6.	70 years	F	5.5	4.4	1.5	0.9		√		√
7.	50 years	F	5.4	3.4	1.2	1.1		√		√
8.	74 years	F	5.6	4.6	1.5	1.0		√		√
9.	57 years	M	3.5	2.2	1.3	1.2		√		√
10.	72 years	M	4.6	3.0	1.0	1.2	√		√	
11.	34 years	M	5.2	3.5	1.0	1.2	√	√		
12.	65 years	F	4.0	2.6	0.9	0.9	√		√	
13.	47 years	M	4.2	2.7	1.1	1.0	√	√		
14.	65 years	M	4.2	2.9	1.4	1.0		√		√
15.	31 years	M	4.7	3.2	0.9	0.9	√			√
16.	47 years	F	5.3	3.5	1.5	0.8		√		√
17.	22 years	M	4.5	2.9	1.2	0.7	√		√	
18.	73 years	F	4.5	2.9	0.7	0.7	√		√	
19.	75 years	M	4.5	2.4	1.6	0.5		√		√
20.	60 years	M	5.3	4.5	1.6	0.8		√		√
21.	75 years	M	4.4	3.0	1.4	1.0		√		√

[1] M: Male; F:Female

No	Age	Gender[1]	L V demission		Septum demission		Follow up		Medication	
			LVED	LVSD	IVC	PW	Yes	No	Yes	No
22.	52 years	M	5.0	3.2	1.4	1.2		√		√
23.	68 years	M	4.4	2.8	1.5	0.8		√		√
24.	32 years	M	5.0	3.4	1.2	0.9		√		√
25.	80 years	M	4.3	2.9	1.5	1.4		√		√
26.	65 years	M	4.0	2.6	1.7	1.5		√		√
27.	35 years	F	4.4	2.9	0.8	0.9	√		√	
28.	70 years	F	4.8	3.3	1.5	1.2	√		√	
29.	68 years	F	5.3	4.3	0.8	0.8		√		√
30.	33 years	F	4.6	3.2	1.0	1.0	√		√	√
31.	83 years	F	4.1	2.6	1.3	0.9		√		√
32.	72 years	F	4.1	2.2	1.5	1.6		√		√
33.	68 years	M	5.2	4.2	1.7	1.6		√		√
34.	60 years	M	4.5	2.4	1.3	1.3		√	√	
35.	64 years	M	4.3	2.5	1.5	1.6		√		
36.	55 years	F	4.3	2.8	1.5	1.3		√		√
37.	40 years	M	3.7	2.2	1.5	1.3	√		√	
38.	62 years	M	4.3	2.5	1.5	1.7		√		√
39.	60 years	F	3.9	2.6	1.3	1.2	√		√	√
40.	75 years	F	4.3	3.0	1.3	1.2		√		√
41.	51 years	M	6.8	5.3	1.3	1.7		√		√
42.	65 years	M	5.9	4.6	0.8	0.6		√		√
43.	43 years	M	4.0	2.6	1.3	0.9	√		√	
44.	44 years	F	4.7	3.0	1.3	0.9	√			√
45.	59 years	F	4.2	2.9	1.2	1.2	√			√
46.	50 years	M	5.5	3.8	1.4	1.1		√	√	

No	Age	Gender[1]	L V demission		Septum demission		Follow up		Medication	
			LVED	LVSD	IVC	PW	Yes	No	Yes	No
47.	33 years	M	6.3	5.3	1.3	1.4		√		√
48.	55 years	F	5.0	4.7	1.5	1.4	√		√	
49.	67 years	F	4.8	3.4	1.3	1.3	√		√	
50.	35 years	M	5.3	3.9	1.3	0.7		√	√	

Appendix (c)

Echo finding

Serial	LVDD	LVSD	LV dimension	IVC	PW	LVH	Echo finding
1	4.4	2.9	Normal	1.2	1.1	Mild	mild LVH
2	4.2	2.4	Normal	1.4	1.2	Mild	mild LVH
3	4.6	2.7	Normal	1.4	1.0	Mild	mild LVH
4	4.4	2.9	Normal	1.1	0.7	NORMAL	Normal echo study
5	4.9	2.3	Normal	1.5	1.0	moderate	moderate LVH
6	5.5	4.4	Dilated	1.5	0.9	moderate	dilated LV& moderate LVH
7	5.4	3.4	Mild Dilated	1.2	1.1	mild LVH	Dilated LV & mild LVH
8	5.6	4.6	Dilated	1.5	1.0	Moderate	Dilated LV & moderate LVH
9	3.5	2.2	SMALL	1.3	1.2	Mild	mild LVH&small LV
10	4.6	3	Normal	1	1.2	Normal	Normal echo study
11	5.2	3.5	Mild Dilated	1.0	1.2	Normal	Mild Dilated LV
12	4	2.6	Normal	0.9	0.9	Normal	Normal echo study
13	4.2	2.7	Normal	1.1	1.0	Normal	Normal echo study
14	4.2	2.9	Normal	1.4	1.0	Mild	mild LVH
15	4.7	3.2	Normal	0.9	0.9	Normal	Normal echo study
16	5.3	3.5	Mild Dilated	1.5	0.8	Moderate	Mild Dilated LV & Moderate LVH
17	4.5	2.9	Normal	1.2	0.7	MILD	MILD LVH
18	4.5	2.9	Normal	0.7	0.7	Normal	Normal echo study
19	4.5	2.4	Normal	1.6	0.5	MODERATE	MODERATE LVH
20	5.3	4.5	Dilated	1.6	0.8	Moderate	DILATED LV& Moderate LVH
21	4.4	3.0	Normal	1.4	1.0	Mild	mild LVH
22	5.0	3.2	Normal	1.4	1.2	Mild	mild LVH
23	4.4	2.8	Normal	1.5	0.8	Moderate	moderate LVH
24	5.0	3.4	Normal	1.2	0.9	Mild	mild LVH
25	4.3	2.9	Normal	1.5	1.4	Moderate	Concentric Moderate LVH
26	4.0	2.6	Normal	1.7	1.5	Moderate	Concentric MODERATE LVH
27	4.4	2.9	Normal	0.8	0.9	Normal	Normal echo study
28	4.8	3.3	Normal	1.5	1.2	Moderate	MODERATE LVH
29	5.3	4.3	Dilated	0.8	0.8	Normal	Dilated LV
30	4.6	3.2	Normal	1	1	Normal	Normal echo study
31	4.1	2.6	Normal	1.3	0.9	Mild	mild LVH
32	4.1	2.2	SMALL	1.5	1.6	Moderate	Concentric moderate LVH &small LV
33	5.2	4.2	Dilated	1.7	1.6	Moderate	Dilated LV &MODERATE Concentric LVH
34	4.5	2.4	Normal	1.3	1.3	Mild	Concentric mild LVH
35	4.3	2.5	Normal	1.5	1.6	Moderate	moderate Concentric LVH
36	4.3	2.8	Normal	1.5	1.3	Moderate	moderate Concentric LVH
37	3.7	2.2	SMALL	1.5	1.3	Moderate	moderate Concentric LVH&small LV
38	4.3	2.5	Normal	1.5	1.7	Moderate	moderate Concentric LVH
39	3.9	2.6	Normal	1.3	1.2	MILD	MILD LVH
40	4.3	3.0	Normal	1.3	1.2	Mild	mild Concentric LVH
41	6.8	5.3	DILATED	1.3	1.7	Mild	Concentric LVH &dilated LV
42	5.9	4.6	Dilated	0.8	0.6	NORMAL	Dilated LV
43	4	2.6	Normal	1.3	0.9	MILD	MILD LVH
44	4.7	3.0	Normal	1.3	0.9	Mild	mild LVH
45	4.2	2.9	Normal	1.2	1.2	Mild	mild LVH
46	5.5	3.8	Dilated	1.4	1.1	Mild	mild LVH & Dilated LV
47	6.3	5.3	Dilated	1.3	1.4	Mild	DILATED LV&mild LVH
48	5	4.7	Mild Dilated	1.5	1.4	MODERATE	MILD DILATED LV & Concentric MODERATE LVH
49	4.8	3.4	Normal	1.3	1.3	MILD	MILD Concentric LVH
50	5.3	3.9	Mild Dilated	1.3	0.7	Mild	Mild Dilated LV & mild LVH