Sudan University of Science and Technology

College of Graduate Studies

Evaluation of Renal Changes in Diabetic and Hypertensive Patients using Ultrasound and Laboratory Findings

تقويم التغيرات الكلوية لدى مرضى السكري وارتفاع ضغط الدم بالموجات فوق الصوتية والفحوصات المعملية

A thesis submitted for the fulfillment of PhD degree in Medical Diagnostic Ultrasound

By

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.....Evaluation of renal changes in Diabetic

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.....and laboratory findings.....

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إقرار

أنا الموقع أدناه أقر بأننى المؤلف الوحيد لرسالة الدكتوراه المعنونة تقويم التغيرات الكلويه لدى مرضى السكري وارتفاع ضغط الدم بالموجات فوق الصوتيه والمعلمات المعمليه

وهى منتج فكري أصيل . وباختياري أعطى حقوق طبع ونشر هذا العمل لكلية الدراسات العليا جامعه السودان للعلوم والتكنولوجيا ،عليه يحق للجامعه نشر هذا العمل للأغراض العلمية .

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الآية

قال تعالى:

﴿قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ

أَنْتَ الْعَلِيمُ الْحَكِيمُ ﴾

سورة البقرة الاية (32)

Dedication:

To my parents

To my teachers

To my brothers and sisters

To my wife

And To my kids

Acknowledgement

Iam deeply grateful to God who gave me patience and power to complete my study; then to my supervisor, Dr. Mohammed Elfadil Mohammed - (associate professor)- for his great support and valuable guidance. Specially appreciation to Dr. Suzan Omer, consultant radiologist at Fedail hospital and to Dr. Mahmoud Salih Babikir, assistant Professor at Taibah University; for their unlimited support and valuable advices and comments.

Abstract

Diabetes mellitus and hypertension are considered the main causes of chronic renal failure in Sudan. These diseases may lead to some renal changes in morphology and function, so they need special care. The aim of this study was to correlate ultrasound findings with lab values in early detection of the disease. The study was conducted in Khartoum state during the period from July 2012 to February 2016. Two hundred and one patients were included in the study; they were divided into three groups: 59 (29.4 %) in diabetic group (DM), 41(20.4%) in hypertensive group (HTN) and 101 (50.2%) in diabetic-hypertensive group (DM-HTN). Hundred participants were included in the control group. All patients had been scanned with ultrasound for measuring renal dimensions and cortical thickness bilaterally and to characterize renal echogenicity. A data collection sheet was designed to include general information of the patient, ultrasound findings and lab values for (serum creatinine, BUN and eGFR). The study revealed that, there were significant differences between the normal group and the other groups in renal dimensions and volume and significant differences among (DM, HTN, and DM-HTN). Inconclusive results were found in cortical thickness cortical and echogenicity among the mentioned groups. Renal function damage was detected only by estimated glomerular filtration rate (eGFR). The study showed that 67 (33%) were normal, 93 (46%) had mild reduction, 39 (19.4) had moderate reduction and 2 (1.0%) had severe reduction in renal function. In conclusion, the study revealed that, ultrasound cannot detect renal function loss earlier before lab investigations.

المستخلص

يعتبر مرضي السكري وارتفاع ضغط الدم من الاسباب الرئيسية التي تؤدي للاصابة بالفشل الكلوي المزمن في السودان. هذه الامراض قد تحدث بعض التغيرات في شكل ووظيفة الكلية لذا تحتاج إلى عناية خاصة. وكان الهدف من هذه الدراسة لربط نتائج الموجات فوق الصوتية مع القيم المخبرية في الكشف المبكر عن علامات الفشل الكلوي المزمن. وقد أجريت هذه الدراسة في ولاية الخرطوم خلال الفترة من يوليو 2012 إلى فبراير عام 2016. وأدرجت مائتان واحد مشارك في الدراسة؛ وقد تم تقسيمهم إلى ثلاث مجموعات:المجموعة الاولى تضمنت 59(29.4%) مصاب بالسكري ،المجموعة الثانية41(20.4%) مصاب بارتفاع ضغط الدم والمجموعة الثالثة تضمنت 101 (50.2٪) مصاب بارتفاع ضغط الدم و السكري. تضمنت المجموعة الضابطة مائة (100) من المشاركين . وقد تم فحص جميع المرضى بالموجات فوق الصوتية لقياس أبعاد الكلى وسمك القشرية للكليتين. تم تصميم ورقة جمع البيانات لتشمل المعلومات العامة للمريض، نتائج الموجات فوق الصوتية والقيم المخبرية وهي مصل الكرياتينين في الدم، البولينا ومعدل الترشيح الكبيبي المقدر في الدقيقة الواحدة. وكشفت الدراسة أن هناك فروقا ذات دلالة إحصائية بين المجموعة الضابطة والمجموعات الأخرى في أبعاد وحجم الكلى وفروق ذات دلالة إحصائية فيما بين المجموعات الثلاثة سالفة الذكر. تم العثور على نتائج غير حاسمة في سمك القشرية وال echogenicity بين المجموعات المذكورة.

لقد تم اكتشاف الضرر الذي اصاب الكلى بواسطة الفحص المعملي (معدل الترشيح الكبيبي) فقط. وأظهرت الدراسة أن 67 حالة (33%) كانت طبيعية،93 (46٪) كانت ذات انخفاض طفيف، 39 (19،4) كانت ذات انخفاض معتدل و(2 = 1.0٪) كانت ذات انخفاض حاد في وظائف الكلى.

في الختام، كشفت الدراسة أن الموجات فوق الصوتية لا يمكنها الكشف المبكر الذي يحدث في فقدان وظائف الكلى قبل الفحوصات المخبرية.

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Abbreviations

SBP	Systolic Blood Pressure
ACP	American College of Physicians
ADH	Anti Diuretic Hormone
AKI	Acute Kidney Injury
ASN	American Society of Nephrology
BUN	Blood Urea Nitrogen
C/M	Cortico-Medullary
CKD	Chronic Kidney Disease
CRF	Chronic Renal Failure
DM	Diabetes Mellitus
HTN	Hypertension
ESRD	End-Stage Renal Disease
eGFR	Estimated Glomerular Filtration Rate
FSGS	Focal and Segmental Glomerulosclerosis
GFR	Glomerular Filtration Rate
HIV	Human Immunodeficiency Virus
HUS	Hemolytic-Uremic Syndrome
KDOQI	Kidney Disease Outcomes Quality Initiative
NHANES	National Health and Nutrition Examination Survey
NIDDK	National Institute of Diabetes and Digestive and Kidney Diseases
NKF	National Kidney Foundation

NSAIDs	No Steroidal Anti-Inflammatory Drugs
PTH	Parathyroid Hormone
USRDS	United States Renal Data System
RR L	Right Renal Length
LR L	Left Renal Length
RR W	Right Renal Width
LR W	Left Renal Width
RR D	Right Renal Depth
LR D	Left Renal Depth
RR V	Right Renal Volume
LR V	Left Renal Volume
Rt_C_ Echo	Right Cortical Echogenicity
Lt_C_Echo	Left Cortical Echogenicity
Rt_CMD	Right Cortico-Medullary Differentiation
Lt_CMD	Left Cortico-Medullary Differentiation

Chapter one
Introduction

1.1 Introduction:

Diabetes mellitus and hypertension may lead to renal changes in morphology and function. They are considered the main causes of chronic renal failure in Sudan (Gameraddin M, et al, 2014). Often, chronic renal failure is diagnosed as a result of screening of people known to be at risk of kidney problems, such as those with high blood pressure or diabetes and those with a blood relative with chronic kidney disease. Chronic renal failure is a progressive loss in renal function over a period of months or years. Morbidity and mortality rates of the (CRF) patients on dialysis are elevated. The symptoms of worsening kidney function are unspecific, and might include feeling generally unwell and experiencing a reduced appetite. Chronic renal failure may also be identified when it leads to one of its recognized complications, such as cardiovascular disease, anemia or pericarditis.

Chronic renal failure is identified by a blood test for creatinine. Higher levels of creatinine indicate a falling glomerular filtration rate and as a result a decreased capability of the kidneys to excrete waste products. Creatinine levels may be normal in the early stages of CRF, and the condition is discovered if urinalysis (testing of a urine sample) shows that the kidney is allowing the loss of protein or red blood cells into the urine. To fully investigate the underlying cause of kidney damage, various forms of medical imaging, blood tests and often renal biopsy (removing a small sample of kidney tissue) are employed to find out if there is a reversible cause for the kidney malfunction.(Sarnak MJ, Levey AS 2000) .Recent professional guidelines classify the severity of chronic renal failure in five stages, with stage 1 being the mildest and usually causing few symptoms and stage 5 being a severe illness with poor life expectancy if untreated. Stage 5 CRF is also called established chronic kidney disease and is synonymous with the now outdated terms end-stage renal disease (ESRD), chronic renal failure (CRF). (Sarnak MJ, Levey AS 2000)

Definition of Chronic renal failure includes functional and/or structural kidney damage for more than three months with or without decrease in glomerular filtration rate (GFR). The changes are either pathological changes or abnormalities of the markers of kidney damage in the blood or urine composition or imaging tests. The decrease of the GFR below 60 ml/min/1.73 m2 for three months with or without damage for more than three months is another sufficient definition for CRF. Most chronic kidney diseases tend to progress and worsen over time. (Bethesda, MD, 1998. & Am J Kidney Dis, 2002)

1.2 Problem of the study:

Renal failure go indication in laboratory investigations, therefore it has been adopt as first method but it got it's limitation concerning the morphological features. Ultrasound might detect renal changes in parenchyma and size in diabetic and hypertensive patients before the appearance of clinical symptoms and lab values of renal function; i.e. signs of prognosis of CRF, therefore adoption of ultrasound as a first investigation method might solve many problems.

1.3 Objective of the study:

The general objective of this study was to evaluate renal changes in diabetic and hypertensive patients using ultrasound and laboratory investigations to reveal the potential of ultrasound in exploring the ramification of chronic renal failure signs before it can appear in laboratory investigations.

Specific objectives:

- To determine the normal renal size by measuring the Length , width and depth of the kidney .(control group)
- To determine the normal renal cortical thickness and evaluate renal parenchymal echogenicity and C/M differentiation. (Control group).
- To characterize the changes in the renal size and parenchyma

- To find normal values (reference) using lab investigation results (serum creatinine, BUN and eGFR) for Sudanese people.
- To correlate the ultrasound findings with the lab findings (serum creatinine level, BUN and eGFR)
- To find the right and Left renal parenchyma (cortical echogenicity).

1.4. Significance of the study:

Early detection of sonographic signs of chronic renal failure in renal size and parenchyma can help us avoiding or slowing down the progression of CRF.

1.5 overview of the study:

This study falls into five chapters; chapter one is an introduction, which includes the problem of the study, objectives, significance of the study, and overview. Chapter two is a literature review which includes theoretical background and previous studies. While chapter three is a methodology that includes material and methods, and chapter four includes the results presentation and finally chapter five includes discussion, conclusion and recommendations.

Chapter two

Theoretical background & Literature review

2.1 Anatomy of urinary organs:

The urinary organs comprise the kidneys, which secrete the urine, the ureters, or ducts, which convey urine to the urinary bladder**,** where it is for a time retained; and the urethra**,** through which it is discharged from the body. (Gray, 1918)

2.1.1The Kidneys:

The kidneys are situated in the posterior part of the abdomen, one on either side of the vertebral column, behind the peritoneum, and surrounded by a mass of fat and loose areolar tissue. Their upper extremities are on a level with the upper border of the twelfth thoracic vertebra, their lower extremities on a level with the third lumbar. The right kidney is usually slightly lower than the left, probably on account of the vicinity of the liver. The long axis of each kidney is directed downward and lateral ward; the transverse axis backward and lateral ward. (Gray, 1918)

Each kidney is about 11.25 cm. in length, 5 to 7.5 cm. in breadth, and rather more than 2.5 cm. in thickness. The left is somewhat longer, and narrower, than the right. The weight of the kidney in the adult male varies from 125 to 170 gm., in the adult female from 115 to 155 gm. The combined weight of the two kidneys in proportion to that of the body is about 1 to 240.

2.1.1.1Macro-Anatomy:

The kidney has a characteristic form, and presents for examination two surfaces, two borders, and an upper and lower extremity.

The anterior surface (facies anterior): of each kidney is convex, and looks forward and lateralward. Its relations to adjacent viscera differ so completely on the two sides that separate descriptions are necessary (Gray, 1918).

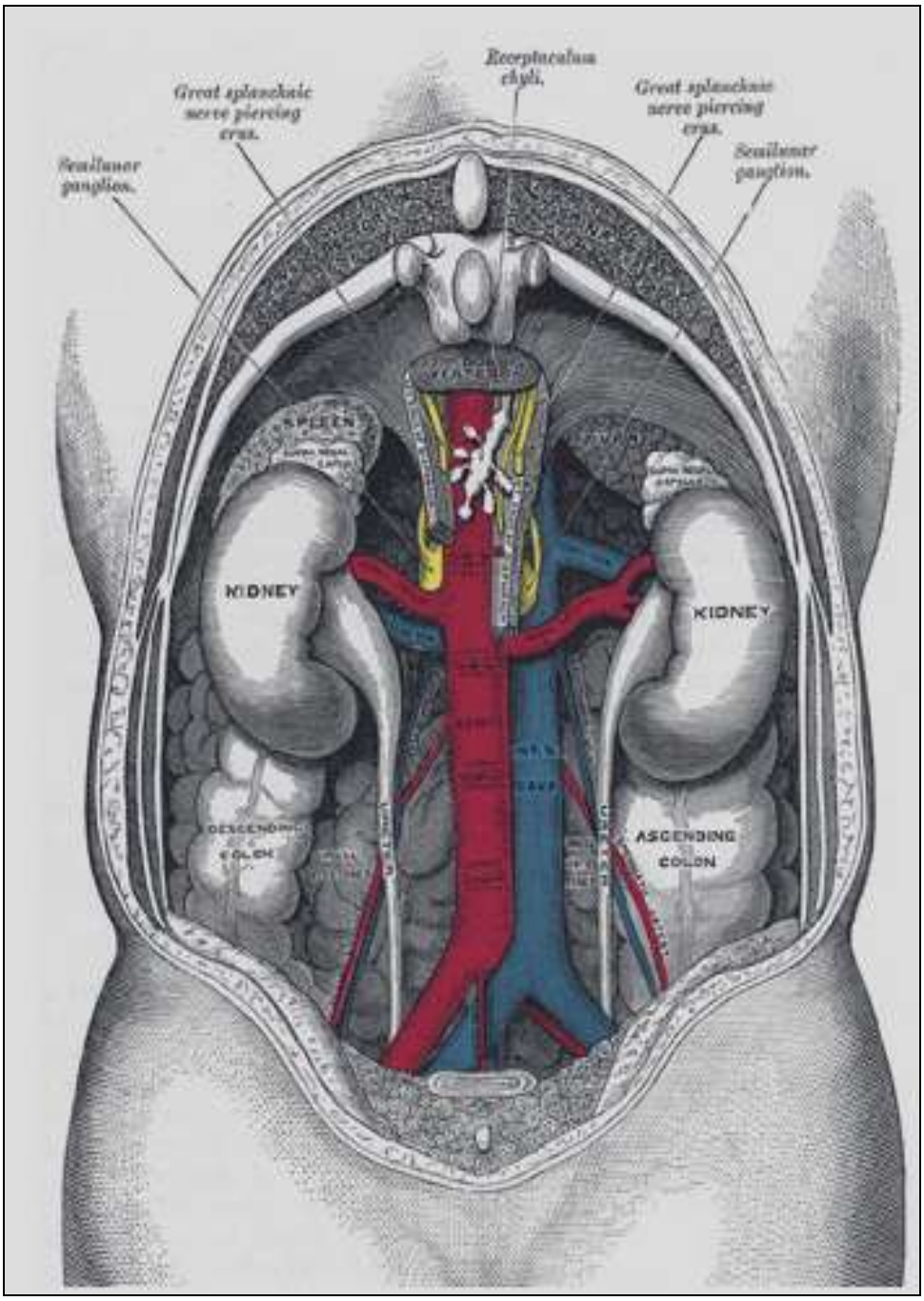

Figure (2.1) the relations of the viscera and large vessels of the abdomen.

(Gray, 1918)

The Posterior Surface (facies posterior): the posterior surface of each kidney is directed backward and medialward. It is imbedded in areolar and fatty tissue and entirely devoid of peritoneal covering. It lies upon the diaphragm, the medial and lateral lumbocostal arches, the Psoas major, the Quadratus lumborum, and the

tendon of the Transversus abdominis, the subcostal, and one or two of the upper lumbar arteries, and the last thoracic, iliohypogastric, and ilioinguinal nerves. The right kidney rests upon the twelfth rib, the left usually on the eleventh and twelfth. The diaphragm separates the kidney from the pleura, which dips down to form the phrenicocostal sinus, but frequently the muscular fibers of the diaphragm are defective or absent over a triangular area immediately above the lateral lumbocostal arch, and when this is the case the perinephric areolar tissue is in contact with the diaphragmatic pleura.

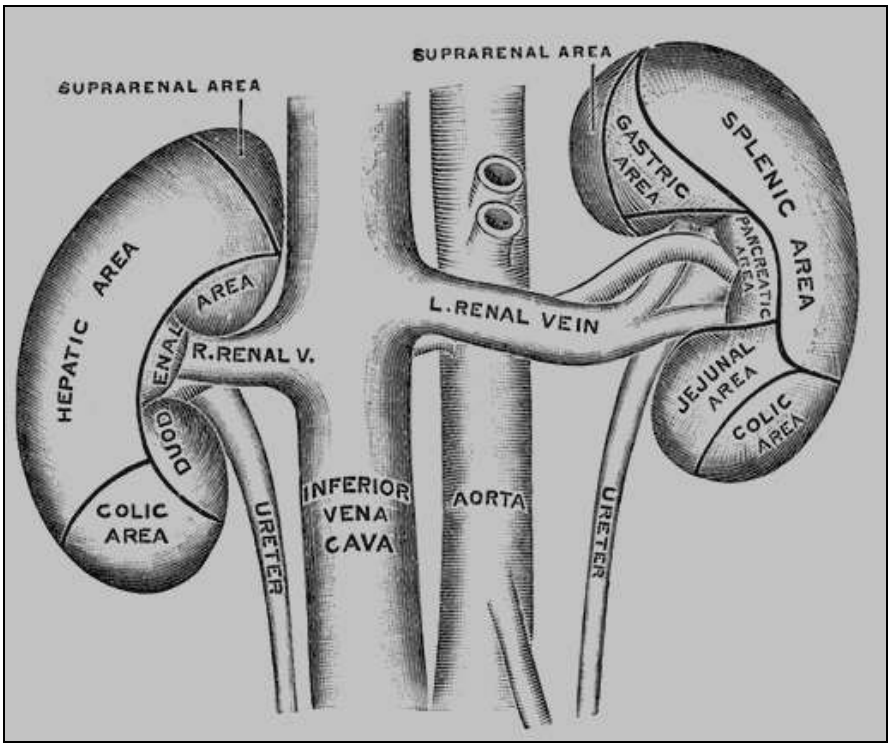

Figure (2.2) the anterior surfaces of the kidneys, showing the areas of contact of neighboring viscera. **(**Gray, 1918)

Borders: The lateral border (external border) is convex, and is directed toward the postero-lateral wall of the abdomen. On the left side it is in contact at its upper part, with the spleen.

The medial border (internal border) is concave in the center and convex toward either extremity; it is directed forward and a little downward. Its central part presents a deep longitudinal fissure, bounded by prominent overhanging anterior

and posterior lips. This fissure is named the hilum, and transmits the vessels, nerves, and ureter. Above the hilum the medial border is in relation with the suprarenal gland; below the hilum, with the ureter.

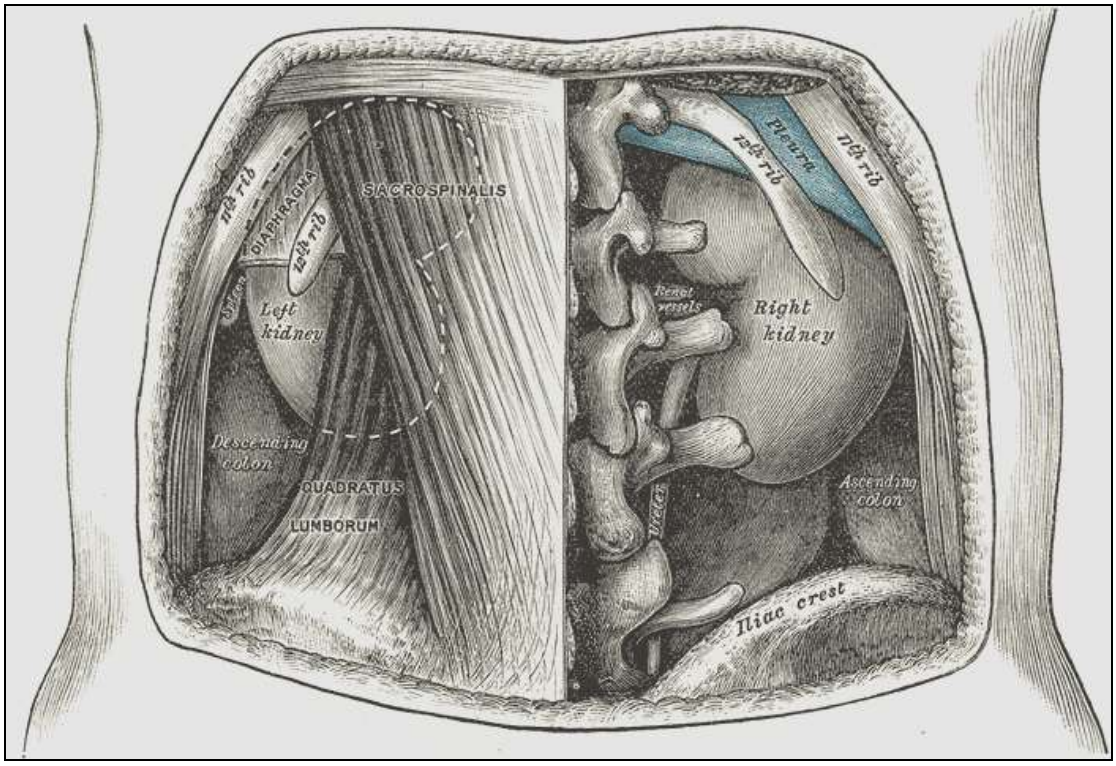

Figure: (2.3) the relations of the kidneys from behind. (Gray,1918)

General Structure of the Kidney: the kidney is composed of an internal medullary and an external cortical substance. The medullary substance (substantia medullaris) consists of a series of red-colored striated conical masses, termed the renal pyramids, the bases of which are directed toward the circumference of the kidney, while their apices converge toward the renal sinus, where they form prominent papillæ projecting into the interior of the calyces.

The cortical substance (substantia corticalis) is reddish brown in color and soft and granular in consistence. It lies immediately beneath the fibrous tunic, arches over the bases of the pyramids, and dips in between adjacent pyramids toward the renal sinus. The parts dipping in between the pyramids are named the renal columns (Bertin), while the portions which connect the renal columns to each other and intervene between the bases of the pyramids and the fibrous tunic are called

the cortical arches. If the cortex be examined with a lens, it will be seen to consist of a series of lighter-colored, conical areas, termed the radiate part, and a darker-colored intervening substance, which from the complexity of its structure is named the convoluted part. The rays gradually taper toward the circumference of the kidney, and consist of a series of outward prolongations from the base of each renal pyramid.

2.1.1.2 Minute Anatomy:

The renal tubules: of which the kidney is for the most part made up, commence in the cortical substance, and after pursuing a very circuitous course through the cortical and medullary substances, finally end at the apices of the renal pyramids by open mouths, so that the fluid which they contain is emptied, through the calyces, into the pelvis of the kidney. If the surface of one of the papillæ be examined with a lens, it will be seen to be studded over with minute openings, the orifices of the renal tubules, from sixteen to twenty in number, and if pressure be made on a fresh kidney, urine will be seen to exude from these orifices. The tubules commence in the convoluted part and renal columns as the renal corpuscles, which are small rounded masses of a deep red color, varying in size, but of an average of about 0.2 mm. in diameter. Each of these little bodies is composed of two parts: a central glomerulus of vessels, and a membranous envelope, the glomerular capsule (capsule of Bowman), which is the small pouch-like commencement of a renal tubule.

The glomerulus: is a lobulated net-work of convoluted capillary blood vessels, held together by scanty connective tissue? This capillary net-work is derived from a small arterial twig, the afferent vessel, which enters the capsule, generally at a point opposite to that at which the latter is connected with the tubule; and the resulting vein, the efferent vessel, emerges from the capsule at the same point. The afferent vessel is usually the larger of the two. The glomerular or Bowman's capsule, which surrounds the glomerulus, consists of a basement membrane, lined on its inner surface by a layer of flattened epithelial cells, which are reflected from

the lining membrane on to the glomerulus, at the point of entrance or exit of the afferent and efferent vessels. The whole surface of the glomerulus is covered with a continuous layer of the same cells, on a delicate supporting membrane. Thus between the glomerulus and the capsule a space is left, forming a cavity lined by a continuous layer of squamous cells; this cavity varies in size according to the state of secretion and the amount of fluid present in it. In the fetus and young subject the lining epithelial cells are polyhedral or even columnar.

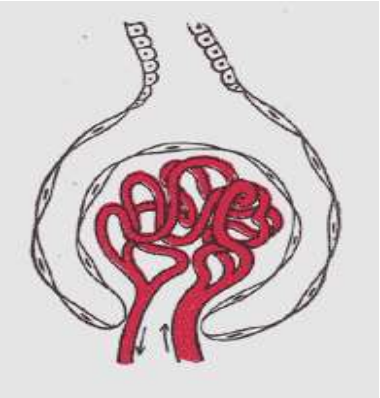

Figure: (2.4) Glomerulus (Gray, 1918)

The renal tubules, commencing in the renal corpuscles, present, during their course, many changes in shape and direction, and are contained partly in the medullary and partly in the cortical substance. At their junction with the glomerular capsule they exhibit a somewhat constricted portion, which is termed the neck. Beyond this the tubule becomes convoluted, and pursues a considerable course in the cortical substance constituting the proximal convoluted tube. After a time the convolutions disappear, and the tube approaches the medullary substance in a more or less spiral manner; this section of the tubule has been called the spiral tube. Throughout this portion of their course the renal tubules are contained entirely in the cortical substance, and present a fairly uniform caliber. They now enter the medullary substance, suddenly become much smaller, quite straight in direction, and dip down for a variable depth into the pyramids, constituting the descending limb of Henle's loop. Bending on themselves, they form what is termed the loop of Henle, and reascending, they become suddenly enlarged,

forming the ascending limb of Henle's loop, and reënter the cortical substance. This portion of the tubule ascends for a short distance, when it again becomes dilated, irregular, and angular. This section is termed the zigzag tubule; it ends in a convoluted tube, which resembles the proximal convoluted tubule, and is called the distal convoluted tubule. This again terminates in a narrow junctional tube, which enters the straight or collecting tube.

The collecting tubes commence in the radiate part of the cortex, where they receive the curved ends of the distal convoluted tubules. They unite at short intervals with one another, the resulting tubes presenting a considerable increase in caliber, so that a series of comparatively large tubes passes from the bases of the rays into the renal pyramids. In the medulla the tubes of each pyramid converge to join a central tube (duct of Bellini) which finally opens on the summit of one of the papillæ; the contents of the tube are therefore discharged into one of the calyces. (Gray, 1918)

2.1.2The ureter:

The ureter is a long (30-34 cm), mucosal-lined conduit that delivers urine from the renal pelvis to the bladder. Each ureter varies in diameter from 2 to 8 mm**.** as it enters the pelvis, the ureter passes anterior to the common (external) iliac artery. The ureter has an oblique course through the bladder wall. (Netter FH. Anatomy, structure, and embryology)

2.1.3The bladder:

The bladder is positioned in the pelvis, inferior and anterior to the peritoneal cavity and posterior to the pubic bones. (Netter FH. Anatomy, structure, and embryology) superiorly, the peritoneum is reflected over the anterior aspect of the bladder. Within the bladder, the ureteric and urethral orifices demarcate an area known as the trigone; the urethral orifice also marks the bladder neck. The bladder neck and trigone remain constant in shape and position; however, the remainder of the bladder will change shape and position depending on the volume of urine within it. Deep to the peritoneum covering the bladder is a loose, connective tissue

layer of subserosa that forms the adventitial layer of the bladder wall. Adjacent to the adventitia are three muscle layers: the outer (longitudinal), middle (circular), and internal longitudinal layers. Adjacent to the muscle, the innermost layer of the bladder is composed of mucosa. The bladder wall should be smooth and of uniform thickness. The wall thickness depends on the degree of bladder distention. (Netter FH. Anatomy, structure, and embryology)

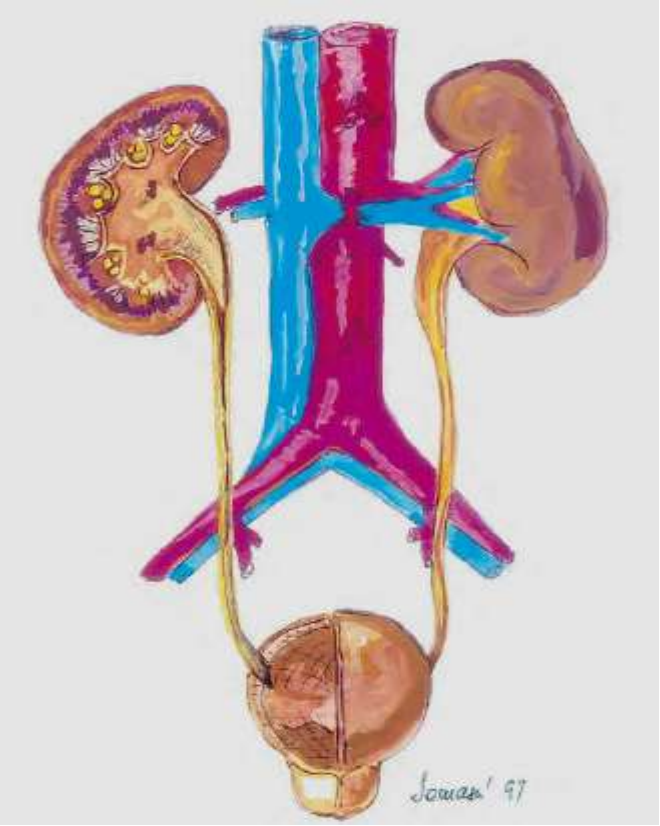

Figure: (2.5) Kidney, ureter and bladder (Carol M. Rumack, et al 2011)

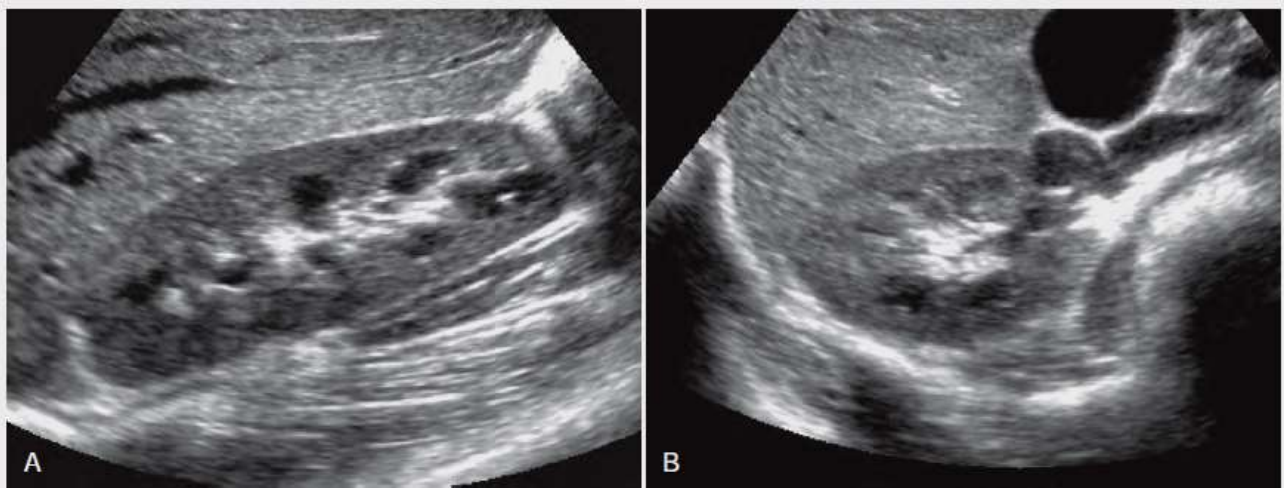

Figure: (2.6): normal sonographic image of kidney: **A,** Sagittal, and **B,** transverse, sonograms of normal anatomy with corticomedullary differentiation show

relatively hypoechoic medullary pyramids, with cortex slightly less echogenic than the liver and spleen. (Carol M. Rumack, et al 2011)

2.2 Physiology:

The kidneys are responsible for urine formation, filtration and elimination of waste products, and regulation of fluids and electrolytes. The kidneys are also vital in maintaining the acid-base balance in the body. Hormone production and synthesis of vitamin D are also essential kidney functions (Scalon.2011).

2.2.1 Urine Formation:

Urine formation is directly related to the glomerular filtration rate (GFR). Urine formation involves filtration, reabsorption, and secretion.

- Filtration: is the transfer of dissolved substances and water and mostly occurs due to hydrostatic pressure in the glomerular capillaries. Fluid is filtered out of the capillaries when the hydrostatic pressure pushes blood against the walls of the capillaries (Scanlon, 2011).
- Reabsorption**:** occurs by active and passive transport. Passive transport is accomplished via the process of osmosis and diffusion. Active transport requires energy (such as the sodium/potassium pump) and a substance to carry the molecules (Scanlon, 2011).
- Secretion: is the movement of a substance from the capillaries that is not needed by the body (Scanlon, 2011).

2.2.2 Elimination of waste products (creatinine and BUN):

- **Creatinine:** excretion of metabolic waste products in the urine can be measured to determine how well the kidneys are functioning. Serum creatinine, a waste product of muscle metabolism, can be used to measure renal function because it is excreted only by the kidneys. The amount of creatinine produced per day is constant and dependent upon the body's muscle mass. It is freely filtered so that production should equal excretion. Because of this, measuring one's serum creatinine is a very reliable indicator of renal function (Scanlon.2011).
- **Blood Urea Nitrogen:** Blood urea nitrogen is a waste product from protein metabolism. It is filtered and reabsorbed along the length of the entire (Scanlon,

2011). It is not as reliable at measuring kidney function because it is dependent upon: urine flow**,** renal blood flow **,** protein metabolism **,** drugs and diet

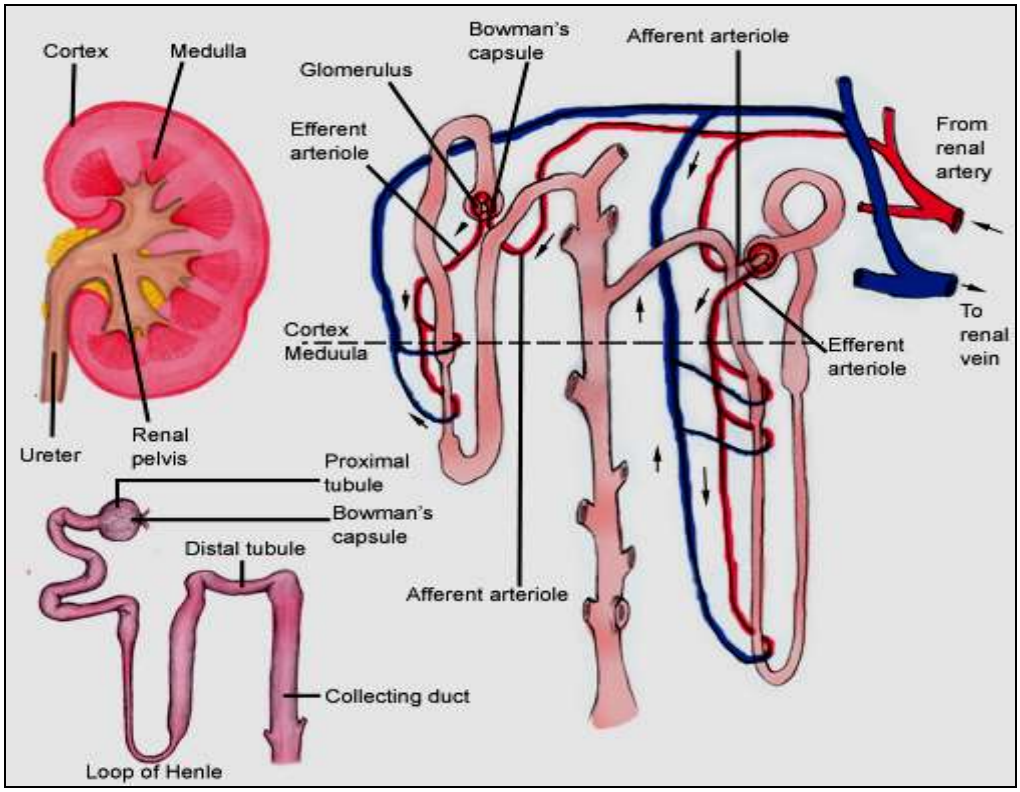

Figure: (2.7) Microanatomy of the nephron

(http://emedicine.medscape.com.kidney anatomy: microanatomy of nephron)

2.2.3 Fluid Regulation:

The body has several mechanisms for regulating fluids. One of the first ways the body regulates fluid is via the hypothalamus. The hypothalamus is the body's thirst center. When cells in the hypothalamus become dehydrated, it causes the brain to tell you that you are thirsty, so you drink more water.

Antidiuretic hormone (ADH) is also made in the hypothalamus, and released from the posterior pituitary.

2.2.4Electrolytes and Factors influencing Excretion and Re-Absorption:

The kidneys also play a vital role in the regulation of certain electrolytes. These electrolytes and the factors that influence their secretion are outlined in the table below.

Table (2.1) Electrolyte Factors Influencing Excretion and Re-absorption

Electrolyte	Factors Influencing Excretion and Re-absorption
Sodium	As GFR increases, sodium re-absorption decreases – so more is excreted. As GFR decreases, sodium re-absorption increases – so less is excreted. When aldosterone is released, it caused the kidneys to reabsorb sodium
Potassium	Elevations in potassium levels High urine flow (increased intake or diuretics) increase potassium excretion Aldosterone
Calcium	Parathyroid hormone – PTH - (stimulated by decrease in Ca). Vitamin D (stimulate Ca absorption from GI tract
Phosphate	PTH (inhibits re-absorption of phosphorus) GFR (as GFR increases, phosphate re-absorption decreases and visa versa)
Magnesium	Sodium dependant
Chloride	Acid Base Balance. In acidosis, bicarbonate is reabsorbed while chloride is excreted. In alkalosis, bicarbonate is excreted, while chloride is reabsorbed

(Hall, Schmidt & Wood, 2005)

2.3. Changes in System and Renal Function due to aging:

- **Changing in system due to aging:**

As we age, our nephrons decrease in number. Additionally, the overall amount of kidney tissue decreases. Blood supply to the kidney can be impacted by atherosclerosis, and GFR decreases. The bladder muscles weaken, resulting in incomplete emptying when urinating. Prostatic changes in men may impede the outward flow of urine. For women, bladder or vaginal prolapse may also block the urethra. Because the bladder does not completely empty, aging persons have an increased risk for urinary tract infections. Since the kidneys have so much reserve capacity, normal age-related changes do not impact our daily renal function. Elderly patients may also have impaired thirst mechanisms or intentionally decrease fluid intake to reduce bladder control issues. Bladder and prostate cancer are also more prevalent in older individuals (Jarvis, 2008; Scanlon, 2011).

- **Aging and renal function:**

The biologic process of aging initiates various structural and functional changes within the kidney. (Hallan SI, et al. 2012, Boer IH. 2012). Renal mass progressively declines with advancing age, and glomerulosclerosis leads to a decrease in renal weight. Histologic examination is notable for a decrease in glomerular number of as much as 30-50% by age 70 years. The GFR peaks during the third decade of life at approximately 120 mL/min/1.73 m2; it then undergoes an annual mean decline of approximately 1 mL/min/y/1.73 m2, reaching a mean value of 70 mL/min/1.73 m2 at age 70 years.

2.4 Blood supply:

The kidneys receive blood from the renal arteries, left and right, which branch directly from the abdominal aorta. Despite their relatively small size, the kidneys receive approximately 20% of the cardiac output. (Walter F, 2004).

Each renal artery branches into segmental arteries, dividing further into interlobar arteries which penetrate the renal capsule and extend through the renal columns between the renal pyramids. The interlobar arteries then supply blood to the arcuate arteries that run through the boundary of the cortex and the medulla. Each arcuate artery supplies several interlobular arteries that feed into the afferent arterioles that supply the glomeruli.

The interstitum (or interstitium) is the functional space in the kidney beneath the individual filters (glomeruli) which are rich in blood vessels. The interstitum absorbs fluid recovered from urine. Various conditions can lead to scarring and congestion of this area, which can cause kidney dysfunction and failure.

After filtration occurs the blood moves through a small network of venules that converge into interlobular veins. The interlobular provide blood to the arcuate veins then back to the interlobar veins which come to form the renal vein exiting the kidney for transfusion for blood.

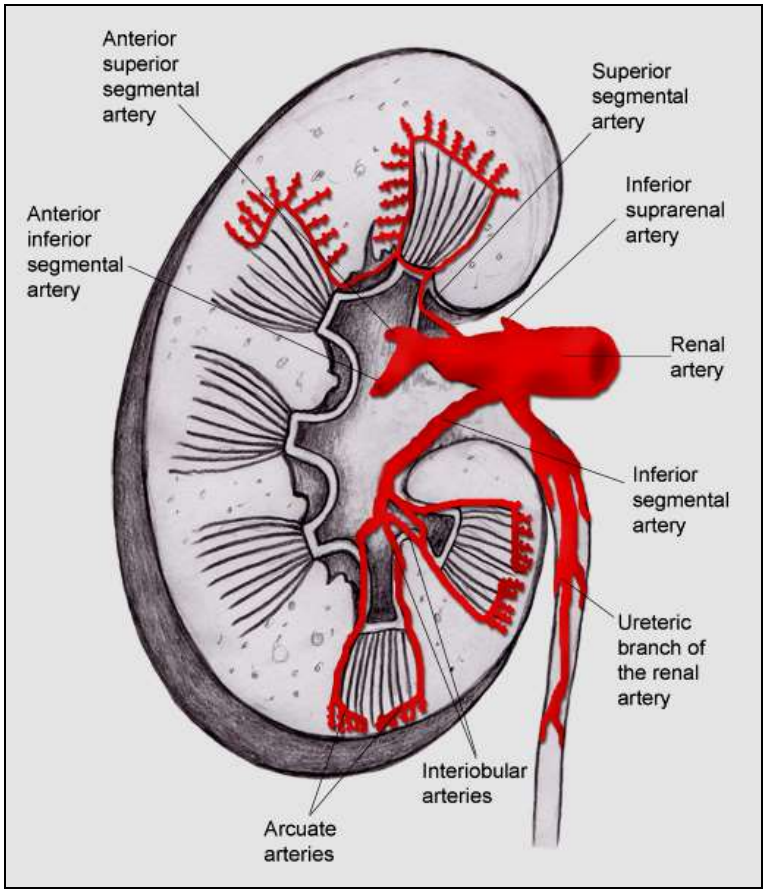

Figure: (2.8): Intrarenal arteries

(http://emedicine.medscape.com.kidney anatomy: intrarenal arteries)

2.5 Pathology:

2.5.1background

Chronic kidney disease (CKD)—or chronic renal failure (CRF), as it was historically termed—is a term that encompasses all degrees of decreased renal function, from damaged–at risk through mild, moderate, and severe chronic kidney failure.

The Kidney Disease Outcomes Quality Initiative (KDOQI) of the National Kidney Foundation (NKF) established a definition and classification of CRF. (Levey AS, et al 2003) .These guidelines have allowed better communication among physicians and have facilitated intervention at the different stages of the disease.

The KDOQI defines CRF as either kidney damage or a decreased glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m2 for 3 or more months. Whatever the underlying etiology, once the loss of nephrons and reduction of functional renal mass reaches a certain point; the remaining nephrons begin a process of irreversible sclerosis that leads to a progressive decline in the GFR.

2.5.2 Staging:

The different stages of CRF form a continuum. The KDOQI classification of the stages of CRF is as follows (Levey AS, et al 2003).

- Stage 1: Kidney damage with normal or increased GFR (>90 mL/min/1.73 m^2)
- Stage 2: Mild reduction in GFR (60-89 mL/min/1.73 m^2)
- Stage 3: Moderate reduction in GFR (30-59 mL/min/1.73 m^2)
- Stage 4: Severe reduction in GFR (15-29 mL/min/1.73 m^2)
- Stage 5: Kidney failure (GFR < 15 mL/min/1.73 m^2 or dialysis)

In stage 1 and stage 2 CRF, reduced GFR alone does not clinch the diagnosis, because the GFR may in fact be normal or borderline normal. Other markers of kidney damage, including abnormalities in the composition of blood or urine or structural abnormalities visualized by imaging studies, establish the diagnosis in such cases. Hypertension is a frequent sign of CRF but should not by itself be considered a marker of it, because elevated blood pressure is also common among people without chronic renal failure.

In an update of its CRF classification system, the National Kidney Foundation (NKF) advised that GFR and albuminuria levels be used together, rather than separately, to improve prognostic accuracy in the assessment of CRF.(Levey AS , et al 2003,Waknine Y. 2012).

2.6 Pathophysiology:

A normal kidney contains approximately 1 million nephrons, each of which contributes to the total glomerular filtration rate (GFR). In the face of renal injury

(regardless of the etiology), the kidney has an innate ability to maintain GFR, despite progressive destruction of nephrons, as the remaining healthy nephrons manifest hyper filtration and compensatory hypertrophy. This nephron adaptability allows for continued normal clearance of plasma solutes. Plasma levels of substances such as urea and creatinine start to show measurable increases only after total GFR has decreased to 50%.

The plasma creatinine value will approximately double with a 50% reduction in GFR. For example, a rise in plasma creatinine from a baseline value of 0.6 mg/dL to 1.2 mg/dL in a patient, although still within the adult reference range, actually represents a loss of 50% of functioning nephron mass. (Levey AS, et al 2003).

2.7 Etiology:

Causes of chronic renal failure (CRF) include the following:

- Diabetic kidney disease
- Hypertension
- Vascular disease such as renal artery stenosis and renal vein thrombosis.
- Glomerular disease (primary or secondary)
- Cystic kidney diseases
- Tubulointerstitial disease
- Urinary tract obstruction or dysfunction
- Recurrent kidney stone disease
- Unrecovered acute kidney injury

2.7.1 Diabetes Mellitus:

Diabetes is a chronic disease in which the body cannot regulate the amount of sugar in the blood.

2.7.1.1Diabetes causes:

Insulin is a hormone produced by the pancreas to control blood sugar. Diabetes can be caused by too little insulin, resistance to insulin, or both.

To understand diabetes, it is important to first understand the normal process by which food is broken down and used by the body for energy. Several things happen when food is digested:

- A sugar called glucose enters the bloodstream. Glucose is a source of fuel for the body.
- An organ called the pancreas makes insulin. The role of insulin is to move glucose from the bloodstream into muscle, fat, and liver cells, where it can be stored or used as fuel.

People with diabetes have high blood sugar because their body cannot move sugar from the blood into muscle and fat cells to be burned or stored for energy, and because their liver makes too much glucose and releases it into the blood. This is because either:

- Their pancreas does not make enough insulin
- Their cells do not respond to insulin normally
- Both of the above

2.7.1.2 Diabetes types:

There are two major types of diabetes. The causes and risk factors are different for each type:

- Type 1 diabetes can occur at any age, but it is most often diagnosed in children, teens, or young adults. In this disease, the body makes little or no insulin. This is because the pancreas cells that make insulin stop working. Daily injections of insulin are needed. The exact cause is unknown
- Type 2 diabetes is much more common. It most often occurs in adulthood, but because of high obesity rates, teens and young adults are now being diagnosed with this disease. Some people with type 2 diabetes do not know they have it.

- Gestational diabetes is high blood sugar that develops at any time during pregnancy in a woman who does not have diabetes (cannot be classified as type 1 or type 2)

If your parent, brother, or sister has diabetes, you may be more likely to develop the disease. **(http://www.nlm.nih.gov/medlineplus)**

2.7.1.3 Symptoms of diabetes:

A high blood sugar level can cause several symptoms, including:

- Blurry vision
- Excess thirst
- Fatigue
- Frequent urination
- Hunger
- Weight loss

Because type 2 diabetes develops slowly, some people with high blood sugar have no symptoms.

Symptoms of type 1 diabetes develop over a short period. People may be very sick by the time they are diagnosed.

After many years, diabetes can lead to other serious problems. These problems are known as diabetes complications, and include:

- Eye problems, including trouble seeing (especially at night), light sensitivity, and blindness
- Sores and infections of the leg or foot, which untreated can lead to amputation of the leg or foot
- Damage to nerves in the body, causing pain, tingling, a loss of feeling, problems digesting food, and erectile dysfunction
- Kidney problems, which can lead to kidney failure

- Weakened immune system, which can lead to more frequent infections
- Increased chance of having a heart attack or stroke

2.7.1.4 Exams and tests for diabetes:

1. A urine analysis test: may show high blood sugar. But a urine test alone does not diagnose diabetes.

2. Blood tests:

- Fasting blood glucose level : diabetes is diagnosed if the fasting glucose level is higher than 126 mg/dL on two different tests. Levels between 100 and 126 mg/dL are called impaired fasting glucose or pre-diabetes. These levels are risk factors for type 2 diabetes.
- Hemoglobin A1c (A1C) test:

  - Normal: Less than 5.7%
  - Pre-diabetes: 5.7% - 6.4%
  - Diabetes: 6.5% or higher

- Oral glucose tolerance test : diabetes is diagnosed if the glucose level is higher than 200 mg/dL 2 hours after drinking a sugar drink.
- Screening for type 2 diabetes in people who have no symptoms is recommended for:

  - Overweight children who have other risk factors for diabetes, starting at age 10 and repeated every 3 years
  - Overweight adults (BMI of 25 or higher) who have other risk factors
  - Adults over age 45, repeated every 3 years

2.7.1.5 Treatment of diabetes:

Type2 diabetes may be reversed with lifestyle changes, especially losing weight with exercise and by eating healthier foods. Some cases of type 2 diabetes can also be improved with weight-loss surgery.

Treating either type 1 diabetes or type 2 diabetes involves medicines, diet, and exercise to blood sugar level.

Getting better control over your blood sugar, cholesterol, and blood pressure levels helps reduce the risk of kidney disease, eye disease, nervous system disease, heart attack, and stroke.

2.7.1.6 Prevention from diabetes:

Keeping an ideal body weight and an active lifestyle may prevent or delay the start of type 2 diabetes. (**http://www.nlm.nih.gov/medlineplus)**

2.7.1.7 Diabetes and kidney disease (diabetic nephropathy):

Kidney disease or kidney damage that occurs in people with diabetes is called diabetic nephropathy. This condition is a complication of diabetes.

- **Causes of diabetic nephropathy:**

Each kidney is made of hundreds of thousands of small units called nephrons. These structures filter the blood, help remove waste from the body, and control fluid balance.

In people with diabetes, the nephrons slowly thicken and become scarred over time. The kidneys begin to leak protein (albumin) passes into the urine. This damage can happen years before any symptoms begin.

Kidney damage is more likely if you:

- Have uncontrolled blood sugar
- Have high blood pressure
- Have type 1 diabetes that began before you were 20 years old
- Have family members who also have diabetes and kidney problems
- Smoke
- Are African American, Mexican American, or Native American

- **Symptoms of diabetic nephropathy:**

Often, there are no symptoms as the kidney damage starts and slowly gets worse. Kidney damage can begin 5 to 10 years before symptoms start.

People who have more severe and long-term (chronic) kidney disease may have symptoms such as:

- Fatigue most of the time
- General ill feeling
- Headache
- Nausea and vomiting
- Poor appetite
- Swelling of the legs

- **Exams and Tests for diabetic nephropathy:** to detect signs of kidney problems.

- Microalbuminuria test: A urine test looks for a protein called albumin leaking into the urine. (It measures small amounts of albumin). Too much albumin in the urine is often a sign of kidney damage.
- BUN test
- Serum creatinine test
- Check the blood pressure. (if you have diabetic nephropathy, you likely also have high blood pressure)
- A kidney biopsy (to confirm the diagnosis or look for other causes of kidney damage)

- **Treatment of diabetic nephropathy:**

When kidney damage is caught in its early stages, it can be slowed with treatment. Once larger amounts of protein appear in the urine, kidney damage will slowly get worse.

1. Control blood pressure:

    - Keeping the blood pressure under control (below 130/80) is one of the best ways to slow kidney damage.
    - Take the prescribed medicines to lower your blood pressure and protect your kidneys from more damage
    - Taking these medicines, even when your blood pressure is in a healthy range, helps slow kidney damage.

2. Control blood sugar level:

    You can also slow kidney damage by controlling your blood sugar level, which you can do by:

    - Eating healthy foods
    - Getting regular exercise
    - Taking medicine or insulin as instructed by your health care provider
    - Checking your blood sugar level as often as instructed and keeping a record of your blood sugar numbers so that you know how meals and activities affect your level

3. Other ways to protect your kidneys:

    - Before having an MRI, CT scan, or other imaging test in which you receive a contrast dye, tell the health care provider who is ordering the test that you have diabetes. Contrast dye can cause more damage to your kidneys.
    - Before taking an NSAID pain medicine, such as ibuprofen or naproxen, ask your health care provider if there is another kind of medicine that you can take instead. NSAIDs can damage the kidneys, especially when you use them often.
    - Know the signs of urinary tract infections and get them treated right away. (http://www.nlm.nih.gov/medlineplus)

2.7.1.8 Diabetes and Obesity:

The International Diabetes Foundation (IDF) says that, "Diabetes and obesity are the biggest public health challenge of the 21st century.

Is there a link between obesity and diabetes?

The people diagnosed with type II diabetes, about 80 to 90 percent are also diagnosed as obese in USA. This fact provides an interesting clue to the link between diabetes and obesity.

Being overweight can cause your body to become resistant to insulin. (Body less sensitive to the insulin)

2.7.2 Hypertension:

2.7.2.1What is high blood pressure?

Blood pressure is the force of blood pushing against blood vessel walls as the heart pumps out blood, and high blood pressure is an increase in the amount of force that blood places on blood vessels as it moves through the body. Factors that can increase this force include higher blood volume due to extra fluid in the blood and blood vessels that are narrow and stiff.

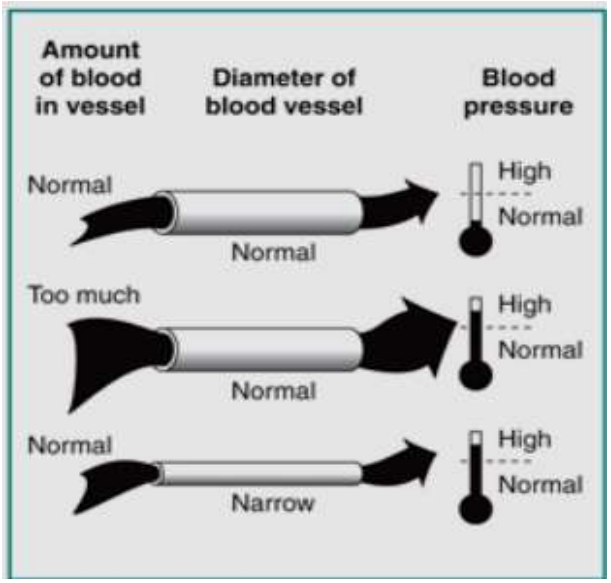

Figure (2.9) blood pushing against blood vessel walls as the heart pumps out blood.

(www.nhlbi.nih.gov/health/health-topics/topics/hbpLink, 2013)

Blood pressure test results are written with two numbers separated by a slash. For example, 120/80 (Blood pressure is measured in units of "millimeters of mercury"; written mm Hg for short). "120 over 80." The top number is called the systolic pressure and represents the pressure as the heart beats and pushes blood through the blood vessels. The bottom number is called the diastolic pressure and represents the pressure as blood vessels relax between heartbeats.

Most people without chronic health conditions have a normal blood pressure if it stays below 120/80. Pre hypertension is a systolic pressure of 120 to 139 or a diastolic pressure of 80 to 89. High blood pressure is a systolic pressure of 140 or above or a diastolic pressure of 90 or above. (www.nhlbi.nih.gov/health/health-topics/topics/hbpLink, 2013).

2.7.2.2 What are the kidneys and what do they do?

The kidneys are two bean-shaped organs, each about the size of a fist. They are located just below the rib cage, one on each side of the spine. Every day, the two kidneys filter about 120 to 150 quarts-(unit volume), three of these kinds of quarts remain in current use, all approximately equal to one litre -of blood to produce about 1 to 2 quarts of urine, composed of wastes and extra fluid.

Kidneys work at the microscopic level. The kidney is not one large filter. Each kidney is made up of about a million filtering units called nephrons. Each nephron filters a small amount of blood. The nephron includes a filter, called the glomerulus, and a tubule. The nephrons work through a two-step process. The glomerulus lets fluid and waste products pass through it; however, it prevents blood cells and large molecules, mostly proteins, from passing. The filtered fluid then passes through the tubule, which sends needed minerals back to the bloodstream and removes wastes. The final product becomes urine.

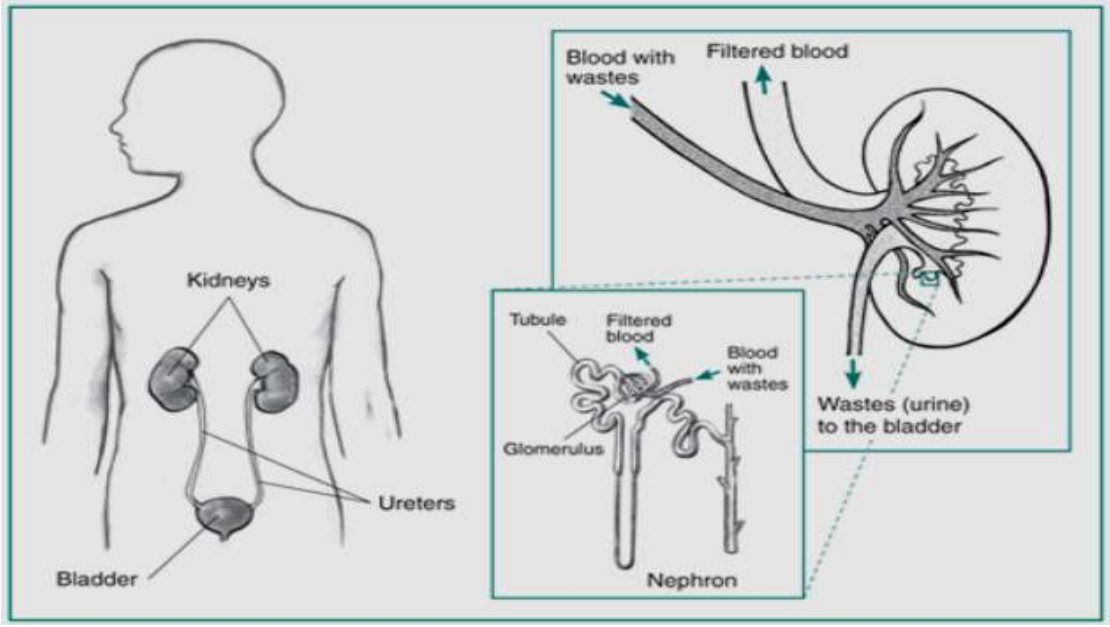

Figure (2.10) filtering units of kidney (nephrons)

(www.cdc.gov/nchs/data/hus/2011/051.pdf.2013)

2.7.2.3 How does high blood pressure affect the kidneys?

High blood pressure can damage blood vessels in the kidneys, reducing their ability to work properly. When the force of blood flow is high, blood vessels stretch so blood flows more easily. Eventually, this stretching scars and weakens blood vessels throughout the body, including those in the kidneys. If the kidneys' blood vessels are damaged, they may stop removing wastes and extra fluid from the body. Extra fluid in the blood vessels may then raise blood pressure even more, creating a dangerous cycle.

High blood pressure is the second leading cause of kidney failure in the United States after diabetes, as illustrated in the figure (2.12) below. In addition, the rate of kidney failure due to high blood pressure increased 7.7 percent from 2000 to 2010. (*Renal Data System 2012 Annual Data Report).*

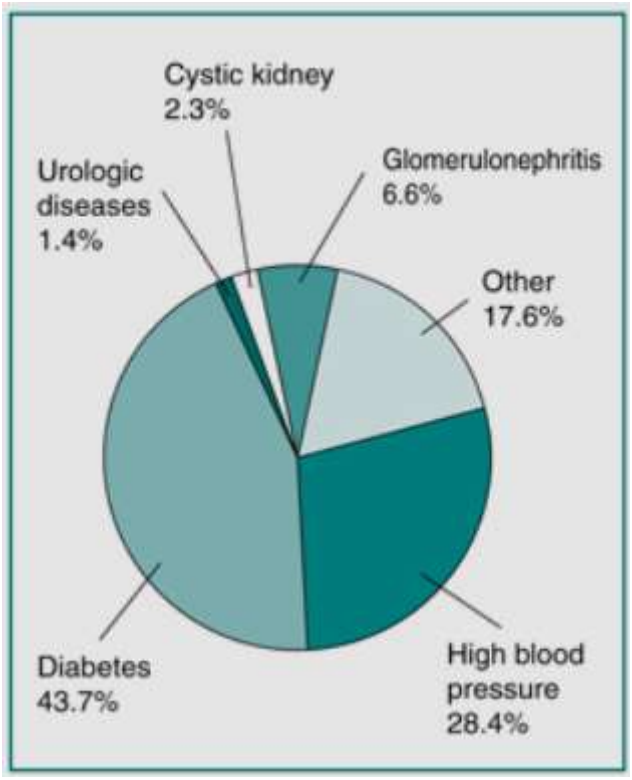

Figure (2.11) Causes of kidney failure in the United States (www.cdc.gov/nchs/data/hus/2011/051.pdf. 2013)

2.7.2.4 What are the symptoms of high blood pressure and kidney disease?

Most people with high blood pressure do not have symptoms. In rare cases, high blood pressure can cause headaches.

Kidney disease also does not have symptoms in the early stages. A person may have swelling called edema, which happens when the kidneys cannot get rid of extra fluid and salt. Edema can occur in the legs, feet, or ankles and less often in the hands or face. Once kidney function decreases further, symptoms can include:

- appetite loss
- nausea
- vomiting
- drowsiness or feeling tired
- trouble concentrating
- sleep problems
- increased or decreased urination
- generalized itching or numbness

- dry skin
- headaches
- weight loss
- shortness of breath
- chest pain

2.7.2.5 How are high blood pressure and kidney disease diagnosed?

1. Multiple blood pressure tests:

High blood pressure diagnosed by multiple blood pressures tests—show that a systolic blood pressure is consistently above 140 or a diastolic blood pressure is consistently above 90. It is measured with a blood pressure cuff.

Kidney disease is diagnosed with urine and blood tests.

Figure (2.12) Health care providers measure blood pressure with a blood pressure cuff.

2. Urine tests:

- Dipstick test for albumin: A dipstick test performed on a urine sample can detect the presence of albumin in the urine. Albumin is a protein in the blood that can pass into the urine when the kidneys are damaged. A technician places a strip of chemically treated paper, called a dipstick, into the urine. Patches on the dipstick change color when blood or protein is present in urine.
- Urine albumin-to-creatinine ratio: Is used to determine the ratio between the albumin and creatinine in the urine. Creatinine is a waste product in the blood

that is filtered in the kidneys and excreted in the urine. A urine albumin-to-creatinine ratio above 30 mg/g may be a sign of kidney disease.

3. Blood test:

A blood test to estimate how much blood the kidneys filter each minute, called the estimated glomerular filtration rate (eGFR). The results of the test indicate the following:

- eGFR of 60 or above is in the normal range
- eGFR below 60 may indicate kidney damage
- eGFR of 15 or below may indicate kidney failure

2.7.2.6 How can people prevent or slow the progression of kidney disease from high blood pressure?

The best way to slow or prevent kidney disease from high blood pressure is to take steps to lower blood pressure. These steps include a combination of medication and lifestyle changes, such as

- Healthy eating: reduce salt intake (4- 5mg per day).
- Physical activity: Regular physical activity can lower blood pressure and reduce the chances of other health problems. Most people should try to get at least 30 to 60 minutes of activity most or all days of the week.
- Maintaining a healthy weight: Overweight is defined as a body mass index (BMI)—a measurement of weight in relation to height—of 25 to 29. A BMI of 30 or higher is considered obese. A BMI lower than 25 is the goal for keeping blood pressure under control.
- Quitting smoking: smoking can damage blood vessels; raise the chance of high blood pressure.
- Managing stress: learning how to manage stress, relax, and cope with problems can improve emotional and physical health.
- Medication: medications that lower blood pressure can also significantly slow the progression of kidney disease. Two types of blood pressure-lowering medications, angiotensin-converting enzyme (ACE) inhibitors and

angiotensin receptor blockers (ARBs), have been shown effective in slowing the progression of kidney disease.

Figure (2.13) obesity is a risk factor for hypertension and other cardiovascular conditions (www.heart.org/HEARTORG/GettingHealthy/WeightManagement/BodyMassIndex)

People with kidney disease should keep their blood pressure below 140/90. (James PA, Oparil S, Carter BL, et al. 2014)

2.8 Epidemiology:

In the United States, the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) reports that 1 in 10 American adults has some level of chronic renal failure (CRF**)**.According to the NIDDK, the incidence of recognized CRF in people aged 20-64 years in the United States rose only slightly from 2000−2008 and remains less than0.5%. In contrast, the incidence of recognized CRF in people aged 65 years or older more than doubled between 2000 and 2008, from approximately 1.8% to approximately 4.3(http ://kidney.niddk.nih.gov/

kudiseases/pubs/kustats.17). In National Health and Nutrition Examination Survey (NHANES), the distribution of estimated GFRs for the stages of CRF was similar in both sexes. (Renal Data System (USRDS) 2011) Annual Data Report).

2.9 Lab investigations:

2.9.1Blood tests:

Serum creatinine:

Creatinine is a nitrogenous compound formed as an end product of muscle metabolism. It is formed in muscle in relatively small amounts, passed into the blood and excreted in the urine. Blood creatinine level measures renal function. Normally it is produced in regular consistently small amounts. There for an elevation means a disturbance in renal function. Renal impairment is virtually the only cause of creatinine elevation.

- **Normal results of serum creatinine level:**

A normal result is 0.7 to 1.3 mg/dL for men and 0.6 to 1.1 mg/dL for women. Women usually have a lower creatinine level than men. This is because women usually have less muscle mass than men. The examples above are common measurements for results of these tests. Normal value ranges may vary slightly among different laboratories. Some labs use different measurements or test different samples. Talk to your doctor about the meaning of your specific test results.

- **Higher than normal level may be due to:**

  - Blocked urinary tract
  - Kidney problems, such as kidney damage or failure, infection, or reduced blood flow
  - Loss of body fluid (dehydration).
  - Muscle problems, such as breakdown of muscle fibers (rhabdomyolysis)
  - Problems during pregnancy, such as seizures (eclampsia), or high blood pressure caused by pregnancy (preeclampsia)

- **Lower than normal level may be due to:**

  - Conditions involving the muscles and the nerves that control them (myasthenia gravis)
  - Muscle problems, such as late stage muscle loss (muscular dystrophy)

The blood urea nitrogen (BUN) and serum creatinine levels will be elevated in patients with CRF. Serum albumin levels may also be measured, as patients may have hypoalbuminemia as a result of urinary protein loss or malnutrition. A lipid profile should be performed in all patients with CRF because of their risk of cardiovascular disease.

BUN (blood urea nitrogen)

- Urea nitrogen is what forms when protein breaks down.
- A test can be done to measure the amount of urea nitrogen in the blood.
- The BUN test is often done to check kidney function.

- **Normal Results of BUN:**

The normal result is generally (6 – 20) mg/dL

- **Higher-than-normal levels may be due to:**

  - Congestive heart failure
  - Excessive protein levels in the gastrointestinal tract
  - Gastrointestinal bleeding
  - Hypovolemia (dehydration)
  - Heart attack
  - Kidney disease, including glomerulonephritis, pyelonephritis, and acute tubular necrosis
  - Kidney failure
  - Shock

- Urinary tract obstruction

- **Lower-than-normal levels may be due to:**

  - Liver failure
  - Low protein diet
  - Malnutrition
  - Over-hydration

(Landry DW, Basari H. 2011)

Estimated Glomerular Filtration Rate (eGFR):

Glomerular filtration rate (GFR) is a test used to check how well the kidneys are working. Specifically, it estimates how much blood passes through the glomeruli each minute. Glomeruli are the tiny filters in the kidneys that filter waste from the blood. Among the many physiologic roles of the renal system, GFR is considered the best indicator of overall kidney function and therefore its assessment has become an important clinical tool in the daily care of patients.

- **Normal results of eGFR**

According to the National Kidney Foundation, normal results range from 90 - 120 mL/min/1.73 m^2. Older people will have lower normal GFR levels, because GFR decreases with age**.**

What abnormal results of mean?

Levels below 60 mL/min/1.73 m^2 for 3 or more months are a sign of chronic kidney disease. GFR result lower than 15 mL/min/1.73 m^2 is a sign of kidney failure and requires immediate medical attention.

- **Why the test is performed:**

  - To measures how well the kidneys are filtering the blood.
  - To see how far kidney disease has progressed.

The GFR test is recommended for people with chronic kidney disease. It is also recommended for persons who may get kidney disease due to:

- Diabetes
- High blood pressure
- Family history of kidney disease
- Frequent urinary tract infections
- Heart disease
- Urinary blockage**.**

GFR cannot be measured directly, but instead it can be assessed by the renal clearance of filtration markers (Stevens LA, Levey AS. 2005).

- **The Cockcroft-Gault formula: (eGFR formula)**

It is for estimating creatinine clearance (CrCl) and eGFR; it should be used routinely as a simple means to provide a reliable approximation of residual renal function in all patients with CRF. The formulas are as follows:

- CrCl (male) = ([140-age] × weight in kg)/(serum creatinine × 72)
- CrCl (female) = CrCl (male) × 0.85

Alternatively, the Modification of Diet in Renal Disease (MDRD) Study equation could be used to calculate the glomerular filtration rate (GFR). This equation does not require a patient's weight. (Levey AS, et .al, 1999).

2.9.2 Urinalysis: (urine test).

In adult patients who are not at elevated risk for CRF, screening with total protein can be done with a standard urine dipstick, according to guidelines from the National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (KDOQI). If the dipstick test is positive (1+ or greater), patients should undergo testing for confirmation of proteinuria.

(http://www.kidney.org/professionals/KDOQI/guidelines. 2012).

For screening patients at elevated risk, the KDOQI recommends using an albumin-specific dipstick; this is because albuminuria is a more sensitive marker than total protein for CRF from diabetes, hypertension, and glomerular diseases. A positive dipstick test should be followed by calculation of the albumin-to-creatinine ratio, with a ratio greater than 30 mg/g followed by a full diagnostic evaluation. (http://www.kidney.org/professionals/KDOQI/guidelines, 2012.).

2.10Methods of diagnosis for CRF:

Renal Ultrasonography:

Renal ultrasonography is useful to screen for hydronephrosis, which may not be observed in early obstruction, or involvement of the retroperitoneum with fibrosis, tumor, or diffuse adenopathy. Small, echogenic kidneys are observed in advanced renal failure.

In contrast, kidneys usually are normal in size in advanced diabetic nephropathy, in which affected kidneys are initially enlarged from hyperfiltration. Structural abnormalities, such as those indicative of polycystic kidneys, also may be observed on ultrasonogram.

Others methods of diagnosis:

- **Radiography:**

A retrograde pyelogram may be indicated if a high index of clinical suspicion for obstruction exists despite a negative finding on renal ultrasonography. Intravenous pyelography is not commonly performed, because of the potential for renal toxicity from the intravenous contrast; however, this procedure is often used to diagnose renal stones. Plain abdominal radiography is particularly useful to look for radio-opaque stones or nephrocalcinosis, while a voiding cystourethrogram (VCUG) is the criterion standard for diagnosis of vesicoureteral reflux.

- **CT, MRI, and Radionuclide Scans:**

Computed tomography (CT) scanning can better define renal masses and cysts usually noted on ultrasonography. Also, CT scanning is the most sensitive test for

identifying renal stones. Intravenous (IV) contrast–enhanced CT scans should be avoided in patients with renal impairment to avoid acute renal failure; this risk significantly increases in patients with moderate to severe CRF. Dehydration also markedly increases this risk.

Magnetic resonance imaging (MRI) is very useful in patients who would otherwise undergo a CT scan but who cannot receive IV contrast. This imaging modality is reliable in the diagnosis of renal vein thrombosis, as are CT scanning and renal venography.

Magnetic resonance angiography (MRA) is becoming more useful for the diagnosis of renal artery stenosis, although renal arteriography remains the criterion standard. However, MRI contrast is problematic in patients with existing chronic kidney disease (CRF) because they have a low, but potentially fatal, risk of developing nephrogenic systemic fibrosis.

A renal radionuclide scan can be used to screen for renal artery stenosis when performed with captopril administration; it also quantitates differential renal contribution to total glomerular filtration rate (GFR). However, radionuclide scans are unreliable in patients with a GFR of less than 30 mL/min/1.73 m^2.

- **Renal Biopsy:**

Percutaneous renal biopsy is performed most often with ultrasonographic guidance and the use of a spring-loaded or other semi-automated needle. This procedure is generally indicated when renal impairment and/or proteinuria approaching the nephrotic range are present and the diagnosis is unclear after an appropriate workup.

Biopsies are also indicated to guide management in already-diagnosed conditions, such as lupus, in which the prognosis is highly dependent on the degree of kidney involvement. Biopsy is not usually indicated when renal ultrasonography reveals small, echogenic kidneys on ultrasonography, because this finding represents severe scarring and chronic, irreversible injury.

Renal histology in CRF reveals findings compatible with the underlying primary renal diagnosis. In some cases, a biopsy may show nonspecific changes, with the exact diagnosis remaining in doubt.

2.11Delaying or Halting Progression of Chronic Renal Failure:

Measures indicated to delay or halt the progression of chronic renal failure (CRF) are as follows:

- Aggressive blood pressure control to target values per current guidelines
- Treatment of hyperlipidemia to target levels per current guidelines
- Aggressive glycemic control per the American Diabetes Association (ADA) recommendations (target hemoglobin A1c [HbA1C] < 7%)
- Avoidance of nephrotoxins, including intravenous (IV) radiocontrast media, nonsteroidal anti-inflammatory agents (NSAIDs), and aminoglycosides
- Use of renin-angiotensin system (RAS) blockers among patients with diabetic kidney disease (DKD) and proteinuria
- Use of angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin-receptor blockers (ARBs) in patients with proteinuria.

2.12 previous studies:

Many studies were published to compare between ultrasound evaluation and laboratory findings worldwide. Some countries have already established their normal renal length and volume; some of these studies were conducted to provide reference values for renal size so as to differentiate between normal and abnormal Kidneys early and quickly by using ultrasound. Therefore, these data could be important to diagnose the problem in kidneys easy and to give the suitable treatment in a short time.

A recent study carried out by Zeb S et al. In their work entitles sonographic measurement of renal dimensions in adults; a survey to establish age and sex based diagnostic reference values in Pakistan. The study assessed the normal range of values for renal dimensions in a symptomatic adult population with various age

groups. The mean kidneys lengths were 9.7 cm on right side and 10cm on the left side. The mean width was 4.6 cm, cortical size was 1.46 cm with estimated average kidney volume was 135.7 cm3 **(**Zeb S et al**,** 2012**)**

A study by Brandt TD et al. (ultrasound assessment of renal dimensions) in (Chicago), confirms the accuracy and reliability of sonographic assessment of renal size. They showed that sonographic dimensions are smaller than those obtained radiology. With improve position, the mean renal length was found to be 10.7 cm in the right side and 11.1cm in the left side (Brandth, 1982).

Study by (Emamian et al.) was performed on 665 adult volunteers using renal sonography. It showed a median renal length 11.2 cm on the left side and 10.9cm on the right side. The median renal volume was 146 cm^3 on the left side and 136cm^3 in the right. Renal size was found to be decreased with age, increased due to parenchymal reduction. (Emamian,1993).

Glodny B et al in their study "normal Kidney size and its influencing factors" were applied normal ultrasound values for pole-to-pole kidney length. Cortical size was also recorded. The length was 10.8 cm for the right and 11.13 for the left kidney. The cortical size was same in the right and in the left (6.6mm) the most significant independent predictors for the length and cortical size were body size, Body mass index age and gender. (Glodny B, 2009).

Mujahid Raza et al. in their study "ultrasonographic assessment of renal size and volume and its correlation with BMI in adults" assessed renal size by ultrasound in 4.035 adult subjects without renal disease. The mean renal length on the right side was 10.16cm, width as 4.2 and thickness was 4.4 cm. on the left side the mean length was 10.27, width was 4.4 and thickness was 5.1cm. The mean renal volume on the right was 99.8 cm^3 and 124cm^3 on the left. The study concluded that the volume in the left side was significantly larger than the right in both gender. (Mujahid, 2011).

Adeela Arooj et al. in their study (comparison of renal size among different ethnicities) were used two dimensional ultrasound machine for one hundred university students to assess the renal size. Before, starting the exam, height and weight were taken. The image was taken in supine position. The mean length for the right and left were 9.7cm, and 9.9 cm respectively. The mean width was 5.6 cm and 6.09 for the right and left respectively (Adeela, 2011).

(Mohammed A. Ali Omer, et al) in Their study entitled " ultrasonographic characteristics of diabetes impact in kidneys' morphology " they found that the diabetes has direct impact in kidney morphology in view of renal volume enlargement and cortical thickening in early stage then atrophied and became echogenic in late stage. Also there is significant correlation between kidney size and the BMI and diabetic duration. (Mohammed A. Ali Omer, et al 2014)

(A.M. Abd Elgyoum, H. Osman et al) in their study " ultrasonography patterns for diabetic nephropathy according to the body shape " they found that renal changes in diabetic patients are detectable by conventional ultrasound only in very advanced stages of the disease.**(**A.M. Abd Elgyoum, H. Osman et al 2014).

(Alsafi Ahmed Abdella et al) in their study recorded "ultrasound finding of renal failure patients and creatinine Serum level relationship" the study revealed that kidneys volume decreases as the creatinine serum level increases**. (**Alsafi Ahmed Abdella et al, 2011)

(Gameraddin M, et al.) in their study enrolled "the sonographic estimation of renal length and determination of the main causes of chronic renal failure" the study confirmed that diabetes, hypertension and glomerulonephritis were the main causes of chronic renal failure which represented 91.9%, 83.8% and 94.6% respectively. **(**Gameraddin M, et al.2014).

(Mazin Abdulla et .al) in their study entitled " Establishment of Reference Values for Renal Length and Volume for Normal Adult Sudanese using MRI Disc Summation Method" The study showed that the kidneys 'length measured for normal Sudanese subjects were 10.08±0.46, 10.67±0.47 for right and left,

respectively. The mean volumes were 101.6 cm^3 and 104 cm^3 for right and left kidney, respectively. (Mazin Abdulla et .al. 2014)

(Abubakr. A. Sanusi et al.**),** in their study " recorded relationship of ultrasonographically determined kidney volume with measured GFR, calculated creatinine clearance and other parameters in chronic kidney disease" the results showed a weak but positive correlation between kidney volume (KV) and various indices of GFR (Abubakr. A. Sanusi et al. 2009).

(Jagdeesh K. et al) in their study entitled " Correlation of ultrasonographic parameters with serum creatinine in Chronic Kidney Disease revealed" it revealed that renal echogenicity and its grading correlates better with serum creatinine in established CRF than other sonographic parameters such as longitudinal size, parenchymal thickness, and cortical thickness. Hence, renal echogenicity is a better parameter than serum creatinine for estimating renal function in CRF, and has the added advantage of irreversibility. (Jagdeesh K. et al,2013)

(Beland MD, et al), in their study entitled "renal cortical thickness measured at ultrasound: is it better than renal length as an indicator of renal Function in chronic kidney disease?" they found that cortical thickness measured on ultrasound appears to be more closely related to eGFR than renal length. (Beland MD, et al 2010),

(Tangri et al,2012), developed and validated a model in adult patients that uses routine laboratory results to predict progression from CKD (stages 3-5) to kidney failure They reported that lower estimated glomerular filtration rate (GFR), higher albuminuria, younger age, and male sex pointed to a faster progression of kidney failure.

In patients with chronic renal failure, the renal cortical echogenicity increases at ultrasound (Khati NJ, Hill MC, Kimmel PL, the essential ultrasound 2005). In addition, the renal cortex often becomes thinned (Morghazi S, Jones E. et al. Kidney int, 2005).

Chapter three

Materials and methods

3.1 design of the study:

It is an analytical study of case control type in which abdominal US examination and lab investigations were done for hypertensive, diabetic and normal population.

3.2 population of the study:

The target population for this research defined to include Sudanese hypertensive and diabetic patients and normal population. Diabetic and hypertensive patients of duration less than (5) years were excluded in order to give an opportunity for renal changes in size and parenchyma to appear.

3.3 study sample and type:

The study was a prospective study in which a group of (201) diabetic and hypertensive patients were drown for renal US examination and lab investigations (serum creatinine, BUN and eGFR). Another group of (100) healthy volunteers were selected as a control group and gray scale US procedure was done for them in order to establish some preliminary data of the population.

3.4 Area and duration of the study:

The study was carried out in Khartoum state at Fedail Specialized Hospital in Khartoum city, Al-dosougi Specialized Hospital and Salamat Medical Center in Omdurman city. The study started in July 2012 and finished in February, 2016.

3.5 Equipment used in the study:

Three US machines were used in the study; the first one is (logio p5) - General electric (GE) company, USA- and the second and third ones were Diggi Prince 6600 and UMT 150 - Mindray Company, china. The probe which was used was curve linear multihertzs probe. An US gel was used and it was put at the top of the transducer to avoid reflection of ultrasound and to maintain a good transmission of US beam inside the body figure (3.3).

3.5.2 Other equipment:

Standiometer was used for height measurement graduated in centimeter. Salter scale was used for purpose of weight measurement and it was graduated in kilogram.

3.6 Technique and protocol of the study

3.6.1: Anthropometric measurement:

Height was measured for each patient to the nearest 0.1 cm using a height measuring board (Standiometer). The head position in horizontal plane heals together and arms hang free .weight was also measured using Salter's scale graduated in kilograms.

3.6.2. Abdominal US examination:

For the purpose of decreasing gas in the abdomen, the patient fasted overnight (6 hours) before US examination. The examination began with subject supine. The para-aortic region was examined to exclude the presence of horse show kidneys. Length, width, depth and cortical thickness of the kidneys were measured. The longitudinal dimensions of the kidneys were measured in a section visually estimated to represent the longest longitudinal section. Both lower and upper poles were defined. If the long axis of the kidney cannot be obtained with the patient supine, Coronal or Sagittal view with the patient in decubitus position should be obtained. The patient was asked to elevate the ipsilateral arm above the head and take a deep breath and hold it to have a good view for both poles. Supero –inferior (pole to pole) measurement was taken in that view, figure (3.5) then the width and depth were measured in a section perpendicular to the long axis of the kidney as assessed from the longitudinal image. The probe was thus not necessary perpendicular to the skin. The transverse section was intended to be placed quite close to the hilum of the kidney but at the same time free of pelvis figure (3.6). Width and depth were then measured in two orthogonal directions; renal volume was estimated from the three orthogonal measurements on the base of ellipsoid formula. The cortical thickness was also measured for each kidney in centimeters.

The echogenicity of the cortex for each kidney was compared with the liver in the right side and spleen in the left side to detect cortico-medullary differentiation.

Then laboratory investigations (serum creatinine and BUN) were taken. Estimated glomerular filtration rate (eGFR) was calculated using (Cockcroft & Gault) formula.

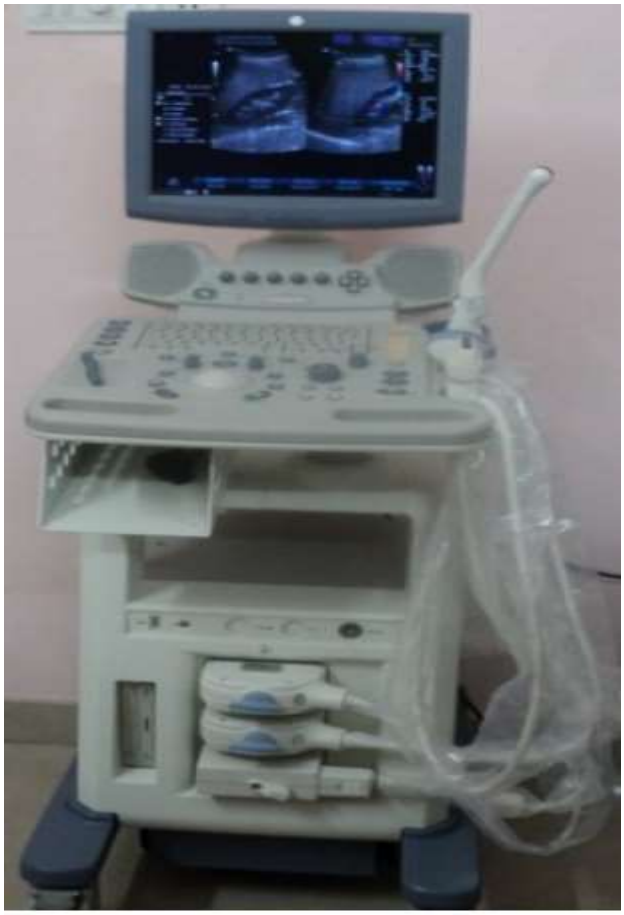

Figure (3.1) Ultrasound machine (www.providianmedical.com)

Figure (3.2) Ultrasound transducer 3.5MHz sample

Figure (3.3) Ultrasound gel

Figure (3.4) Standiometer (quickmedical.com)

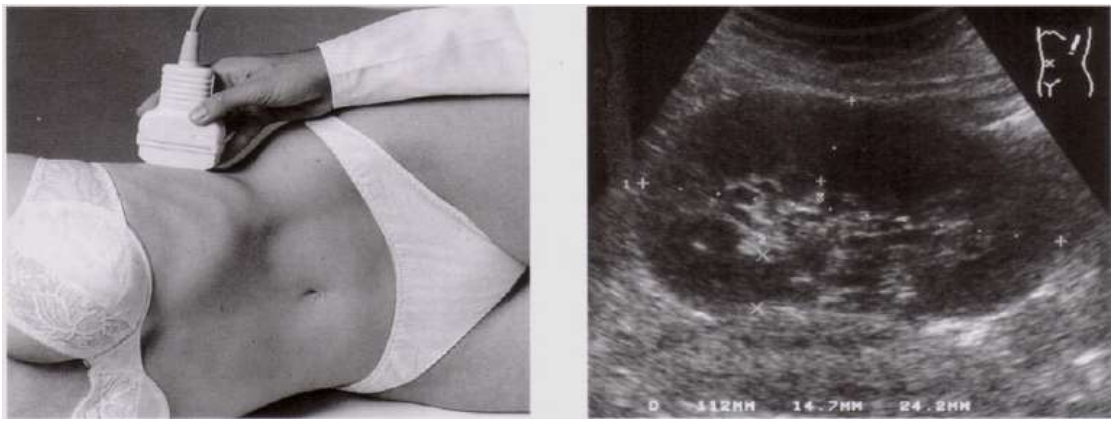

Figure (3.5) (pole to pole) measurement of the left kidney

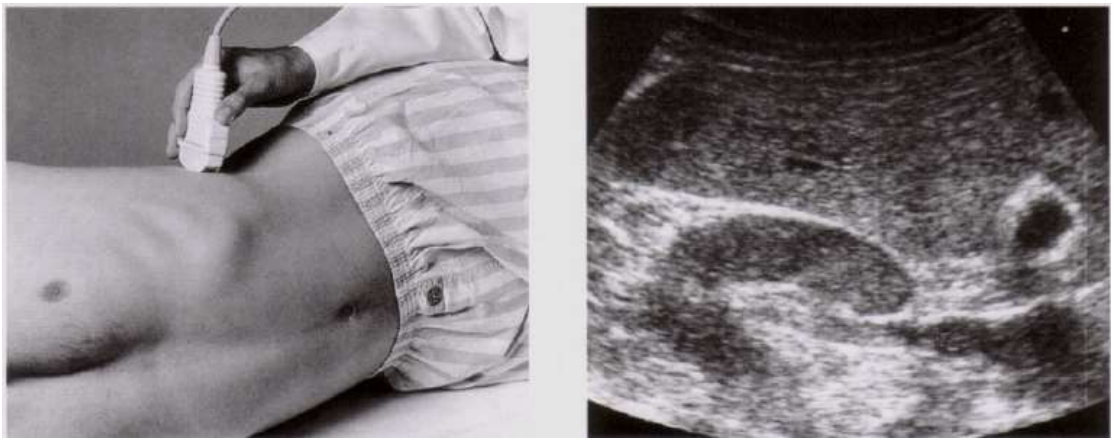

Figure (3.6) Normal left t Kidney. Transverse at the mid-kidney level.
The renal cortex is less echogenic than the adjacent normal liver
(Matthias Hofer.1998 3rd edition**)**

Chapter four

Results

Section one: patient data (general information)

Table (4.1) Distribution of frequency among (DM, HTN and DM-HTN)

Group	Frequency	Percentage
DM	59	29.4
HTN	41	20.4
DM-HTN	101	50.2
Total	201	100.0

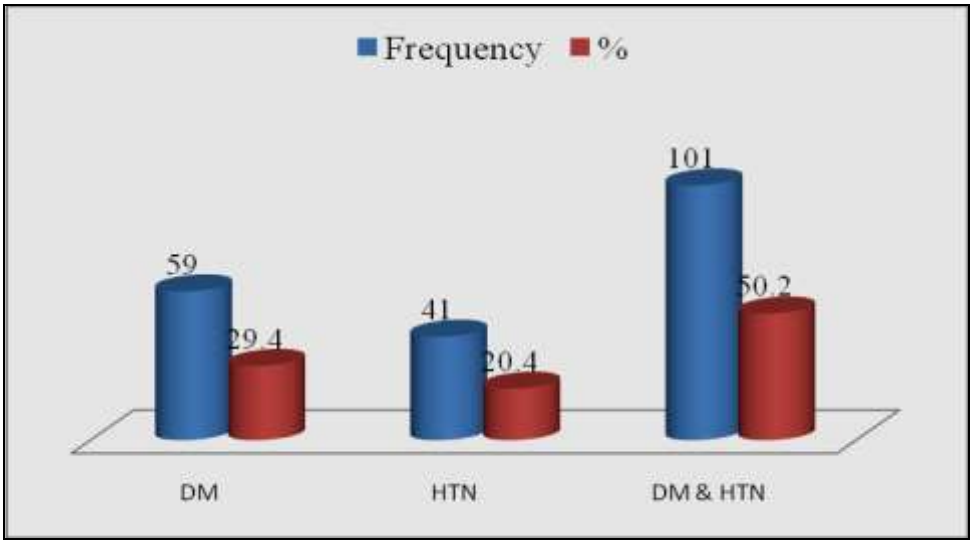

Figure (4.1) bar graph show the distribution of frequency among the **(**DM, HTN and DM-HTN)

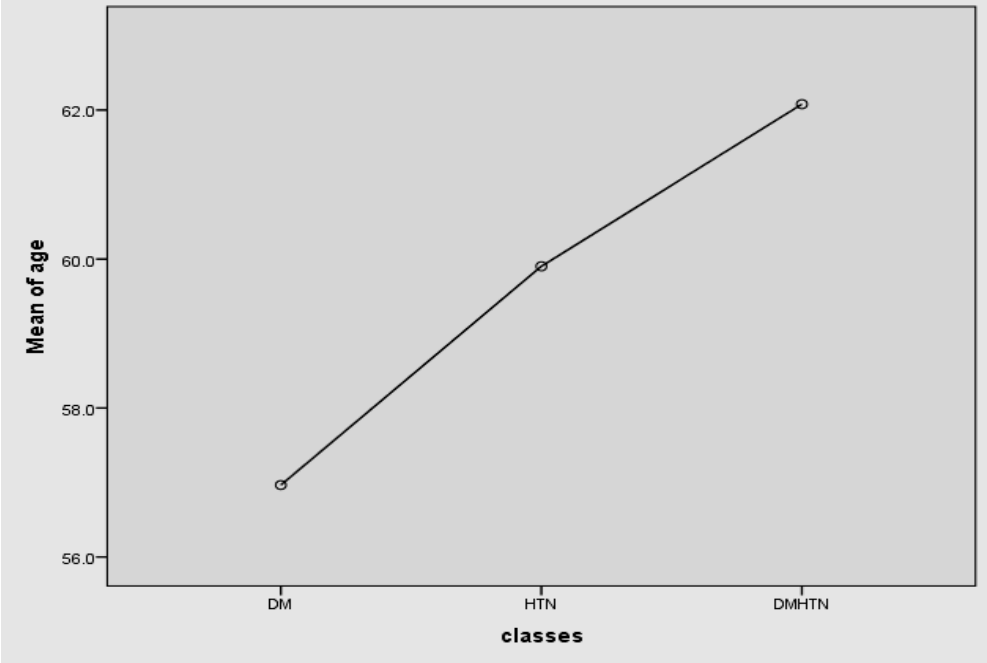

Figure (4.2) Line graph show mean of age for DM, HTN and DM-HTN

Table (4.2) cross-tabulation table of gender distribution among (DM, HTN and DM-HTN)

Gender	Classes			Total
	DM	HTN	DM-HTN	
Male	26	16	30	72
Female	33	25	71	129
Total	59	41	101	201

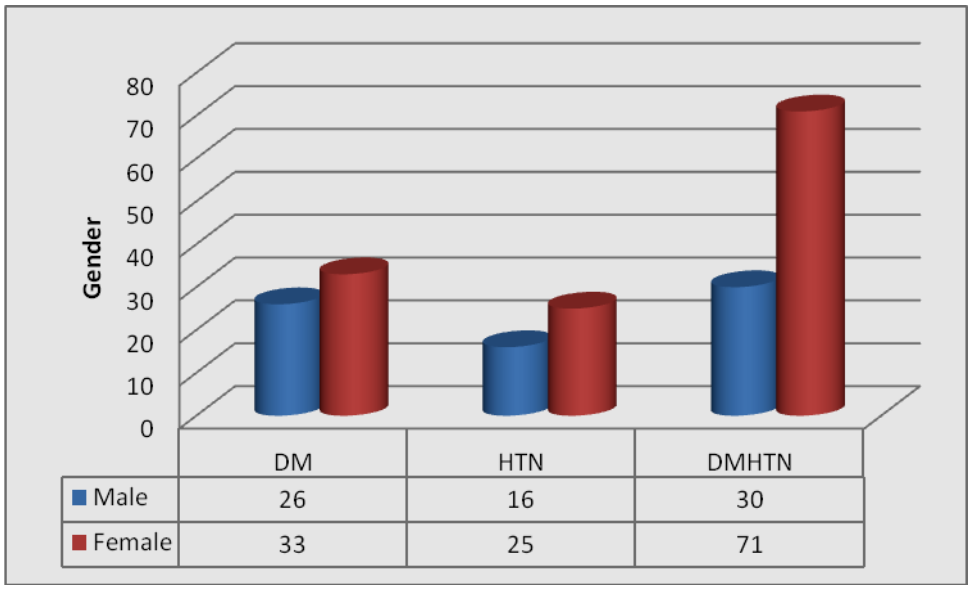

Figure (4.3) bar graph show the distribution of gender among the **(**DM, HTN and DM-HTN)

Table (4.3) cross-tabulation table of occupation distribution among (DM, HTN and DM-HTN)

Classes	Occupation					Total
	Housewife	Teacher	Employee	worker	Others	
DM	30	5	7	12	5	59
HTN	24	3	5	5	4	41
DMHTN	61	9	7	11	13	101
Total	115	17	19	28	22	201

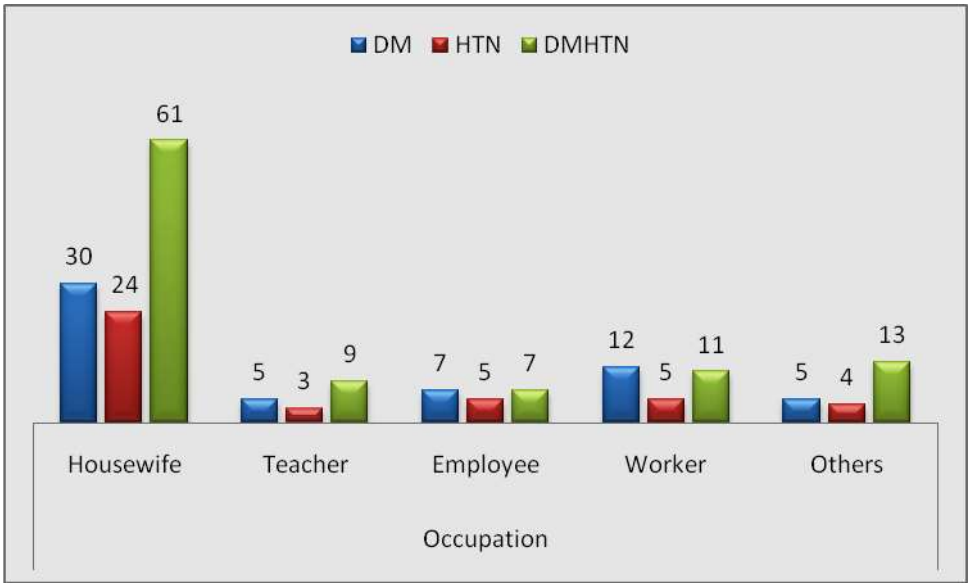

Figure (4.4) bar graph show the distribution of occupation among **(**DM, HTN and DM-HTN)

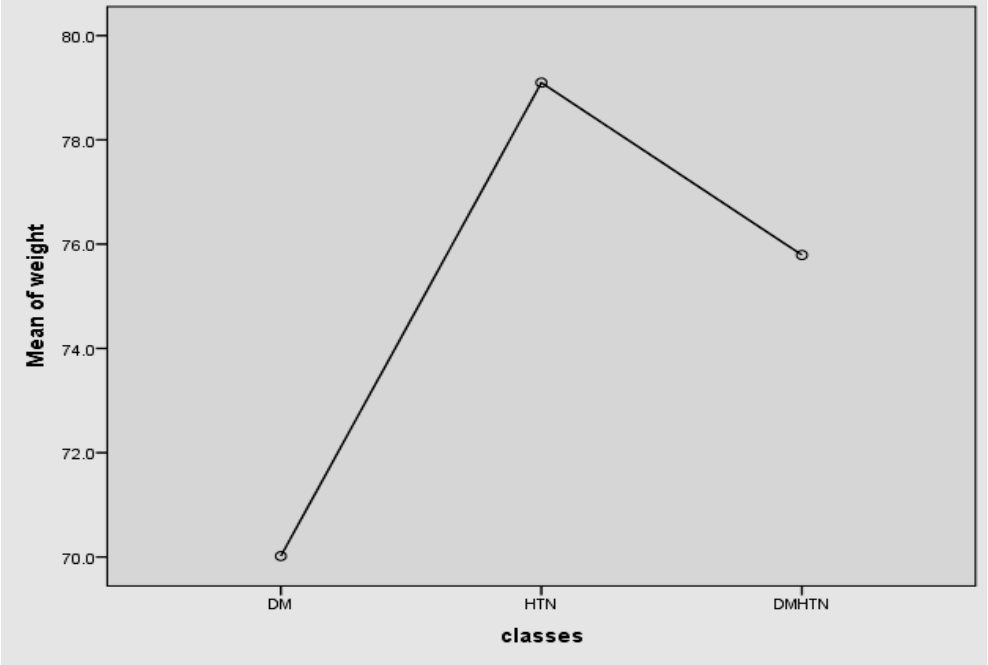

Figure (4.5) Line graph show mean of weight for DM, HTN and DM-HTN

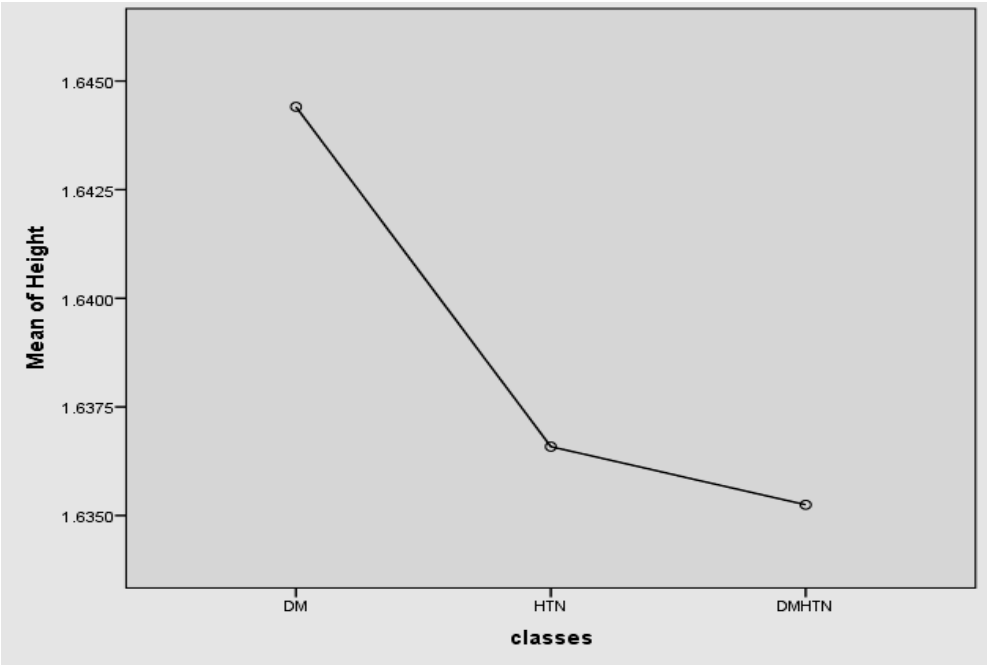

Figure (4.6) Line graph show mean of height for DM, HTN and DM-HTN

Table (4.4) distribution of family history among DM, HTN and DM-HTN

Family history	Frequency	Percentage (%)
With no family history	12	6
With family history	189	94

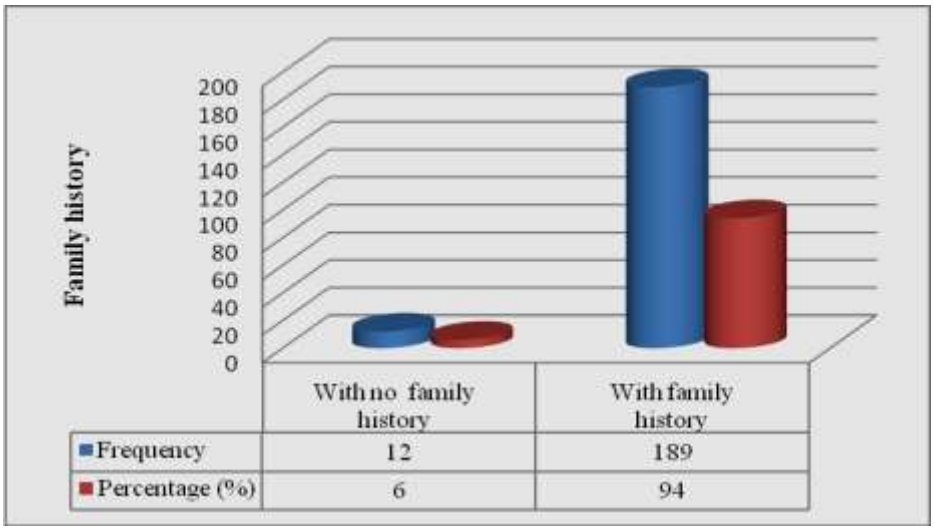

Figure (4.7) bar graph show the distribution of family history among **(**DM, HTN and DM-HTN)

Table (4.5) cross-tabulation table of others distribution among (DM, HTN and DM, HTN)

Classes	Others				Total
	No	IBS	Renal cyst	Hydronephrosis	
DM	49	8	2	0	59
HTN	33	3	4	1	41
DMHTN	80	12	8	1	101
Total	162	23	14	2	201

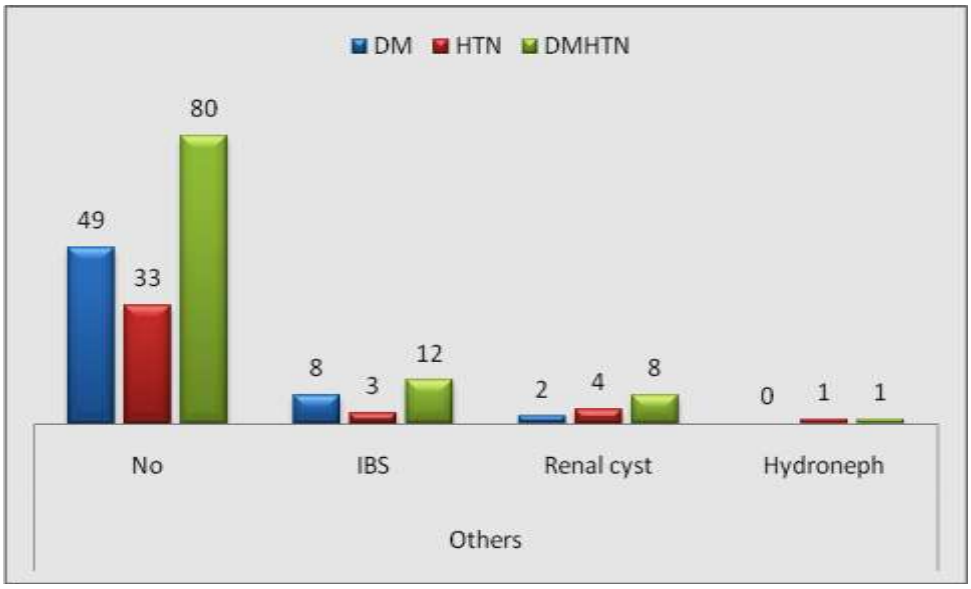

Figure (4.8) bar graph show the distribution of others among the (DM, HTN and DM-HTN)

Section two: sonographic evaluation

Table (4.6) the means values of the measured variables for the right kidney for (DM, HTN, DM-HTN and normal)

Right kidney	RR L	RR W	RR D	RR V	Rt_C_thickness
Normal	10.214	4.945	3.786	101.224	1.474
DM	10.107	4.558	4.098	100.320	1.527
HTN	9.876	4.400	3.878	88.641	1.429
DM-HTN	10.209	4.491	4.016	98.281	1.502

Table (4.7) the means values of the measured variables for the left kidney for (DM, HTN, DM- HTN and normal)

Left kidney	LR L	LR W	LR D	LR V	Lt_C_thickness
Normal	10.389	4.882	4.372	117.97	1.742
DM	10.476	4.549	4.514	114.441	1.729
HTN	10.029	4.346	4.383	100.856	1.637
DM-HTN	10.434	4.590	4.668	117.642	1.748

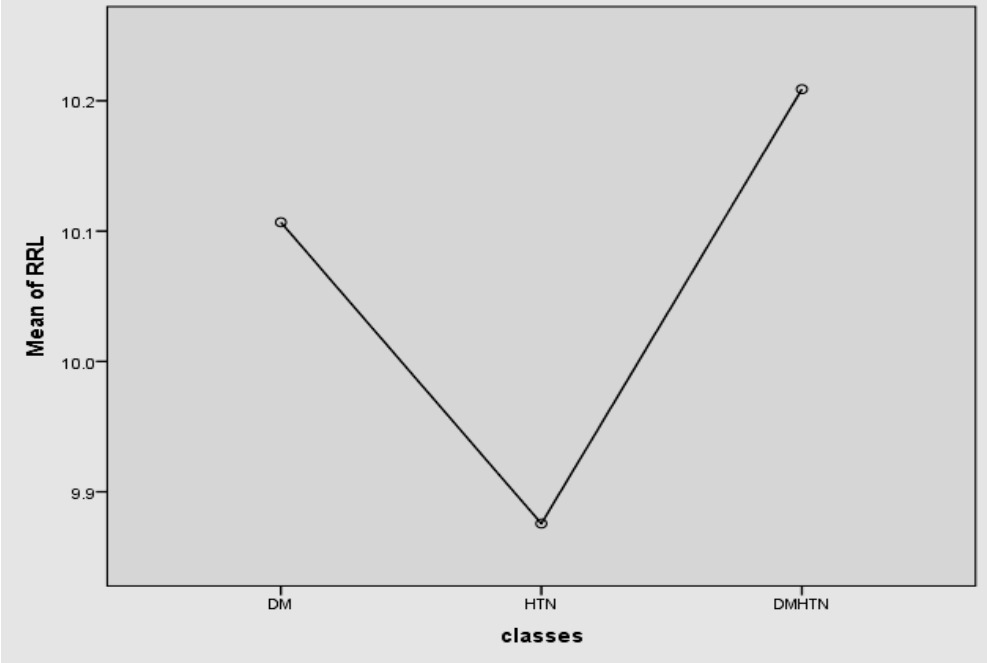

Figure (4.9) Line graph show mean of right renal length for DM, HTN&DM-HTN

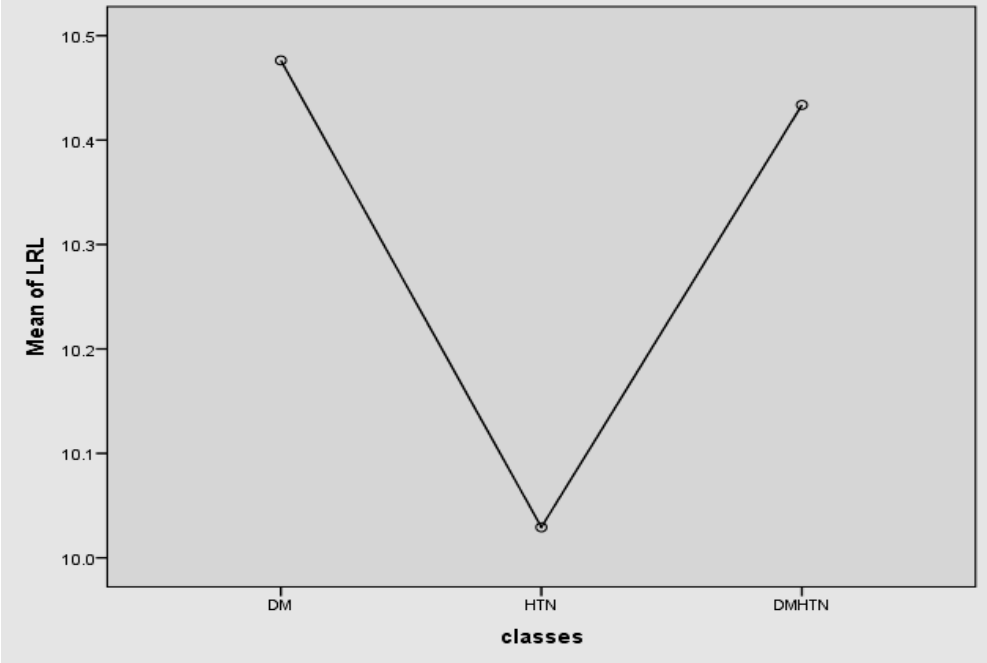

Figure (4.10) Line graph show mean of left renal length for DM, HTN&DM-HTN

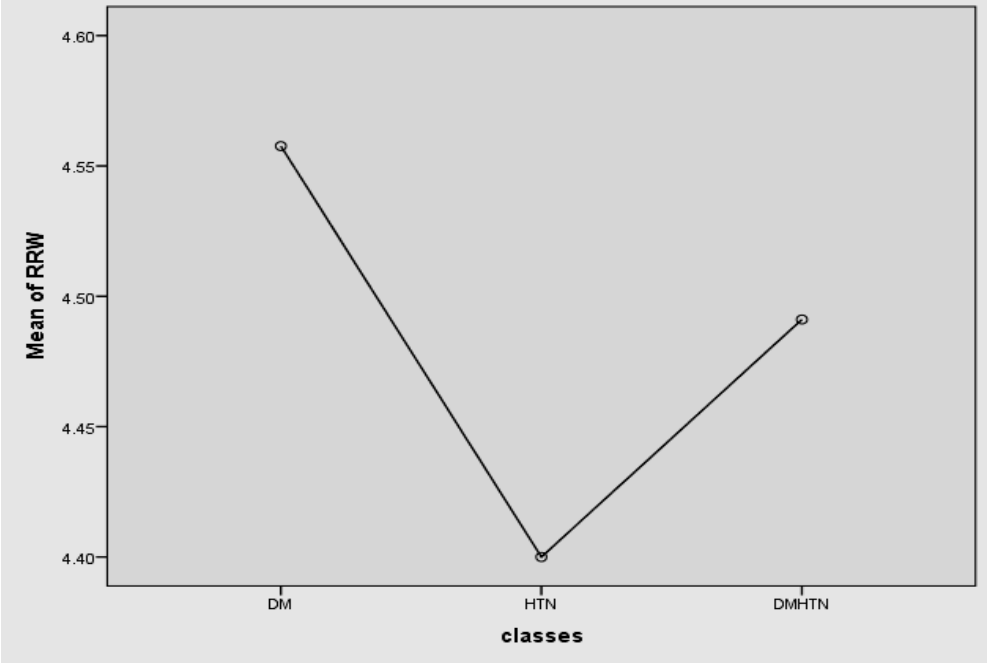

Figure (4.11) Line graph show mean of right renal width for DM, HTN&DM-HTN

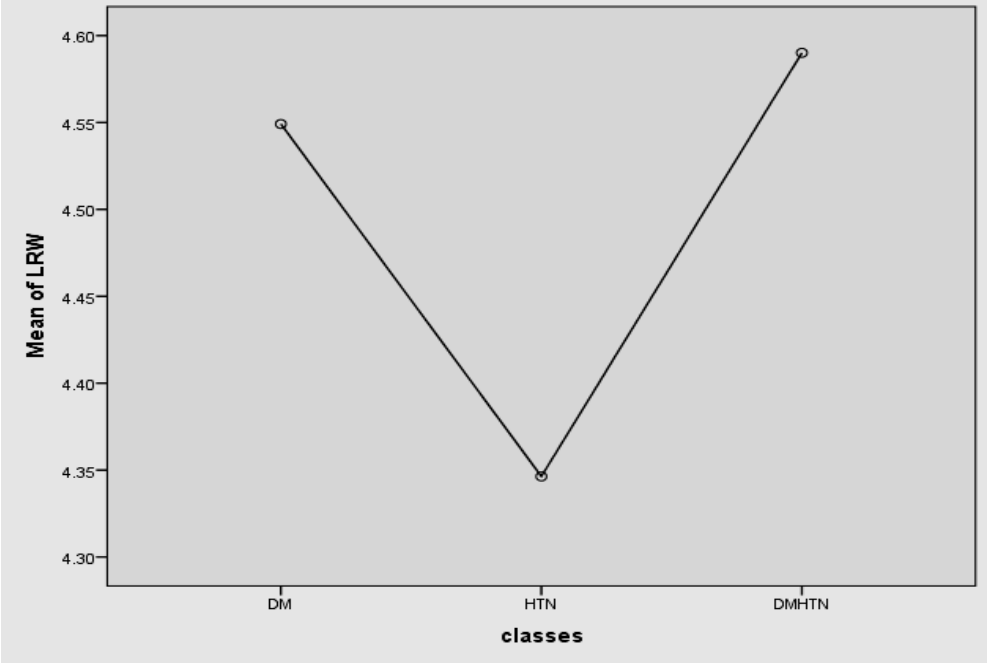

Figure (4.12) Line graph show mean of left renal width for DM, HTN and DM-HTN

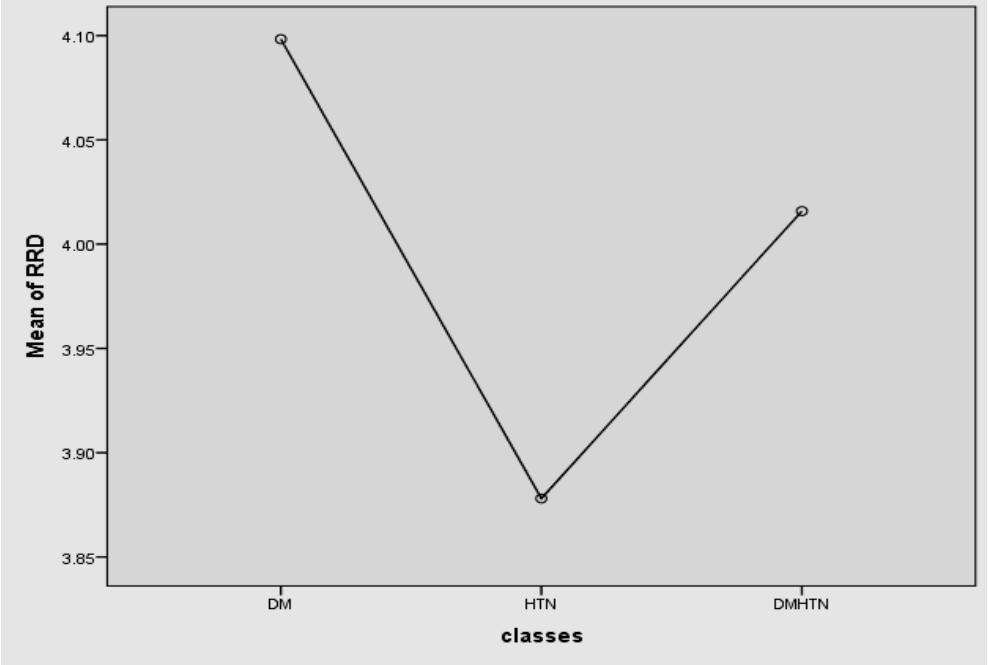

Figure (4.13) Line graph show mean of right renal depth for DM, HTN & DM-HTN

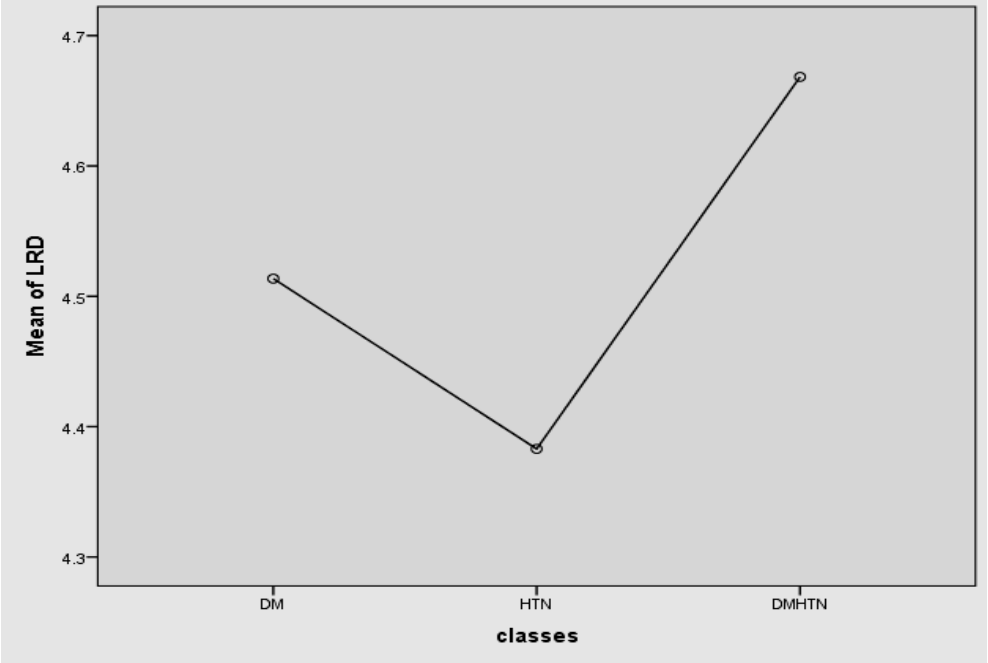

Figure (4.14) Line graph show mean of left renal depth for DM, HTN &DM-HTN

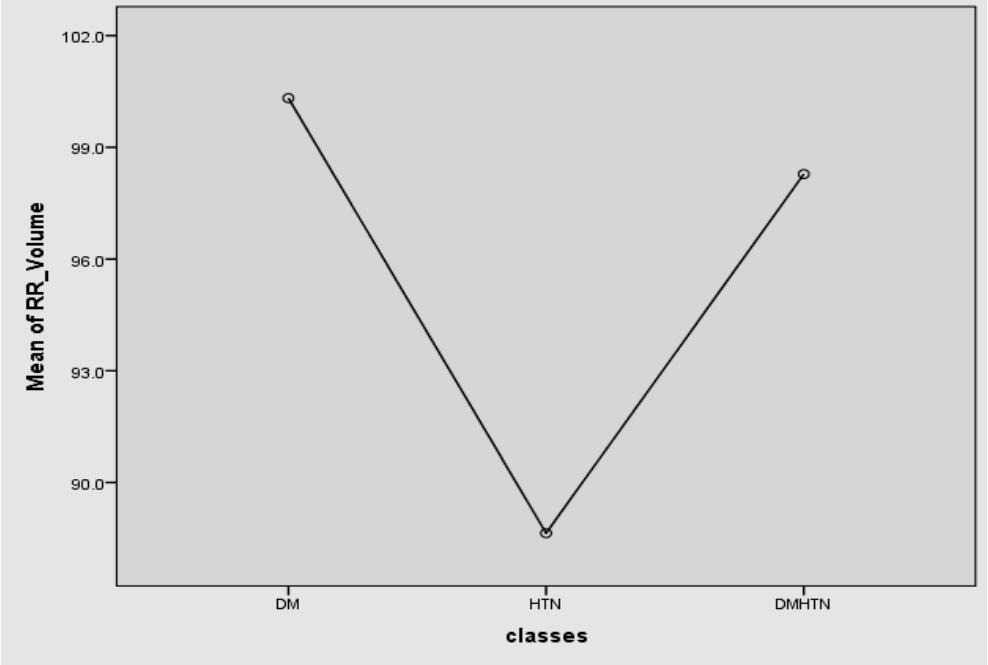

Figure (4.15) Line graph show mean of right renal volume for DM, HTN, DM-HTN

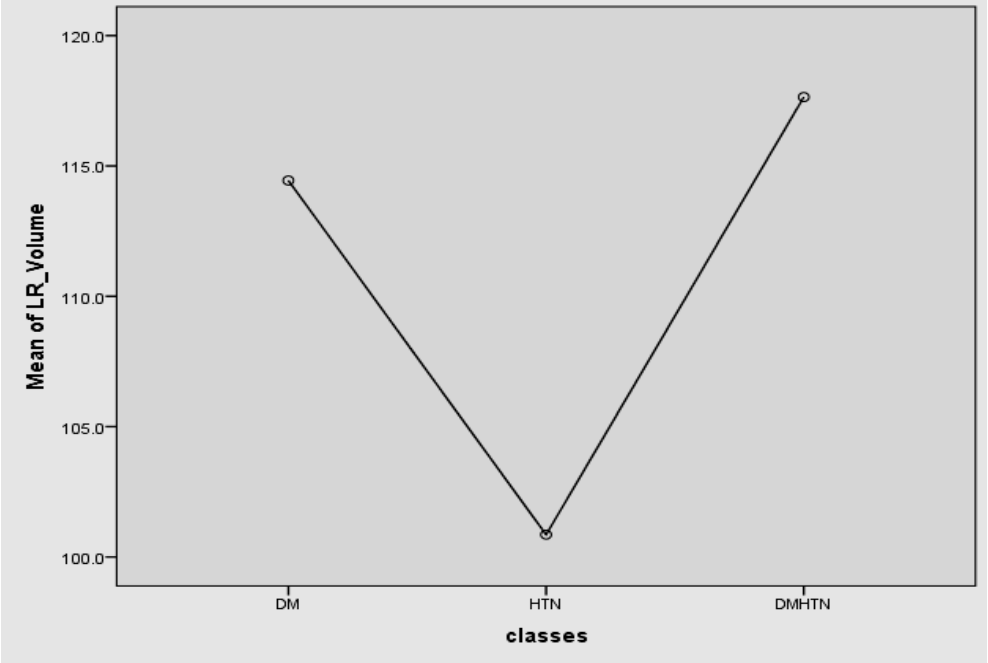

Figure (4.16) Line graph show mean of left renal volume for DM, HTN&DM-HTN

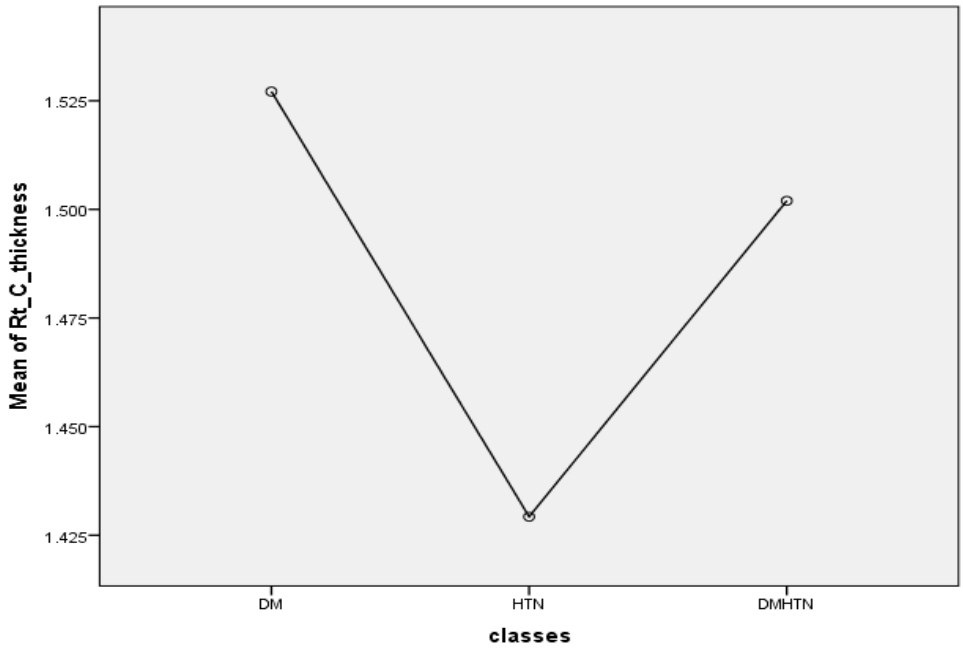

Figure (4.17) Line graph show mean of right cortical thickness for DM, HTN and DM-HTN

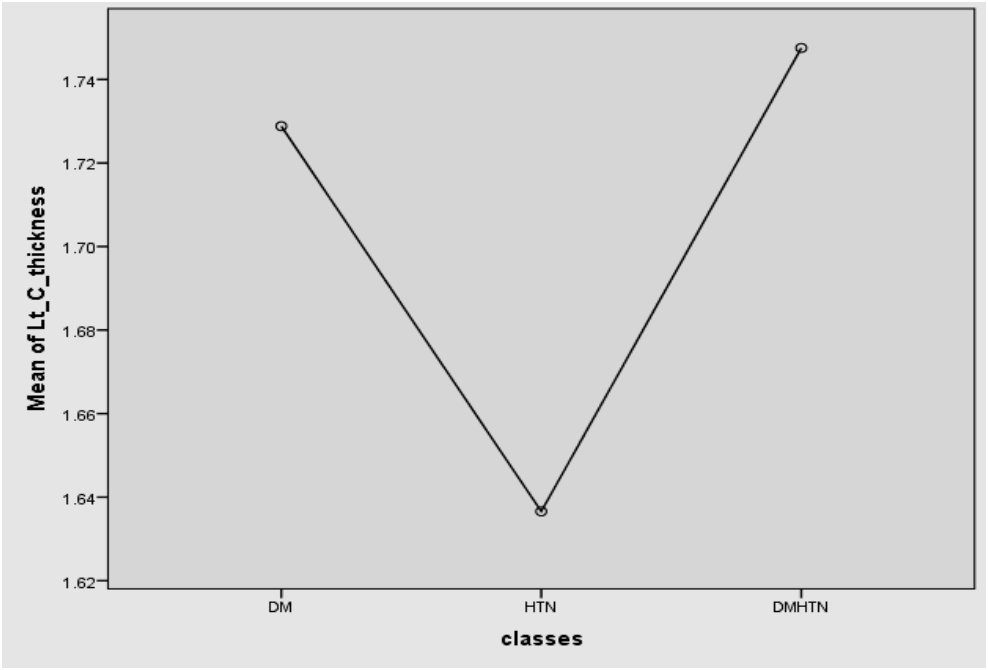

Figure (4.18) Line graph show mean of left cortical thickness for DM, HTN and DM-HTN

Table (4.8) cross-tabulation table of right cortico-medullary differentiation (Rt_CMD) among (DM, HTN and DM-HTN)

Classes	Rt_CMD		Total
	Normal	Mild loss	
DM	58	1	59
HTN	39	2	41
DMHTN	96	5	101
Total	193	8	201

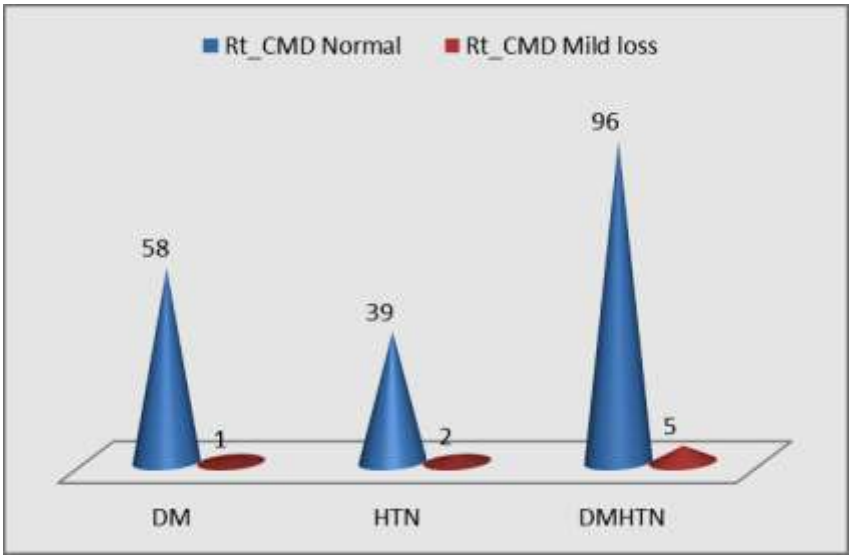

Figure (4.19) bar graph show the distribution of right cortico-medullary differentiation among (DM, HTN, DM-HTN)

Table (4.9) cross-tabulation table of left cortico-medullary differentiation (Lt_CMD) among (DM, HTN and DM-HTN)

Classes	Lt_CMD		Total
	Normal	Mild loss	
DM	58	1	59
HTN	38	3	41
DMHTN	98	3	101
Total	194	7	201

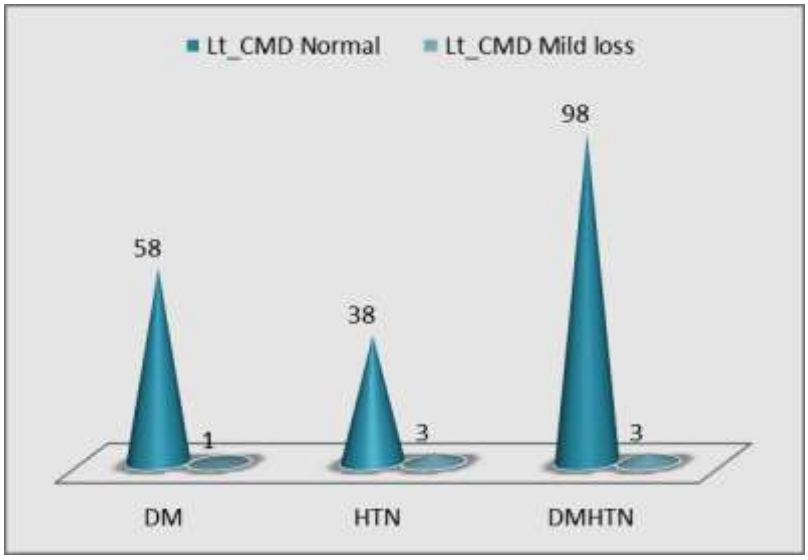

Figure (4.20) bar graph show the distribution of left cortico-medullary differentiation among (DM, HTN and DM-HTN)

Table (4.10) cross-tabulation table of right cortical echogenicity (Rt_C_Echo) among (DM, HTN and DM-HTN)

Classes	Rt_C_Echo		Total
	Normal	Increased	
DM	58	1	59
HTN	39	2	41
DMHTN	96	5	101
Total	193	8	201

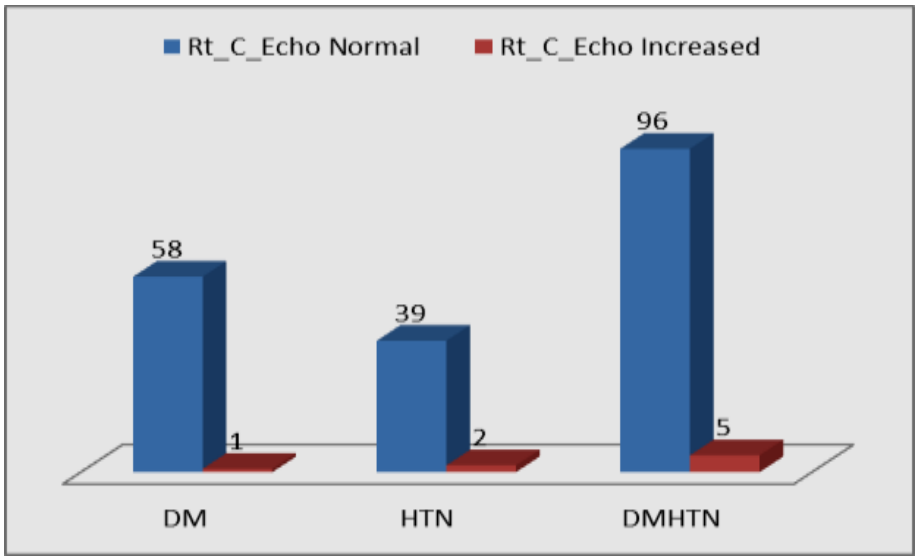

Figure (4.21) bar graph show the distribution of right cortical echogenicity among (DM, HTN and DM-HTN)

Table (4.11) cross-tabulation table of left cortical echogenicity (Lt_C_Echo) among (DM, HTN and DM-HTN)

Classes	Lt_C_Echo		Total
	Normal	Increased	
DM	57	2	59
HTN	38	3	41
DMHTN	98	3	101
Total	193	8	201

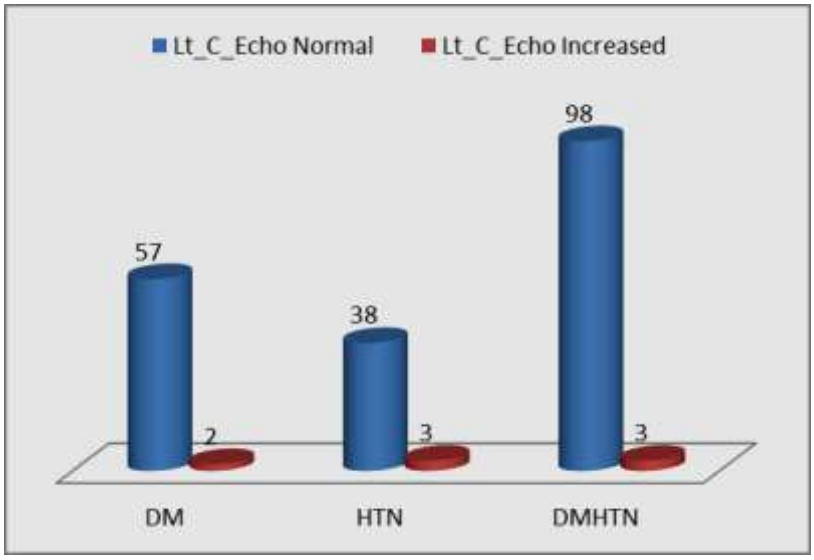

Figure (4.22) bar graph show the distribution of left cortical echogenicity among (DM, HTN and DM-HTN)

Section three: laboratory findings

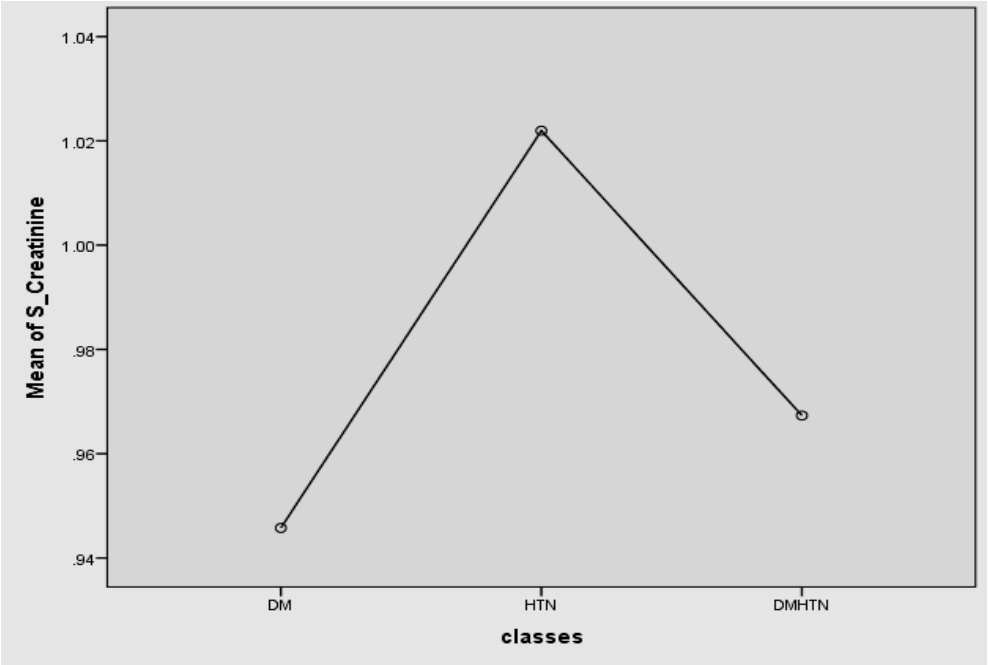

Figure (4.23) Line graph show mean of serum creatinine for DM, HTN& DM-HTN

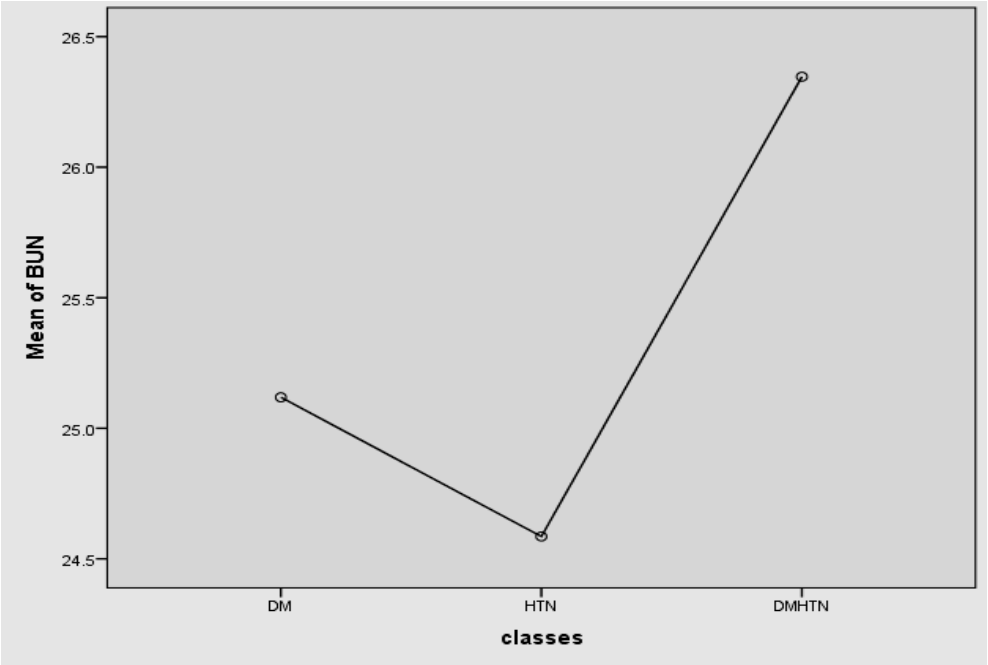

Figure (4.24) Line graph show mean of BUN for DM, HTN and DM-HTN

Table (4.12) eGFR levels and renal function stages for the whole cases

eGFR levels		Frequency	Percent %
Normal	(`>90)	67	33.3
Mild reduction	(60-89)	93	46.3
Moderate reduction	(30-59)	39	19.4
Severe reduction	(15-29)	2	1.0

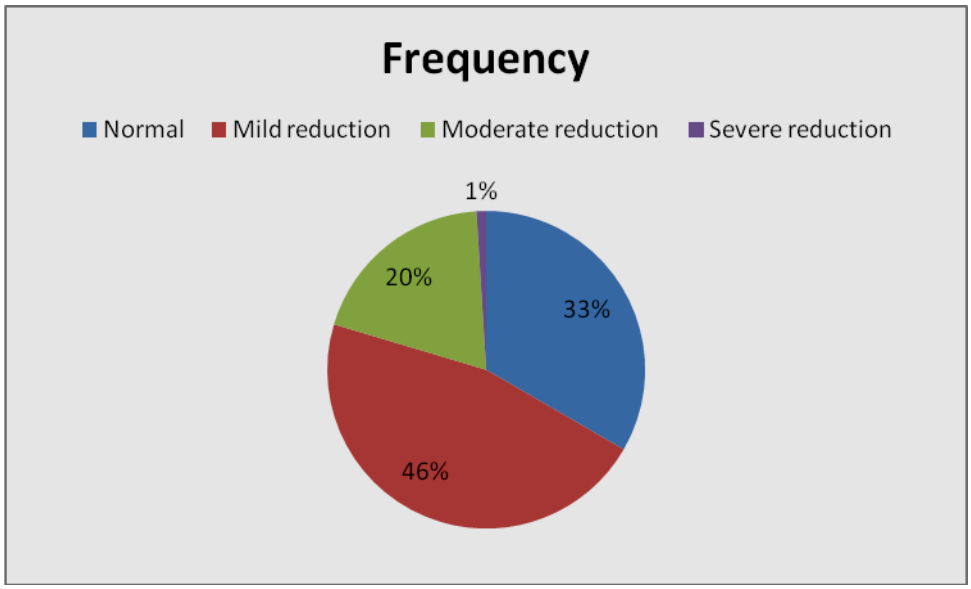

Figure (4.25) bar graph shows the distribution of eGFR levels through the whole cases

Table (4.13) eGFR levels and renal function stages for (DM, HTN, and DM-HTN)

eGFR level	DM	HTN	DM-HTN	Total
Normal (`>90)	16	16	35	67
Mild reduction (60-89)	33	18	42	93
Moderate reduction (30-59)	9	6	24	39
Severe reduction (15-29)	1	1	0	2
Total	59	41	101	201

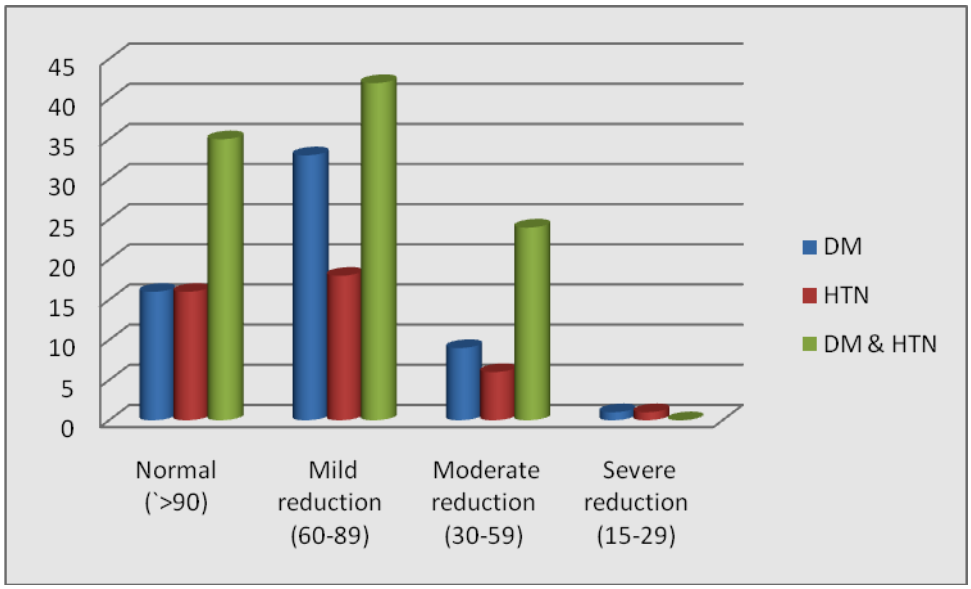

Figure (4.26) bar graph shows the distribution of eGFR levels among (DM, HTN, and DM-HTN)

Table (4.14) one way Anova - test table for the difference in means between DM & normal concerning measured variables

DM and Normal			
Measurement	Classes	Mean	Std. Deviation
Rt_C_thickness	DM	1.527	.2935
	Normal	1.474	.3368
Lt_C_thickness	DM	1.729	.3538
	Normal	1.742	.3715
RRL	DM	10.107	1.2795
	Normal	10.214	.7716
LRL	DM	10.476	1.0575
	Normal	10.389	.8427
RRW	DM	4.558	.5899
	Normal	4.945	.6328
LRW	DM	4.549	.5597
	Normal	4.882	.6908
RRD	DM	4.098	.5161
	Normal	3.786	.5244
LRD	DM	4.514	.5594
	Normal	4.372	.6020
RR_Volume	DM	100.320	29.8570
	Normal	101.224	25.8012
LR_Volume	DM	114.441	28.2872
	Normal	117.097	29.6686

Table (4.15) t- test table for the difference in mean between DM & normal concerning measured variables

	t- test for Equality of Means	
DM &Normal	T	Sig. (2 tailed)
Rt_C_thickness	1.01	0.316
Lt_C_thickness	0.22	0.826
RRL	0.66	0.510
LRL	0.57	0.567
RRW	3.82	**0.000**
LRW	3.14	**0.002**
RRD	3.65	**0.000**
LRD	1.47	0.144
RR_Volume	0.20	0.841
LR_Volume	0.55	0.580

Table (4.16) one way Anova - test table for the difference in mean between HTN & normal concerning measured variables

HTN and Normal			
Measurement	Classes	Mean	Std. Deviation
Rt_C_thickness	HTN	1.429	.2629
	Normal	1.474	.3368
Lt_C_thickness	HTN	1.637	.3192
	Normal	1.742	.3715
RRL	HTN	9.876	.7446
	Normal	10.214	.7716
LRL	HTN	10.029	.9642
	Normal	10.389	.8427
RRW	HTN	4.400	.4370
	Normal	4.945	.6328
LRW	HTN	4.346	.5065
	Normal	4.882	.6908
RRD	HTN	3.878	.5672
	Normal	3.786	.5244
LRD	HTN	4.383	.5937
	Normal	4.372	.6020
RR_Volume	HTN	88.641	20.7662
	Normal	101.224	25.8012
LR_Volume	HTN	100.856	26.5461
	Normal	117.097	29.6686

Table (4.17) t-test table for the difference in mean between HTN & normal concerning measured variables

Independent Samples Test		
	t test for Equality of Means	
HTN & Normal	T	Sig. (2 tailed)
Rt_C_thickness	0.76	0.448
Lt_C_thickness	1.59	0.114
RRL	2.39	**0.018**
LRL	2.21	**0.029**
RRW	5.04	**0.000**
LRW	4.49	**0.000**
RRD	0.92	0.357
LRD	0.10	0.922
RR_Volume	2.77	**0.006**
LR_Volume	3.04	**0.003**

Table (4.18) one way Anova - test for the difference in mean between (DM-HTN) and normal concerning measured variables

DM-HTN and Normal			
Measurements	Classes	Mean	Std. Deviation
Rt_C_thickness	DMHTN	1.502	.3426
	Normal	1.474	.3368
Lt_C_thickness	DMHTN	1.748	.5478
	Normal	1.742	.3715
RRL	DMHTN	10.209	1.0187
	Normal	10.214	.7716
LRL	DMHTN	10.434	1.0405
	Normal	10.389	.8427
RRW	DMHTN	4.491	.5171
	Normal	4.945	.6328
LRW	DMHTN	4.590	.5801
	Normal	4.882	.6908
RRD	DMHTN	4.016	.6302
	Normal	3.786	.5244
LRD	DMHTN	4.668	.6372
	Normal	4.372	.6020
RR_Volume	DMHTN	98.281	29.7306
	Normal	101.224	25.8012
LR_Volume	DMHTN	117.642	34.7830
	Normal	117.097	29.6686

Table (4.19) t-test table for the difference in mean between (DM-HTN) and normal concerning measured variable

Independent Samples Test		
	t test for Equality of Means	
DM-HTN & Normal	T	Sig. (2 tailed)
Rt_C_thickness	0.58	0.560
Lt_C_thickness	0.08	0.933
RRL	0.04	0.968
LRL	0.33	0.739
RRW	5.57	**0.000**
LRW	3.25	**0.001**
RRD	2.81	**0.005**
LRD	3.39	**0.001**
RR_Volume	0.75	0.455
LR_Volume	0.12	0.905

Chapter five
Discussion, Conclusion and recommendations

5.1 Discussion

Diabetes mellitus and hypertension may lead to renal changes in morphology and function, and chronic renal failure is identified as a progressive loss in renal function over a period of months or years. Often, chronic renal failure is diagnosed as a result of screening of people known to be at risk of kidney problems, such as those with high blood pressure or diabetes. (Sarnak and Levey, 2000)

This study was conducted to detect early renal changes in size and parenchyma, in diabetic and hypertensive patients in Sudan using ultrasound and laboratory findings.

Three hundred and one participants were included in this study; they were divided into four groups; 59 (29.4%) diabetic (DM), 41(20.4%) hypertensive (HTN), 101(50.2%), diabetic with hypertensive (DM-HTN) and 100 control (normal) group, table (4.1). The overall average age was 60.134, range from 26 to 80 years old. The oldest age noted in (DM-HTN) and the youngest in diabetic group (DM); which dictates that, diabetes mellitus approximately affect Sudanese people earlier before hypertension. The study included 72 males (35.8%) and 129 females (64.2%), table (4.2).The participant's occupations included: housewives, teachers, employees, workers and others; the most affected were housewives 115 (57.2%) and less affective were teachers, 17 (8.4%) table (4.3). This study showed that diabetic patients were had lighter weight (weight loss) and hypertensive patients were the fattest (obese) as illustrated in Figure (4.5); and that might be due to resistance to insulin in diabetic patients (prevent glucose to enter cells). The study also revealed that diabetic patients (DM) were the tallest (long in height) and diabetic-hypertensive patients (DM-HTN) were the shortest; as illustrated in Figure (4.6). This study showed that most of the participants have family history of diabetes and hypertension (94%) as shown in table (4.4); that means diabetes and hypertension are inherited diseases in Sudan.

Decrease of renal size and function are seen with many diseases such as, renal artery stenosis, late stage of renal vein thrombosis and chronic renal failure as revealed in study carried out by (Alsafi. et al,2011), the Kidneys volumes decreases as the creatinine serum level increases in Sudanese population . On the other hand there is increment in renal size in early stage of diabetes mellitus and renal inflammation (Abd Elgyoum et al. 2014) in Sudan. The number of nephrons in a kidney correlates with the physical dimensions and size of the organ. The mean lengths of kidneys were (10.1, 9.8, and 10.2 cm) for the right and were (10.4, 10.0 and 10.4 cm) for the left for DM, HTN and (DM-HTN) in tables (4.6) and (4.7) respectively. The longest right renal length noted in (DM-HTN) group and the shortest one in hypertensive group as illustrated in figure (4.9); while the longest dimension in the left kidney noted in diabetic group (DM) as illustrated in figure (4.10). There were significant differences between right and left renal lengths for the normal and hypertensive group (HTN) at P=0.05 using t-test with T= (2.39) and P= (0.018) for the right and with (T=2.21) and P= (0.029) for the left as illustrated in table (4.17). There were significant differences between right and left renal volumes for the normal and hypertensive group (HTN) at P=0.05 using t-test with T= (2.77) and P= (0.006) for the right and with (T=3.04) and P= (0.003) for the left as illustrated in table (4.17). Hypertension affected elderly people; and as we age nephrons decrease in number. Additionally, the overall amount of kidney tissue decreases. (Jarvis, 2008; Scanlon, 2011). No significant changes were found in renal lengths and volumes bilaterally between DM, (DM-HTN) and normal. Although all the means of renal volumes in the study fell in the normal range of Sudanese population (80-140 cm^3) but there were slight differences among the groups. The mean renal volumes of kidneys were (100.3, 88.6 and 98.2 cm^3) for the right and were (114.4, 100.8 and 117.6 cm^3) for the left for DM, HTN and (DM-HTN) in tables (4.6) and (4.7) respectively. The largest right renal volume noted in diabetic group (DM) and the smallest one in hypertensive group (HTN); while the largest left renal volume noted in diabetic-hypertensive (DM-HTN) and the smallest one in hypertensive group (HTN) as illustrated in figures

(4.15) and (4.16) respectively. Throughout the study, there was a marked and significant difference between length and volume of left and right kidney; with the left kidney appeared larger than the right in all groups as illustrated in tables (4.6) and (4.7). This difference had been reported by other investigators (Zeb et al 2012 and Mujahid, 2011). The explanation of this, is that spleen is smaller than liver, so the left kidney has more space to grow and it is also found that, the left renal artery is shorter than the right, so increased blood flow in the left renal artery may result in relatively increase in volume. Other authors have described the renal volume as a better predictor of renal function than renal length (Emamian et al, 1993) and (Jones TB et al 1983) The means cortical thicknesses of kidneys were (1.52, 1.42, and 1.50cm) for the right and were (1.72, 1.63 and 1.74cm) for the left for DM, HTN and (DM-HTN) respectively. The thickened cortex of the right kidneys for the three groups was noted in diabetic group (DM) and the thinnest one was noted in hypertensive group (HTN); the thickened cortex of the left kidneys for the three groups was noted in group (DM-HTN) and the thinnest one was noted in hypertensive group (HTN), see tables (4.6) and (4.7).

In this study there were no significant changes in cortico-medullary differentiation bilaterally, see figures (4.19) and (4.20). Also no significant changes were noted in the renal cortical echogenicity and cortical thickness in the right or left kidney in DM, HTN or (DM-HTN), Figures (4.21) and (4.22). In patients with chronic renal failure, the renal cortical echogenicity increases at ultrasound (Khati NJ, Hill MC, Kimmel PL, the essential ultrasound 2005). In addition, the renal cortex often becomes thinned (Morghazi S, Jones E. et al. Kidney int, 2005 .No sonographic evidence of chronic renal failure changes were noted in this study and this agreed with the result of the study carried out by Abd Elgyoum AM et al (2014) in Sudan (renal changes in diabetic patients are detectable by conventional ultrasound only in very advanced stages of the disease).

Increasing of serum creatinine level is a bad indicator for renal function loss because it is excreted only by the kidneys (Scanlon.2011). In this study, the highest level of serum creatinine was found in hypertensive group and the lowest level in

diabetic group, see figure (4.23). Increasing of serum creatinine level associated with decreasing in renal volume (Alsafi et al, 2011). The highest and the lowest level of blood urea nitrogen (BUN) were seen in diabetic-hypertensive (DM-HTN) and hypertensive (HTN) group respectively. BUN is not a reliable at measuring kidney function because it is dependent upon many factors such as protein metabolism, renal blood flow and diet (Scanlon, 2011). GFR is considered the best indicator of overall kidney function (Stevens LA, Levey AS. 2005). Decreasing in the estimated glomerular filtration rate is indicator of renal function loss. (National Kidney Foundation's Guide lines). Regarding eGFR levels there were (67) of participants: normal, (93) had mild reduction, (39) had moderate reduction and (2) had severe reduction in renal function as illustrated in table (4.13). That means eGFR level is the best lab test to know the stage of renal damage. Diabetes and hypertension are considered the main causes of chronic renal failure in Sudan, but their early effects on kidney function cannot be demonstrated by ultrasound before lab investigations. (Gameraddin et al, 2014) and Abd Elgyoum AM et al, 2014)

5.2 Conclusion:

Generally the normal sonographic values of renal measurements depend on age, and gender. The study established the normal values for renal dimensions in the adult population. Although all sonographic renal values in the study fell in the normal range of Sudanese population, but there were slight differences among the groups (DM, HTN and (DM-HTN).

In agreement with published studies, the study showed that renal volume is larger in the left than in the right kidney. The largest right renal volume noted in diabetic group (DM) and the largest left renal volume noted in diabetic-hypertensive (DM-HTN); while the smallest ones noted in hypertensive group bilaterally. The study also showed that the longest right renal length noted in (DM-HTN) group and the longest dimension in the left kidney noted in diabetic group (DM); while the shortest one noted in hypertensive group (HTN) bilaterally. More studies are needed to know the cause.

Throughout the study, no significant sonographic changes were observed in cortical thickness, cortical echogenicity and cortico-medullary differentiation. Estimated glomerular filtration level is the best lab investigation to detect and classify the renal function loss. Serum creatinine level played an important role in calculation of estimated glomerular filtration level (eGFR). We cannot depend on blood urea nitrogen (BUN) alone in detection or determination of renal function loss, because it relies on many other factors. Micro-albuminurea is the best lab test to detect small amount of albumin (protein) in urine, and this consider the early sign of renal function loss.

In conclusion lab investigations have superiority to ultrasound in detection and classification of renal function loss.

5.3 Recommendations:

- When performing ultrasound**,** dependability of renal size on age and gender has to be considered by the operator so as to differentiate between a pathological and normal small size and larger kidney.
- Regular ultrasound scanning for kidneys is recommended for diabetic and hypertensive patients to detect renal changes in size and parenchyma
- The current clinical practice of using renal length measurement by ultrasound can be improved on by volume measurement to provide accurate data and decision making.
- The largest right renal length was noted in diabetic-hypertensive group **(DM-HTN)**; while the largest left renal length was noted in diabetic group **(DM)**. More studies are needed to know the cause.
- The smallest renal volume and length were noted in hypertensive group **(HTN)** bilaterally, further investigations are needed to evaluate multifactorial effects on blood pressure and kidney size.
- Micro-albuminurea test is a more sensitive marker than total protein for CRF from diabetes and hypertension, so we recommend this lab test for early detection of renal function loss.
- Estimated Glomerular filtration Rate (eGFR) is considered the best indicator of overall kidney function and therefore its assessment has become an important clinical tool in detection of early renal function damage.
- Ultrasound cannot detect the early signs of chronic renal failure before lab investigations, so we recommend using beside lab.

References:

Abd Elgyoum A.M, H. Osman et al (Ultrasonography Patterns for Diabetic Nephropathy according to the Body Shape), Scholars Journal of Applied Medical Sciences (SJAMS) 2014; 2(5C):1649-1652

Abdullah M B, Garelnabi M E l, Ayad C E and Ahmed E: Establishment of Reference Values for Renal Length and Volume for Normal Adult Sudanese using MRI Disc Summation Method. Global Journal of Medical Research 2014; 14(2): 27-37

Abubakr.A. Sanusi *et al*. relationship of ultrasonographically determined kidney volume with measured GFR, calculated creatinine clearance and other parameters in chronic kidney disease (CKD**)**.Nephrol Dial Transplant (2009) 24: 1690–1694

Adeela Arooj, Jostinah Lam, Yeoh J.Wui, Eko Supriyanto, Comparison of Renal Size among Different Ethnicities, International Journal Of Biology And Biomedical Engineering 2011, Volume 5, Issue 4: 221- 229

Alsafi Ahmed Abdella et al, ultrasound finding of renal failure patients and creatinine serum level relationship, international journal of medical diagnostic ultrasound-Lahore-Pakistan 2011; 2 (1): 4-7

American Medical Association. Published online December 18, 2013.
at http://www.medscape.com/viewarticle/776940. Accessed Jan/ 9/ 2013.

Beland MD, Walle NL, Machan JT and Cornan JJ: Renal cortical thickness measured at ultrasound: is it better than renal length as an indicator of renal function in chronic kidney disease. AJR. 2010; 195(2): 146-149.

Brandt TD, et al, Ultrasound assessment of normal renal dimensions, Ultrasound Med. 1982 Mar;1(2):49-52.

Carol M. Rumack, et al: diagnostic ultrasound volume one, 4th edition by Mosby, pages 319 -321.

Choi AI, Rodriguez RA, Bacchetti P, Bertenthal D, Hernandez GT, O'Hare AM. White/black racial differences in risk of end-stage renal disease and death. *Am J Med*. Jul 2009; 122(7):672-8.

Emamian SA, Nielson MB, Pedersen JF, Ytte L. Kidney dimensions at sonography: correlation with age, sex, and habitus in 665 adult volunteers. AJR1993; 160:83–86

Gameraddin M, Ismeal M, Yousef M, Tamboul J, Salih S. The Sonographic Estimation of Renal Length and Determination of the Main Causes of Chronic Renal Failure. *Life Sci J* 2014;11(5):222-225].

Glodny B, et al, Normal kidney size and its influencing factors - a 64-slice MDCT study of 1.040 asymptomatic patients , BMC Urol. 2009 Dec 23;9:19

Hall, J., Schmidt, G., & Wood, L. (2005). *Principles of critical care* (3rd ed.). New York: McGraw-Hill.

Hallan SI, Matsushita K, Sang Y, Mahmoodi BK, Black C, Ishani A, et al. Age and association of kidney measures with mortality and end-stage renal disease. *JAMA*. Dec 12 2012;308(22):2349-60.hemodialysis. *BMC Nephrol*. Jan 19 2012;13(1):3.

James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults—report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *The Journal of the*

Jarvis, C. (2008). *Physical examination and health assessment*, (5th ed). St. Louis: W.B. Saunders.

Jones TB, Riddick LR, Harpen MD, Dubuisson RL, Samuels D. (Ultrasonographic determination of renal mass and renal volume). J Ultrasound Med1983; 2:151–154

Khati NJ, Hill MC, Kimmel PL. The Role of Ultrasound in Renal Insufficiency.The Essential Ultrasound Q 2005; 21:227-244

Kidney Disease Statistics for the United States. National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC). Available at http://kidney.niddk.nih.gov/kudiseases/pubs/kustats/17. Accessed September 5, 2012.

Levey AS, Adler S, Caggiula AW, et al. Effects of dietary protein restriction on the progression of moderate renal disease in the Modification of Diet in Renal Disease Study. *J Am Soc Nephrol*. Dec 1996;7(12):2616-26.

Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med*. Mar 16 1999;130(6):461-70.

Levey AS, Coresh J, Balk E, Kausz AT, Levin A, Steffes MW, et al. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Ann Intern Med*. Jul 15 2003;139(2):137-47.

Matthias Hofer. ultrasound teaching manual1998 3rd edition

McNamara D. More frequent dialysis increases risk for complications. February 13, 2013. Medscape Medical News. Available at http://www.medscape.com/viewarticle/ 779265. Accessed August 29, 2013.

Mohammed A. Ali omer, et al (ultrasonographic characteristics of diabetes impact in kidneys' morphology) international journal of science and research (IJSR) volume 3 issue 10 ,October 2014

Molina P, Górriz JL, Molina MD, Peris A, Beltrán S, Kanter J, et al. The effect of cholecalciferol for lowering albuminuria in chronic kidney disease: a prospective controlled study. *Nephrol Dial Transplant*. Aug 24 2013

Morghazi S, Jones E,Schroepple J et al.(Correlation of Renal Histopathology with Sonographic Findings).Kidney Int 2005; 67 : 1515-1520

Mujahid Raza, Amina Hameed, M Imran Khan, Ultrasonographic Assessment of Renal Size and Its Correlation with Body Mass Index in Adults without Known Renal Disease,J Ayub Med Coll Abbottabad 2011;23(3): 64-68

National Institute of Diabetes and Digestive and Kidney Diseases. United States Renal Data System 2012 Annual Data Report: Volume 2: Atlas of End-Stage Renal Disease in the United States. Washington, D.C.: U.S. Government Printing Office; 2012. Report.

National Kidney Foundation's Kidney Disease Outcomes Quality Initiative. Chronic Kidney Disease: Evaluation, Classification, and Stratification. Available at http://www.kidney.org/professionals/KDOQI/guidelines_ ckd/toc.htm. Accessed September 6, 2012.

Netter FH. Anatomy, structure, and embryology. In: The CIBA collection of medical illustrations, vol. 6. Kidneys, ureters, and urinary bladder. CIBA Pharmaceutical; 1987. p. 2-35.

Sarnak MJ, Levey AS: Cardio-vascular disease and chronic renal disease: A new paradigm. Am J Kidney Dis, 2000; 35(1):117-131.

Scanlon, V. (2011). *Essentials of anatomy and physiology* (6th ed.). Philadelphia: F.A. Davis Co.

Siddappa JK, Singla S, Al Ameen M, Rakshith SC, Kumar N. Correlation of Ultrasonographic Parameters with Serum Creatinine in Chronic Kidney Disease. J Clin Imaging Sci 2013;3:(2) 1-6
Stevens LA, Levey AS. Measurement of kidney function. Med Clin North Am. 2005, 89: 457-473.).

United States Renal Data System. 2011 Annual Data Report. Available at http://www.usrds.org/adr.aspx. Accessed Sept 6, 2012.

US Renal Data System. USRDS 1998 Annual Data Report, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases. Bethesda, MD, 1998.

Waknine Y. Kidney Disease Classification to Include Albuminuria Medscape Medical News. Dec 31 2012. Available at http://www.medscape.com/viewarticle/ 779265. Accessed August 29, 2013.

Wald R, Yan AT, Perl J, et al. Regression of left ventricular mass following conversion from conventional hemodialysis to thrice weekly in-centre nocturnal
Walter F., PhD. Boron (2004). Medical Physiology: A Cellular And Molecular Approach. Elsevier/Saunders. ISBN 1-4160-2328-3.

Wong CS, Pierce CB, Cole SR, Warady BA, Mak RH, Benador NM, et al. Association of proteinuria with race, cause of chronic kidney disease, and

glomerular filtration rate in the chronic kidney disease in children study. *Clin J Am Soc Nephrol*. Apr 2009;4(4):812-9.

www.cdc.gov/nchs/data/hus/2011/051.pdf .Accessed December 20, 2013

www.emedicine.medscape.com.kidney anatomy: Overview, Gross Anatomy, Microscopic Anatomy) , accessed on July 23/ 2014

www.en.wikipedia.org/ 9/2015

www.heartfoundation.org.au/healthy-eating/Pages/bmi-calculator.aspx.Accessed on June 14/2015)

www.medicinenet.com/ 7/2015

www.nhlbi.nih.gov/health/health-topics/topics/hbpLink. Accessed December 20, 2013.

www.nlm.nih.gov/ 25,2014

Zeb S, et al: Sonographic Measurement of Renal Dimensions in Adults and its Correlates, International Journal of Collaborative Research on. Internal Medicine & Public Health. 2012; 4 (9): 1630-1631.

Appendix I: Data collection sheet

Evaluation of Renal Changes in Diabetic and Hypertensive Patients using Ultrasound and laboratory findings

Patient data:

1. Age ☐ 2. Gender ☐

3. Occupation ☐

4. Weight …….. 5. Height ……..

P.M.H (past medical history):

1. Duration of the disease.....

2. Diabetic: yes ☐ No ☐

3. HTN : yes ☐ No ☐

4. Family history: yes ☐ No ☐

Lab findings:

Lab findings	Last value	Reference value
Serum Creatinine		
BUN		
eGFR		

Ultrasound parameters:

C/M differentiation:

Right kidney: Mild ☐ Moderate ☐ evere ☐

Left kidney: Mild ☐ Moderate ☐ vere ☐

Cortical echogenicity:

RT kidney: increased ☐ Normal ☐

LT kidney: increased ☐ Normal ☐

Cortical thickness: Right kidney= …….Left kidney =……..

Renal volume: RT kidney (LxWxD 0.523)……

LT kidney (LxWxD0.523)…….

- Stones : yes No ☐ ☐
- Obstruction : yes No ☐ ☐
- **Others**……...……

……

Comment……

Appendix II

PUBLICATIONS

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Ultrasonographic Renal Length and Parenchymal Thickness in Normal Sudanese Population

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Abstract: ***This study was intended to measure the ultrasound normative values of renal length and parenchymal thickness in adult Sudanese population in order to establish their reference value for Sudanese population while no recorded reference value in literature for them. Ultrasonographic kidney measurements were performed on 77 adult patients without known kidney lesions. Measurements included length and parenchymal thickness. The effect of age, gender, site (left and right side) and height was statistically analyzed. All normal patient was included in this study while any renal disease were excluded. This study was include (35) males and (42) females. The mean renal lengths were (10.15± 0.78) and (10.33± 0.80) cm for the right and left kidney respectively. The mean parenchymal thicknesses were (1.4714± 0.33cm) for the right kidney and (1.7169± 0.36cm) for the left kidney. These result were correlated with age, gender, site, and height which showed that there is no significant difference between right and left renal length, but there was significant difference between right and left parenchymal thicknesses. The significant effect of age was found only in left renal length. The significant effect of gender was noted only in the right parenchymal thicknesses. No significant difference among height groups for renal length, but there was significant difference in right parenchymal thickness. The study concluded that normal value of left renal length was affected with age and normal parenchymal thickness was affected with site (left or right). The right parenchymal thickness was affected with gender and height. Establishment of normal renal values of renal length and parenchymal thickness in Sudanese population will help us in evaluation of patients with chronic renal disease.***

Keywords: Sonography, renal length, Parenchymal Thickness, Adults.

1. Introduction

Renal length and parenchymal thickness are valuable diagnostic parameters in urological and nephrology practice. In the adult, each kidney measures approximately 11 cm long, 2.5 cm thick, and 5 cm wide (M. Rumack, et al, 2011). The normal parenchymal thickness range from (14- 18mm). Further review of the literature shows that renal length varies with age, gender, body mass index and pregnancy (Shcherbak et.al 1989 and Guzman RP, et.al 1994). Renal infections/inflammations, nephrologic disorders, diabetes mellitus and hypertension are the most important co-morbid conditions affecting renal length (Yamaguchi S and Yamada-H. et.al 1992). Since the renal length and parenchymal thickness are affected by various factors, it is necessary to first establish the normal values. The information available in the West may not be extrapolated to Sudanese's population since the renal length and parenchymal thickness may differ between ethnic groups and according to body size (Emamian Sa, 1993 and Wang F, 1989). The current study determined the Ultrasonographic renal length and parenchymal thickness in a group of individuals without known renal disease and assessed the effect of age, gender, side and height.

2. Material and Methods

This prospective observational study was conducted in the department of diagnostic radiology, Fedail Specialized Hospital in Khartoum city- Sudan. Renal length and parenchymal thickness were assessed by ultrasound in Seventy seven healthy participants, having normal renal function tests, between January 2014 and July 2015. Participant's age ranged from (22- 79) years. Pregnant females, subjects with known diabetes and hypertension and the participant who were unable to change posture for accurate assessment of kidneys during US examination were excluded from the study. Height was taken in meters (m). Participants required stopping having food for 6 hours before exam in order to reduce bowel gas. Ultrasound procedure performed according to the protocol of renal U/S scanning as mentioned by Sandra (Sandra L. H (2001). All the US examinations and measurements were performed using two-dimensional Real Time US machine with curvilinear transducer of (3.5–6 MHz). Once the kidney was located, the transducer was rotated slightly to determine the longest renal axis and renal length was measured as the maximum bipolar dimension in longitudinal plane. Then the renal parenchymal thickness was measured as the distance between outer renal margin and renal sinus. Correlation of renal length and parenchymal thickness with age, gender and height of the subjects were determined.

Data was analyzed on SPSS-16. Descriptive statistics were applied on the available data. Mean ±SD was presented for age, height, right renal length (RR L), left renal length (LR L), right parenchymal thickness and left parenchymal thickness. Frequencies and percentages were computed for gender and age groups.

3. Result

Table (1): Distribution of renal length and parenchymal thickness means according to participant's side (right and left) through the whole cases.

Variable	Renal	Mean	Std. D	P-Value
Renal Length	Right	10.1506	0.78348	0.16
	Left	10.3312	0.80447	
parenchymal	Right	1.4714	0.33001	**0.00**

thickness	Left	1.7169	0.36252	

Table 2: Renal length and parenchymal thickness according to participant's age**:**

Age group	RR L	LR L	Rt p. thickness	Lt p. thickness
20-30	10.07	9.92	**1.58**	1.90
31-40	10.05	10.69	1.51	1.76
41-50	10.23	10.50	1.36	1.62
51-60	**9.95**	**9.67**	1.56	1.61
61-70	10.28	10.60	**1.32**	**1.58**
71-80	**11.07**	**10.77**	1.53	**2.00**

Table 3: Relationship of renal length and parenchymal thickness with gender:

Renal parameter	Gender	Mean	Std. D	P-Value
RR L	Male	10.075	0.749	0.43
	Female	10.215	0.814	
L R L	Male	10.394	0.655	0.52
	Female	10.279	0.915	
Rt parenchymal thickness	Male	1.566	0.304	0.02
	Female	1.39	0.334	
Lt parenchymal thickness	Male	1.76	0.378	0.31
	Female	1.68	0.349	

Table 4: Relationship of renal length and parenchymal thickness according to participant's height:

participant's height	RR L	LR L	Rt p. thickness	Lt p. thickness
1.40-1.50	10.4250	10.2250	**1.2500**	1.8000
1.51-1.60	10.2833	10.1833	1.4056	**1.6167**
1.61-1.70	**9.9594**	10.3063	1.3875	1.6563
1.71-1.80	10.2455	**10.5182**	1.6773	1.8636
1.81-1.90	**10.7000**	**10.1000**	**1.7000**	**1.9000**

4. Discussion

The mean of renal lengths were (10.15) and (10.33 cm) for the right and left kidney respectively. The mean of renal parenchymal thickness were (1.47) and (1.71 cm) for the right and left kidney respectively. Although there was no significant difference between the right and left renal length but the study showed that the left kidney was slightly larger than the right one same result noted by Zeb Saeed et al, (2012). The study also revealed that, the left parenchyma was thicker than the right one, with significant difference between the right and left parenchymal thickness at (P= 0.00) using one way Anova, and that agreed with some authors such as (Emamian, 1993), as in table (1).

The smallest mean of RR L was (9.94cm), noted in the age group (51- 60) and the largest mean was (11.06cm), noted in age group (71-80). The study revealed that there was no significant difference in right renal length through age groups at (P=0.33), this finding agreed with El-Reshaid et al 2014), they found that right renal length was 10.68 ± 1.4 (p = 0.56) without a significant change with age.

The smallest mean of LR L was (9.67cm), noted in the age group (51- 60) and the largest mean was (10.76cm), noted in age group (71-80), with significant difference at (P=0.00). This result consistent with Glodny et al, 2009), they found that renal length affected with age significantly.

The thinnest right parenchymal thickness (1.32 cm) was noted in the age group (61-70) and the thickest one (1.58cm) was noted the age group (20-30). No significant difference at (P=0.26).

The thinnest left parenchymal thickness (1.58cm) was noted in the age group (61-70) and the thickest one (2.00cm) was noted the age group (71-80). The study revealed that, no significant difference at (P=0.09), that meant cortical thickness did not vary significantly with age as shown in (Wael El-Reshaid et al 2014**)**. While we observed that both the thinnest right and left parenchymal thickness were found in the same group of age (61-70), that means parenchymal thickness decrease with age, increased reduction in parenchymal thickness due to age was noted in the study carried out by (Emamian et al,1993) as in table (2).

The mean of right renal lengths were (10.07) and (10.21 cm) for male and female respectively. The mean of left renal lengths were 10.39 and 10.27 for male and female respectively. The study revealed that no significant difference in renal length (right and left) between male and female at (P= 0.43), (P=0.52) for right and left respectively, this finding consistent with [Luyckx VA 2010]; Some studies, however, show that renal length is greater in males than in females [Buchholz NP ,2000, Wang F, 1989] and other study found that renal length was similar for both genders (9.82 cm) in males and (9.88 cm) in females (Saeed et al, (2012). Also no significant difference noted in left parenchymal thickness between male and female at (P= 0.31).The only significant difference was noted in the right parenchymal thickness at (P = 0.02) using one way Anova, but bilaterally, the parenchyma in males was thicker than females. As in table (3).

- **Renal length according to participant's height**

The smallest mean of RR L was (9.95cm), noted in the height group (1.61-1.70) and the largest mean was (10.7cm) noted in height group (1.81-1.90). The smallest mean of LR L was (10.1cm), noted in the height group (1.81-1.90) and the largest mean was (10.51cm) noted in height group (1.71-1.80). The study revealed that, there was no significant difference among height groups at (P= 0.45) for right renal length and at (P= 0.74) for left renal length. The study showed that renal length did not correlate with height, this result agreed with El-Reshaid et.al 2014, table (4).

- **Parenchymal thickness according to participant's height**

The thinnest right parenchymal thickness (1.25 cm) was noted in the height group (1.40-1.50) and the thickest one (1.70 cm) noted in the height group (1.81-1.90). Right renal parenchymal thickness in the current study exhibited strong positive correlations with height at (**P=0.00)**. This result is consistent with previous findings in (Emamian, 1993, Weisenbach J 2001 and Charles 2014). The thinnest left parenchymal thickness (1.61 cm) was noted in the height group (1.51-1.60) and the thickest one (1.90 cm) was noted the height group (1.81-1.90). The study showed that no significant difference among height groups for left

parenchymal thickness at (P=0.17). We observed that, the left parenchyma was thicker than the right. Table (4).

5. Conclusion and Recommendations

The study concluded that normal value of left renal length was affected with age and normal parenchymal thickness was affected with side. The right parenchymal thickness was affected with gender and height. The right parenchymal thickness was the most affected renal parameters, so the study recommended using in evaluation of patients with chronic renal disease in addition to left renal length. Establishment of normal renal values of renal length and parenchymal thickness in Sudanese population will help us detecting renal changes early.

References:

[1] Buchholz NP, Abbas F, Biyabani SR, et al: Ultrasonographic renal size in individuals without known renal disease. J Pak Med Assoc 2000; 50: 12–16.
[2] Carol M. Rumack, et al: Diagnostic Ultrasound Volume one, 4th edition, 2011 by Mosby, page 319.
[3] Emamian SA, Nielsen BM, Peterson FI, Ytte L. Kidney dimension at sonography: Correlation with age, sex and habitus in 665 adult volunteers. American Journal of Roentgenology 1993; 160: 83-86.
[4] Eze Charles; Okoye Joy; Agwu Kenneth: Normative ultrasound values of renal parenchymal thickness among adults in Enugu, South-East Nigeria African Health Sciences. 2014 Sep; Vol 14 (3): p 689-97.
[5] Guzman RP, Zierler RE, Isaacson JA, el al: Renal atrophy and arterial stenosis. A prospective study with duplex ultrasound. Hypertension. 1994:23:346-47.
[6] Luyckx VA, Brenner BM: The clinical importance of nephron mass. J Am Soc Nephrol 2010; 21: 898–910.
[7] Shcherbak Al. Angiographic criteria in the determination of indications for organ preserving surgery in renal artery occlusion. Klin. Khir. 1989; 2: 5.
[8] Wael El-Reshaid and Husam Abdul-Fattah' Sonographic Assessment of Renal Size in Healthy Adults, Med Princ Pract 2014; 23:432–436.
[9] Wang F, Cheok SP, Kuan BB: Renal size in healthy Malaysian adults by ultrasonography. Med J Malaysia 1989; 44: 45–51.
[10] Weisenbach J, Horvahth M, Jeges S, Adanovich K, Huszar T. Normal percentiles of the kidney size in children as measured by ultrasonography. ORV Hetil. 2001; 142 (2): 71-4.
[11] Yamada-H Hishida-A, Kumagai-H. et al. Effects of' age. Renal diseases and diabetes mellitus on the renal size reduction accompanied by the decrease of renal function. Nippon-Jinzo-Gakkai-Shi., 1992:34:1071-75.
[12] Yamaguchi S. Fujii H, Kaneko S. et.al Ultrasonographic study in patients with chronic renal failure. Part 1. Ultrasonic measurement of renal size and analysis of renal ultrasonotomograms. Nippon Hinyokika Gakkai Zasshi, 1990; 81: 1175-77.
[13] Zeb S, Waseem M, Raza S , Adil S, Iffat Y. and Syed A. H. Sonographic Measurement of Renal Dimensions in Adults and its Correlates. International Journal of Collaborative Research on. Internal Medicine & Public Health. 2012; 4 (9): 1630-1631.

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Sonographic Screening for Signs of Renal Failure in Diabetic & Hypertensive Sudanese Patients

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A B S T R A C T

The objective behind this study was to diagnose early changes in renal size & cortical thickness be ultrasound and to correlate the ultrasound findings with creatinine & BUN results among the diabetic & hypertensive Sudanese patients. In screening study 100 diabetic & hypertensive participants with mean age of 59 ±1 were studied. (27% of 100) were diabetic, (22% of 100) were hypertensive while (51% of 100) were both diabetic & hypertensive. The mean of Rt. renal length was 10.07 & 9.85 cm for the diabetic & hypertensive respectively. While the mean of Lt. renal length was 10.37 & 10.01 cm respectively. The mean cortical thickness was 1.45± 0.31& 1.42± 0.23 in the Rt. and was 1.65± 0.34 & 1.59± 0.28 in the Lt. for the diabetic and the hypertensive respectively. The hypertensive values of both creatinine and BUN were increased more than of diabetic CI= 95%. The mean creatinine in the hypertensive was 0.98± 0.2 while it was 0.91± 0.155 in the diabetic. The mean BUN in the hypertensive was 24.2± 7.7 & 23.29± 6.7 in the diabetic. The statistical analysis revealed positive correlations of Lt. renal length with creatinine and BUN, P = 0.002 and 0.000 respectively. The study concluded that it is difficult to diagnose early signs of renal failure by ultrasound. Moreover, it concluded that the hypertensive Sudanese patients may have the high risk for ARF signs more than the diabetic.

Introduction

(Acute renal failure (ARF), characterized by sudden loss of the ability of the kidneys to excrete wastes, concentrate urine, conserve electrolytes, and maintain fluid balance) (Robert W. Schrier et al (2004).

In this study, the researchers attempted to study changes could occur to the kidneys' length or cortical thickness of known hypertensive & diabetic Sudanese patients.

Acute renal failure is characterized by a rapid but usually rever sible decline in glomerular filtration rate (GFR) that can occur in patients with normal renal function or in those with pre- existing chronic renal disease [1]. Various criteria have been used to define acute renal failure, primarily GFR or adjusted creat inine, and urine output as proposed by the International Acute Dialysis Quality Initiative [1,2].

Usually the diagnosis of RF (renal failure) obtained through using different modalities; the first line is Plain X-ray for kidney, ureter and bladder (KUB), then Intravenous urography (IVU), Nuclear medicine, Cystography, Computerized tomography (CT), and Magnetic resonance imaging (MRI). In clinical practice, Lab. Tests for Blood Urea Nitrogen (BUN) and creatinine serum level were used to confirm the diagnosis. Some researchers proposed that (an early diagnosis of renal changes may provide the potential to prevent progression to established ARF) (Robert W. Schrier et al (2004). The researchers in the current research used diagnostic ultrasound (US) to evaluate the renal size, renal cortical thickness and renal echogenicity to check early renal changes could occur in known hypertensive & diabetic patients. The US finding was correlated with creatinine & BUN Lab. Tests. (Rahman M et al (2012) has mentioned most of these modalities that used in RF diagnosis).

As some researchers suggested (the renal failure increases rapidly among Sudanese native during the last decade and no published epidemiological study is available to be correlated with renal failure in Sudan) (Bala A. A et al (2011). For this the researchers conducted this study and the hypothesis was; an early signs of renal failure in known diabetic & hypertensive Sudanese can be diagnosed by ultrasound. The objective of this study is to check the possibility of early diagnosis of renal failure signs in diabetic & hypertensive Sudanese patients. An early diagnosis of RF signs may be helpful in national health promotion program in Sudan.

Materials and Methods

In a correlative descriptive study, a hundred Sudanese patients of renal failure were involved. All participants were selected through convenient simple selection, the inclusion criteria was patients of diabetes & hypertension. The study was conducted in Khartoum during 2014 – 2015. A Mindray Digi prince ultrasound machine model(DP)- 6600) with curve linear 3.5MHz probe was used to scan kidneys of all participants. Ultrasound procedure performed according to the protocol of renal U/S scanning as mentioned by Sandra (Sandra L. H (2001). All participants were scanned in supine and lateral decubitus positions using the liver and spleen as acoustic window to scan both kidneys in transverse or longitudinal plane (Sagittal plane) during deep inspiration.

The US measurements included; the right and left kidney volume which has been determined out of length (L) multiplied by width (W) multiplied by depth (D) multiplied by the constant factor (0.523).These measurements obtained by certain ultrasound technique in which the transducer in perpendicular position, just inferior to the most lateral edge of the right costal margin or by moving the transducer in medial and inferior sections until the right kidney is located. Another US parameters were taken such as; Parenchymal (cortical) thickness, cortical echogenicity (compared to liver for the right kidney) or cortical echogenicity (compared to spleen for the left kidney).

The standard references for U/S measurements were as follow; The kidney is considered normal in size when the length: 9–12 cm, Width: 5cm and the Depth (Thick): 2.5 cm) (Sandra L. H (2001).
The size of the parenchyma is measured from the convex outer edge to the tip of a papilla, the normal value is 13–18 mm (Singer (2006).

A lab. Tests; creatinine serum level and blood urea nitrogen (BUN) using enzymatic method were applied in the study to be correlated with US findings. The reference for creatinine normal levels in the blood are approximately 0.6 to 1.2 milligrams (mg) per deciliter (dL) in adult males and 0.5 to 1.1 milligrams per deciliter in adult females (Banfi G et al (2010), (www.medicinenet.com). For the BUN in general, 7 to 20 mg/dL (2.5 to 7.1 mmol/L) is considered normal (www.mayoclinic.org), (McMillan J.I (20015).

A data collection sheet was used to organize the ultrasound & lab findings with other patients' data (age, gender, weight and height). Statistical Package for the Social Sciences (SPSS) soft word program was used to analyze the data of the study, descriptive analysis and correlations were applied to achieve the statistical values.

Results and Discussion

Table.1 & Figure.1 summarize the means, Std. Deviations and the frequencies of the study variables, the mean age of the participants was 59 ±1 (range from 36 – 80 years old), 65% of the participants were female and 35% were male. One of the participants had single kidney (Lt. renal agenesis), the mean renal length for the Rt. & Lt. kidneys was 10 ± 1 & 10.2 ± 1.4 cm, respectively (ranged from 8.7 to 12.5 cm). The mean cortical thickness for both Rt. & Lt. kidneys was 1.45± 0.3 & 1.7± 0.5 cm respectively (ranged from 1.1 to 2.2 cm). The mean serum creatinine was 0.93 ± 0.19 mg/dl (ranged from 0.6 – 1.7). The mean BUN was 23.9 ± 6.8 mg/dl (ranged from 12-41), which was the obvious elevated indicator in this study. (27% of 100) were diabetic, (22% of 100) were hypertensive while (51% of 100) were both diabetic & hypertensive. Table2 summarizes the correlations of renal length between diabetic & hypertensive. The mean of Rt. renal length was 10.07 & 9.85 cm for the diabetic & hypertensive respectively. While the mean of Lt. renal length was 10.37 & 10.01 cm respectively. Tables3 summarizes the correlations of renal cortex between diabetic & hypertensive participants, the mean cortical thickness was 1.45± 0.31& 1.42± 0.23 in the Rt. and was 1.65± 0.34 & 1.59± 0.28 in the Lt. for the diabetic and the hypertensive respectively. Tables4&5 summarize the correlations between renal length and creatinine & between renal length and BUN, the statistical analysis revealed positive correlations of Lt. renal length with creatinine and BUN, P = 0.002 and 0.000 respectively. There is no statistical association between renal length and BMI P = 0.1 & 0.7 for Rt. & Lt. kidneys respectively. While the association between renal length and age is positive P = 0.02.

Figure2 through 3 summarize the measurements of renal size & cortical thickness. While figure 4& 5 summarize the correlation between diabetes & hypertension with creatinine & BUN.

The results of this study showed that the mean renal length for the Rt. & Lt. kidneys was 10 ± 1 & 10.2 ± 1.4 cm, respectively, Table1. These findings were considered around the normal renal length in Sudanese when compared with the standard renal size and with a previous study for renal length in normal Sudanese (Abdullah M B et al(2014) (who showed that the kidneys' length

measured for normal Sudanese subjects were 10.08±0.46, 10.67±0.47 for Rt. & Lt. respectively). Also the study showed statistical association between Lt. renal length and the creatinine (P = 0.002) and statistical association between BUN and renal length (P= 0.000), (Tables4&5). These findings agreed with some authors' findings (Giacomo Di Zazzo et al (2011) who proposed that (a significant correlation was found between kidney size and serum creatinine (P < 0.0001) and between kidney size and serum blood urea nitrogen, P < 0.002).

Table.1 Means & Std. Deviations for the variables of the 100 study participants.

Variable	Mean	Std. Deviation
Age	59.4900	1.00408
Body mass index (BMI)	28.3650	5.11937
Rt. cortical thickness/ cm	1.4580	.30291
Lt. cortical thickness/cm	1.7200	.55176
Rt. Renal length/cm	10.0880	1.02640
Lt. Renal length/cm	10.2536	1.49643
Creatinine/mg/dl	.9370	.19157
BUN/mg/dl	23.9100	6.85211

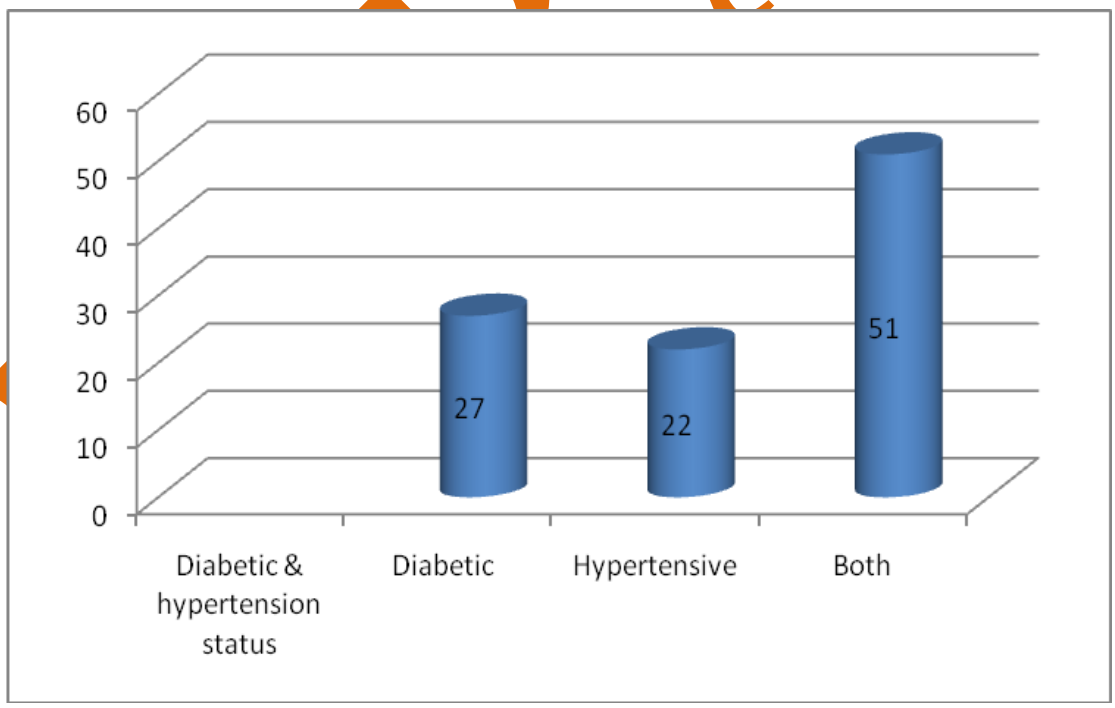

Figure.1 The status of the participants

Table 2: Correlations of renal length between diabetic & hypertensive participants.

	Diabetes & Hypertension			Statistic	Std. Error
Rt. renal length	Diabetic	Mean		10.0704	.22951
		95% Confidence Interval for Mean	Lower Bound	9.5986	
			Upper Bound	10.5421	
		Std. Deviation		1.19255	
		Minimum		8.30	
		Maximum		12.20	
	Hypertensive	Mean		9.8500	.18064
		95% Confidence Interval for Mean	Lower Bound	9.4743	
			Upper Bound	10.2257	
		Std. Deviation		.84726	
		Minimum		8.10	
		Maximum		11.60	
Lt. renal length	Diabetic	Mean		10.3778	.21030
		95% Confidence Interval for Mean	Lower Bound	9.9455	
			Upper Bound	10.8101	
		Std. Deviation		1.09275	
		Minimum		8.30	
		Maximum		12.50	
	Hypertensive	Mean		10.0136	.21565
		95% Confidence Interval for Mean	Lower Bound	9.5652	
			Upper Bound	10.4621	
		Std. Deviation		1.01150	
		Minimum		8.20	
		Maximum		12.10	

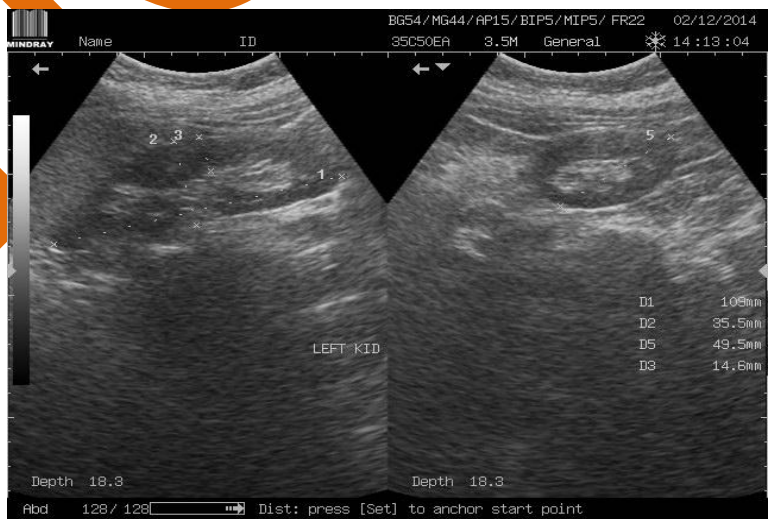

Figure2: Renal measurements (length, cortical thickness and volume)

Table3: Correlation of renal cortical thickness between diabetes & hypertension.

	Diabetes & Hypertension			Statistic	Std. Error
Rt. cortical thickness	Diabetic	Mean		1.4519	.06118
		95% Confidence Interval for Mean	Lower Bound	1.3261	
			Upper Bound	1.5776	
		Std. Deviation		.31789	
		Minimum		.90	
		Maximum		2.10	
	Hypertensive	Mean		1.4273	.05019
		95% Confidence Interval for Mean	Lower Bound	1.3229	
			Upper Bound	1.5316	
		Std. Deviation		.23540	
		Minimum		1.00	
		Maximum		1.80	
Lt. cortical thickness	Diabetic	Mean		1.6556	.06674
		95% Confidence Interval for Mean	Lower Bound	1.5184	
			Upper Bound	1.7927	
		Std. Deviation		.34678	
		Minimum		.70	
		Maximum		2.50	
	Hypertensive	Mean		1.5909	.06027
		95% Confidence Interval for Mean	Lower Bound	1.4656	
			Upper Bound	1.7162	
		Std. Deviation		.28269	
		Minimum		1.10	
		Maximum		2.10	

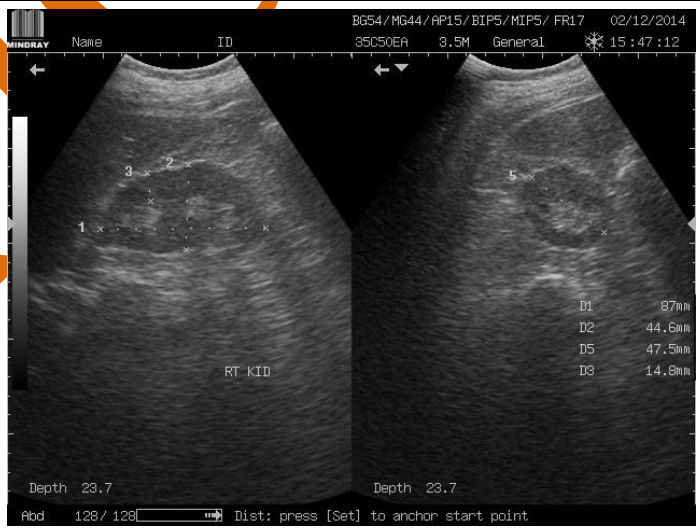

Figure3: Rt. Kidney measurements (Length, cortical thickness and volume) which is increased in the hypertensive

Table 4: Correlations between renal length and creatinine for the participants.

		Rt. renal length	Lt. renal length	Creatinine
Rt. renal length	Pearson Correlation	1	.271	-.188
	Sig. (2-tailed)		.006	.060
	N	100	100	100
Lt. renal length	Pearson Correlation	.271	1	-.302
	Sig. (2-tailed)	.006		.002
	N	100	100	100
Creatinine	Pearson Correlation	-.188	-.302	1
	Sig. (2-tailed)	.060	.002	
	N	100	100	100

Table 5: Correlations between renal length and BUN for the participants.

		Rt. renal length	Lt. renal length	BUN
Rt. renal length	Pearson Correlation	1	.271	-.194
	Sig. (2-tailed)		.006	.053
	N	100	100	100
Lt. renal length	Pearson Correlation	.271	1	-.402
	Sig. (2-tailed)	.006		.000
	N	100	100	100
BUN	Pearson Correlation	-.194	-.402	1
	Sig. (2-tailed)	.053	.000	
	N	100	100	100

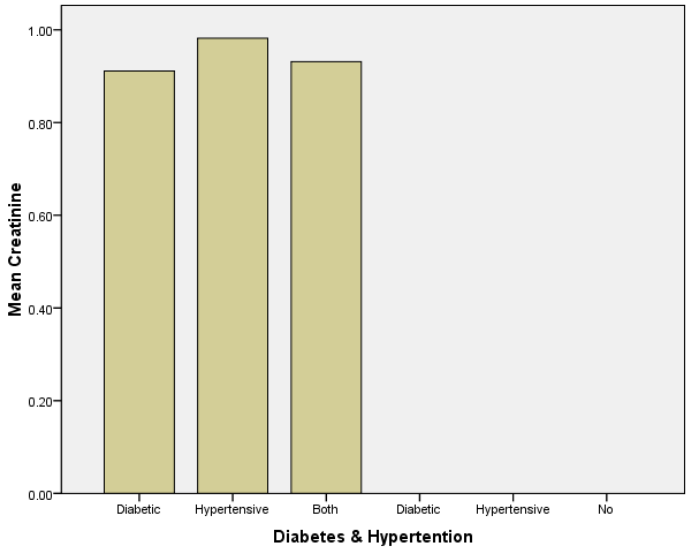

Figure4: Correlation between diabetes & hypertension with creatinine for the 100 participants, which is increased in the hypertensive.

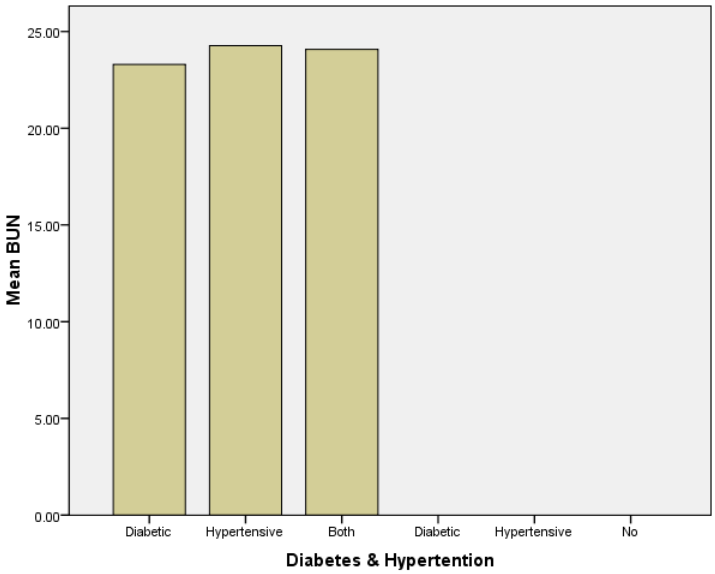

Figure5: Correlation between diabetes & hypertension with BUN for the 100 participants which is increased in the hypertensive.

This study also showed that the decrease renal size & parenchyma thickness and the increase of creatinine & BUN levels were more prominent in hypertensive patients than in the diabetic (Tables2 through3 & Figures4,5). To some extent these findings agreed with (Zeb Saeed et al (2012) who indicated that (It was observed that both hypertensives and diabetics had larger kidney sizes than the healthy subset; only diabetes was associated with statistically significant increase in kidney length of 10.2

± 0.76 cm when compared to 9.90 ± 0.80 cm of the remaining individuals (P =0.019).

Although some other authors (Leblanc M et al(2005) suggested that diabetics with baseline renal insufficiency represent the highest risk subgroup for acute renal failure. Finally in this study the statistical analysis indicated that there is no statistical association between renal length and BMI P = 0.1 & 0.7 for Rt. & Lt. kidneys respectively. While the association between renal length and age is positive P = 0.02. These findings were agreed – to some extend- with some authors' suggestions (Mohammed A. A. O. et al(2014) who proposed that in Sudanese diabetic (The BMI of diabetic patients has been significantly (R2 = 0.6) decreasing following aging. The kidney size increases significantly as R2 = 0.75 and 0.6 for left and right kidney respectively).

Conclusion

The study showed that all US findings regarding renal length and cortical thickness were within the normal range. Although, the hypertensive Sudanese patients might have the high risk for ARF signs more than the diabetic. Moreover it showed that the general mean BUN for the participants was elevated.

Limitation & recommendation for this study includes the sample size of this study was not big enough, further studies in the same field in Sudan may be needed. It is better to be a cohort study for good follow up of hypertensive & diabetic.

References

Abdullah M B, Garelnabi M E l, Ayad C E and Ahmed E.2104. Establishment of Reference Values for Renal Length and Volume for Normal Adult Sudanese using MRI Disc Summation Method. Global Journal of Medical Research. 14(2): 27-37

Banfi G, Melegati G , Melzi G D and Barassi A., 2010. Evaluation of equations for measuring eGFR based on serum creatinine and 6 cystatin C values in top level rugby players. Journal of Human Sports and exercise. 5(3): 370- 378.

BUN test, available from: http://www.mayoclinic.org/tests-procedures/blood-urea-nitrogen/basics/results/PRC-20020239, accessed on 20/4/2015.

Creatinine in the blood Test, available from: http://www.medicinenet.com/creatinine_blood_test/page2.htm, accessed on 20/4/2015.

Giacomo Di Zazzo, Gilda Stringini, Maria Chiara Matteucci, Maurizio Muraca, Saverio Malena and Francesco Emma. 2011. Serum Creatinine Levels Are Significantly Influenced by Renal Size in the Normal Pediatric Population. Clin J Am Soc Nephrol. 6(1): 107–113.

Leblanc M, Kellum JA, Gibney RT, Lieberthal W, Tumlin J and Mehta R., 2005. Risk factors for acute renal failure: inherent and modifiable risks. Curr Opin Crit Care . 11(6): 533-6.

Mayo Clinic Staff. Acute kidney failure: Tests and diagnosis. Available from http://www.mayoclinic.org/diseases-conditions/kidney-failure/basics/tests-diagnosis/con-20024029. accessed on 31/5/2015.

McMillan J.I. Acute Kidney Injury (AKI) (Acute Renal Failure). Available from http://www.merckmanuals.com/professional/genitourinary-disorders/acute-kidney-injury/acute-kidney-injury-aki, accessed on 28/4/2015.

Mohammed A. A O., Ayman H. E, Mohamed E. M. G, Mustafa Z. M, Muatasim E and Ghada A. E., 2014.Ultrasonographic Characteristics of Diabetes Impacts in Kidneys' Morphology. Open Journal of Radiology. 4: 301-308.

Rahman M, Shad F and Smith M. C.,2012. Acute Kidney Injury: A Guide to Diagnosis and Management. Am Fam Physician. 86(7): 631-639.

Robert W. S, Wei W, Brian P., and Amit M., 2004. Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest. 114 (1): 5–14.

Sandra L. H., 2001.Textbook of Diagnostic Ultrasonogrphy.,vol.1,fifth ed. Mosby, USA. Pp.

Singer. 2006. Renal Ultrasound: Sonography of the Kidneys. Available from http://www.urology-textbook.com/renal-ultrasound.html, accessed on 27/4/2015.

Zeb S, Waseem M, Raza S , Adil S, Iffat Y. and Syed A. H., 2012. Sonographic Measurement of Renal Dimensions in Adults and its Correlates. International Journal of Collaborative Research on Internal Medicine & Public Health. 4 (9): 1630-1631.

Appendix III: Ultrasound images

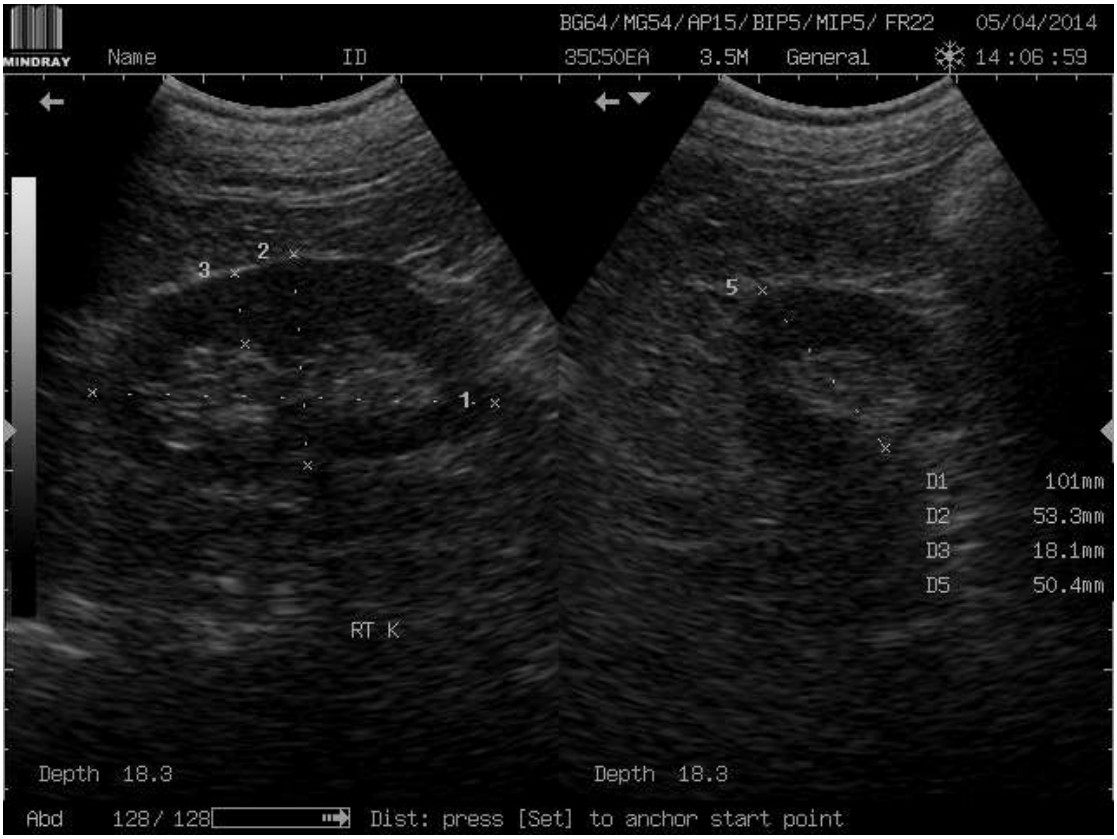

Image (1):

Patient age: 75 (male)

Duration of the disease: DM for 35 y, HTN for 15 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : good

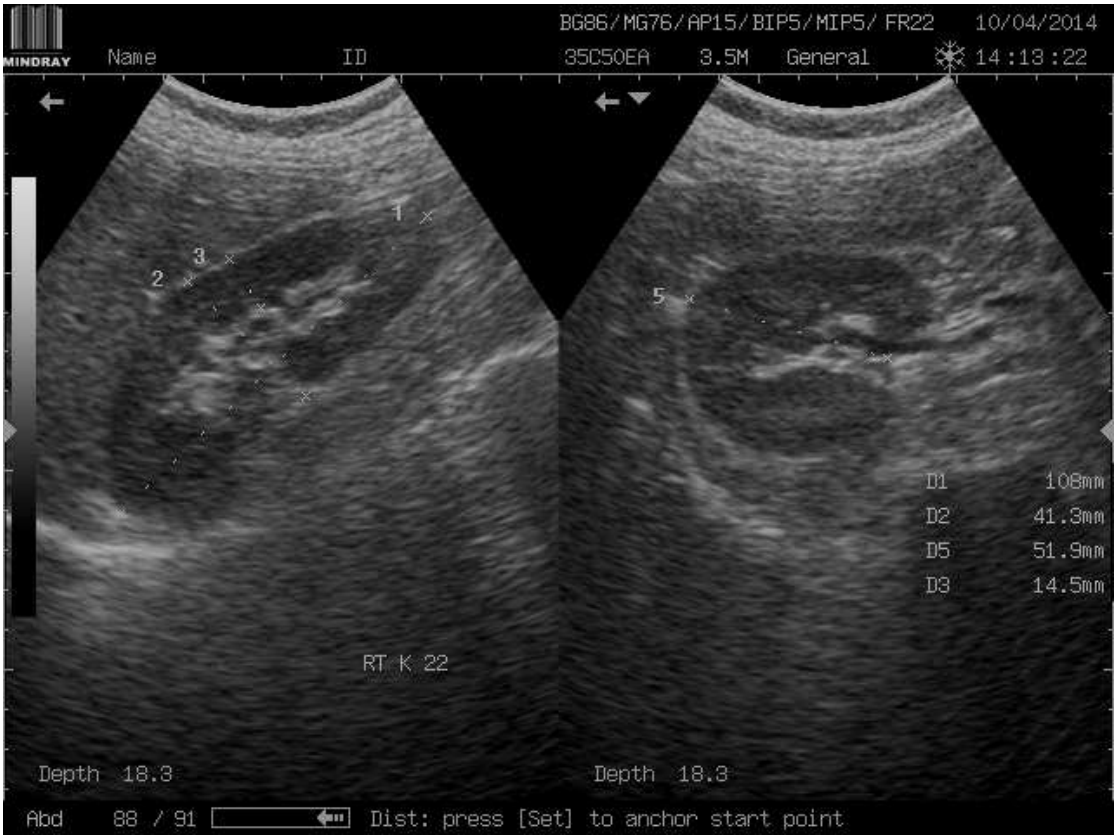

Image (2):

Patient age: 65 (female)

Duration of the disease: DM for 10 y, HTN for 30 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : good

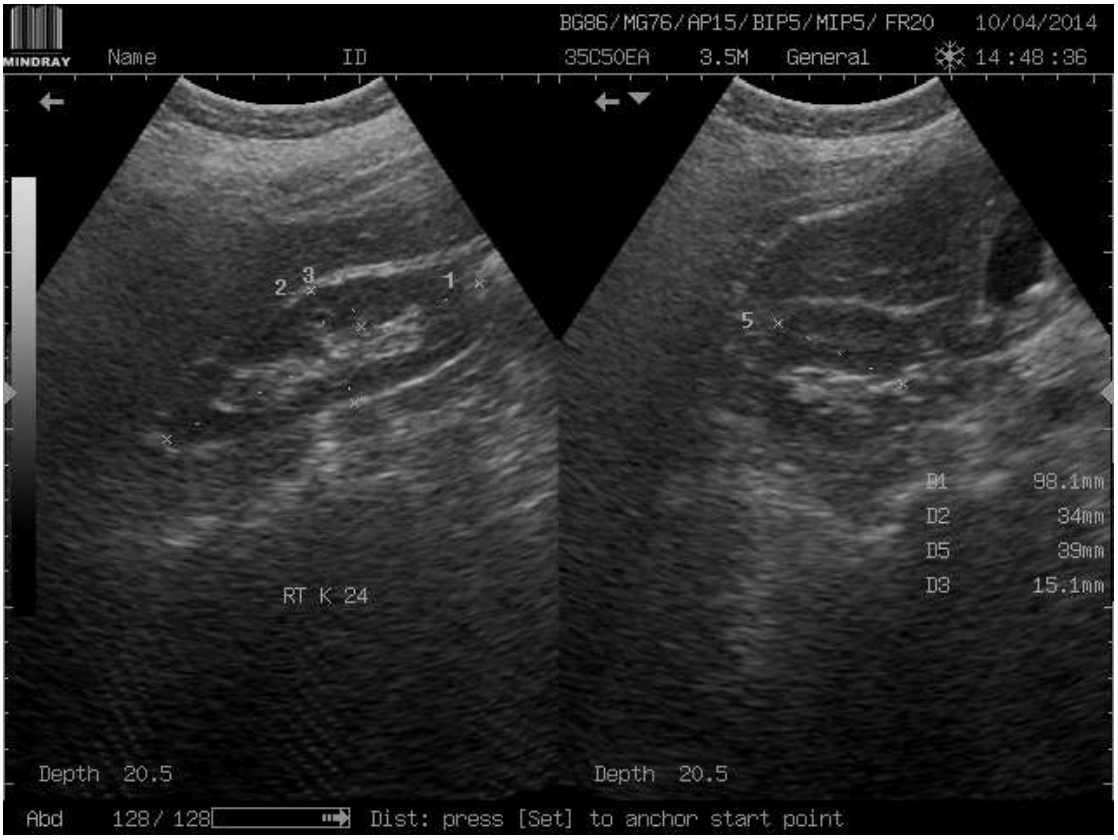

Image (3):

Patient age: 60 (Female)

Duration of the disease: HTN for 28 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : good

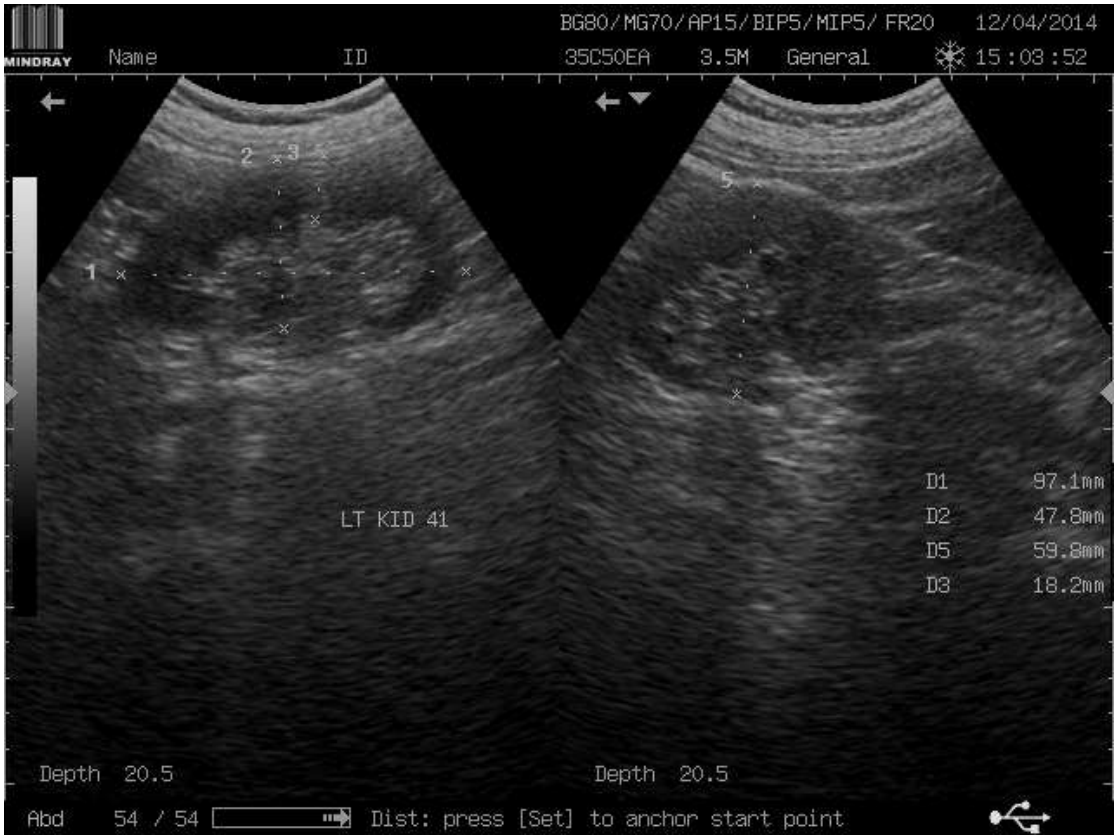

Image (4):

Patient age: 68 (male)

Duration of the disease: DM for 29 y, HTN for 10 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

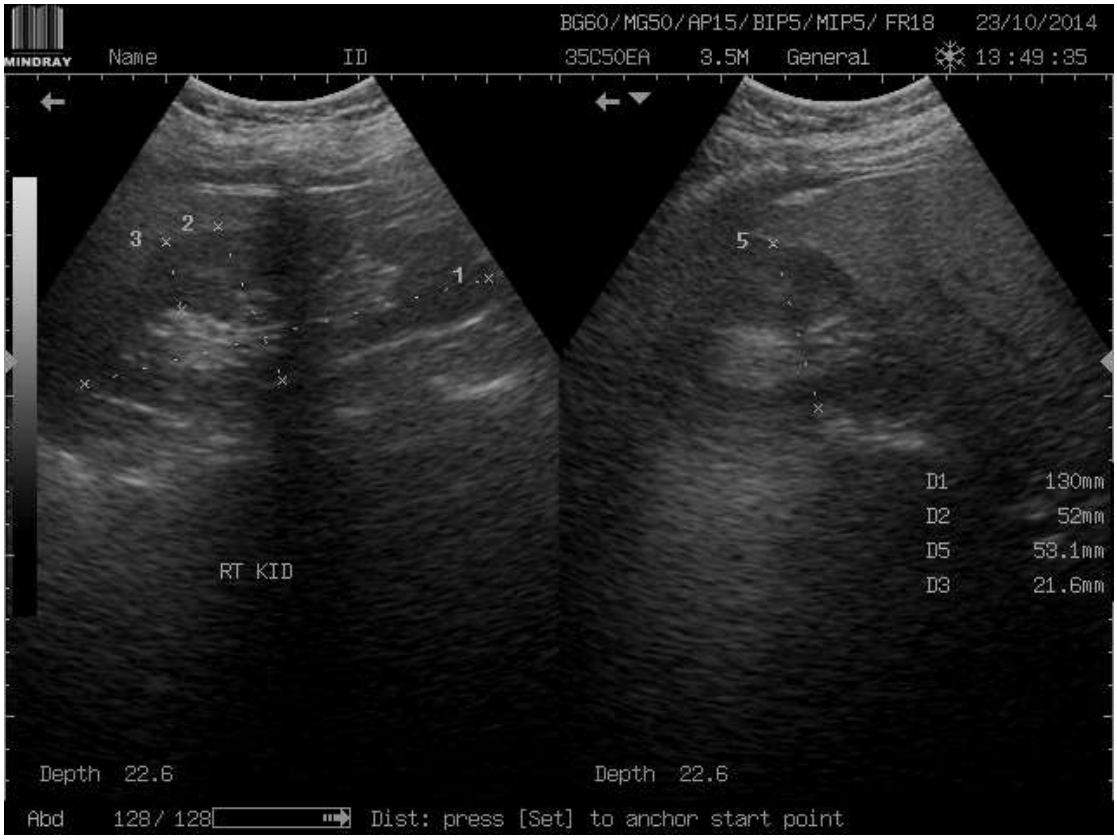

Image (5):

Patient age: 60 (Female)

Duration of the disease: DM for 5 y, HTN for 25 years

Cortical echogenicity: normal

Renal length : increased

C/M differentiation : normal

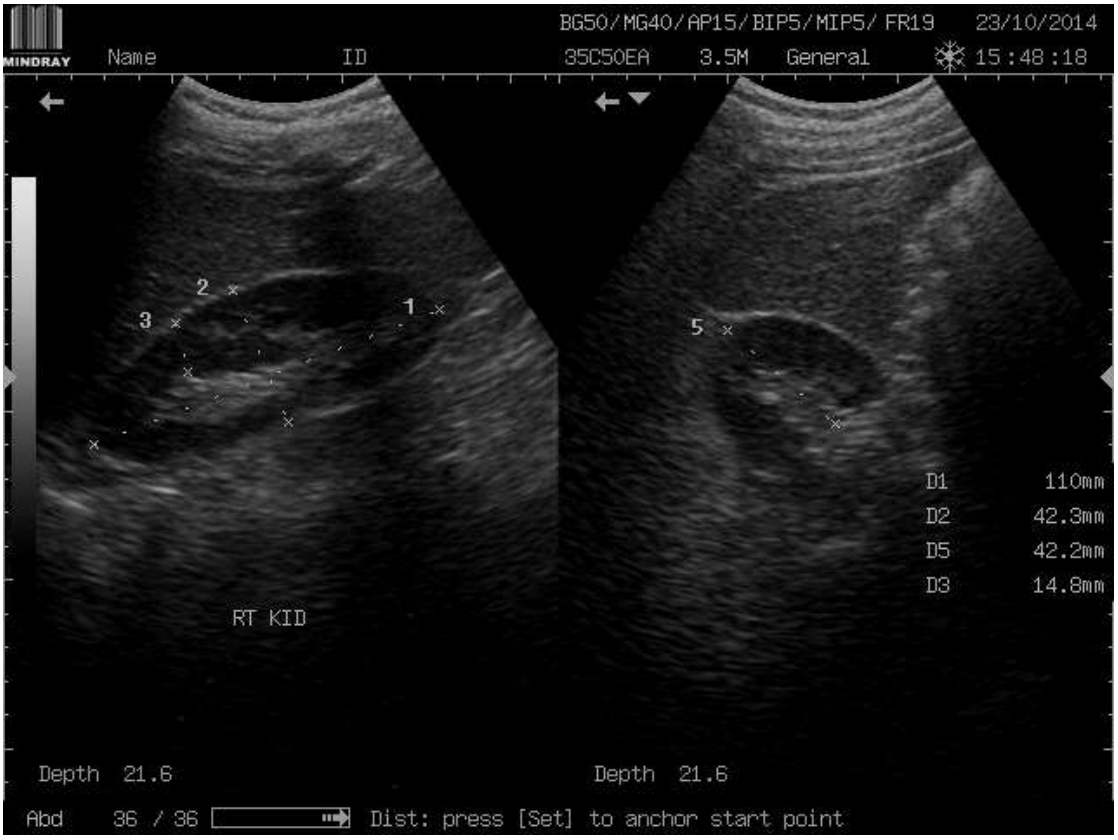

Image (6):

Patient age: 60 (male)

Duration of the disease: DM for 17 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : good

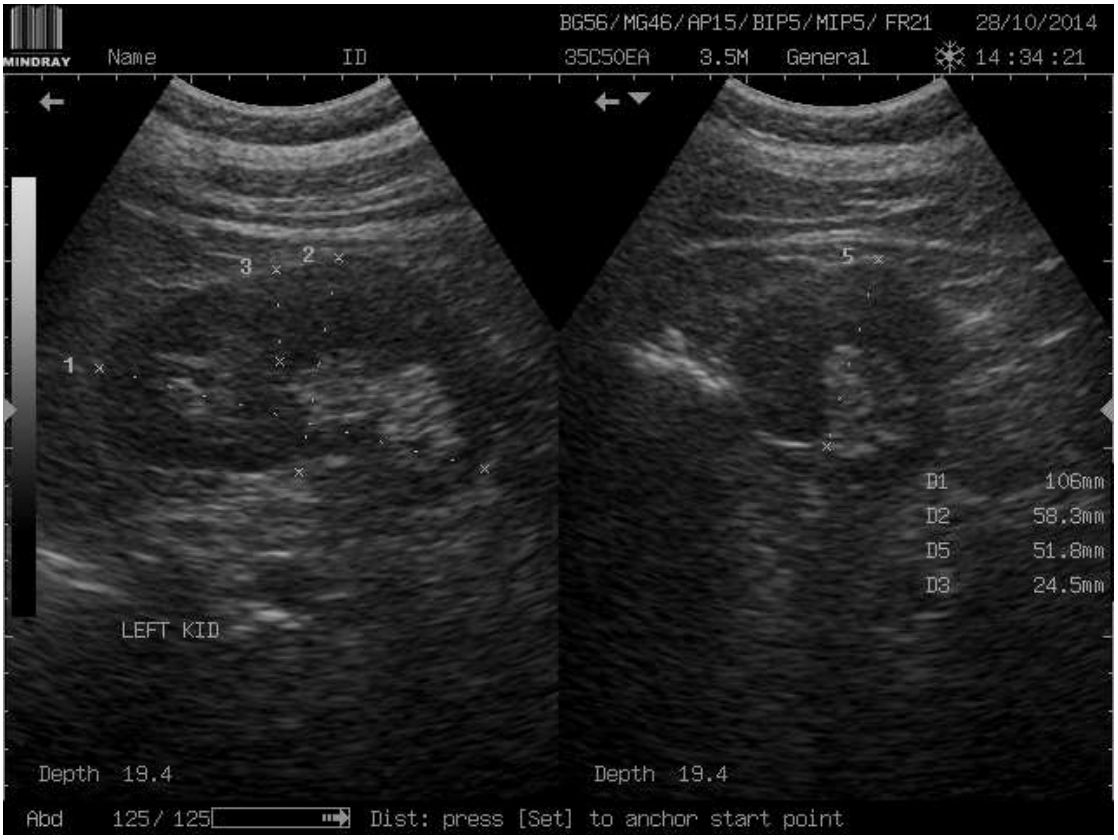

Image (7):

Patient age: 66 (male)

Duration of the disease: DM for 30 y, HTN for 5 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

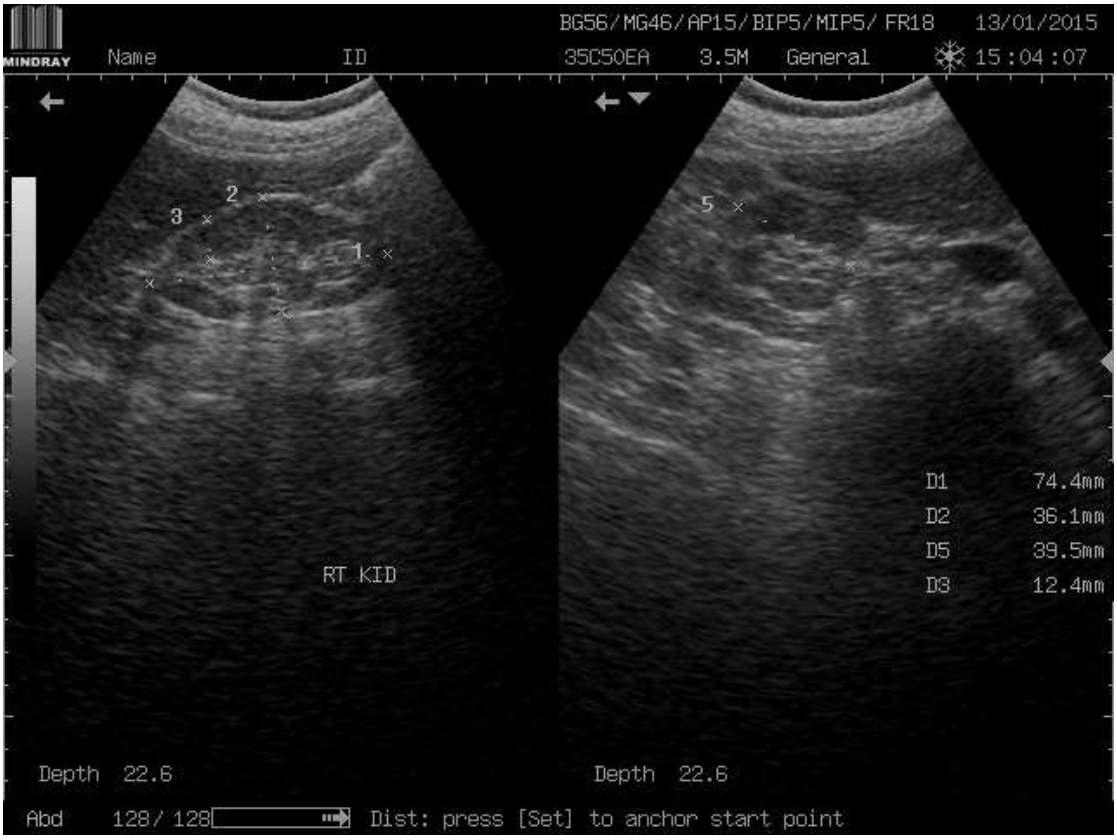

Image (8):

Patient age: 71 (male)

Duration of the disease: DM for 21 years

Cortical echogenicity: normal

Renal length : small (7.4 cm)

C/M differentiation : normal

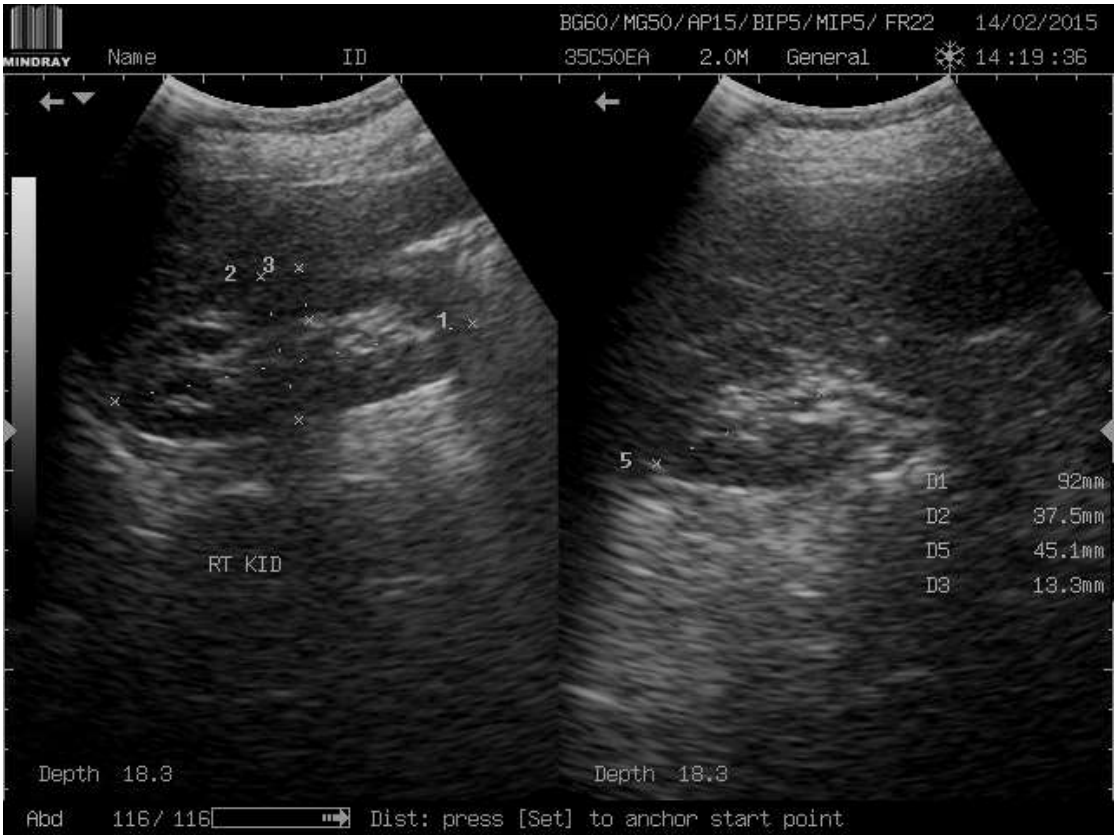

Image (9):

Patient age: 74 (male)

Duration of the disease: DM for 29 y, HTN for 13 years

Cortical echogenicity: increased

Renal length : normal

C/M differentiation : mild loss

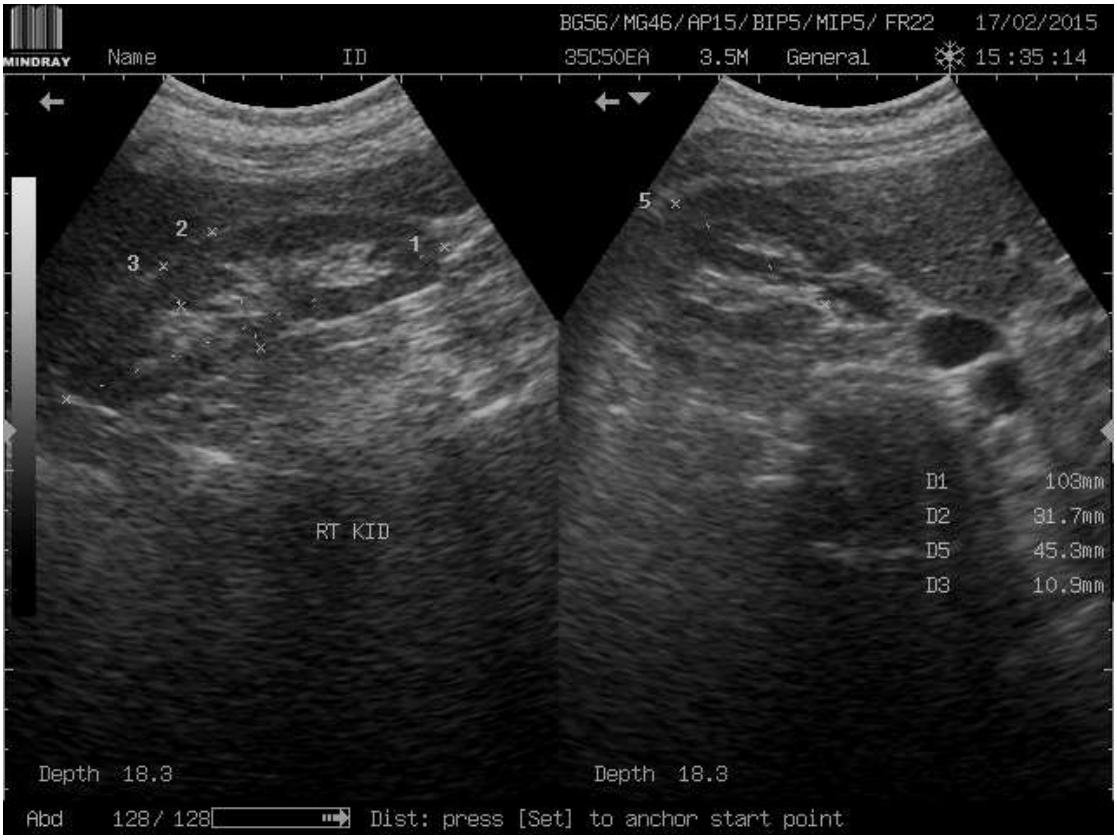

Image (10):

Patient age: 70 (Female)

Duration of the disease: DM for 15 y, HTN for 33 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

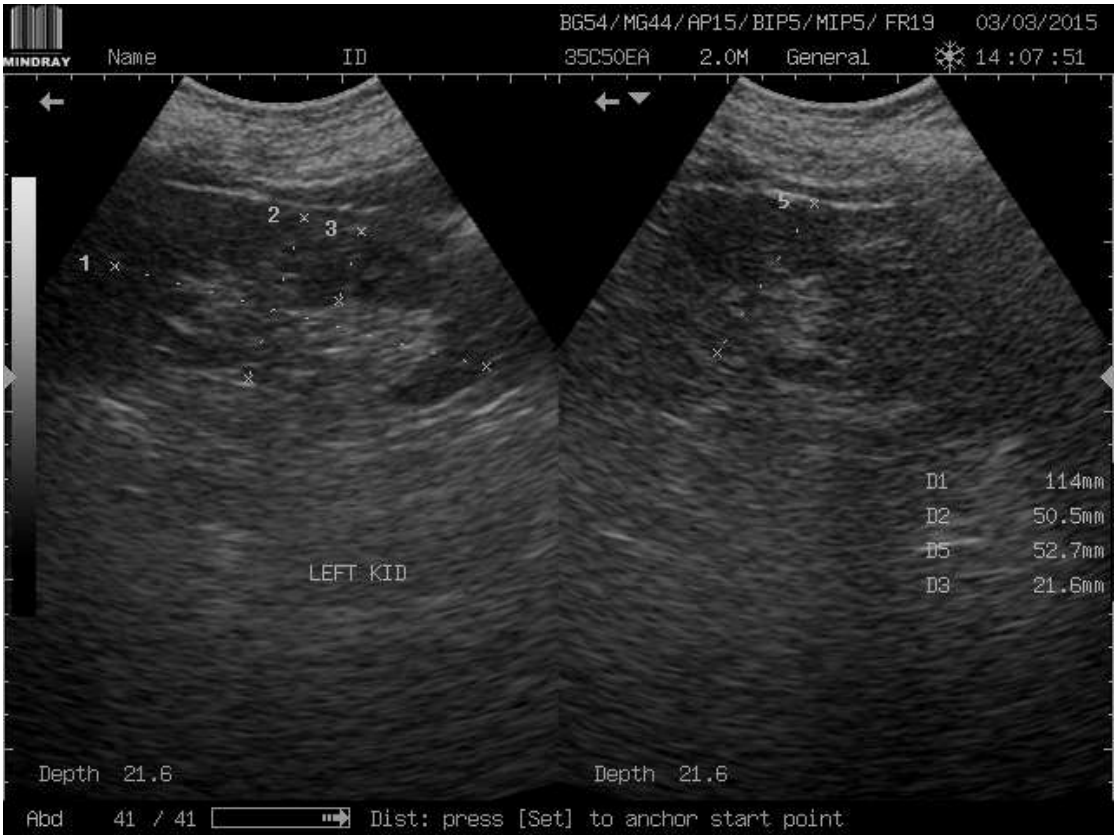

Image (11):

Patient age: 68 (female)

Duration of the disease: DM for 22 y, HTN for 22 years

Cortical echogenicity: increased

Renal length : normal

C/M differentiation : mild loss

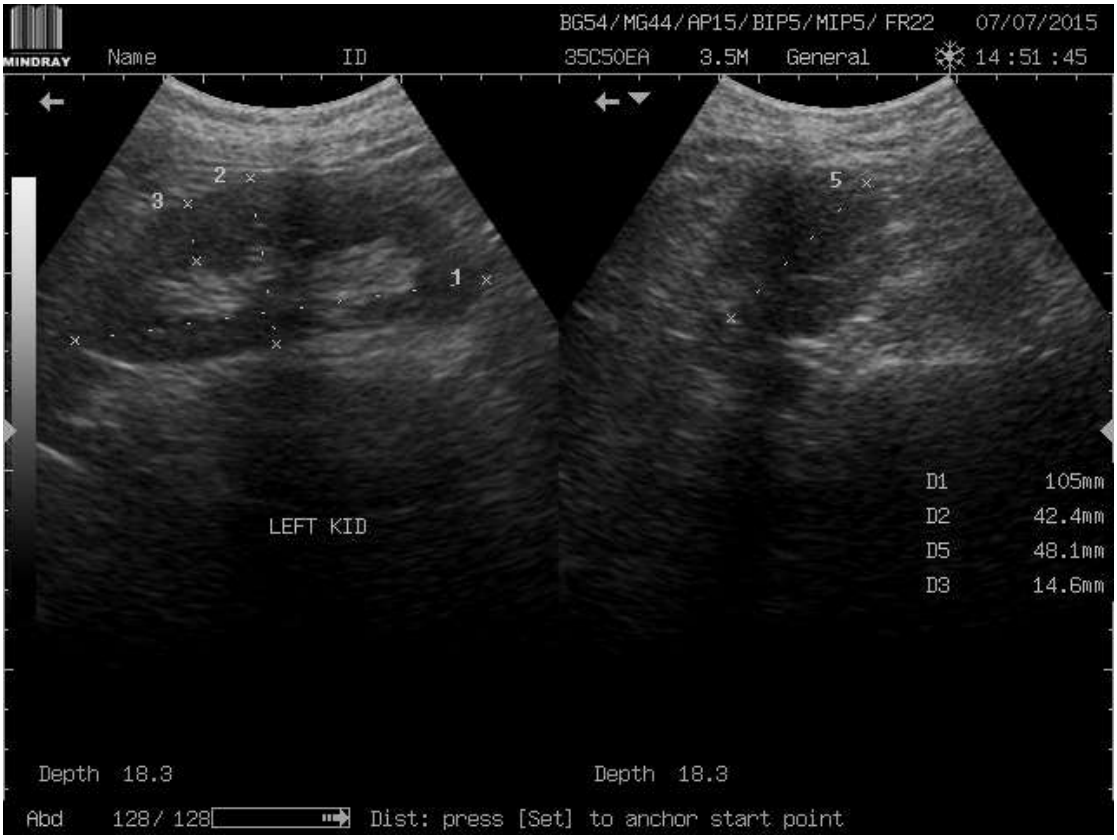

Image (12):

Patient age: 76 (female)

Duration of the disease: DM for 22 y, HTN for 10 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : good

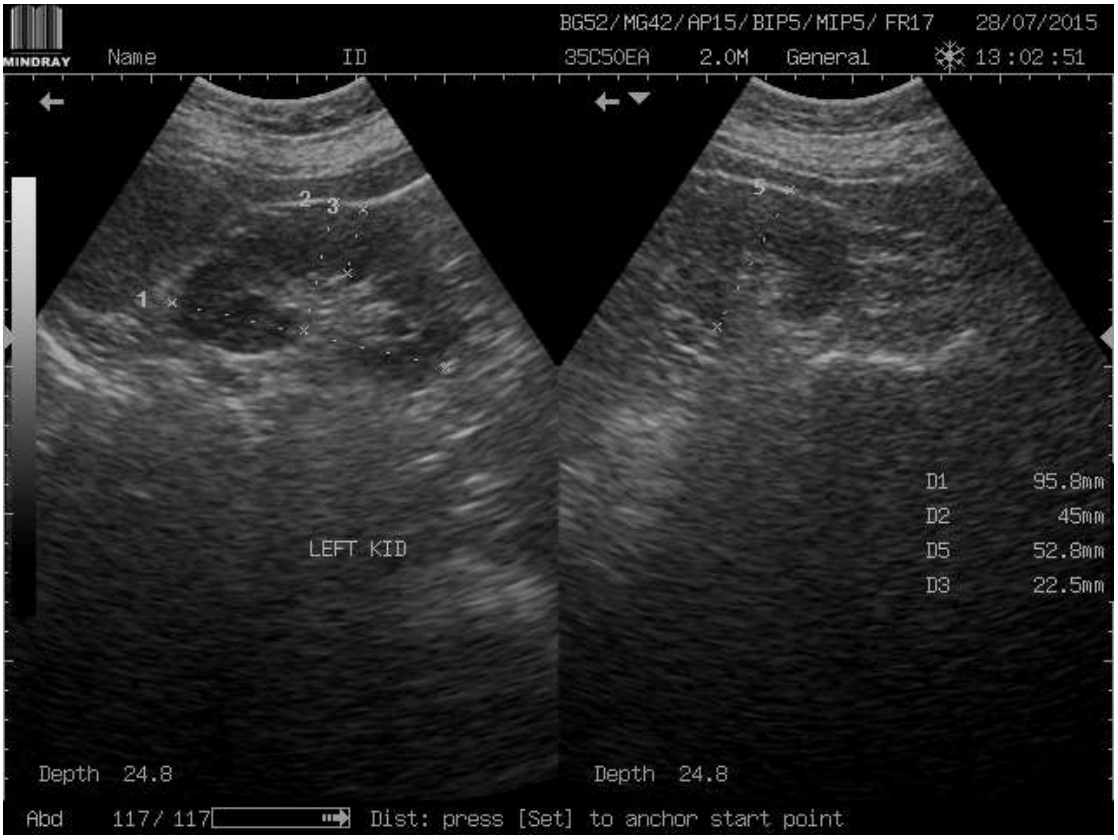

Image (13):

Patient age: 56 (female)

Duration of the disease: DM for 25 y, HTN for 26 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : good

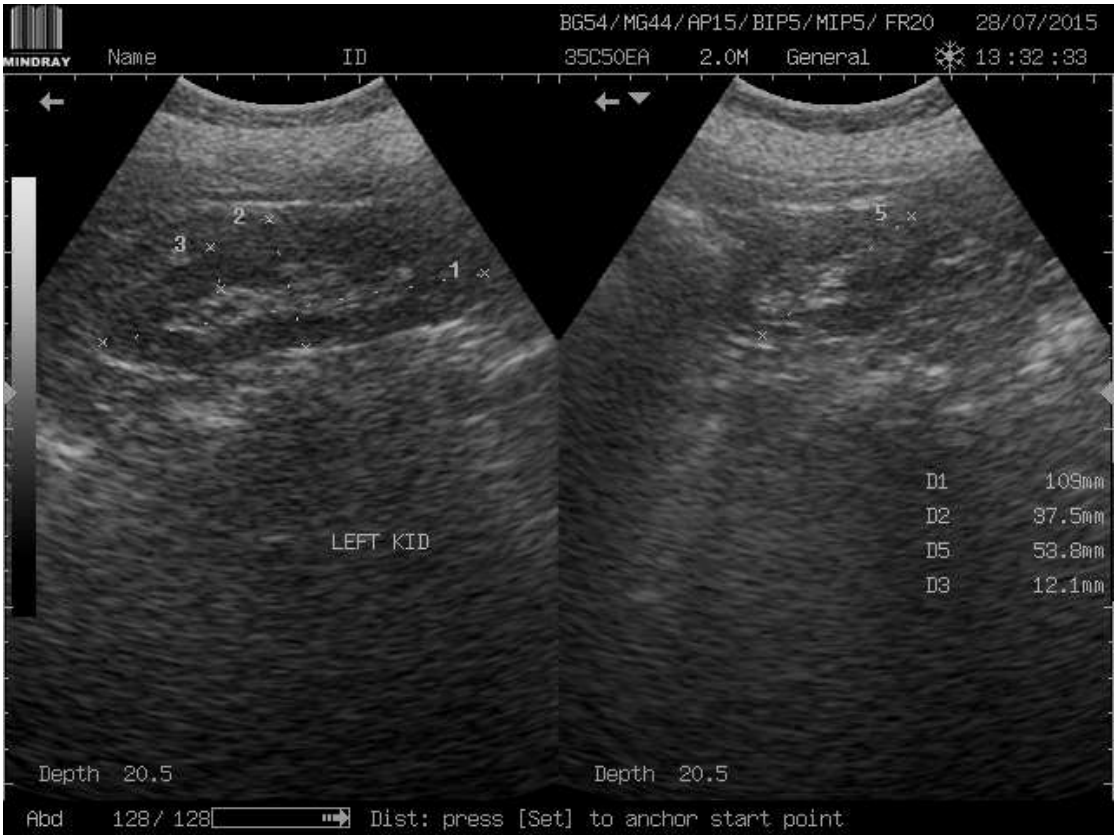

Image (14):

Patient age: 56 (female)

Duration of the disease: HTN for 15 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

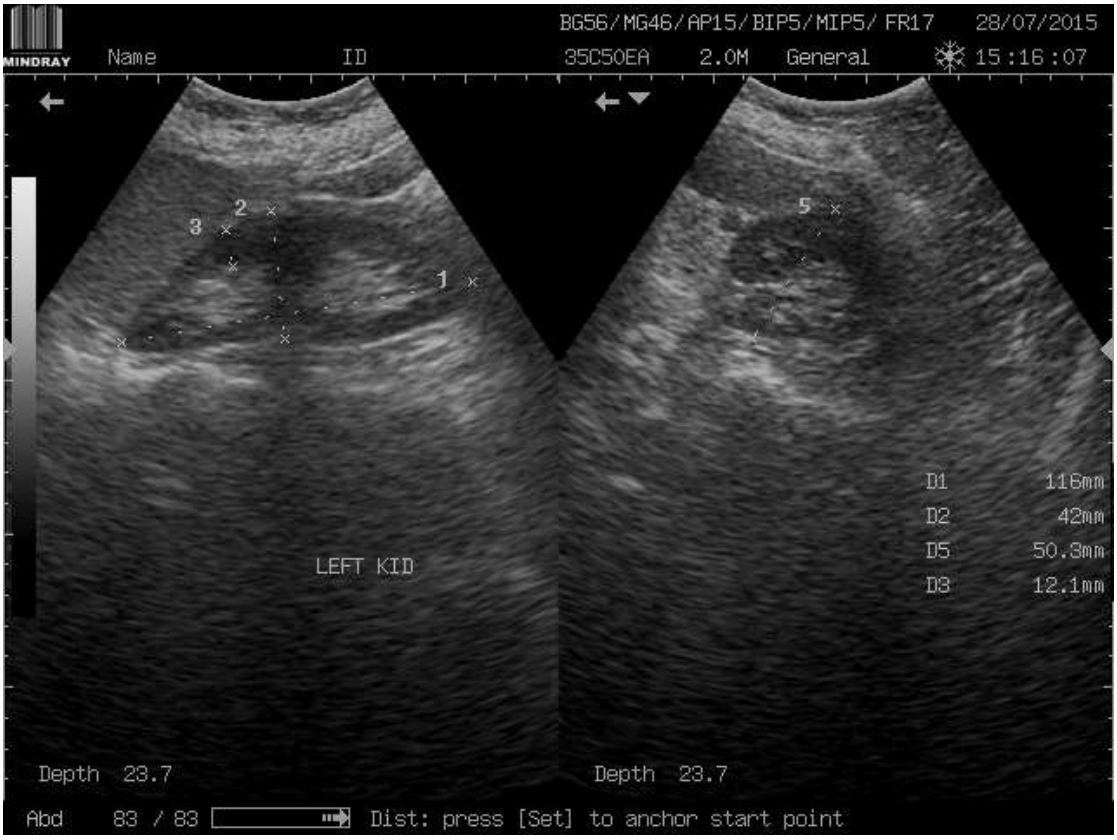

Image (15):

Patient age: 50 (female)

Duration of the disease: DM for 12 y, HTN for 10 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

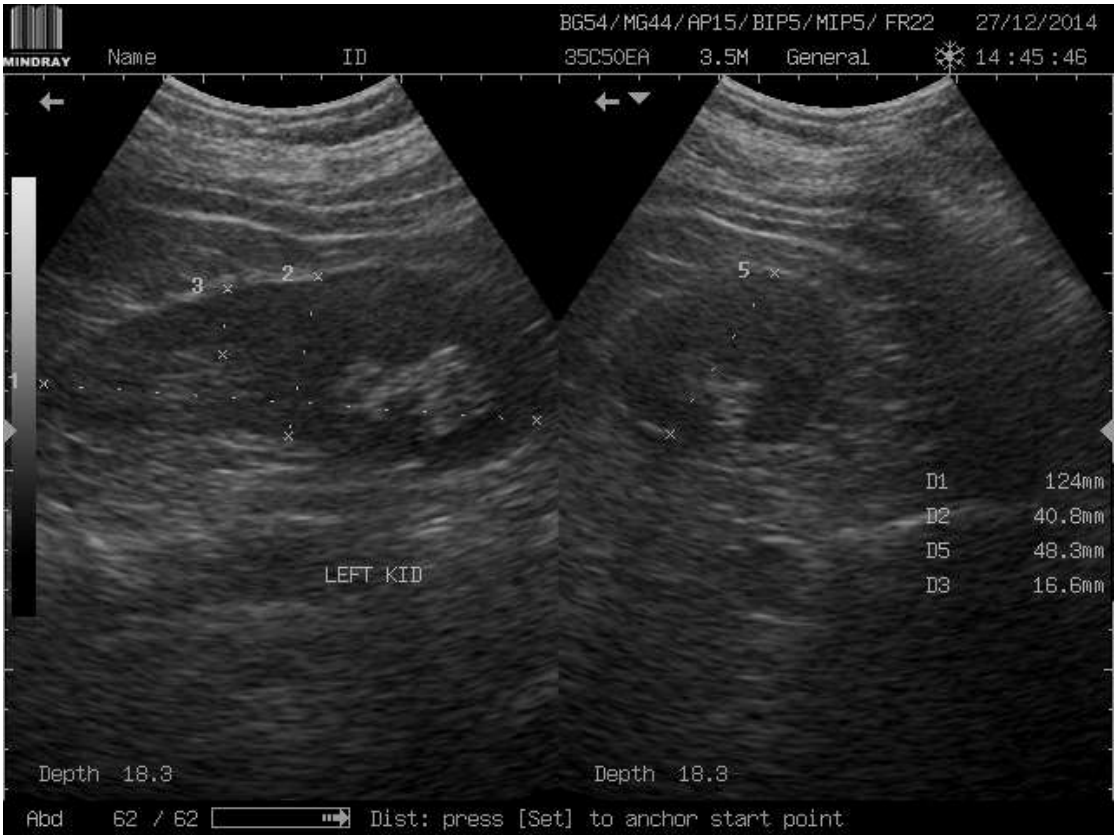

Image (16):

Patient age: 72 (male)

Duration of the disease: DM for 26 years

Cortical echogenicity: normal

Renal length : increased

C/M differentiation : normal

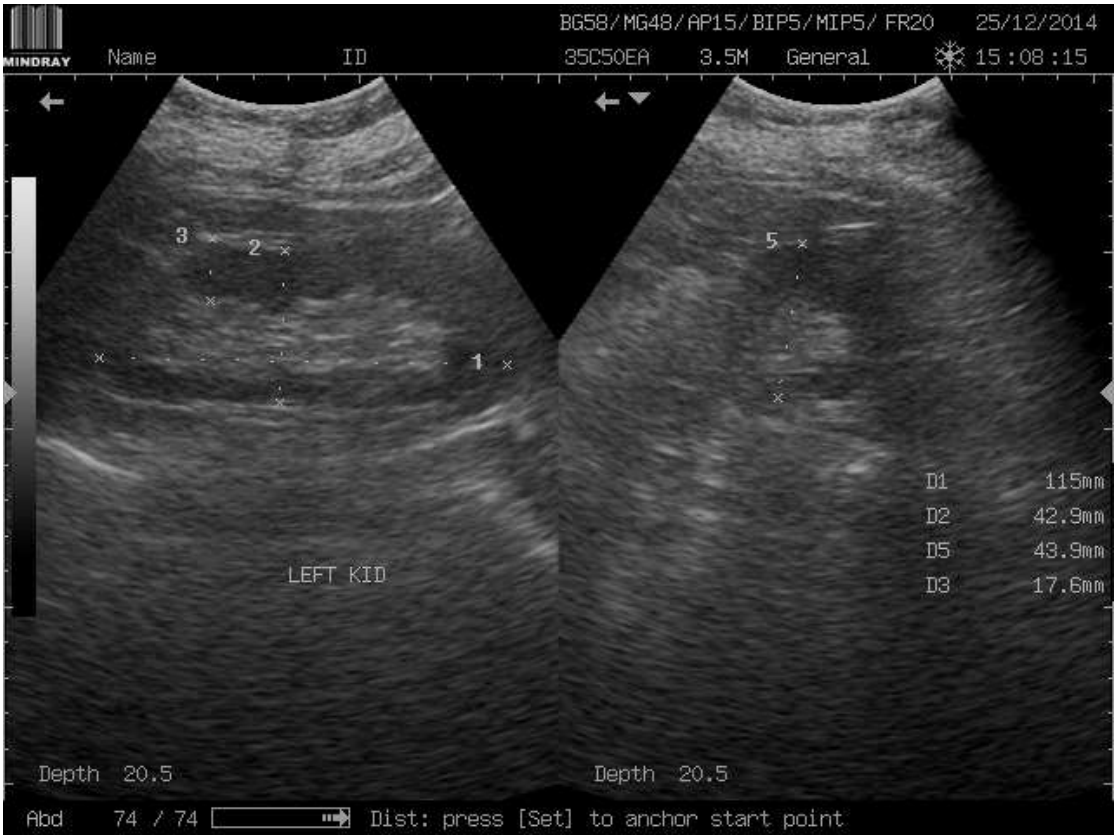

Image (17):

Patient age: 70 (female)

Duration of the disease: DM for 20 y, HTN for 21 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

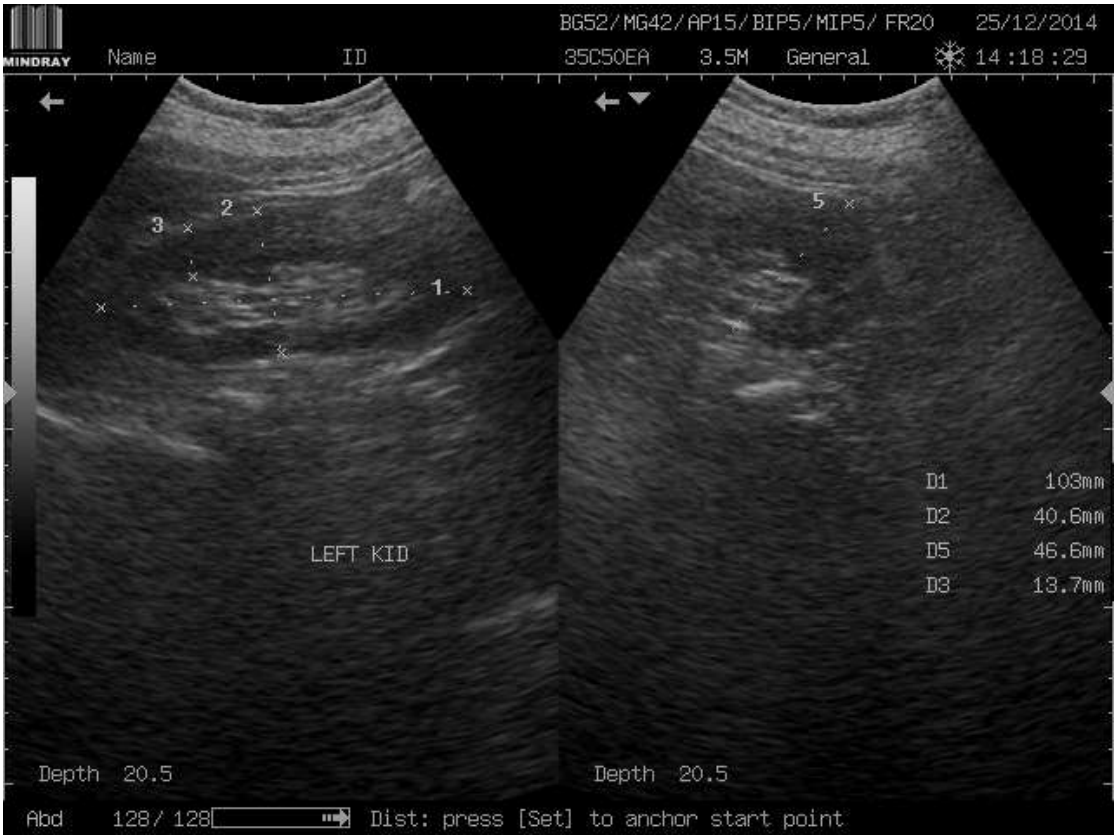

Image (18):

Patient age: 73 (female)

Duration of the disease: DM for 21 y, HTN for 7 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

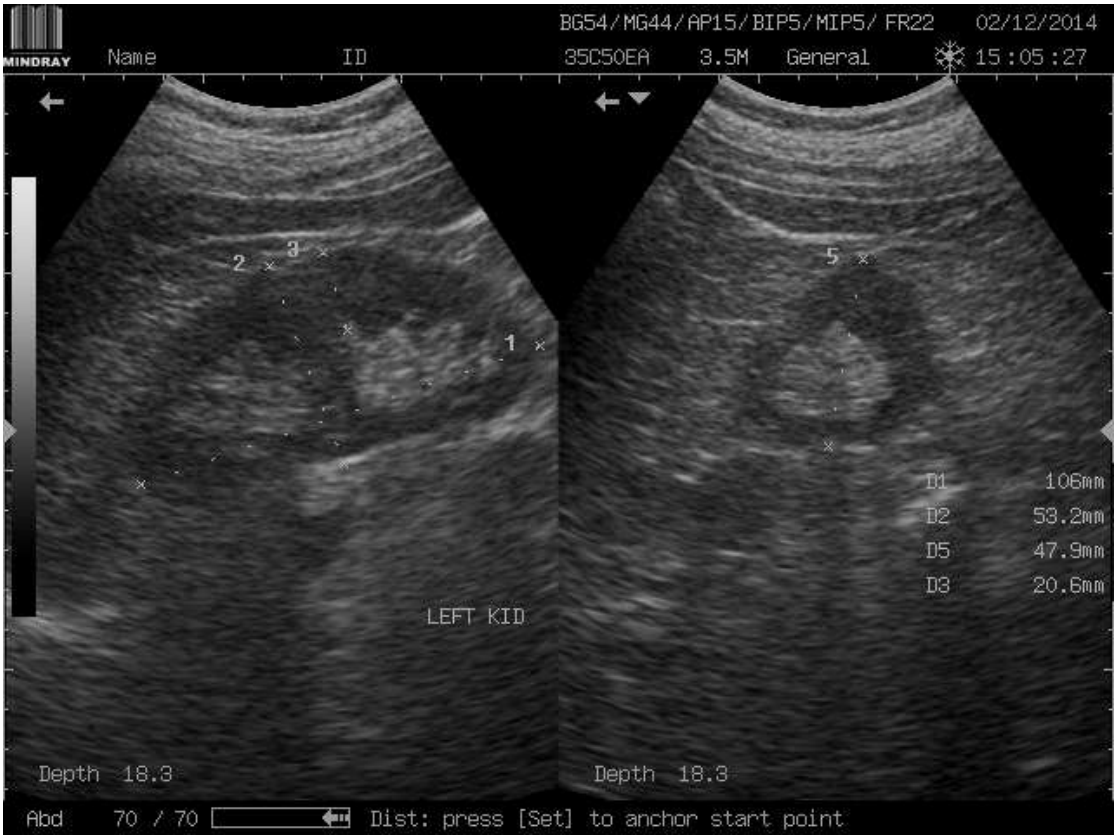

Image (19):

Patient age: 58 (male)

Duration of the disease: DM for 10 y, HTN for 5 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal

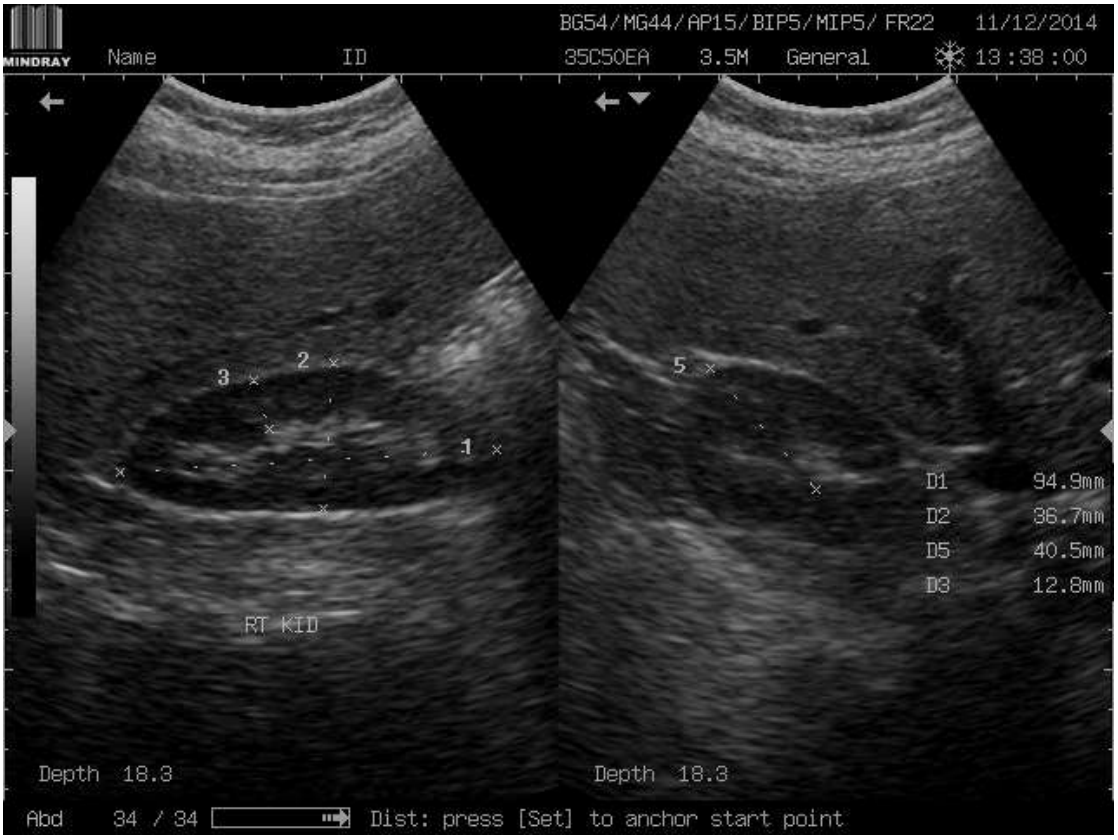

Image (20):

Patient age: 62 (female)

Duration of the disease: DM for 8 y, HTN for 6 years

Cortical echogenicity: normal

Renal length : normal

C/M differentiation : normal