Sudan University of Sciences and Technology

College of Graduate Studies

Assessment of Cardiac Thoracic Ratio For Hypertensive Patients By Using Computed Tomography

تقييم بين ابعاد القلب إلى الصدر لمرضى الضغط الدم باستخدام الأشعة المقطعية المحوسبة

A Thesis submitted for Partial Fulfillment for the Degree of Masters(M. Sc) in Diagnostic Radiologic Technology

By

Rayan Salah EldienAltayebElsafi

Supervisor

Dr/ Caroline Edward Ayad

(Associate Professor)

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الآيـــــة

بسم الله الرحمن الرحيم

قال تعالى:

قَالُوا۟ سُبْحَٰنَكَ لَا عِلْمَ لَنَآ إِلَّا مَا عَلَّمْتَنَآ ۖ إِنَّكَ أَنتَ ٱلْعَلِيمُ ٱلْحَكِيمُ ﴿٣٢﴾

صدق الله العظيم

(سورة البقرة، الآية (٣٢))

Dedication

To;

My parents...

My Family……..

my friends……..

And My teachers

Acknowledgment

First of all, I thank Allah the Almighty for helping me complete this project. I thank Dr. Caroli ne Edward Ayad, my supervisor, for her help and guidance.

I would li ke to express my gratitude to the whole staff of the Radi ology department of AlamalMedical Centre and Doctor Clinical H ospital, for thei r great help and support.

I am greatly indebted to my mom..,Nimat Mubarak Osman for bearing with me during the past several months.

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

List of Contents

List of tables

List of Figures

List of Abbreviation

The Abbreviation	The full word
PHT	Pulmonary Hypertension
CT	Computed Tomography
HRCT	High Resolution Computed Tomography
CTR	Cardiac Thoracic Ratio
AV	atrio ventricular
CHF	Congestive Heart failure
LAE	Left atrial enlargement
PT	Patient
BP	Blood Pressure

Abstract

The CT scan is one of the methods to assess the heart enlargement in the hypertensive patients, the objective of this study was to the assessthe relationship between high blood pressure and cardiothoracic ratio of the Heart.

The study took place during the period from June to December 2014 at Radiology department ofAlamal Medical Centre and Doctor Clinical Hospital.

Cardiac thoracic ratio were taken in Patients who were diagnosed as hypertension Patient, Both gender were included 17 female and 23 were male with mean of age were (54-80).

The CTR was measured in axial CT chest at the level of T5, CTR values for both control and hypertensive were calculated.

The mean range of CTR for hypertensive patient were between (0.8-1) where the control were found to be between (0.5-0.6)

The relation between the control and hypertensive were found to significant at level p value 0.051.

There is significant relation between CTR hypertensive and high blood pressure value p=<0.05.

The results showed that a significant relationship between the cardiac thoracic ratio value with age, weight, medication duration of hypertensive patients.

مستخلص البحث

الاشعه المقطعيه هي واحده من الطرق لتقيم حجم القلب لمرضى ضغط الدم .

الهدف من هذه الدراسه تقيم العلاقه مابين نسبه قياس القلب الي الصدر للمرضي ضغط الدم، استغرقت فترة الدراسة مابين يونيو- ديسمبر ٢٠١٤ م .

تم قياس نسبه القلب الي الصدر في مقاطع محوريه بالاشعه المقطعيه للصدر في مستوي الفقره الصدريه الخامسه للمجموعهمن المرضي تكونت من ٤٠ مريض ضغط دم شمله نساء ورجال بلغ عدد الرجال ٢٣ وعدد النساء ١٧ وللمجموعه الضبط بلغت ١٩ من النساء والرجال.

تم حساب قيمه نسبه القلب الي الصدر للمجموعتين حيث بلغت قيمه متوسط نسبه قياس محيط القلب للصدر للمرضي ضغط الدم مابين ٠.٨الي ١ بينما بلغت نسبه متوسط قياس القلب الي الصدر للمجموعة المضبوطة مابين ٠.٥ الي ٠.٦ ووجد ان هنالك فرق معنوي مابين المجوعتين.

ووجد ان هنالك علاقه ربط مابين نسبه قياس القلب الي الصدر وارتفاع ضغط الدم.

تم مقارنة النتائج المدروسه لأوزان وأعمار وأنواع العلاج المستخدمه والفتره الزمنيه للمرض وجد أن نسبه زياده بين ابعاد القلب الي الصدر ليس له علاقه بعمر ووزن المريض ، بينما اتوجد علاقه مباشره بين ارتفاع ضغط الدم وابعاد القلب الي الصدر.

Chapter one

Introduction

Chapter One

1.1 Introduction

Hypertension is a major risk factor for cardiovascular morbidity and mortality. The presence of hypertension more than doubles the risk for coronary heart disease, including acute myocardial infarction and sudden death, and more than triples the risk of congestive heart failure as well as strokesPatientswith high blood pressure frequently have abnormalities of cardiac structure or function, including left ventricular hypertrophy, systolic and diastolic dysfunction and in extreme cases, overt heart failure.

There may also be concomitant or related coronary heart disease and an increased risk of arrhythmias and sudden death (Kannel and Cobb. 1992).

Many of these factors are inter-related and their individual contributions are difficult to quantify. There is, however, some debate as to whether 'hypertensive Cardiomyopathy' exists as a separate entity septic to hypertension. The term 'cardiomyopathy', however, would normally be reserved for intrinsic myocardial Disease, where underlying causes such as hypertension and coronary artery disease have been excluded. Therefore, the preferred term should perhaps be 'hypertensive Heart disease', and given the many mechanisms by which the heart may be abnormal in hypertension, the term 'hypertensive heart disease' is probably not so Controversial (Kannel, Cobb. 1992).

Pulmonary arterial hypertension is a progressive disease characterized by raised pulmonary artery pressure above 25 mm hand pathological changes in the pulmonary precapillary vessels. New therapeutic strategies have considerably improved survival. However, the diagnosis of PHT can be missed because of nonspecific signs and symptoms. Echocardiography is used for screening and diagnosis, but the gold standard measurement of pulmonary artery pressure and

response to vasodilators is done by right heart catheterization. Computerized tomography angiography and high resolution CT are commonly used for the diagnosis of pulmonary embolism and underlying lung parenchymal disease (Devereux et al 1994).

Chronic elevation of pulmonary artery pressure causes dilation of the pulmonary arteries right atrium and ventricle

The correlation between the ratio of the main pulmonary artery/ascending aorta diameters and the pressure measurement by right heart catheterization, Asymmetric dilation of the pulmonary arteries, calcified thrombi and bronchial collaterals are also considered signs of chronic thromboembolic disease

Deviation of the interventricular septum can be found on echocardiography and was reported on CT as a subjective sign of raised right heart pressure. HRCT of the chest can show a mosaic perfusion pattern, pulmonary scars, and underlying pulmonary disease (Devereux 1994).

Alular heart disease is one of the most important heart disorders that causes heart failure in children and it can be congenital or acquired. Almost all acquired alular heart diseases are rheumatic in origin. Mitral valve involvement occurs in about three quarters of all cases of rheumatic heart disease and aortic valve involvement in about one quarter (Kannel et al 1996) .

Mitral regurgitation (MR) is the most common alular involvement in children with rheumatic heart disease and aortic regurgitation (AR) is less common. Apart from rheumatic heart disease, the major causes of MR and AR are infective endocarditis, collagen-vascular disease, cardiomyopathy, congenital heart disease and annular abnormalities. The left ventricle initially compensates in acute MR, in part by emptying more completely and in part by increasing preload, i.e., by use of the Frank-Starling principle. As regurgitation, particularly severe regurgitation,

becomes chronic, the left ventricular end-diastolic volume increases and the endsystolic volume returns to normal (Kannel1996).

Chest roentgenogram (CR) and echocardiographic examinations are essential parts of cardiac evaluation. Chest roentgenogram is often used to detect cardiac enlargement. Overall heart size can be evaluated from chest films in a variety of ways; two of the most popular ways are calculation of the cardiothoracic ratio from posterior anterior (PA) films alone, and determination of the total cardiac silhouette volume utilizing both PA and lateral projections. Two-dimensional and Doppler echocardiographic studies are useful for the measurement Of left ventricular end-diastolic and end-systolic dimensions, volumes, shortening fraction, ejection fraction and mass (Flohr 2005).

Computed tomography (CT) is an imaging technique which produces a digital topographic image from diagnostic x-ray. In the early 1970s a major innovation was introduced into diagnostic imaging. This innovation, x-ray computed tomography (CT), is recognized today as the most significant single event in medical imaging since the discovery of x-rays (William Russell, 2002).

Computed Tomography (CT) was invented by a British engineer, Sir Godfrey Hounsfield who also won the Nobel Prize because of His invention. CT was first introduced in the clinical practice in 1972 which was only limited to the brain scan. Prior to that, X-ray planar radiography and fluoroscopy systems were the main contributors of radiation in imaging (Floret 2005).

Computed tomography (CT) is in its fourth decade of clinical use and has proved invaluable as a diagnostic tool for many clinical applications, from cancer diagnosis trauma to osteoporosis screening. CT was the first imaging modality that made it

possible to probe the inner depths of the body, slice by slice. Since 1972, when the first head CT scanner was introduced, CT has matured greatly and gained technological sophistication. Concomitant changes have occurred in the quality of CT images. The first CT scanner, an EMI Mark 1, produced images with 80 X 80 pixel resolution (3-mm pixels), and each pair of slices required approximately 4.5 minutes of scan time and 1.5 minutes of reconstruction time. Because of the long acquisition times required for the early scanners and the constraints of cardiac and respiratory motion, it was originally thought that CT would be practical only for head scans (Flohr , et al , 2005).

Cardiothoracic ratio (CTR)is cardiac size is measured by dropping parallel lines down both sides of the heart, at the most lateral points on each side, and measuring between them. Thoracic width is measured by dropping parallel lines down the inner aspect of the widest points of the rib cage, and measuring between these. The cardio-thoracic ratio can then be stated.

Here the CTR is approximately 15 : 33 (arbitrary units) and is therefore within the normal limit (expressed as a percentage) of 50% .The cardiac silhouette normally occupies less than 50% of the transverse diameter of the chest on a frontal cut. If this cardiothoracic ratio Exceeds 50%, the cardiac silhouette may be enlarged (Zierler 1998).

On upright Patients with high blood pressure frequently have abnormalities of cardiac structure or function, including left ventricular hypertrophy, systolic and diastolic dysfunction and in extreme cases, overt heart failure. There may also be concomitant or related coronary heart disease and an increased risk of arrhythmias and sudden death many of these factors are inter-related and their individual contributions are difficult to quantify. There is, however, some debate as to whether ‘hypertensive cardiomyopathy’ exists as a separate entity specific to Hypertension.

The term ‘cardiomyopathy’, however, would normally be reserved for intrinsic myocardial disease, where underlying causes such as hypertension and coronary artery disease have been excluded. Therefore, the preferred term should perhaps be ‘hypertensive heart disease’, and given the many mechanisms by which the heart may be abnormal in hypertension, the term ‘hypertensive heart disease’ is probably not so controversial (Zierler 1998).

1.2 Problem of the study:

There is no study done for Sudanese patients to detect the effect of the hypertation and the CTR which was be one of the causes that may change the heart measurement , therefore this study took place

1.3. Objectives of the study:

*** General objective: To**assess of cardiothoracic ratio for hypertensive patient using computed tomography

Specific objectives:

To assess the CTR in control group and hypertensive patient

To correlate the CTR,Age,Weight and bold pressure in hypertensive patient

To compare the CTR of hypertation patient with the control group

1.4: Overview of this study:

This study was consist of five chapters, chapter one will be an introduction introduce briefly this thesis and it was contain, general introduction about the CT chest and how to get the cardiothoracic ratio value and Hyperion as general , general and specific objectives, significant of the study in addition to the overview of the study. Chapters two was the literature review which contain the general theoretical background and previous study about the correlation between the heart size and the hypertensive factors age, Wight .history and type of treatments used. Chapter three will describe the methodology (materials, methods) was used in this

study. Chapter four was including result of presentation of final finding of study. Chapter five was including discussion, conclusion and recommendation for future scope in addition to references and appendices.

Chapter Two

Literature Review

Chapter Two

2.1 Theoretical background

Pulmonary hypertension may primarily affect either the arterial (pre- capillary) or the venous (postcapillary) pulmonary circulation. Pulmo- nary arterial hypertension may be idiopathic or arise in association with chronic pulmonary thromboembolism; pulmonary embolism caused by tumor cells, parasitic material, or foreign material; parenchymal lung disease; liver disease; vasculitis; human immunodeficiency virus infection; or a left-to-right cardiac shunt.

Its histologic characteristics include vascular changes—medial hypertrophy, intimal cellular pro- life ration, intraluminal thrombosis, and the development of plexiform lesions— that manifest primarily in the muscular pulmonary arteries. Features of pulmonary arterial hypertension that may be seen at com- puted tomography (CT) are central pulmonary artery dilatation, abrupt narrowing or tapering of peripheral pulmonary vessels, right ventricular hypertrophy, right ventricular and atrial enlargement, dilated bronchial arteries, and a mosaic pattern of attenuation due to variable lung perfu- sion. Pulmonary venous hypertension may result from pulmonary veno- occlusive disease, pulmonary venous compression by extrinsic lesions (eg, mediastinal fibrosis), left-sided cardiac disease, or pulmonary vein stenosis.

Its histologic hallmarks include venous intimal cellular prolifer- ation, medial hypertrophy, and thickening of the internal elastic lamina; capillary congestion and proliferation; interlobular septal thickening; lymphatic dilatation; and, sometimes, venous infarction and vascular changes characteristic of pulmonary arterial hypertension. CT scans in patients with pulmonary venous hypertension show pulmonary intersti- tial and alveolar edema with signs of pulmonary arterial hypertension. High-resolution CT with standard axial and angiographic acquisitions

is useful for identifying underlying disorders and differentiating among the various causes of secondary pulmonary hypertension (GhaliJK , et al 1991).

2.1.1 Anatomy of heart:

The Heart resides in the Anterior Mediastinum, behind the Sternum, slightly left of Midline, surrounded by the Lungs, in sac called the Pericardium the heart is Normally about the size of clenched fist, and weighs 250-300 grams in females and 300-350 grams in males Heart (front view) apex base superior vena cava right Atrium (behind and lateral) pulmonary artery aorta pulmonary veins (4 totals) left Anterior descending (polissar2006)

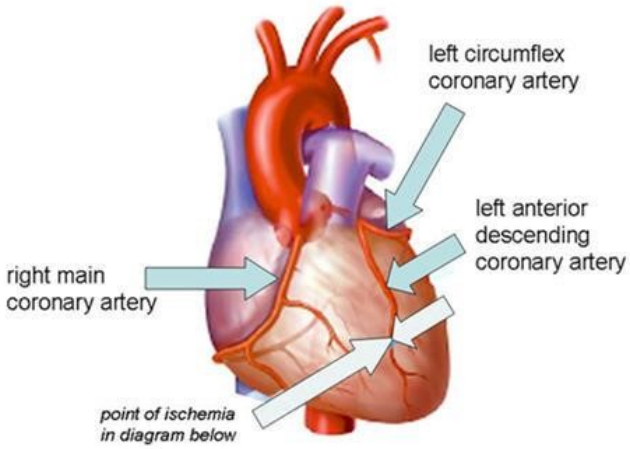

Figure 2-1 Show the front view (Human Morphology) (polissar2006).

External Anatomy of the Hear Has four hollow chambers, two smaller atria, two larger ventricles, Heart is rotated such that its right side (right atrium and ventricle)is located more anteriorly, while its left side (left atrium and ventricle) is located more posterior. The poster superior surface of the heart, formed primarily by the left atrium, is called the base. Related to T5 to T8 vertebrae in lying position.

The apex of the heart is the most anterior and inferior portion of the left Ventricle. This is the most mobile portion of the heart, and produces the "apex beat". The Surfaces: Anterior (stern costal) surface, the inferior (diaphragmatic) surface, has

contributions from both ventricles 2/3 by the left and 1/3 by the right. The posterior surface: is the least mobile portion of the heart. The Borders: the right margin or border is formed by, the right atrium the superior and inferiorvenae cave left margin or border is oblique and formed by, the left ventricle with contributions from the pulmonary trunk and the left auricle,

The superior borders oblique formed by:2 atria but mainly the left The inferior border is horizontal and formed by, Vessels returning blood to the heart include: Superior and inferior venae cave, right and left pulmonary veins, vessels conveying blood away from the heart: Coverings of the Heart (Pericardium) Pericardium: is a double-walled tough connective tissue sac enclosing the heart and roots of the great vessels (in middle mediastinum) Composed of, superficial fibrous pericardium tough, dense connective tissue, a deep two-layer serous pericardium thin, double-layered serous membrane The parietal layer lines the internal surface of the fibrous pericardium, the visceral layer or epicedium lines the surface of the heart.

They are separated by the fluid-filled pericardial cavity called the pericardial cavity Heart wall structure, three distinctive layers: .external epicedium. Middle myocardium .internal endocardium Internal anatomy of the heart, there are four heart chambers, right atrium, right ventricle, left atrium , left ventricle Each plays a role in the continuous process of blood circulation(polissar2006) .

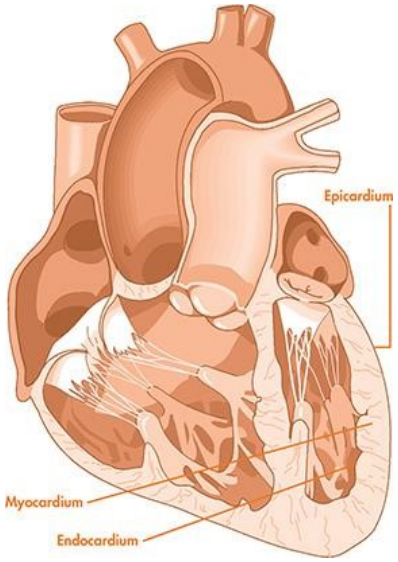

Figure 2.2 (Layers of the heart) (polissar 2006)

Valves permit the passage of blood in one direction and prevent its back flow, Heart Valves, heart valves ensure unidirectional blood flow through the heart Two major types ,(atrioventricular) valves semi lunar (valvesatrioventricular) (AV) valves lie between the atria and the ventricles R-AV valve = tricuspid valve-AV valve = bicuspid or mitral valve valves prevent backflow of blood into the atria when ventricles (polissar2006) .

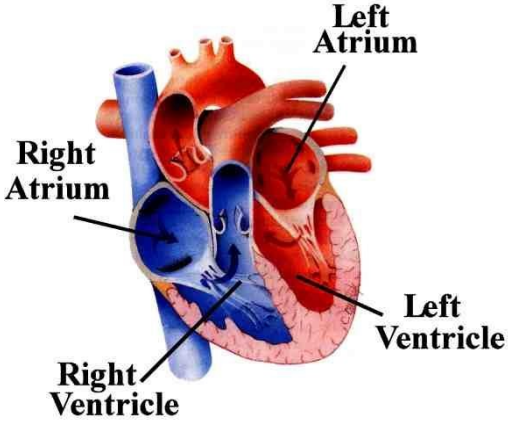

Figure 2.3 The champers of the heart (polissar2006)

2.3 The physiology:

Composed of the heart and the vascular system (blood vessels).The heart is a muscle pump. The function of the heart is to pump blood to all parts of the body and vary

that amount of blood according to the metabolic need of the body, the pumping action of the heart creates a pressure which pushes blood ahead into
The blood vessels.

The Blood Vessels, three types of blood vessels Arteries (and Arterioles).Carry blood away from heart to capillaries Carry blood away from heart to capillaries Permit exchange of materials within the tissues Veins (and venues).
Return blood from capillaries to heart, Heart is a double pump, right ventricle Pumps blood to lungs (through the pulmonary artery) Left ventricle pumps blood .

To body (through the aorta) this is a much harder task, so walls on left side of heart is larger than right side Blood pressure is also greater in the aorta more than Pulmonary artery. Heart beats, each heartbeat is called a cardiac cycle first the two atria contract at same time, then the two ventricles contract at same time, then all chambers relax Systole—heart muscle contraction Diastole relaxation of heart muscle Heart contracts (beats) approximately 70 times a minute; each beat lasts about 0.8 second the volume of blood pumped by each ventricle per beat is normally 70 ml. This is termed Stroke volume. The volume at the end of diastole is called EDV 120 ml (Verdecchia 1996).
Control of the heart beats, Intrinsic Control of heart beat Rhythmic contraction of Atria and ventricles are due to intrinsic conduction system of heart Nodal tissue, specialized cardiac muscle, located in two regions of heart SA (senatorial) node AV (atrioventricular) node SA node initiates heartbeat and automatically sends out an excitation impulse every 0.8 second Causes atria to contract When impulses reach AV node,
there is a slight delay that allows atria to finish their contraction before ventricles begin their contraction Signal for ventricles to contract travels from AV node through two branches of AV bundle before reaching numerous and smaller Purkinje

fibers. Extrinsic Control of Heartbeat Medulla oblongata, portion of brain that controls internal organs, can alter heart beats by way of autonomic nervous system Autonomic Nervous System has two subdivisions Parasympathetic System Decreases SA and AV nodal activity when we are inactive Sympathetic System Increases SA and AV nodal activity when we are active or excited
(Verdecchia, 1996).

Cardiac output, Stroke vol. x Heart Rate = Cardiac output.

Is the volume of blood pumped by each ventricle per minute? Controlled through Control of Stroke volume Heart rate (Human anatomy and physiology .sixth edition)

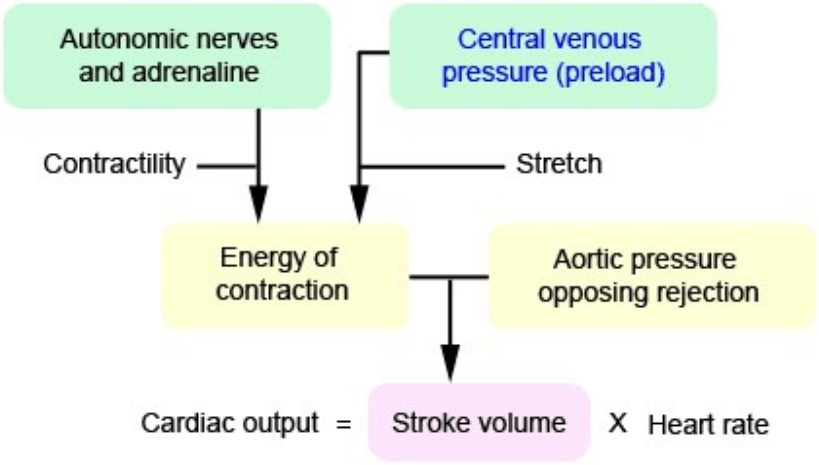

Figure 2.4 (Stroke volume can be raised by one or more of the following)

-Increasing the force of contraction of the myocardium (polissar2006)

-Lowering the arterial blood pressure opposing ejection

2.3.1 The common pathological disorders:

Hypertensive heart disease includes a number of complications of high blood pressure that affect the heart. While there are several definitions of hypertensive Heart disease in the medical literature (Verdecchia, 1996)**.**

2.3.2 Rheumatic heart diseases:

Acute Rheumatic Fever is apancardities, involving allayers of the heart. Pericarditis and myocarditis often responsible for initial symptoms.

Pericarditis – fibrous myocarditis aschoff bodies: Per vascular nodules of inflammatory cells including multinucleated Asch off cells, Anitschkowcells, lymphocytes, and plasma cells Myocarditis can lead to CHF and even death

endocarditis- initially results in tiny vegetation's along lines of closure of mitral and aortic valves, with little functional significance

2.3.3 Ischemic heart disease:

Although atherosclerosis of the coronary arteries is the most common mechanism Responsible for myocardial ischemia, other less common mechanisms can also Cause ischemia (Verdecchia, 1996).

2.3.4 The Congenital Heart Disease:

Left ventricular hypertrophy: is usually symptomless. Symptoms and signs of Chronic heart failure can include: Fatigue Irregular pulse or palpitations swelling Of feet and ankles Weight gain Nausea Shortness of breathe Difficulty sleeping flat in bed (orthopnea) Bloating and abdominal pain Greater need to urinate at night altered mentation (in severe cases) An enlarged heart (cardiomegaly) (Charles - 2006) .

Cardiomegaly: is a medical condition wherein the heart is enlarged, cardiac dilation is where mainly one or more heart chambers is mainly located at must

This regions, Ventricular enlargement Atrial enlargement Left atrial enlargement Right atria enlargement Clinical Inform Abnormal enlargement of the hear, enlargement of the heart, usually indicated by a cardiothoracic ratio above 0.50. Heart enlargement may involve the right, the left, or both heart ventricles or heart atria. Cardiomegaly is a nonspecific symptom seen in patients with chronic systolic heart failure (Charles - 2006).

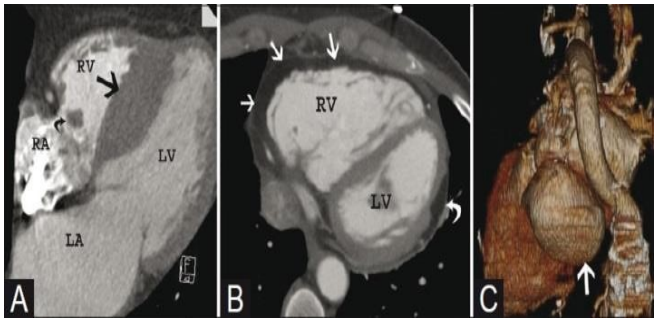

Figure 2.5 Short-axis CT image shows severe thickening of the mitral leaflets (arrows) in a severe left atrial enlargement.

Hypertrophy or enlargement of the heart. Left atrial enlargement (LAE) or left atrial dilation refers to enlargement of the left atrium (LA) of the heart, and is a form of cardiomegaly in the general population, obesity appears to be the most important risk factor for LAE.[1] LAE has been found to be correlated to body size, independent of obesity, meaning that LAE is more common in people with a naturally large body size (Tans -2009) Heart imaging:

2.4. CT Chest:

2.4.1 The indication

- A CT scan of the chest may be performed to assess the chest and its organs for tumors and other lesions, injuries, intrathoracic bleeding, infections, unexplained chest pain, obstructions, or other conditions, particularly when another type of examination, such as X-rays or physical examination, is not conclusive.
- A CT scan of the chest may also be used to evaluate the effects of treatment of thoracic tumors. Another use of chest CT is to provide guidance for biopsies and/or aspiration of tissue from the chest.
- The imaging protocol:
- Scan gram (scout view)

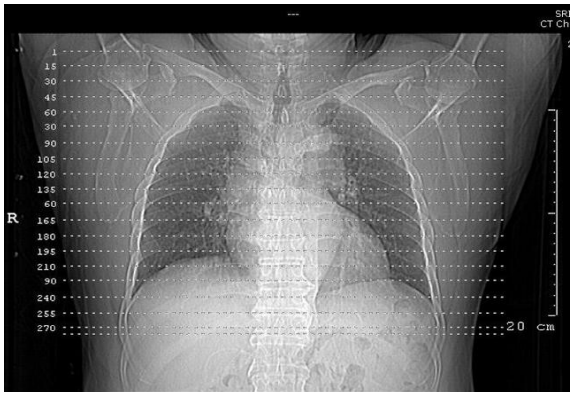

Figure 2-6 Show scout view (Mercy-2012) -Axial

Axil cut of CT images

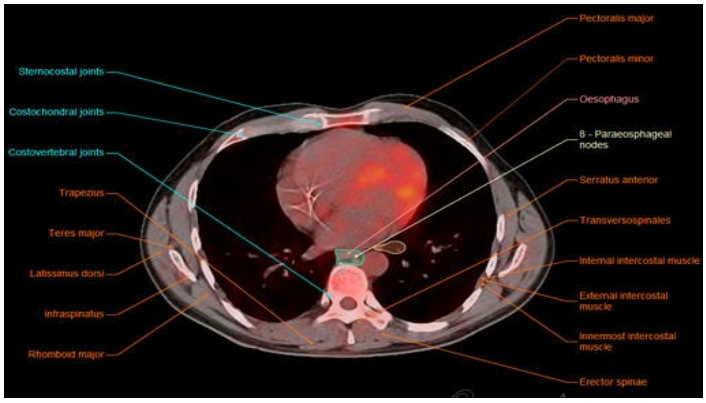

Figure 2-7 Show axial view (Mercy-2012)

2.4.2 Chest x ray:

All chest views are taken at 72” SID to minimize magnification.

All chest view are taken using high kVpto obtain a broad scale of contrast.

Routine: P-A & Lateral, Supplemental: Apical Lordotic, Anterior, Oblique Views

36.5 P-A Chest, Measure: P-A at mid chest, Protection: Half Apron, SID: 72” Bucky ,No Tube Angle, Film: 14” x 17” regular I.D. up Portrait unless wider than 35 cm, Marker: Pronated

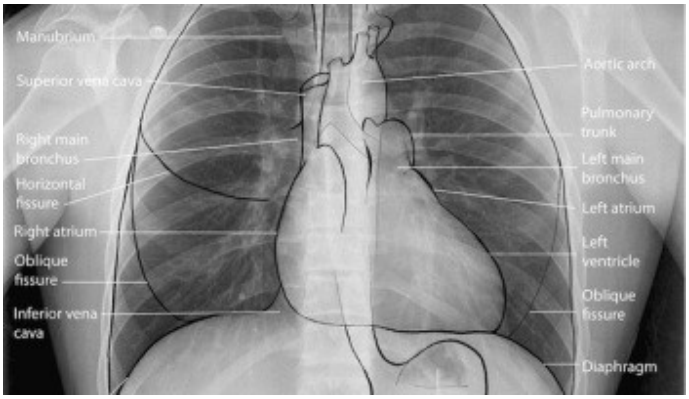

Figure 2.5 show PA chest x-ray(Mercy-2012)

Pulmonary arterial hypertension is a progressive disease characterized by raised pulmonary artery pressure above 25 mmHg and pathological changes in the pulmonary precapillary vessels. New therapeutic strategies have considerably improved survival. However, the diagnosis of PHT can be missed because of nonspecific signs and symptoms. Echocardiography is used for screening and diagnosis, but the gold standard measurement of pulmonary artery pressure and response to vasodilators is done by right heart catheterization. Computerized tomography angiography and high resolution CT are commonly used for the diagnosis of pulmonary embolism and underlying lung parenchyma disease (Messerli1984).

Chronic elevation of pulmonary artery pressure causes dilation of the pulmonary arteries right atrium and ventricle.

The emergence of new treatments and the close follow-up required for patients with PHT emphasize the need for non-invasive imaging studies. The aim of this study was to assess the additional capability of CT angiography and HRCT to diagnose and estimate the severity of PHT compared to right heart catheterization, echocardiography and pulmonary function tests, Hypertension is a major risk factor for cardiovascular morbidity and mortality. The presence of hypertension more than doubles the risk for coronary heart disease, including acute myocardial infarction

and sudden death, and more than triples the risk of congestive heart failure as well as strokes (Messerli-1984).

Patients with high blood pressure frequently have abnormalities of cardiac structure or function, including left ventricular hypertrophy, systolic and diastolic dysfunction and in extreme cases, overt heart failure. There may also be concomitant or related coronary heart disease and an increased risk of arrhythmias and sudden death many of these factors are inter-related and their individual contributions are difficult to quantify. There is, however, some debate as to whether 'hypertensive cardiomyopathy' exists as a separate entity specific to Hypertension. The term 'cardiomyopathy', however, would normally be reserved for intrinsic myocardial disease, where underlying causes such as hypertension
And coronary artery disease has been excluded. Therefore, the preferred term should perhaps be 'hypertensive heart disease', and given the many mechanisms by which the heart may be abnormal in hypertension, the term 'hypertensive heart disease' is probably not so controversial (Messerli-1984).

The purpose of this review is to describe the various mechanisms whereby the heart is abnormal in hypertension and to discuss the possibility of a discrete entity Called hypertensive cardiomyopathy (Moraes-, 1997).

2.5 Left ventricular hypertrophy:

Left ventricular hypertrophy has long been recognized as an important clinical prognostic entity. Epidemiological research has shown that left ventricular hypertrophy itself is associated with increased mortality and mortality for myocardial infarction, heart failure and stroke. There is a continuous graded relationship between left ventricular mass and the development of cardiovascular

Disease with no distinct threshold separating the postulated ‘compensatory’ from ‘pathological’ left ventricular hypertrophy. In normotensive adults, for example, left ventricular mass is directly related to the risk of developing later hypertension., raising the possibility that left ventricular hypertrophy may also be involved in the development of hypertension in the first place, as well as being a consequence of raised systemic pressure. There may also be a continuous graded relationship between left ventricular mass and blood pressure, with no critical threshold of blood pressure, beyond which left ventricular hypertrophy develops (Hey JC, Scharf SM. 2003) .

Left ventricular hypertrophy also has been linked to the development of atrial fibrillation, ventricular arrhythmias and sudden cardiac death, Left ventricular hypertrophy is also associated with a three- to four-fold increase in the risk of stroke, a two- to three-fold increase in coronary heart disease (CHD) and a threefold increase in peripheral arterial disease. However, the pathophysiological basis linking left ventricular hypertrophy with these adverse cardiovascular events has not been fully elucidated. In several epidemiological studies, left ventricular hypertrophy is an independent risk factor for cardiovascular morbidity and mortality (Hey JC, Scharf. 2003).

The gradient of risk factor, adjusted for age, of cardiovascular disease in
Men was 1·49 for each 50g.m _1(corrected for height) and in women, 1·57 This graded correlation between echocardiographically determined left ventricular mass And the development of cardiovascular disease appears to be without a critical mass, separating the sometimes postulated ‘compensatory’ from ‘pathological’ hypertrophy. Although a much less sensitive measure for thpresence of left ventricular hypertrophy, the ECG is also indicative of increased cardiovascular morbidity and mortality. In the Framingham Study, for example, ECG–left

ventricular hypertrophy increased the risk of cardiovascular disease from three- to seven-fold depending on age and sex of the patient (Hey JC, Scharf SM. 2003) .

2.5.1 Types of left ventricular hypertrophy:

There are two common types of left ventricular hypertrophy:
Concentric and eccentric. The thickening of the left ventricular wall relative to the internal cavity is referred to as 'concentric' left ventricular hypertrophy.
Less common is the disproportionate (relative to the posterior wall) thickening of the intraventricular septum, referred to as asymmetrical or eccentric left ventricular hypertrophy. These different patterns of left ventricular hypertrophy have deferent features, haemo- . For example, concentric left ventricular hypertrophy is normally
Associated with moderate to severe hypertension and is more common in the middle aged and elderly than in young patients. Cardiac output is normal or low in
Concentric left ventricular hypertrophy, but is high in eccentric left ventricular hypertrophy.

However, some investigators have reported that these geometric patterns do not add much additional prognostic information beyond that offered by the simple degree of left ventricular hypertrophy and traditional cardiovascular risk Factors dynamic relations, and prognostic implications (Ng CS , et al , 1999). In patients with normal blood pressure (systolic blood pressure <140 mmHg) the risk of having left ventricular hypertrophy is 1·3%–1·6%, whilst with mild hypertension (systolic blood pressure 140 mmHg–160 mmHg) the risk is 2·7%– 5·6%, and with definite hypertension (systolic blood pressure >180 mmHg) the risk is 11·8%– 18·8% .In one series, the prevalence of left ventricular hypertrophy using the Sokolow–Lyon ECG criteria in malignant phase hypertension, which is the most severe form of hypertension, is 75·6%–82·6% (Ng CS , et al , 1999). In the Framingham study, after adjusting for age, diastolic blood pressure and body mass index, isolated systolic hypertension was found to be associated with a 2·6%- to 5·9-fold risk of developing

echocardiographic evidence of left ventricular hypertrophy. Also increased wall thickness tended to predominate in women, whilst left ventricular dilatation predominates in men However, the correlation between blood pressures measured in the clinical environment and left ventricular mass is poor . By contrast, 24 h ambulatory blood pressure monitoring provides a close correlation between average daytime arterial blood pressure and left ventricular mass (Ng CS , et al , 1999).

Non-dippers are considered to have a greater 24 h blood pressure ‘load’ when compared to dippers, as well as greater end-diastolic volumes . However, one meta-analysis of 19 studies published before 1994 found that the left ventricular mass was only weakly associated with the day–night blood pressure deference.

It has also been suggested that nocturnal falls in pressure could induce myocardial ischemia in hypertensive's with left ventricular hypertrophy and impaired coronary vasodilator reserve, perhaps contributing to the so-called ‘J curve’, which has been previously observed in some retrospective studies where diastolic blood pressure was lowered below 85 mmHg, resulting in increased coronary events , also found that more magnetic resonance imaging scan evidence of silent cerebrovascular disease was present in extreme ‘dippers’, whose fall in nocturnal systolic pressure was greater than 20%. By contrast, the recent large prospective Hypertension Optimal Treatment (HOT) trial showed no evidence of a J curve in the short-term amongst hypertensive patients including those with previous coronary heart disease (Ghali-1999).

2.5.2 Pathophysiology of left ventricular hypertrophy:

The progression from a structurally normal heart to left ventricular hypertrophy is not solely a consequence of increased afterload imposed by hypertension, with an Increased total peripheral resistance. Many mechanisms are now known to be involved in the growth regulation of the heart, including neurogenic, humeral,

anticrime, peregrine and possibly endocrine factors, all of which have important therapeutic implications (RunoJ , et al , 2003).

2.5.3 Pressure overload:

Both aortic stenosis and arterial hypertension lead to pressure overload of the left ventricle. Therefore if pressure overload was to be the sole stimulus for left Ventricular hypertrophy the results should be identical. Nonetheless, different models inducing left ventricular hypertrophy showed heterogeneity of myocardial remodeling with varying quantity and distribution of fibrillar collagen, in addition to varying degrees of intramyocardial arterial adaptation (RunoJ , et al , 2003).

. The intraventricular pressure overload of hypertension as wells aortic stenos is results in myocytic hypertrophy and increased perimyocytic fibrosis. However, intramyocardial arteriole wall-thickening and enhanced perivascular fibrosis are distinctive features of hypertension that are not seen in aortic stenos is (RunoJ , et al , 2003).

2.5.4 Renin–angiotensin–aldosterone system:

Several growth factors have been implicated in initiating and maintaining myocardial hypertrophy, In particular, the renin–angiotensin–aldosterone system is likely to have an important role in hypertensive heart disease, and both angiotensin-II and aldosterone are known to cause myocardial fibrosis .

Hypertension-induced left ventricular hypertrophy results in increased myocardial fibrosis, but chronic pressure overload per se does not cause myocardial fibrosis, as demonstrated with experiments of infrarenal aortic banding causing left ventricular hypertrophy without fibrosis , Only after activation of the renin–angiotensin–aldosterone by suprarenal banding would fibrosis be found in the hypertensive, hypertrophied left ventricle. Similar changes can be observed after simulated renin–angiotensin–aldosterone activation with intravenous administration of angiotensin II

or aldosterone, suggesting an important role of the renin– angiotensin–aldosterone in the development of left ventricular hypertrophy and fibrosis (Humbert- 2007).

Myocardial fibrosis is associated with increased expression of angiotensin converting enzyme and bradykinin receptor binding at sites of repair. It is also recognized that there are both circulating and tissue renin–angiotensin systems. Tissue renin–angiotensin– aldosterone components are found in the lungs, Myocardium, brain, kidneys, and testes, and in or around blood vessels with possible vasoconstriction effects (Humbert -2007).

An important role for the renin–angiotensin system is suggested by the impressive effect of angiotensinconverting enzyme (ACE) inhibitors, and more recently the angiotensin II receptor antagonists in causing regression of hypertensive left ventricular hypertrophy , and in preventing remodeling and improving prognosis after myocardial infarction , there is a close correlation between circulating renin–angiotensin levels and left ventricular mass .The renin–angiotensin–aldosterone may also explain some clinical observations related to the heart in hypertension.

2.6 Previous Study:

(Ayres SM, 1998).was measured The cardiothoracic ratio in 410normal fetuses and in a group of 73 fetuses with functional or structural heart disease. In normal fetuses it was fairly constant through-out pregnancy, but of those with congenital heart disease it was raised in cases of Ebstein'sanomaly, tricuspid dysplasia.

. There was a significant positive correlation between the cardiothoracic ratio and fetal hydrops in the group of 15 fetuses with supraventriculartachycardias. In these fetuses the cardiac size decreased significantly once the fetus reverted to sinus

(Rich S 2006) investigated the effects of cardiac remodeling on left ventricular (LV) diastolic function, as evaluated by tissue Doppler and blood-pool indices, with respect to loading as expressed by wall stress.

Conclusions DD appears early in hypertensive disease, before the onset of abnormal remodeling or LV hypertrophy. With progression of the remodeling process and the advance of LVH, diastolic function proaggressively deteriorates. Tissue Doppler indices are better correlated with clinical and echo cardio graphic parameters of LV remodeling compared to blood-pool indices .

(Ayres SM 1991) assessed the Left ventricular hypertrophy has been suggested to mediate the relation between hypertension and left atrial enlargement, with associated risks of atrial fibrillation and stroke.

In logistic regression analysis, left atrial enlargement was related to left ventricular hypertrophy and eccentric geometry; greater body mass index, systolic blood pressure, and age; female gender; mitral regurgitation; and atrial fibrillation (all, P 0.05). Thus, left atrial size in hypertensive patients with electrocardiographic left ventricular hypertrophy is influenced by gender, age, obesity, systolic blood pressure, and left ventricular geometry independently of left ventricular mass and presence of mitral regurgitation or atrial fibrillation.

(Caballero L 2007) Measured the association of radiographic of heart size with mortality from coronary heart disease.
heart rate, smoking, cholesterol, angina and ECG ischaemia had little effect, reducing the rate ratio to 1.65 (95% CI 1.01-2.70). Similar rate ratios were observed for relative heart volume.

(Levy D 1995) studied the increased left atrial size has been identified as a precursor of atrial fibrillation and of stroke once atrial fibrillation is manifest. Conflicting data exist regarding the effect of high blood pressure on left atrial size. Our objective was to evaluate the association of contemporary and long-term measures of blood pressure with

Overall, in this population-based study sample, increased levels of systolic and pulse pressures (but not diastolic or mean arterial pressures) were significantly associated with increased left atrial size. However, the magnitude of these associations was quite modest, particularly after controlling for age and body mass index.

Chapter Three

Material &Methodology

Chapter Three Material

and method

3.1 Material:

3.1.1 .Patients :

Patients who were diagnosed as hypertension PT, males & females at age between (45-80.)

Study includes 40 patients with Hypertension with Congestive obstruction pulmonary diseases or Congestive heart disease can affect the cardiothoracic ratio and 19 control group

3.1.2 Instrumentations:

CT Toshiba 64 slices and Philip 64 slices machines.

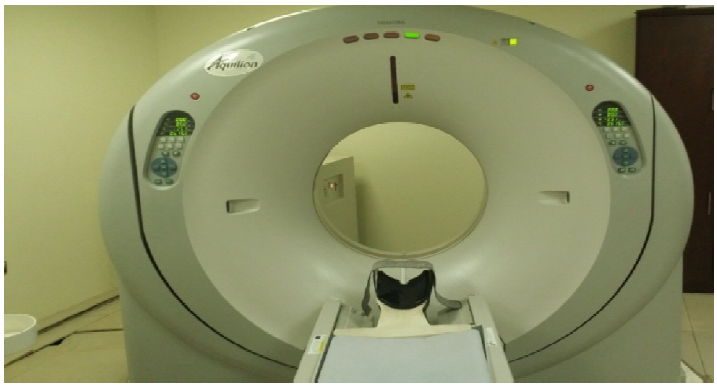

Figure 3.1 Gantry computer tomography

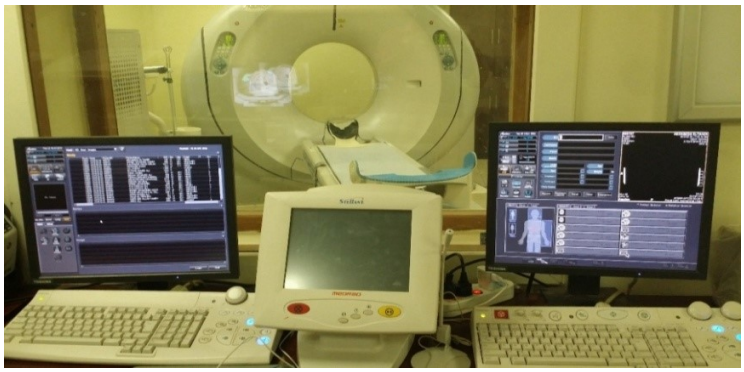

Figure 3.2 Control computer tomography

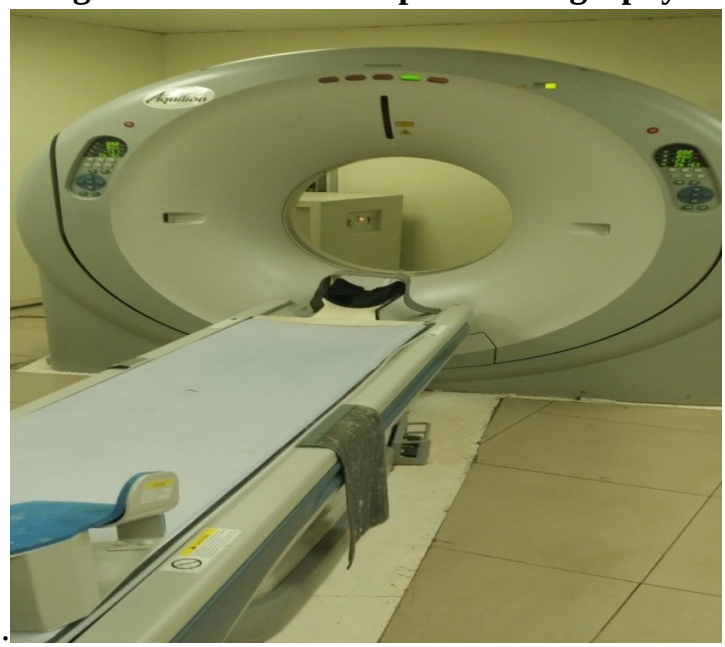

Figure 3.3 Machine table

3. 2 Methods :

The data was collected by master data sheets using the variables of age, Gender, Age, Pb, type of Medication, duration of disease, and the CRT value Data analysis using statistical software (SPSS) and Microsoft Excel.

3.2.1: computed tomography technique:

Patient instructiment to stop eating and drinking for period time before the exam or taken any type of medication at least one day before the exam.

Female patient will be asked for the probability of positive pregnant test.

On exam room asked the patient to lying on supine position on CT table after wearing the hospital gown and remove all the metallic forging body.

CT chest Scan, Most protocols of the heart and lung and thoracic cage are performed while the patient lies in a supine position on the scan table with the arms elevated above the head. In a few instances changing the patient position and obtaining additional slices can provide added information. Head first and suspended inspiration (KV =120 , MA =300 , Time of rotation = 0,75s), Axial cut with slice thickness 2 mm

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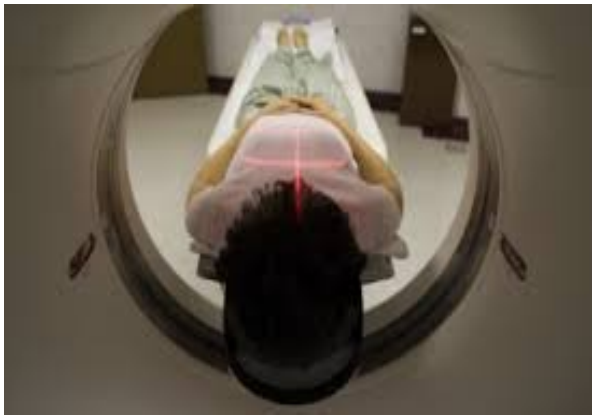
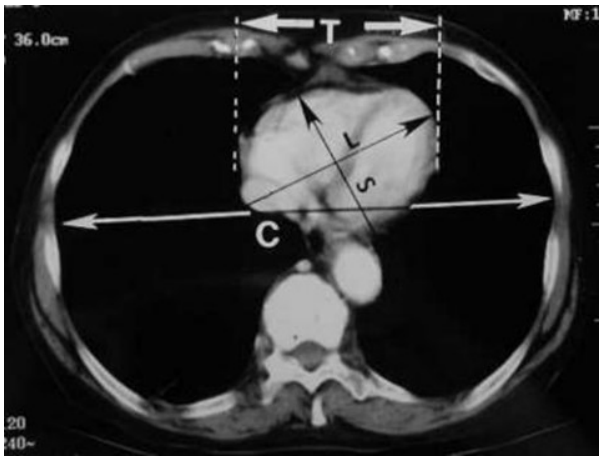


Figure 3.4 show patient position for CT chest

3-2.2 – cardiac thoracic ratio measurement (CTR) :

Measurement process done from outer to outer myocardium

On axial CT image divided by transvers greatest thoracic diameter from inner to inner chest wall on axial image. As shown below.



Figurer 3.5 various methods of deriving CTR on HCT.

CTR using the maximum Transverse cardiac diameter, T (superior white arrows), is an attempt to replicate.

Radiographic CTR (Transverse CTR=T/C). L, the long cardiac axis (black arrow pointing to left ventricular apex) and S, the short axis (black arrow pointing medially) are used to derive.

HCT CTR index (CTR index=0.5(L+S)/2). The Transverse thoracic diameter, C (inferior white horizontal arrows).

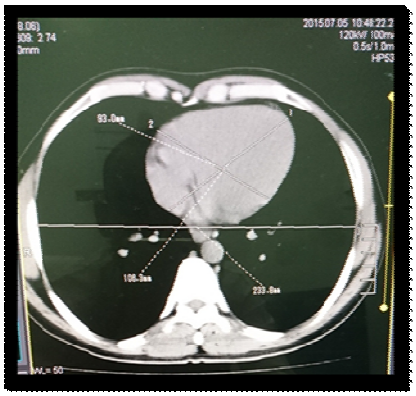


Figure 3.6 Show the CTR Measurement on Axial CT Chest Cut

CTR =T/C(S+L)

T= Transverse cardiac diameter

L= the long cardiac axis

S= the short axis

C=transverse thoracic diameter

Chapter Four

Results

Chapter Four
Results

The following tables and figures presented the data obtained from 40 patients who were examined for CT Chest, 19 control Group at Radiology department of Alamal Medical Centre and Doctor Clinical Hospital.

Cardio Thoracic Ratio (CRT), blood pressure, Medication used and medication duration were evaluated. Patient's age and gender have also been registered. The data were analyzed using SPSS program version16. Frequency tables mean and standard deviations were presented. T-test and Chi square test were used to test the degree of significance. Data were considered significant at p 0.005.

Table (4.1) Variables statistics, Age, Weight, CTR and Medication Duration

	Statistics				
	Age	Weight	Blood Pressure	CTR	Medication Duration
Mean	62.50	70.37	0.82	3.07	5.62
	63.00	75.50	1.00	3.00	5.50
Std. Deviation	4.997	1.06	0.38	0.79	1.58
Minimum	54.00	54.00	0.00	2.00	3.00
Maximum	72.00	85.00	1.00	4.00	9.00
Total Number	40	40	40	40	40
Median					

Table 4.2 the distribution of the sample according to age classes

		Gender			
		Hypertension patients		Control	
		Frequency	Percent	Frequency	Percent
Count	Female	17	42.5%	11	57.9%
	Male	23	57.5%	8	42.1%
	Total	40	100%	19	100%

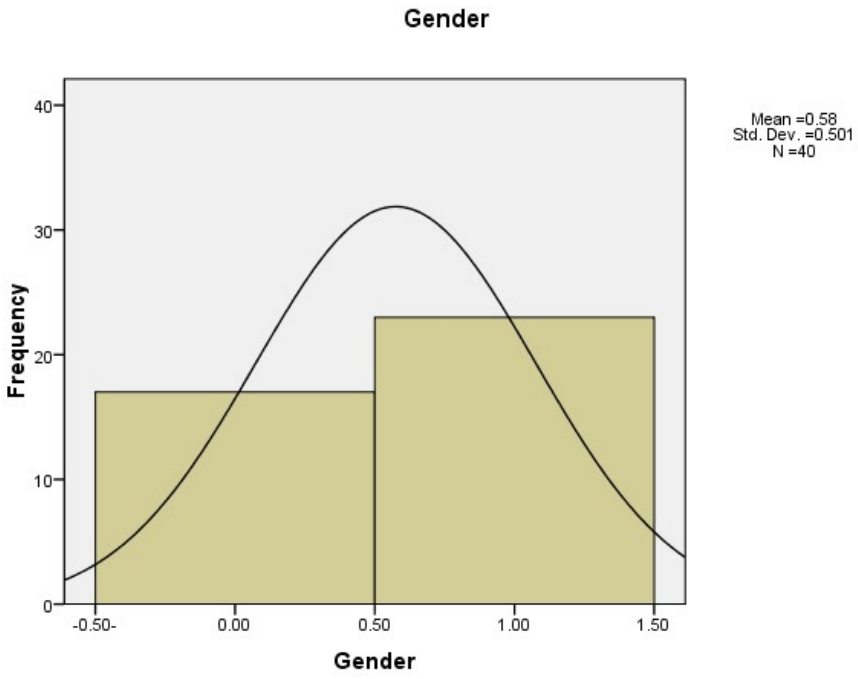


Figure 4.1 the distribution of the sample according to Gender

Table 4.3 The distribution of the sample according to age classes

		Age			
		Hypertensive Patients		Control	
		Frequency	Percent	Frequency	Percent
Class	54-60	16	40%	9	47.4%
	61-67	15	37.5%	6	31.6%
	68-74	9	22.5	4	21%
	Total	40	100%	19	100%

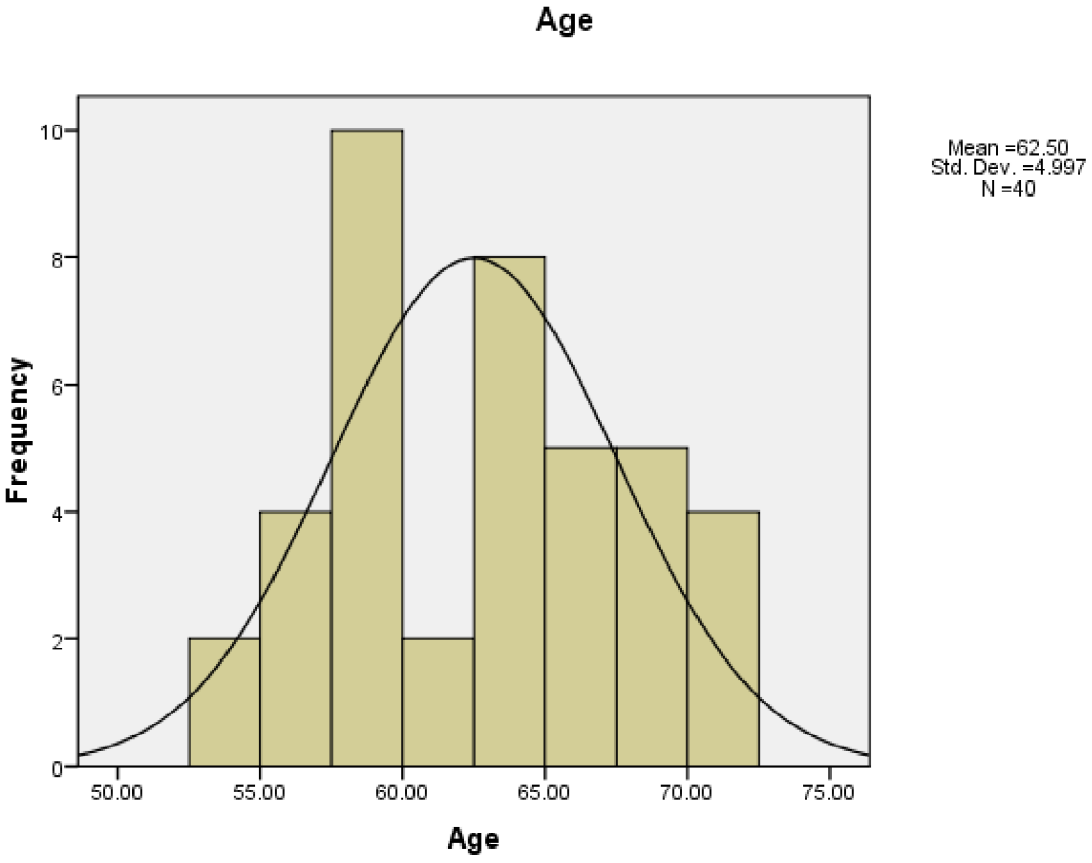


Figure 4.2 The distribution of the sample according to age classes

Table 4.4 The distribution of the sample according to Weight class

		Weight			
		Hypertensive patients		Control	
		Frequency	Percentage	Frequency	Percentage
Class	54-60	13	32.5%	6	31.6%
	61-67	3	7.5%	0	0%
	68-74	4	10%	3	15.8%
	75-81	17	42.5%	9	47.4
	82-88	3	7.5%	1	5.2%
	Total	40	100%	19	100%

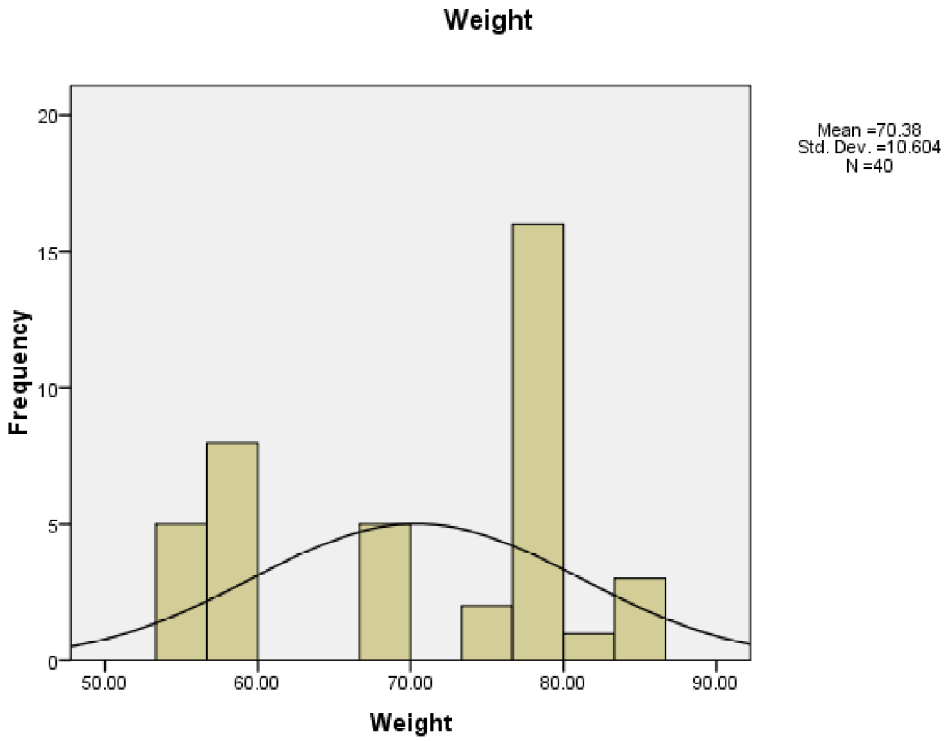


Figure 4.3The distribution of the sample according to weight classes

Table 4.5 distribution of the sample according Blood Pressure

	Blood Pressure		
		Frequency	**Percent**
Value	**Normal**	7	17.5%
	High	33	82.5%
	Total	40	100%

Diastolic value /80 Considered as normal

Diastolic value /90-110 Considered as high

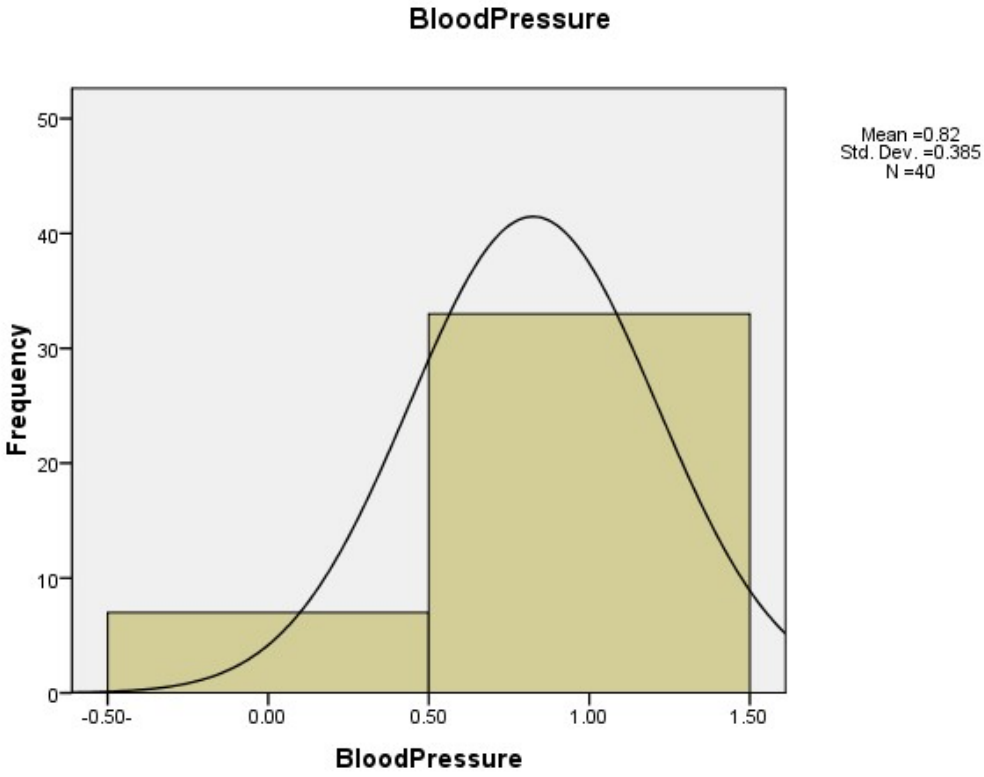


Figure 4.4 The distribution of the sample according to Blood Pressure

Table 4.6 Distribution of the sample according to CTR Value

	CTR/Hypertensive patients			CTR/Control		
		Frequency	Percent		Frequency	Percent
Value	**0.8**	11	27.5% Value	0.5	11	57.9%
	0.9	15	37.5%	0.6	8	42.1%
	1	14	35%	-	-	-
	Total	40	100%	19	19	100%

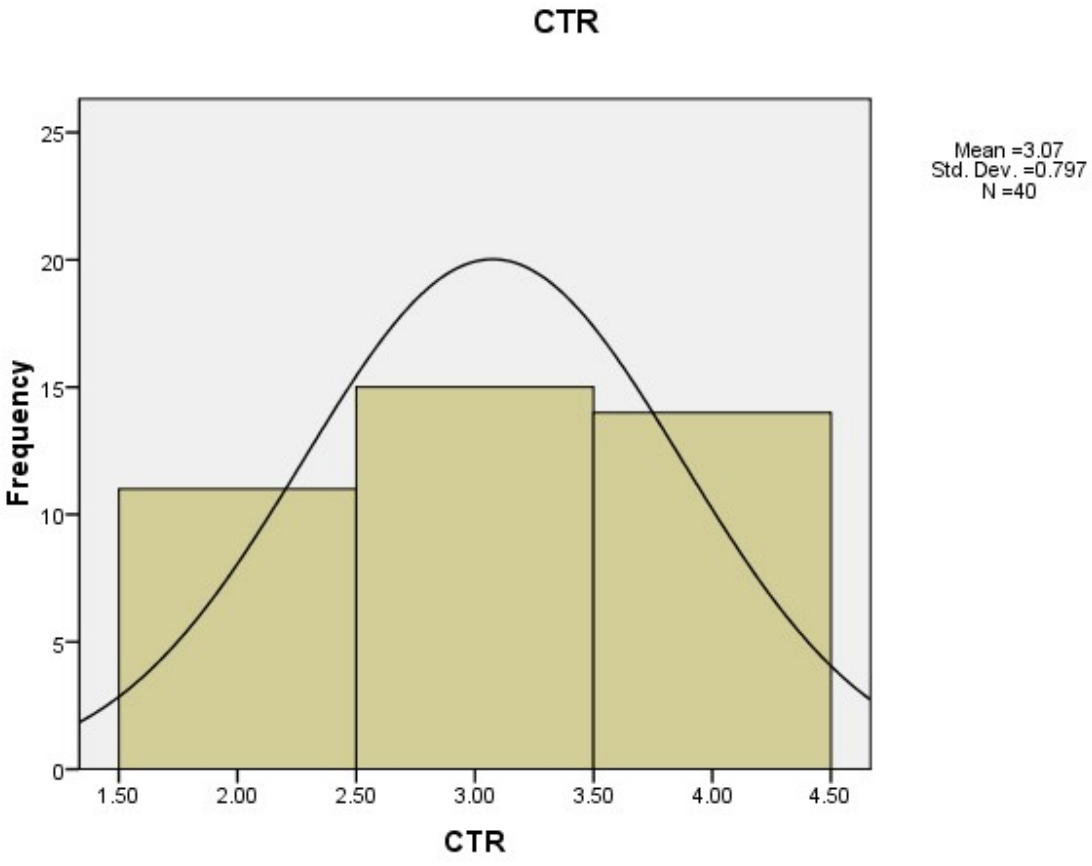


Figure 4.5 The distribution of the sample according to CTR

Table4.7 distribution of the sample according to Medication Duration

	Medication Duration		
		Frequency	Percent
Years	**2**	3	7.5%
	4	7	17.5%
	5	10	25%
	6	10	25%
	7	4	10%
	8	4	10%
	9	2	5%
	Total	40	100%

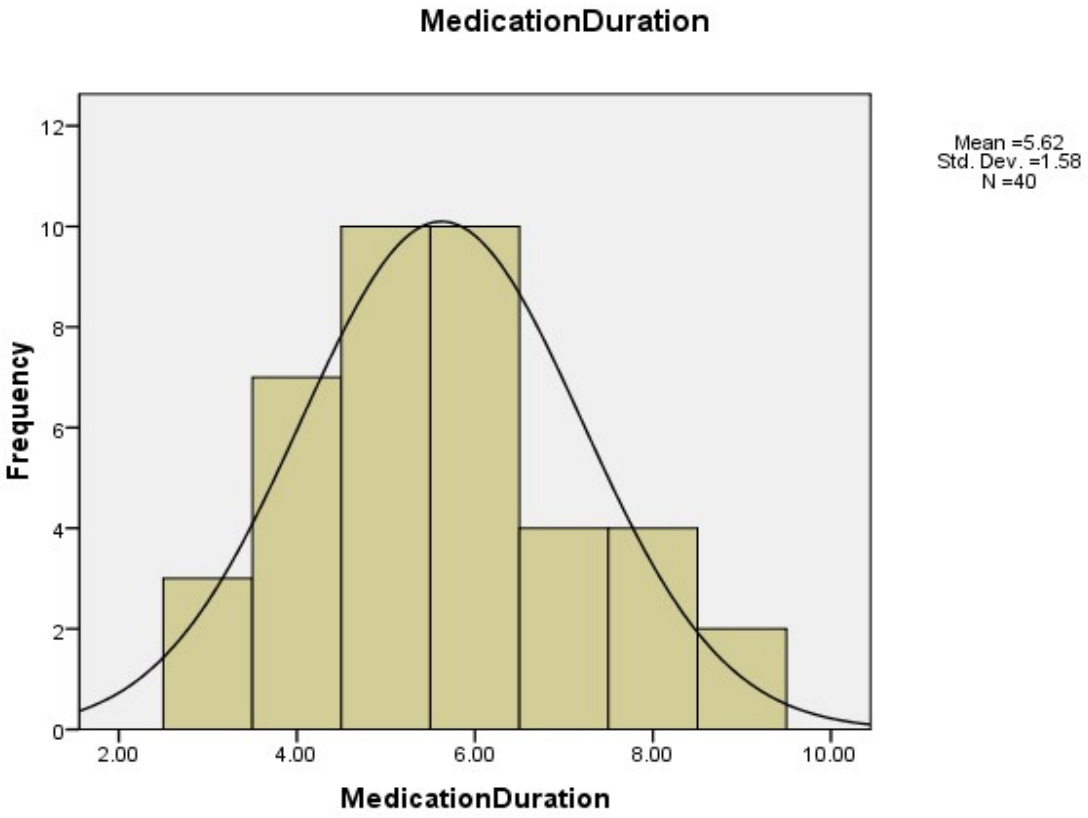


Figure 4.6 The distribution of the sample according to Medication Duration

Table 4-8 The distribution of the sample according to (Medication) Drugs used

	Frequency	Percentages%
LISINOPRIL (15 mg)	4	10%
LOSARTAN (50 mg)	11	27%
AMLODIPINE (20 mg)	7	17%
HYDROTHAIZIAD (500mg)	6	15%
ATENOLOL (700 mg)	8	20%
BISOPROLOL (5 mg)	4	10%
Total	40	

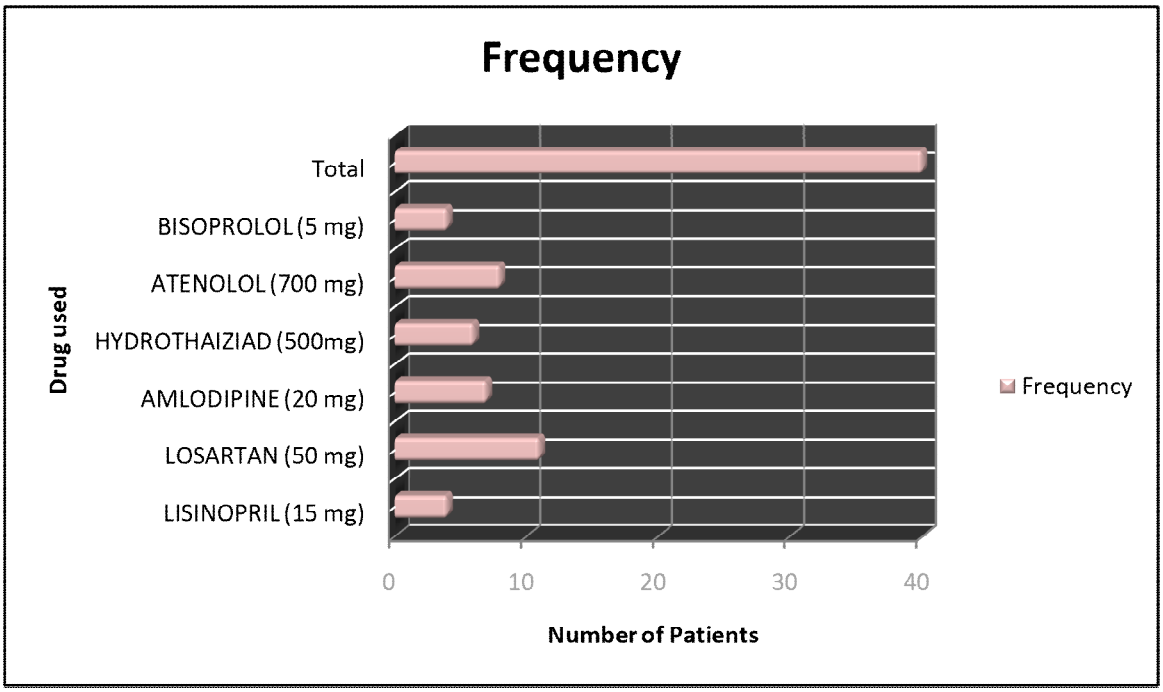


Figure 4.7 The distribution of the sample according to Drug Used.

Table 4.9 the Correlation between, Medication Duration, Blood Pressure with CTR

Correlations				
		Blood Pressure	**CTR**	**Medication Duration**
Blood Pressure	**Pearson Correlation**	1	0.295	.227
	Sig. (2-tailed)		.055	0.160
	N	40	40	40
CTR	**Pearson Correlation**	.295	1	-.140-
	Sig. (2-tailed)	0.055		0.389
	N	40	40	40
Medication Duration	**Pearson Correlation**	.227	-.140-	1
	Sig. (2-tailed)	.160	.389	
	N	40	40	40

*. Correlation is significant at the 0.05 level (2-tailed).

Table 4.10 the Correlation between ages, weight, and gender with CTR

Correlations					
		CTR	**Gender**	**Age**	**Weight**
CTR	**Pearson Correlation**	1	-.175-	.113	-.100-
	Sig. (2-tailed)		.280	.489	.537
	N	40	40	40	40
Gender	**Pearson Correlation**	-.175-	1	-.118-	-.037-
	Sig. (2-tailed)	.280		.469	.822
	N	40	40	40	40
Age	**Pearson Correlation**	.113	-.118-	1	-.260-
	Sig. (2-tailed)	.489	.469		.105
	N	40	40	40	40
Weight	**Pearson Correlation**	-.100-	-.037-	-.260-	1
	Sig. (2-tailed)	.537	.822	.105	
	N	40	40	40	40

Table 4.11 The Cross tabulation Between CTR and Blood Pressure

Blood Pressure * CTR Cross tabulation					
		CTR			Total
		0.8	0.9	1	
Blood Pressure	Normal	5	0	2	7
	High	6	15	12	33
Total		11	15	14	40

Table 4.12 The Cross tabulation Between CTR and Medication Type

MedicationType * CTR Crosstabulation					
		CTR			Total
		0.8	0.9	1	
Medication Type	Lisinopril(15 Mg)	1	1	2	4
	Losartan (50 Mg)	3	6	2	11
	Amlodipine (20 Mg)	1	2	4	7
	Hydrothaiziad(500mg)	2	1	3	6
	Atenolol (700 Mg)	2	5	1	8
	Bisoprolol(5 Mg)	2	0	2	4
Total		11	15	14	40

Table 4.13 The Cross tabulation Between CTR and Medication Duration

Medication Duration * CTR Cross tabulation					
		CTR			Total
		0.8	0.9	1	
Medication Duration	2	2	0	1	3
	4	1	3	3	7
	5	2	5	3	10
	6	1	3	6	10
	7	3	1	0	4
	8	1	2	1	4
	9	1	1	0	2
Total		11	15	14	40

Table 4-14 the Correlation between Patient Group and Control Group with CTR

	Correlations			
		CTR Patients	CTR Control	Blood Pressure Patients	Blood Pressure Control
CTR	Pearson Correlation	1	.075	.295	.[a]
	Sig. (2-tailed)		.051	.055	.
	N	40	19	40	19
CTR Control	Pearson Correlation	.075	1	.025	.[a]
	Sig. (2-tailed)	.051		.018	.
	N	19	19	19	19

Table 4-15 Coefficients of variables

Coefficients[a]								
Model		**Unstandardized Coefficients**		**Standardized Coefficients**	**t**	**Sig.**	**95% Confidence Interval for B**	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	**(Constant)**	2.571	.292		8.817	.000	1.981	3.162
	Blood Pressure	.610	.321	.295	1.901	.055	-.040-	1.260
a. Dependent Variable: CTR								

Predictive Equation:

CTR=Blood pressurex2.571+ 0.610

Chapter Five

Discussion, Conclusion

&

Recommendations

Chapter Five

The discussion, Conclusion and Recommendations

5-1 The Discussion:

In order to assess the effect of hypertension on the CTR, analyzed 40 with hypertension42.5% were females, 57.5%and male .19 were Control Group57.9% female and 42.1%.

Cardiac thoracic ratio were taken in Patients who were diagnosed as hypertension Patient, Both gender were included 17 female and 23 were male with mean of age , weight, CTR ,and medication duration which represents in table (4-1).

Table 4.9 showed the relationship between, Medication Duration, Blood Pressure with CTR, There is significant correlation at p value<0.005.

Witch similar to the study bone by(Rich S, 2006) investigated the effects of cardiac remodeling on left ventricular (LV) diastolic function, Cardiac remodeling is the major pathophysiological result of increased blood pressure; diastolic function pro-aggressively deteriorates.

Table 4.10 showed that the no significant relationship between ages, weight, gender with CTR at p<0.00.

Were was not similar to study done by (Ayres SM 1991)who assessed left atrial size by echocardiography in 941 hypertensive patients, age 55 to 80 (mean, 66) years, with electrocardiographic left ventricular hypertrophy.

Thus, left atrial size in hypertensive patients with electrocardiographic left ventricular hypertrophy is influenced by gender, age, obesity, systolic blood

pressure, and left ventricular geometry independently of left ventricular mass and presence of mitral regurgitation or atrial fibrillation.

Table 4 12 showed that, the mean average on CTR which were (0.8-1) on hypertaion group, and were (0.5.0.6) for the control group, therefore, the relationship between Patient Group and Control Group with CTR also shown the the significant relationship between the blood presser and CTR, There is a sginficant deference at p value <0.05.

The result of this study showed that these is a significant relationship between the CTR and hypertension , a new equation established to find the CTR for patient with known blood pressure Predictive Equation:

CTR=Blood pressurex2.571+ 0.610

5-2 The Conclusion

The objective of study was to evaluate the CTR value in hypertensive patient by using computed tomography.

The study conclude that there is no significant between the weight of patients and the of CTR heart.

Also the study showed the There is no significant relation between the medication duration.

Also the results showed significant relationship between the cardiac thoracic ratio and high blood presser, in addition the significant deference between the hypertensive patients and control group.

5-3 The Recommendations

Patient should control bold presser because it away that change the CTR.

Further study should be done to evaluate the heart volume in hypertensive patients with increase the sample size.

To evaluate the cardiothoracic ratio in patient with hypertensive may use other modalities of screening like normal chest x. ray echocardiogram.

References

References

A.Arch Intern Med. 1998;158:761–7.Am J Cardiol 1996; 78: 197–202, Ayres SM.

B.Borgioni C et al. Prognostic value of left ventricular mass and geometry in systemic hypertension with left ventricular hypertrophyBenza, Liao YL.Impact of left ventricular Hansen KJ R.L.;,Badesch, D.B.;Bruder H

C. Craven TE, et al. Prevalence of renovascular disease in the elderly: a population-based study.,Cooper RS,

D.Devereux RB, et al. Recurrent pulmonary oedema in hypertension due to bilateral renal artery stenosis: treatment by angioplasty or surgical revascularization. Lancet. 1988;2:551, D.P.; Barst,,Dunnick NR, Devereux et al

E., Edwards MS

F.Frost, A.E.; McGoon,Flohr TG

G.Ghali JK, Kadakia S, ,Goodman L. Utility of CT scan for predicting pulmonary hypertension in patient with parenchymal lung disease. Chest 1998;113:1250–6

H.hypertrophy on ventricular arrhythmias in the absence of coronary artery disease. J Am CollCardiol 1991; 17: 1277–82. Hypertension. 1991;17:678,Humbert M. The burden of pulmonary hypertension. The European respiratory journal : official journal of the European Society for Clinical Respiratory Physiology. 2007; 30(1):1–2,Himmelstein SI, et al. Renal artery stenosis,Humbert

J. J Vasc Surg. 2002;36:443–51.. . J Am CollCardiol 1995; 25: 879–84.

K.Krumholz HM, ,Kuzo R, Kannel and Cobb.

L. Levy D. Impact of left ventricular structure on the incidence of hypertension. Levy D. Prognosis of left ventricular geometric patterns in the Framingham Heart Study, Larson M, R.J.; Larson MG,LP, Kadir S

M... Miller, M.D. An evaluation of long-term survival from time of diagnosis in pulmonary arterial hypertension from the REVEAL Registry. Chest 2012, 142, 448–456., Multi-detector row CT systems and image-reconstruction techniques.Radiology. 2005;235:756–773.

N.Ng CS , et al ,

O.Ohnesorge BM

P. Post WS, , prevalence and associated risk factors in patients undergoing routine cardiac catheterization, Polissar NL, et al. Carotid and lower extremity arterial disease in patients with renal artery atherosclerosis, Pickering

R.RunoJ , et al, Rich S.

S. Smith LR, Svetkey, , et al. Similar prevalence of renovascular hypertension in selected blacks and whites., Schaller S, Stierstorfer K, Schoepf UJ

T. The Framingham Heart Study. Circulation 1994; 90: 179–8, Tans RT TG, Herman L, ,

V.Verdecchia P, Schillaci G

W. William Russell

Z.Zierler RE, Bergelin RO,

Appendices

Appendix A

THE TABLE OF DATA COLLECTED DURING THE STUDY

Pt No.	Gender/g	Age/A	PB	CTR Value	Weight/W	Type of Medication	Medication Duration