Assessment Causes of Hydronephrosis among Adult Patients using Ultrasonography

تحديد أسباب الاستسقاء الكلوي عند البالغين باستخدام الموجات فوق الصوتية

A thesis Submitted for Partial Fulfillment for the Requirements
M.Sc. Degree in Medical Diagnostic Ultrasound

By:

Imad Eldin Abdala Mohamed Ali Elbashier

Supervisor:

Dr. Babiker Abd Elwahab Awad Alla

2016
قال تعالى:

(بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ (1) الحمد لله رب العالمين (2) الرحمن الرحيم (3) ملك يوم الدين (4) إياك نعبيد وإياك نستعين (5) هذين الصراط المستقيم (6) صراط الذين أنعمنا عليهم غير المغضوب عليهم ولا الضالين (7)

سورة الفاتحة - الآيات (1-7)
Dedication

To my father

To my mother

To my family

To my kids
Acknowledgement

I thank God for enabling me to complete this thesis.

I sincerely thank Dr. Babiker Abd Elwahab the supervisor of my thesis for his continuous help, supervision and guidance.

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

Especial thanks for my colleagues; Mohamed Ahmed Elshiekh.
Abstract
This a descriptive cross sectional study which was done in Omdurman clinics (Khartoum –Sudan) from June (2016) to October (2016). This thesis was conducted to detect the different causes of hydronephrosis among adult patients using ultrasound scan. There were no other research in this area in Sudan to evaluate common presentations of hydronephrosis using ultrasound scan.

To assess different causes of hydronephrosis among adult patients using ultrasound.
To evaluate hydronephrosis among patients in the study area.
To demonstrate the ultrasound changes among patients with hydronephrosis.
To correlate the presence of hydonephrosis between males and females.

There were (50) patients was scanned transabdominaly using ( Sono scape A-5) their ages more than(20)years, all these are inclusive, while less than twenty years, pregnant ladies and infants are exclusive.

This study showed that the most affected age (20 – 30) (31 – 40) 22%. The most common causes of hydronephrosis is right ureteric stone, (17) (34%) followed by left ureteric stone 8 (16%), and BPH 4 (8%). The study found the high percentage of hydronephrosis in male more than female.

Finally, U/S is the first time of investigation at the renal system. It is sensitive and accurate to determine the main causes of hydronephrosis.
مستخلص البحث


وأجري هذا البحث للكشف عن الأسباب المختلفة للاستسقاء الكلوي بين المرضى البالغين باستخدام الفحص بالموجات فوق الصوتية لم تكن هناك بحوث أخرى في هذا المجال في السودان لقييم العروض المشتركة للاستسقاء الكلوي باستخدام الفحص بالموجات فوق الصوتية.

لتقييم أسباب مختلفة من الاستسقاء الكلوي بين المرضى البالغين باستخدام الموجات فوق الصوتية

لتقييم الاستسقاء الكلوي بين المرضى في منطقة الدراسة.

لإظهار تغييرات الموجات فوق الصوتية بين المرضى الذين يعانون من استسقاء الكلية.

لربط وجود الاستسقاء الكلوي بين الذكور والإناث.

هدفت هذه الدراسة تحديد أسباب الاستسقاء الكلوي عند البالغين باستخدام الموجات فوق الصوتية.

اجري المسح البطني بالموجات فوق الصوتية استخدام جهاز سونوسكيب (5-A) على (50) مريض اعمارهم فوق (20) سنة واما المرضى الذين تقل اعمارهم عن (20) سنة والسيدات الحوامل والاطفال خارج نطاق هذه الدراسة.

وأظهرت هذه الدراسة أن الاعمار الأكثر تضارراً بين (20-30) (31-40) 22%، الأسباب الأكثر شيوعا من الاستسقاء الكلوي هي حصوة الحالبلايمن (34) 17%، يليه حصوة الحالب الأيسر (16) 4%، تضخم البروستاتا (8)

وجدت الدراسة أن هناك نسبة عالية من الاستسقاء الكلوي في الذكور أكثر من الإناث. وأخيراً فإن الموجات الصوتية هي الفحص الأولي لتحقيق من الاستسقاء الكلوي، لحساسيتها ودقتها في تحديد الأسباب الرئيسية للاستسقاء الكلوي.
List of contents

<table>
<thead>
<tr>
<th>Title</th>
<th>Page No</th>
</tr>
</thead>
<tbody>
<tr>
<td>الأية</td>
<td>I</td>
</tr>
<tr>
<td>Dedication</td>
<td>II</td>
</tr>
<tr>
<td>Acknowledgment</td>
<td>III</td>
</tr>
<tr>
<td>Abstract (English)</td>
<td>IV</td>
</tr>
<tr>
<td>Abstract (Arabic)</td>
<td>V</td>
</tr>
<tr>
<td>List of contents</td>
<td>VI-VII</td>
</tr>
<tr>
<td>List of tables</td>
<td>VIII</td>
</tr>
<tr>
<td>List of figures</td>
<td>IX</td>
</tr>
<tr>
<td>List of abbreviations</td>
<td>X-XI</td>
</tr>
</tbody>
</table>

Chapter one

1.1 Introduction               1-3
1.2 Problem of study            4
1.3 Objectives                  4
1.3.1 General objectives        4
1.3.2 Specific objectives       4
1.4 Overview of the study       4

Chapter two

**Literature Review & Previous study**

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1 Anatomy</td>
<td>5</td>
</tr>
<tr>
<td>2.1 Histology</td>
<td>12</td>
</tr>
<tr>
<td>2.2 Physiology</td>
<td>15</td>
</tr>
<tr>
<td>2.3 Pathology</td>
<td>22</td>
</tr>
<tr>
<td>2.4 Laboratory test</td>
<td>39</td>
</tr>
<tr>
<td>2.5 Sonographic scanning of Kidneys</td>
<td>40</td>
</tr>
<tr>
<td>Section</td>
<td>Page</td>
</tr>
<tr>
<td>---------</td>
<td>------</td>
</tr>
<tr>
<td>2.6 Previous study</td>
<td>43</td>
</tr>
<tr>
<td><strong>Chapter three</strong></td>
<td></td>
</tr>
<tr>
<td>3.1 Material and methodology</td>
<td>45</td>
</tr>
<tr>
<td><strong>Chapter four</strong></td>
<td></td>
</tr>
<tr>
<td>4.1 Result &amp; analysis</td>
<td>47</td>
</tr>
<tr>
<td><strong>Chapter five</strong></td>
<td></td>
</tr>
<tr>
<td>5.1 Discussion</td>
<td></td>
</tr>
<tr>
<td>5.2 Conclusion</td>
<td>66</td>
</tr>
<tr>
<td>5.3 Recommendations</td>
<td>67</td>
</tr>
<tr>
<td>References</td>
<td>68</td>
</tr>
<tr>
<td>Appendices</td>
<td></td>
</tr>
</tbody>
</table>
### List of tables

<table>
<thead>
<tr>
<th>Title</th>
<th>Page No</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1 shows function of kidney</td>
<td>18</td>
</tr>
<tr>
<td>4.1 Shows frequency distribution of patient’s age group:</td>
<td>47</td>
</tr>
<tr>
<td>4.2 Shows frequency distribution of patient’s gender</td>
<td>48</td>
</tr>
<tr>
<td>4.3 Shows frequency distribution of patient’s complain</td>
<td>49</td>
</tr>
<tr>
<td>4.4 Shows frequency distribution of patient’s other finding</td>
<td>51</td>
</tr>
<tr>
<td>4.5 Shows frequency distribution of ultrasound finding</td>
<td>53</td>
</tr>
<tr>
<td>4.6 Shows frequency distribution of patient’s occupation:</td>
<td>55</td>
</tr>
<tr>
<td>4.7 Shows frequency distribution of final diagnosed by ultrasound</td>
<td>57</td>
</tr>
<tr>
<td>4.8 Shows chi square test between age distribution and final diagnosis</td>
<td>59</td>
</tr>
<tr>
<td>4.9 Shows chi square test between us finding and us final diagnosed</td>
<td>59</td>
</tr>
<tr>
<td>4.10 Shows chi square test between gender distribution and final diagnosis</td>
<td>60</td>
</tr>
<tr>
<td>4.11 Shows chi square test between gender distribution and us finding</td>
<td>61</td>
</tr>
<tr>
<td>4.12 Shows chi square test between occupation distribution and final diagnosis</td>
<td>61</td>
</tr>
</tbody>
</table>
## List of figures

<table>
<thead>
<tr>
<th>Title</th>
<th>Page No</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1 Shows posterior surface relationship of kidneys</td>
<td>6</td>
</tr>
<tr>
<td>2.2 Shows kidney structure</td>
<td>9</td>
</tr>
<tr>
<td>2.3 3D-rendered computed tomography, showing renal arteries and veins</td>
<td>11</td>
</tr>
<tr>
<td>2.4 Shows Microscopic photograph of the renal medulla</td>
<td>13</td>
</tr>
<tr>
<td>2.5 Shows Microscopic photograph of the renal cortex</td>
<td>14</td>
</tr>
<tr>
<td>2.6 Shows function of kidney</td>
<td>16</td>
</tr>
<tr>
<td>2.7 Shows ultrasound image of hydronephrosis caused by a left ureter stone</td>
<td>27</td>
</tr>
<tr>
<td>2.8 Shows normal kidney. Longitudinal A and transverse B gray-scale sonogram.</td>
<td>42</td>
</tr>
<tr>
<td>4.1 Shows frequency distribution of patient’s age group:</td>
<td>47</td>
</tr>
<tr>
<td>4.2 Shows frequency distribution of patient’s gender</td>
<td>48</td>
</tr>
<tr>
<td>4.3 Shows frequency distribution of patient’s complain</td>
<td>50</td>
</tr>
<tr>
<td>4.4 Shows frequency distribution of patient’s other finding</td>
<td>52</td>
</tr>
<tr>
<td>4.5 Shows frequency distribution of ultrasound finding</td>
<td>54</td>
</tr>
<tr>
<td>4.6 Shows frequency distribution of patient’s occupation:</td>
<td>56</td>
</tr>
<tr>
<td>4.7 Shows frequency distribution of final diagnosed by ultrasound</td>
<td>58</td>
</tr>
</tbody>
</table>
# List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADH</td>
<td>Antidiuritic hormone</td>
</tr>
<tr>
<td>ADPRD</td>
<td>Autosomal Dominant Polycystic Renal Disease</td>
</tr>
<tr>
<td>AKI</td>
<td>Acute kidney injury</td>
</tr>
<tr>
<td>AML</td>
<td>Angiomyolipoma</td>
</tr>
<tr>
<td>AOCRF</td>
<td>Acute on chronic renal failure</td>
</tr>
<tr>
<td>ARF</td>
<td>Acute renal failure</td>
</tr>
<tr>
<td>ARPRD</td>
<td>Autosomal Recessive Polycystic Renal Disease</td>
</tr>
<tr>
<td>Bil</td>
<td>Bi lateral</td>
</tr>
<tr>
<td>BPH</td>
<td>Benign prosatatic hyperplasia</td>
</tr>
<tr>
<td>BUN</td>
<td>Blood Urea Nitrogen</td>
</tr>
<tr>
<td>CBC</td>
<td>Complete blood count</td>
</tr>
<tr>
<td>CKD</td>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>CRF</td>
<td>Chronic Renal Failure</td>
</tr>
<tr>
<td>CT</td>
<td>Computed tomography</td>
</tr>
<tr>
<td>EPN</td>
<td>Emphysematous Pyelonephritis</td>
</tr>
<tr>
<td>FENA</td>
<td>Fractional Excretion of Sodium</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>FF</td>
<td>Filtration Fraction</td>
</tr>
<tr>
<td>GFR</td>
<td>Glomerular filtration rate</td>
</tr>
<tr>
<td>IVU</td>
<td>Intera venous urography</td>
</tr>
<tr>
<td>KUB</td>
<td>Kidney Ureter Bladder</td>
</tr>
<tr>
<td>LT</td>
<td>Left</td>
</tr>
<tr>
<td>MCDK</td>
<td>Multicystic Dysplastic Kidney</td>
</tr>
<tr>
<td>PCS</td>
<td>Pelvicalyceal system</td>
</tr>
<tr>
<td>R CC</td>
<td>Renal Cell Cacinoma</td>
</tr>
<tr>
<td>RPF</td>
<td>Renal plasma flow</td>
</tr>
<tr>
<td>RT</td>
<td>Right</td>
</tr>
<tr>
<td>TCC</td>
<td>Transitional Cell Cacinoma</td>
</tr>
<tr>
<td>UPJ</td>
<td>Ureter pelvic junction</td>
</tr>
<tr>
<td>UPJO</td>
<td>Ureteropelvic junction obstruction</td>
</tr>
<tr>
<td>US</td>
<td>Ultrasound</td>
</tr>
<tr>
<td>VCGU</td>
<td>Voidiningcysourethrogram</td>
</tr>
</tbody>
</table>
Chapter One

Introduction
1.1 Introduction:
Hydronephrosis refers to distension and dilation of the renal pelvis and calyces, usually caused by obstruction of the free flow of urine from the kidney. Untreated, it leads to progressive atrophy of the kidney. (Kumar, 2005)

One or both kidneys may be affected. In cases of hydroureter and hydronephrosis, there is distention of both the ureter and the renal pelvis and calices. (Tamparo, 2011)

The signs and symptoms of hydronephrosis depend upon whether the obstruction is acute or chronic, partial or complete, unilateral or bilateral. Hydronephrosis that occurs acutely with sudden onset (as caused by a pain in the ribs). hydronephrosis that develops gradually will generally cause no pain or attacks of a dull discomfort. Nausea and vomiting may also occur. An obstruction that occurs at the urethra or bladder outlet can cause pain and pressure resulting from distension of the bladder. Blocking the flow of urine will commonly result in urinary tract infections which can lead to the development of additional stones, fever, and blood or pus in the urine. If complete obstruction occurs, kidney failure may follow. (Merck, 2009)

Blood tests may show impaired kidney function (elevated urea or creatinine) or electrolyte imbalances such as hyponatremia or hyperchloremic metabolic acidosis. Urinalysis may indicate an elevated pH due to the secondary destruction of nephrons within the affected kidney. Physical examination may detect a palpable abdominal or flank mass caused by the enlarged kidney.

Hydronephrosis is the result of any of several abnormal pathophysiological occurrences. Structural abnormalities of the junctions between the kidney, ureter, and bladder that lead to hydronephrosis can occur during fetal development. Some of these congenital defects have been identified as inherited conditions; however the benefits of linking genetic testing to early
diagnosis have not been determined. (Toka HR, 2010) Other structural abnormalities could be caused by injury, surgery, or radiation therapy. Compression of one or both ureters can also be caused by other developmental defects not completely occurring during the fetal stage such as an abnormally placed vein, artery, or tumor. Bilateral compression of the ureters can occur during pregnancy due to enlargement of the uterus. Changes in hormone levels during this time may also affect the muscle contractions of the bladder, further complicating this condition. Sources of obstruction that can arise from other various causes include kidney stones, blood clots, or retroperitoneal fibrosis. (Koh JS, et al, 1998)

The obstruction may be either partial or complete and can occur anywhere from the urethral meatus to the calyces of the renal pelvis. Hydronephrosis can also result from the reverse flow of urine from the bladder back into the kidneys. This reflux can be caused by some of the factors listed above as well as compression of the bladder outlet into the urethra by prostatic enlargement or impaction of feces in the colon, as well as abnormal contractions of bladder muscles resulting from neurological dysfunction or other muscular disorders. (Merck, 2009)

Diagnostic workup depends on the age of the patient, as well as whether the hydronephrosis was detected incidentally or prenatally or is associated with other symptoms.

Blood tests (such measurement of creatinine) are typically indicated, though they must be interpreted cautiously. Even in cases of severe unilateral hydronephrosis, the overall kidney function may remain normal since the unaffected kidney will compensate for the obstructed kidney. Urinalysis is usually performed to determine the presence of blood (which is typical for kidney stones) or signs of infection (such as a positive leukocyte esterase or nitrite). Impaired concentrating ability or elevated urine pH
(distal renal tubular acidosis) are also commonly found due to tubular stress and injury.

Imaging studies, such as an intravenous urogram (IVU), ultrasound, CT or MRI, are also important investigations in determining the presence and/ or cause of hydronephrosis. (Kay& Robert, 1999)

Ultrasound is non-invasive, inexpensive and repeatable modility and has been used as important and valuable diagnostic tool for detecting renal diseases. An ultrasound evaluation of hydrenalisis has been performed by assessing type of hydronephrosis and causes.

1-2 Problem;

Hydronephrosis may cause renal failure, for this reason early diagnosis, detected of causes and management is important.

1-3 Objectives:

1.3.1 General objective
To assess different causes of hydronephrosis among adult patients using ultrasound.

1.3.2 Specific objectives
- To evaluate hydronephrosis among patients in the study area.
- To demonstrate the ultrasound changes among patients with hydronephrosis.
- To correlate the causes of hydronephrosis between males and females.

1.4 Overview of the study
This study consisted of five chapters with chapter one is an introduction which includes problem and objective of the study, chapter two is a literature review which includes (Anatomy, Physiology, Pathology and previous study), chapter three deal with research methodology, chapter four deal with result and chapter five discussion, conclusion and recommendation.
Chapter Two

Literature Reviews

&background studies
Chapter Two
Literature Reviews

2.1 Anatomy

The kidneys are bean-shaped organs that serve several essential regulatory roles in vertebrates. They remove excess organic molecules from the blood, and it is by this action that their best-known function is performed: the removal of waste products of metabolism. Kidneys are essential to the urinary system and also serve homeostatic functions such as the regulation of electrolytes, maintenance of acid–base balance, and regulation of blood pressure (via maintaining the salt and water balance). They serve the body as a natural filter of the blood, and remove water-soluble wastes which are diverted to the bladder. In producing urine, the kidneys excrete wastes such as urea and ammonium. They are also responsible for the reabsorption of water, glucose, and amino acids. The kidneys also produce hormones including calcitriol and erythropoietin. An important enzyme renin is also produced in the kidneys which acts in negative feedback. Located at the near of the abdominal cavity in the retroperitoneal space, the kidneys receive blood from the paired renal arteries, and drain into the paired renal veins. Each kidney excretes urine into a ureter which empties into the bladder. Renal physiology is the study of kidney function, while nephrology is the medical specialty concerned with kidney diseases. Diseases of the kidney are diverse, but individuals with kidney disease frequently display characteristic clinical features. Common clinical conditions involving the kidney include the nephritic and nephrotic syndromes, renal cysts, acute kidney injury, chronic kidney disease, urinary tract infection, nephrolithiasis, and urinary tract obstruction. (cotan et al 2005) Various cancers of the kidney exist. The most common adult renal cancer is renal cell carcinoma. Cancers, cysts, and some other renal conditions can be managed with removal of the
kidney. This is known as nephrectomy. When renal function, measured by the glomerular filtration rate, is persistently poor, dialysis and kidney transplantation may be treatment options. Although they are not normally harmful, kidney stones can be extremely painful.

Fig(2.1) Shows posterior surface relationship of kidneys (Henary Gray, 1918)
2.1.1 Location:

In humans, the kidneys are located in the abdominal cavity, one on each side of the spine, and lie in a retroperitoneal position at a slightly oblique angle. The asymmetry within the abdominal cavity, caused by the position of the liver, typically results in the right kidney being slightly lower and smaller than the left, and being placed slightly more to the middle than the left kidney. The left kidney is approximately at the vertebral level T12 to L3, and the right is slightly lower. The right kidney sits just below the diaphragm and posterior to the liver. The left sits below the diaphragm and posterior to the spleen. On top of each kidney is an adrenal gland. The upper parts of the kidneys are partially protected by the 11th and 12th ribs. Each kidney, with its adrenal gland, is surrounded by two layers of fat: the perirenal and pararenal fat and the renal fascia. In adult males, the kidney weighs between 125 and 170 grams. In females the weight of the kidney is between 115 and 155 grams. (Walter F 2004)

2.1.2 Structure

The kidney has a bean-shaped structure having a convex and a concave border. A recessed area on the concave border is the renal hilum, where the renal artery enters the kidney and the renal vein and ureter leave. The kidney is surrounded by tough fibrous tissue, the renal capsule, which is itself surrounded by perirenal fat (adipose capsule), renal fascia, and pararenal fat (paranephric body). The anterior (front) surface of these tissues is the peritoneum, while the posterior (rear) surface is the transversalis fascia. The superior pole of the right kidney is adjacent to the liver. For the left kidney, it's next to the spleen. Both, therefore, move down upon inhalation. The kidney is approximately 11–14 cm (4.3–5.5 in) in length, 6 cm (2.4 in) wide and 4 cm (1.6 in) thick. The substance, or parenchyma, of the
kidney is divided into two major structures: the outer renal cortex and the inner renal medulla. Grossly, these structures take the shape of eight to 18 cone-shaped renal lobes, each containing renal cortex surrounding a portion of medulla called a renal pyramid (of Malpighi). (Walter F 2004)

Between the renal pyramids are projections of cortex called renal columns (or Bertin columns). Nephrons, the urine-producing functional structures of the kidney, span the cortex and medulla. The initial filtering portion of a nephron is the renal corpuscle which is located in the cortex. This is followed by a renal tubule that passes from the cortex deep into the medullary pyramids. Part of the renal cortex, a medullary ray is a collection of renal tubules that drain into a single collecting duct.

The tip, or papilla, of each pyramid empties urine into a minor calyx; minor calyces empty into major calyces, and major calyces empty into the renal pelvis. This becomes the ureter. At the hilum, the ureter and renal vein exit the kidney and the renal artery enters. Hilar fat and lymphatic tissue with lymph nodes surrounds these structures. The hilar fat is contiguous with a fat-filled cavity called the renal sinus. The renal sinus collectively contains the renal pelvis and calyces and separates these structures from the renal medullary tissue (Clap, 2003)
Fig(2.2) Shows kidney structure (Piotr Michal, 2006)

1. Renal pyramid  •  2. Interlobular artery  •  3. Renal artery  •  4. Renal vein  
5. Renal hilum  •  6. Renal pelvis  •  7. Ureter  •  8. Minor calyx  •  
9. Renal capsule  •  10. Inferior renal capsule  •  11. Superior renal capsule  •  
15. Major calyx  •  16. Renal papilla
2.1.3 Blood supply

The renal circulation supplies the blood to the kidneys via 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 arteriole that supply the glomeruli. The medullary interstitium is the functional space in the kidney beneath the individual filters (glomeruli), which are rich in blood vessels. The interstitium 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. As with the arteriole distribution, the veins follow the same pattern: 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.
2.1.4 Histology

Renal histology studies the microscopic structure of the kidney. Distinct cell types include: Kidney glomerulus parietal cell, Kidney glomerulus podocyte, Kidney proximal tubule brush border cell, Loop of Henle thin segment cell, Thick ascending limb cell, Kidney distal tubule cell, Collecting duct principal cell, Collecting duct intercalated cell, and Interstitial kidney cells.

The renal artery enters into the kidney at the level of the first lumbar vertebra just below the superior mesenteric artery. As it enters the kidney, it divides into branches: first the segmental artery, which divides into 2 or 3 lobar arteries, then further divides into interlobar arteries, which further divide into the arcuate artery, which leads into the interlobular artery, which form afferent arterioles. The afferent arterioles form the glomerulus (network of capillaries enclosed in Bowman's capsule). From here, efferent arterioles
leaves the glomerulus and divide into peritubular capillaries, which drain into the interlobular veins and then into arcuate vein and then into interlobar vein, which runs into lobar vein, which opens into the segmental vein and which drains into the renal vein, and then from it blood moves into the inferior vena cava.

Fig(2.4) Show.Microscopic photograph of the renal medulla(Human kidney 2007)

2.1.5 Innervation
The kidney and nervous system communicate via the renal plexus, whose fibers course along the renal arteries to reach each kidney. Input from the sympathetic nervous system triggers vasoconstriction in the kidney, thereby reducing renal blood flow. The kidney also receives input from
the parasympathetic nervous system, by way of the renal branches of the vagus nerve (cranial nerve X); the function of this is yet unclear. Sensory input from the kidney travels to the T10-11 levels of the spinal cord and is sensed in the corresponding dermatome. Thus, pain in the flank region may be referred from corresponding kidney (Bard 2003).
2.2 Renal physiology

The kidney participates in whole-body homeostasis, regulating acid-base balance, electrolyte concentrations, extracellular fluid volume, and blood pressure. The kidney accomplishes these homeostatic functions both independently and in concert with other organs, particularly those of the endocrine system. Various endocrine hormones coordinate these endocrine functions; these include renin, angiotensin II, aldosterone, antidiuretic hormone, and atrial natriuretic peptide, among others. Many of the kidney's functions are accomplished by relatively simple mechanisms of filtration, reabsorption, and secretion, which take place in the nephron. Filtration, which takes place at the renal corpuscle, is the process by which cells and large proteins are filtered from the blood to make an ultrafiltrate that eventually becomes urine. The kidney generates 180 liters of filtrate a day, while reabsorbing a large percentage, allowing for the generation of only approximately 2 liters of urine. Reabsorption is the transport of molecules from this ultrafiltrate and into the blood. Secretion is the reverse process, in which molecules are transported in the opposite direction, from the blood into the urine. (Madhero 2010)
Fig(2.6) Shows function of kidney (Madhero 2010)

Excretion = Filtration – Reabsorption + Secretion
2.2.1 Excretion of wastes

The kidneys excrete a variety of waste products produced by metabolism into the urine. These include the nitrogenous wastes urea, from protein catabolism, and uric acid, from nucleic acid metabolism. The ability of mammals and some birds to concentrate wastes into a volume of urine much smaller than the volume of blood from which the wastes were extracted is dependent on an elaborate counter current multiplication mechanism. This requires several independent nephron characteristics to operate: a tight hairpin configuration of the tubules, water and ion permeability in the descending limb of the loop, water impermeability in the ascending loop, and active ion transport out of most of the ascending limb. In addition, passive countercurrent exchange by the vessels carrying the blood supply to the nephron is essential for enabling this function.

2.2.2 Reabsorption of vital nutrients

Glucose at normal plasma levels is completely reabsorbed in the proximal tubule. The mechanism for this is the Na+/glucose cotransporter. (cotran, et al., 2005)

A plasma level of 350 mg/dL will fully saturate the transporters and glucose will be lost in the urine. A plasma glucose level of approximately 160 is sufficient to allow glucosuria, which is an important clinical clue to diabetes mellitus. Amino acids are reabsorbed by sodium dependent transporters in the proximal tubule. Hartnup disease is a deficiency of the tryptophan amino acid transporter, which results in pellagra. Pregnancy reduces the reabsorption of glucose and amino acid (Le Tao 2013)
Table (2.1) shows function of kidney (Le Tao 2013)

### 2.2.3 Acid-base homeostasis

Two organ systems, the kidneys and lungs, maintain acid-base homeostasis, which is the maintenance of pH around a relatively stable value. The lungs contribute to acid-base homeostasis by regulating carbon dioxide (CO₂) concentration. The kidneys have two very important roles in maintaining the acid-base balance: to reabsorb and regenerate bicarbonate from urine, and to excrete hydrogen ions and fixed acids (anions of acids) into urine.
2.2.4 Osmolality regulation

Any significant rise in plasma osmolality is detected by the hypothalamus, which communicates directly with the posterior pituitary gland. An increase in osmolality causes the gland to secrete antidiuretic hormone (ADH), resulting in water reabsorption by the kidney and an increase in urine concentration. The two factors work together to return the plasma osmolality to its normal levels. ADH binds to principal cells in the collecting duct that translocate aquaporins to the membrane, allowing water to leave the normally impermeable membrane and be reabsorbed into the body by the vasa recta, thus increasing the plasma volume of the body. There are two systems that create a hyperosmotic medulla and thus increase the body plasma volume: Urea recycling and the 'single effect.' Urea is usually excreted as a waste product from the kidneys. However, when plasma blood volume is low and ADH is released the aquaporins that are opened are also permeable to urea. This allows urea to leave the collecting duct into the medulla creating a hyperosmotic solution that 'attracts' water. Urea can then re-enter the nephron and be excreted or recycled again depending on whether ADH is still present or not.

The 'Single effect' describes the fact that the ascending thick limb of the loop of Henle is not permeable to water but is permeable to NaCl. This allows for a countercurrent exchange system where by the medulla becomes increasingly concentrated, but at the same time setting up an osmotic gradient for water to follow should the aquaporins of the collecting duct be opened by ADH.
2.2.5 Blood pressure regulation:

Although the kidney cannot directly sense blood, long-term regulation of blood pressure predominantly depends upon the kidney. This primarily occurs through maintenance of the extracellular fluid compartment, the size of which depends on the plasma sodium concentration. Renin is the first in a series of important chemical messengers that make up the renin-angiotensin system. Changes in renin ultimately alter the output of this system, principally the hormones angiotensin II and aldosterone. Each hormone acts via multiple mechanisms, but both increase the kidney's absorption of sodium chloride, thereby expanding the extracellular fluid compartment and raising blood pressure. When renin levels are elevated, the concentrations of angiotensin II and aldosterone increase, leading to increased sodium chloride reabsorption, expansion of the extracellular fluid compartment, and an increase in blood pressure. Conversely, when renin levels are low, angiotensin II and aldosterone levels decrease, contracting the extracellular fluid compartment, and decreasing blood pressure. (Le Tao 2013)

2.2.6 Hormone secretion:

The kidneys secrete a variety of hormones, including erythropoietin, and the enzyme renin. Erythropoietin is released in response to hypoxia (low levels of oxygen at tissue level) in the renal circulation. It stimulates erythropoiesis (production of red blood cells) in the bone marrow. Calcitriol, the activated form of vitamin D, promotes intestinal absorption of calcium and the renal reabsorption of phosphate. Part of the renin–angiotensin–aldosterone system, renin is an enzyme involved in the regulation of aldosterone levels) (Le Tao 2013)
2.3 Pathology

2.3.1 Congenital:

Congenital hydronephrosis, Congenital obstruction of urinary tract, Duplex kidneys, or double kidneys, occur in approximately 1% of the population. This occurrence normally causes no complications, but can occasionally cause urine infections. Duplicated ureter occurs in approximately one in 100 live births. Horseshoe kidney occurs in approximately one in 400 live births. Polycystic kidney disease, Autosomal dominant polycystic kidney disease afflicts patients later in life. Approximately one in 1000 people will develop this condition. Autosomal recessive polycystic kidney disease is far less common, but more severe, than the dominant condition. It is apparent in utero or at birth. Renal agenesis. Failure of one kidney to form occurs in approximately one in 750 live births. Failure of both kidneys to form used to be fatal; however, medical advances such as amnioinfusion therapy during pregnancy and peritoneal dialysis have made it possible to stay alive until a transplant can occur. (Deam et al 2002)

2.3.2 Renal Cystic Disease

2.3.2.1 Simple Renal Cysts

These are true cysts that have a serous epithelial lining and are fluid filled, benign cortical masses. They meet all the ultrasound criteria of a simple cyst: they are spherical, anechoic, thin-walled and have accentuated posterior enhancement. (Deam et al 2002)
2.3.2.2 Atypical Renal Cysts;

An atypical renal cyst is any cyst that does not meet the strict criteria of a simple cyst. Many atypical cysts are simple cysts complicated by hemorrhage or infection. (Deam et al 2002)

2.3.2.3 Parapelvic Cysts;

Parapelvic cysts are cysts of the renal sinus. Most parapelvic cysts are asymptomatic although they may cause hematuria, hypertension, hydronephrosis or become infected. Generally they are anechoic and exhibit posterior acoustic enhancement. Parapelvic cysts are rarely purely spherical, their margins usually being shaped by the margins of the renal sinus. They therefore can mimic a hydronephrosis and particularly a pelviureteric junction obstruction. (Deam et al 2002)

2.3.2.4 Acquired Uremic Cysts, Adenomas and Carcinomas;

“Cysts and neoplasms have been identified with remarkable frequency in the end stage kidneys of chronic hemodialysis or peritoneal dialysis patients . . . Cysts generally do not become visible until the patient has completed 3 years of dialysis. There after, they increase rapidly in number to a prevalence level approaching 100%.” These cysts are prone to hemorrhage, rupture and the creation of perinephric hematomas. Long term hemodialysis patients are also prone to renal adenomas and about 8% of long term dialysis patients develop renal cell carcinoma. The carcinomas do not usually appear until after 3 years or more of dialysis treatment. “Most tumors that develop in dialysis patients are small, low-grade malignancies, and are nonmetastatic. After renal transplantation, the cysts reduce in size and number but the risk of
malignancy persists. Affected patients lack a history of polycystic kidney disease. (Deam et al 2002)

2.3.2.5 Multicystic Dysplastic Kidney;

Multicystic dysplastic kidney disease (MCDK) is a congenital, nonhereditary, cystic renal disease. It is the most common cause of a palpable abdominal mass in a newborn. MCDK is typically unilateral, affecting a single kidney in its entirety, but may be bilateral or segmental. The IVU and nuclear medicine scan would indicate absence of function. (Deam et al 2002)

2.3.2.6 Autosomal Recessive Polycystic Renal Disease (ARPRD);

“Autosomal recessive polycystic kidney disease is an inherited disorder characterized by nephromegaly, microscopic or macroscopic cystic dilatation of the renal collecting tubules, and periportal hepatic fibrosis. The renal abnormalities are seen early in life, while the liver pathology becomes predominant with increasing age. ARPRD is associated with pulmonary hypoplasia. The baby typically presents with palpable renal masses or renal insufficiency. There is a high neonatal mortality rate. Early causes of death are from renal insufficiency or respiratory failure associated with pulmonary hypoplasia. “Most patients who survive early childhood will eventually develop chronic liver disease, resulting in hepatic dysfunction and portal hypertension. There are few adult survivors with this condition. The disease was formerly called infantile polycystic kidney disease. (Deam et al 2002)
2.3.2.7 Autosomal Dominant Polycystic Renal Disease (ADPRD);

This is an autosomal dominant disorder which often lies latent for many years and then manifests itself in the third, fourth, or fifth decades in what had appeared to be normal renal parenchyma. ADPRD consists of numerous cystic lesions in an enlarged kidney. It is a slowly progressive bilateral disorder that eventually develops into renal failure when the normal parenchyma is depleted. The patient may present with hypertension, flank pain from hemorrhage within a cyst, hematuria or renal failure. The disorder was formerly called adult polycystic kidney disease (APKD). ADPRD is associated with hepatic cysts in 30% of affected individuals. Pancreatic (10%), spleen (5%) and seminal vesicle cysts are less common. Cerebral aneurysms occur in 10% of patients with the risk of associated subarachnoid hemorrhage. (Deam et al 2002)

2.3.2. Medullary Cystic Disease;

Medullary cystic disease is an hereditary disorder resulting in cysts located within the medullary portions of the kidney. There is a childhood recessive form and an adult dominant form.

Calyceal Diverticulum; This is an outpouching from the calyx. Stasis of urine may occur predisposing the patient to infection and stone formation. The diverticulum can project into the renal parenchyma. (Deam et al 2002)
2.3.3 Hydronephrosis

Hydronephrosis refers to dilatation of the renal collecting system most frequently caused by incomplete or complete obstruction. Hydroureter is dilatation of the ureter also caused by complete or incomplete obstruction.

Causes - In infants and children ureteropelvic junction obstruction, posterior urethral valves in males and Prune Belly Syndrome are the most common causes of obstruction. Calculi is the most common cause in adults followed by tumors of the kidney, ureter and bladder. Less common causes are inflammatory ureteral strictures, neurogenic bladder and bladder outlet obstruction.

Presentation - “Characteristically acute calculus obstruction presents with symptoms of renal colic, while patients with obstruction of longer standing may present with a more diffuse loin (flank) pain, with a loin mass or, if infection supervenes with fever, malaise as well as focal signs of obstruction. Increased serum creatinine and BUN levels may occur.(Deam et al 2002)

Grading the Society of Foetal Ultrasound has developed a grading system, which was intended for use with neonatal and infant hydronephrosis, which is now used for grading hydrenephrosis.
Grade 0 - no dilation with calyceal walls apposed
Grade 1 (mild) - dilation of the renal pelvis without dilation of the calyces nor parenchymal atrophy
Grade 2 (mild) - dilation of the renal pelvis and calyces without parenchymal atrophy
Grade 3 (moderate) - moderate dilation of the renal pelvis and calyces with blunting of the fornices and flattening of papillae
Grade 4 (severe) - gross dilation/ballooning of the renal pelvis and calyces with loss of borders between the renal pelvis and calyces and renal atrophy seen as cortical thinning (Fernbach SK et al 1994)

Figure (2.7): Show ultrasound picture of hydronephrosis caused by a left ureter stone. (created in Taiwan 2006)

Other Conditions Associated With Hydronephrosis:

Blood in the Collecting System - Trauma to the kidney may result in blood within the collecting system. In time the blood becomes echogenic and is shown to be less echogenic than the surrounding renal sinus echoes.

Pyonephrosis - This is a known and serious complication of hydronephrosis that develops as a direct consequence of urinary stasis and secondary infection. It is defined as the presence of pus in a dilated collecting system. Predisposing factors are diabetes, large calculi and collecting system anomalies. E. coli is the most common infecting organism.

Percutaneous Nephrostomy - Percutaneous nephrostomy is transcutaneous placement of a draining catheter for permanent or temporary urinary diversion. It is the initial treatment of choice for severe obstructive renal
failure and treatment of pyonephrosis. The catheter may also be used for ureteral stent insertion. A ureteral stent is a catheter used to restore internal drainage that extends from the renal pelvis across the ureteral narrowing into the urinary bladder. (Deam et al 2002)

2.3.4 Renal Calculus Disease

Urolithiasis is most prevalent in males aged 20-40 years. Calculi can form in any part of the urinary tract but most form in the kidneys. They may be clinically silent or associated with flank pain. Hematuria (gross or microscopic) and renal colic are most often associated with ureteric calculi. Stones can occur within any part of the kidneys - the renal cortex, medulla, vessels, calyces or renal pelvis. Most calculi arise in the collecting system. Stone formation may be idiopathic or associated with stasis (stagnation) of urine, prolonged ingestion of stone forming substances, chronic urinary infections and climate conditions associated with dehydration. Stasis also predisposes the patient to infection. Ultrasound demonstrates calculi as highly echogenic structures regardless of chemical composition. Shadow detection posterior to the stone depends on stone size, transducer frequency, and transducer focal zone. Tiny calculi will not shadow if they are smaller than the focal zone. (Deam et al 2002)

Collecting System Stones: The reflectivity of stones located in the renal sinus may be equal to that of the renal sinus itself, therefore, it may be beneficial to scan with the patient hydrated if a collecting system calculus is being considered. The fluid outlines the calculus and enables better visualization of the calculus versus the echogenic renal sinus. It is essential to use the highest frequency transducer as possible with the focus at the site of the suspected calculus. A staghorn calculus is a stone that completely fills the entire collecting system (i.e. the pelvis and calyces). It is usually associated with
chronic infection and obstruction. A staghorn calculus appears as a curved echogenic structure in the renal sinus area. The acoustic shadow created by the calculus often hides any associated hydronephrosis. (Deam et al 2002)

*Nephrocalcinosis;* Nephrocalcinosis is the formation of calcium deposits in the renal parenchyma. “It most commonly occurs in the medullary pyramids but calcium salts also may be deposited in the renal cortex - either exclusive of, or in conjunction with, medullary calcification.

A. *Cortical Nephrocalcinosis* causes of cortical nephrocalcinosis include acute cortical necrosis, chronic glomerulonephritis, chronic hypercalcemic states, ethylene glycol poisoning, sickle cell disease, and rejected renal transplants.

B. *Medullary Nephrocalcinosis* "The most common cause of medullary nephrocalcinosis in children is furosemide therapy in premature infants . . . It is administered to neonates for the treatment of heart failure or bronchopulmonary dysplasia . . . The incidence of medullary nephrocalcinosis in premature infants ranges from one-quarter to two-thirds of all those being treated with furosemide. Furosemide causes hypercalciuria. Other causes of medullary nephrocalcinosis in children are also associated with hypercalciuria such as, skeletal metastasis, severe osteoporosis, steroid therapy, vitamin D excess and idiopathic hypercalcemia.In adults the “causes of medullary nephrocalcinosis include hyperparathyroidism (40%), renal tubular acidosis (20%), medullary sponge kidney, bone metastases, chronic pyelonephritis, Cushing’s syndrome, hyperthyroidism, malignancy, renal papillary necrosis, sarcoidosis, sickle cell anemia and gout. (Deam et al 2002)
2.5 Neoplasms

2.5.1 Benign lesions;

*Angiomyolipoma (AML)* AML is a benign solid tumor containing variable amounts of blood vessels (angio), smooth muscle (myo) and fat (lipoma).

AMLs have a tendency to undergo spontaneous hemorrhage resulting in flank pain, hematuria or palpable mass. Focal hypoechoic areas may then be demonstrated in a predominantly hyperechoic mass. AML’s show significant growth when followed by US over time. Tumors range from 1 or 2 cm to more than 20 cm, however tumors less then 4 cm tend to be asymptomatic. AML is difficult to differentiate from RCC (renal cell carcinoma) when the renal tumors are small (<3 cm) and echogenic. Signs that favor RCC or AML include the presence of intratumoral cystic spaces, a peripheral hypoechoic rim, or intratumoral calcification. These findings are all rare in AML. Acoustic shadowing is sometimes seen with AML, but not with RCC. CT is recommended for lesions discovered on US that are believed to be AML. There are two forms of AML: a unilateral solitary mass arising from the renal cortex and multiple bilateral masses, usually in patients with the tuberous sclerosis complex. *Oncocytoma* is a benign solid renal tumor occurring most often in men in their 60's. It is usually asymptomatic and an incidental finding. (Deam et al 2002)

2.5.2 Malignant lesions;

*Renal Cell Carcinoma* is a primary tumor of the renal parenchyma thought to originate from the renal tubular epithelium. It is also called a hypernephroma or a renal adenocarcinoma. Renal cell carcinomas (RCC) are the most common primary malignant renal parenchymal tumors (86%). These
tumors occur most frequently in males between the fifth to the seventh decade. They are usually unilateral and clinically silent until they become large. The most common presenting complaints are painless hematuria, dull flank pain and palpable mass. Weight loss, malaise and hypertension may also be associated with RCC. Renal cell carcinomas may produce: a nonfunctioning kidney due to widespread parenchymal infiltration, obstruction of the collecting system, vascular thrombosis or compression and renal vein occlusion by tumor extension into the lumen. (Deam et al 2002)

*Transitional Cell Carcinoma* (TCC) is a malignancy involving the epithelial lining of the renal collecting system, ureters or bladder. It usually occurs in older age groups between 50 to 70, with a higher incidence in males. Bladder TCC is 50 times more common than renal pelvic TCC because of the large surface area. Painless hematuria is the most common complaint, however if there is ureteral obstruction, there will be flank pain. Most renal TCCs develop in the renal pelvis or major calyces and produce mass lesions within the renal sinus. TCC may occasionally infiltrate the renal parenchyma focally or diffusely causing enlargement of the kidney and loss of function (without the presence of a discrete mass).

*Renal Lymphoma:* is metastatic in origin and occurs by hematogenous dissemination or direct extension of retroperitoneal disease. Renal involvement is common with Non-Hodgkin’s lymphoma and less common with Hodgkin’s lymphoma. The disease is usually widespread by the time renal involvement is evident. Most patients have no urinary symptoms.

*Leukemia* have a predilection for infiltration of the renal parenchyma and often cause focal or diffuse renal enlargement. Acute lymphoblastic leukemia is the most common form to involve the kidney. (Deam et al 2002)
**Nephroblastoma** is a rapidly growing malignant tumor of the kidneys, consisting of embryonal elements. It is also known as Wilm's tumors, Wilm's embryoma or embryonal carcinoma. A nephroblastoma is the most common renal tumor in children. It is seen most commonly in children between 2 to 3 years of age. The child presents with a large palpable abdominal mass. Hypertension may be present due to renin production by the tumor. Hematuria is present in 25% of presenting patients. Metastases to the kidney are usually asymptomatic and demonstrated in patients with a known malignancy which has already metastasized elsewhere. Spread to the kidneys is via the hematogeneous route. The most common primary tumors associated with renal metastases are from the lung, breast and RCC of the contralateral kidney. Less common are colon, stomach, cervix, ovary, pancreas and prostate primaries. (Deam et al 2002)

**2.6 Medical Renal Disease**

This term describes renal disorders that are initially treatable with medicine rather than surgery. (Deam et al 2002)

**2.6.1 Acute Renal Failure (ARF)**

Renal failure is considered acute if it develops over days or weeks, and chronic if it spans months or years. Acute or chronic renal failure may result from insufficient renal perfusion (prerenal causes), intrinsic renal disease (renal causes), or obstructive uropathy (postrenal causes). In the setting of ARF, the main purpose of the US study is to exclude hydronephrosis. (Deam et al 2002)
2.6.2 Chronic Renal Failure (CRF)

Diabetes mellitus is the most common cause of CRF. Other common causes are: glomerulonephritis, chronic pyelonephritis, renal vascular disease, gout and polycystic renal disease. (Deam et al 2002)

2. 7 Renal Infections

Most renal infections occur via the ascending route. They are usually caused by contaminants from the intestinal tract. Instrumentation, stasis, calculi, and vesicoureteral reflux are predisposing factors. Hematogenous infection also occurs as the result of intravenous drug abuse, tuberculosis and in immunocompromised patients. (Deam et al 2002)

2. 7.1 Acute pyelonephritis:

This is most often (85%) caused by an ascending Escherichia coli (E. coli) infection. The E. coli is a normal inhabitant of the bowel and because of the female urethra's close proximity to the anus, these infections are far more common in the female. The remaining 15% of acute pyelonephritis cases are caused by hematogenous seeding (of staphylococcus aureus) from elsewhere in the body. The patient presents with flank pain, fever, chills, leukocytosis, dysuria and bacteriuria. One or both kidneys may be involved. (Deam et al 2002)

2.7.2 Acute Focal Bacterial Nephritis (Lobar Nephronia)

“Studies of pyelonephritic kidneys suggest that each lobe is infected as a unit and that the severity of the infection may vary considerably from one renal lobe to another. The term acute, focal bacterial nephritis is applied to disproportionately severe infection of one or more lobes. The severity of
lobar infections in such cases falls somewhere between the usual form of pyelonephritis and renal abscess.

*Renal Abscess;* "Untreated or inadequately treated acute pyelonephritis may lead to parenchymal necrosis with abscess formation." Renal abscesses may also result from infected cysts. Patients associated with renal abscesses are those with diabetes, infected renal stones, immune compromise, IV drug abuse or debilitating disease. Renal carbuncle is an alternate term for renal abscess. Renal abscesses tend to be solitary and may spontaneously decompress into the collecting system or perirenal space. Small abscesses are treated with antibiotics whereas, larger ones will require percutaneous drainage or surgery. *(Deam et al 2002)*

### 2.7.3 Emphysematous Pyelonephritis (EPN)

“If a gas-forming organism infects the renal parenchyma, gas may accumulate within affected portions of the renal tissue. The term ‘emphysematous pyelonephritis’ is applied to this uncommon but highly dangerous condition, which usually occurs in diabetic patients. EPN may be associated with urinary tract obstruction. The most common infecting organism is E. coli. Patients usually present as extremely ill, with fever, flank pain, hyperglycemia, acidosis, dehydration and electrolyte imbalance. Some (18%) present with only fever of unknown origin. Emergency nephrectomy is the treatment of choice. *Papillary Necrosis* is an infrequent form of pyelonephritis involving necrosis of the renal papillae. The lesion consists of a combination of ischemic and suppurative necrosis at the tips of the renal pyramids. The papillae may shrivel, slough off and drop into the pelvis. Calcification may develop in the necrotic areas. The cortex drained by the necrotic tissue undergoes atrophy. Papillary necrosis is associated with diabetics who develop acute pyelonephritis, analgesic abuse (aspirin,
acetaminophen, codeine) and sometimes as a complication of acute pyelonephritis when there is significant urinary tract obstruction.

Renal Fungal Disease; Candida albicans is the most common fungus to infect the urinary tract and is usually the result of hematogenous spread. Candidiasis most often infects the immunocompromised.

Chronic Pyelonephritis; Chronic Pyelonephritis may be the end-stage of reflux nephropathy or due to recurring infection associated with calculi and chronic obstruction.“Reflux nephropathy is believed to cause 10% to 30% of all cases of end-stage renal disease.”3 Chronic pyelonephritis associated with reflux usually begins in childhood and is more common in females. Reflux into the collecting tubules occurs when the papillary duct orifices are incompetent. Cortical scarring overlies the involved calyx. Both kidneys may be involved although the patterns may be asymmetric.(Deam et al 2002)

2.8 Laboratory Test:

Laboratory studies are an important adjunct to clinical evaluation for assessment of renal function. An initial workup of a patient may include a complete blood count (CBC); serum electrolytes including sodium, potassium, chloride, bicarbonate, calcium, and phosphorus; blood urea, nitrogen and creatinine; blood glucose and glycocylated hemoglobin. Glomerular filtration rate (GFR) can be calculated.

Urine studies may include urine electrolytes, creatinine, protein, fractional excretion of sodium (FENA) and other studies to assist in evaluation of the etiology of a patient's renal disease. Urinalysis is used to evaluate urine for its pH, protein, glucose, specific gravity and the presence of blood. Microscopic analysis can be helpful in the identification of casts, red blood cells, white blood cells and crystals.(Deam et al 2002)
2.9 Radiology

Imaging studies are important in the evaluation of structural renal disease caused by urinary tract obstruction, renal stones, renal cyst, mass lesions, renal vascular disease, and vesicoureteral reflux.

Imaging techniques used most frequently include renal ultrasound and helical CT scan. Patients with suspected vesicoureteral reflux may undergo voiding cystourethrogram (VCUG). (Post TW et al 2012).

2.6 Biopsy:

The role of the renal biopsy is to diagnose renal disease in which the etiology is not clear based upon noninvasive means (clinical history, past medical history, medication history, physical exam, laboratory studies, imaging studies). A detailed description of renal biopsy interpretation is beyond the scope of this article. In general, a renal pathologist will perform a detailed morphological evaluation and integrate the morphologic findings with the clinical history and laboratory data, ultimately arriving at a pathological diagnosis. A renal pathologist is a physician who has undergone general training in anatomic pathology and additional specially training in the interpretation of renal biopsy specimens. Ideally, multiple core sections are obtained and evaluated for adequacy (presence of glomeruli) intraoperatively. A pathologist/pathology assistant divides the specimen(s) for submission for light microscopy, immunofluorescence microscopy and electron microscopy. The pathologist will examine the specimen using light microscopy with multiple staining techniques (hematoxylin and eosin/H&E, trichrome, silver stain) on multiple level sections. Multiple immunofluorescence stains are performed to evaluate for antibody, protein and complement deposition. Finally, ultra-structural examination is
performed with electron microscopy and may reveal the presence of electron-dense deposits or other characteristic abnormalities that may suggest an etiology for the patient's renal disease (Post TW et al 2012).

2.7. Sonographic scanning of the normal kidneys:

The examination begins with the patient in supine position, by using 3 to 5 MHz frequency and the scans are performed in the sagittal and transverse planes. The right kidney is readily demonstrated through the right lobe of the liver. Generally a subcostal approach displays the (more anterior) lower pole to best effect, while an intercostal approach is best for demonstrating the upper pole. The left kidney is not usually demonstrable sagittally because it lies posterior to the stomach and splenic flexure. The spleen can be used as an acoustic window to the upper pole by scanning coronally, from the patient’s left side, with the patient supine or decubitus (left side raised), but, unless the spleen is enlarged, the lower pole must usually be imaged from the left side posteriorly. Coronal sections of both kidneys are particularly useful as they display the renal pelvic calyceal system (PCS) and its relationship to the renal hilum. This section demonstrates the main blood vessels and ureter (if dilated)

2.7.1 Normal ultrasound appearances of the kidneys:

The normal renal outline is smooth, and the cortical thickness between the capsule and cortical medullary junction is uniform with a slight prominence at the pole. The cortex of the normal kidney is slightly hypoechoic when compared to the adjacent liver parenchyma, although this is age-dependent. In young people it may be of similar echogenicity and in the elderly it is not unusual for it to be comparatively hyperechoic and thin. The medullary pyramids are seen as regularly spaced, echo-poor triangular structures between the cortex and the renal sinus and are more prominent in neonates. The tiny reflective structures often seen at the margins of the pyramids are
echoes from the arcuate arteries which branch around the pyramids. The renal sinus containing the PCS is hyperechoic due to sinus fat which surrounds the vessels. The main artery and vein can be readily demonstrated at the renal hilum and should not be confused with a mild degree of PCS dilatation. Colour Doppler can help differentiate. The kidney develops in the fetus from a number of lobes, which fuse. Occasionally the traces of these lobes can be seen on the surface of the kidney, forming fetal lobulations; these may persist into adulthood. (Walsh J 2002)

Fig. 2.8 Show normal kidney. Longitudinal (A) and transverse (B) grayscale sonogram. (from www.springerlink.com)
2.8 Previous Studies


It is a prospective study, the study population composed of 53 female and 47 male who were suspected with renal diseases Results: According to sonographic appearance, hydronephrosis had been classified into mild, moderate and severe hydronephrosis. Mild hydronephrosis is most common (53%), moderate (30%), severe hydronephrosis (13%) and extreme hydronephrosis was 4%. There were various causes of hydronephrosis, ureteric stone 31%, kidney stone 23%, pregnancy 12% and benign prostatic hypertrophy 11%. Most of the study population had history of renal stones (63%) Conclusion: Ultrasound is the first line of investigation of the renal system. It is sensitive and accurate to assess and classify the hydronephrosis and determine the main causes. Metabolic disorders (gout and diabetes) were the most risk factors of renal obstructive disease(International journal of medical imaging 2015)

**AlsheikhBabiker Ahmed Hamid**, 2015.Role of Ultrasound in Evaluating Obstructive Uropathy in SudaneseThis study (100) patients came to ultrasounddepartment check up .This result (65) patients (65% ) obstruction uropathy the causes of obstruction. Main cause stones 75% came next mass 20% , least infection 5% .Types Mild hydronephrosis 29% and Moderate hydronephrosis58% , severehydronephrosis 13%. Ultrasound is sensitive and specific for renal stones , 83% and 100% for hydronephrosis , 83% and 100% , respectively Its sensitivity to pick ureteric stones (40%) and to identify Hydroureter (50%) is low. Addition of Computer tomography (CT) KUB abdomen increases the sensitivity for ureteric stones to 100%.
Ultrasound criteria of obstruction is hydronephrosis dilatation of the calyx ultrasound have high accuracy indetected fluid which results from obstruction the common causes ofobstruction uropathy are stones and masses .prostrate enlargement andposterior urethral valve infant, infections. Ultrasound is non invasive portable no need for contrast media.
Chapter Three
Methodology
Chapter Three
Methodology

3.1 Material:

3.1.1 Study design
Descriptive cross-sectional study

3.1.2 Study area
The study will be conducted at Khartoum State–Sudan

3.1.3 Study duration
The study is going to be conducted over three months, from June 2016 to August 2016.

3.1.4 Study population
All patients diagnosed with hydronephrosis during an ultrasound scan investigation presented at the time of the study.

3.1.5. Inclusion criteria:
All patients diagnosed with hydronephrosis during an ultrasound scan investigation presented at the study area within the period of the study with ages more than (20) years

3.1.6 Exclusion criteria:
Patients who are diagnosed with hydronephrosis with ages less than (20) years, pregnant ladies and infant.

3.1.7 Sample size
50 patients with sign and symptom of hydronephrosis
3.2 Method;

3.2.1 Technique;

The examination begin with the patient in supine position, by using 3 to 5 MHz frequency and the scans are performed in the sagittal and transverse planes.

3.2.2 Data collection:

By data collection sheet.

3.2.3 Data analysis:

The data will be entered into computer program, statistical package of social sciences (SPSS) version 20.0. Frequencies, chi square test and will be used when appropriate. The P value will be considered significant if < 0.05.

3.2.4 Ethical considerations:

- Ethical clearance will be obtained from ethical clearance committee of Sudan University Board.
- Consent will be obtained from all patients before participation in the study
Chapter Four
The Results
Chapter Four

Results

Table (4.1) Frequency distribution of patient’s age group:

<table>
<thead>
<tr>
<th>Age group</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>less than 20 years</td>
<td>8</td>
<td>16.0</td>
<td>16.0</td>
<td>16.0</td>
</tr>
<tr>
<td>20-30 years</td>
<td>11</td>
<td>22.0</td>
<td>22.0</td>
<td>38.0</td>
</tr>
<tr>
<td>31- 40 years</td>
<td>11</td>
<td>22.0</td>
<td>22.0</td>
<td>60.0</td>
</tr>
<tr>
<td>41-50 years</td>
<td>5</td>
<td>10.0</td>
<td>10.0</td>
<td>70.0</td>
</tr>
<tr>
<td>51- 60 years</td>
<td>7</td>
<td>14.0</td>
<td>14.0</td>
<td>84.0</td>
</tr>
<tr>
<td>more than 60 years</td>
<td>8</td>
<td>16.0</td>
<td>16.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Minimum =10 years, maximum = 80 years, mean = 39.16 years, std= 18.88 years

Figure (4.1) shows frequency distribution of age group
Table (4.2) Frequency distribution of patient’s gender

<table>
<thead>
<tr>
<th>Gender</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>22</td>
<td>44.0</td>
<td>44.0</td>
<td>44.0</td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td>56.0</td>
<td>56.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>100.0</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

Figure (4.2) shows frequency distribution of patient’s gender
<table>
<thead>
<tr>
<th>Patient’s complain</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Back pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Bil. Loin pain</td>
<td>4</td>
<td>8.0</td>
<td>8.0</td>
<td>10.0</td>
</tr>
<tr>
<td>Flank pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>12.0</td>
</tr>
<tr>
<td>Frequency</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>16.0</td>
</tr>
<tr>
<td>Haemturia</td>
<td>3</td>
<td>6.0</td>
<td>6.0</td>
<td>22.0</td>
</tr>
<tr>
<td>Low Back pain &amp; L. Iliac fossa</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>24.0</td>
</tr>
<tr>
<td>Low back pain + testicular pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>26.0</td>
</tr>
<tr>
<td>Lower abdominal pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>28.0</td>
</tr>
<tr>
<td>Lower abdominal pain + U.T.I.</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>30.0</td>
</tr>
<tr>
<td>Lt . loin pain</td>
<td>4</td>
<td>8.0</td>
<td>8.0</td>
<td>38.0</td>
</tr>
<tr>
<td>Lt. iliac fossa pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>40.0</td>
</tr>
<tr>
<td>Lt. Loin pain &amp; colic</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>42.0</td>
</tr>
<tr>
<td>Lt. lower abdominal pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>44.0</td>
</tr>
<tr>
<td>Nocturea Dysurea</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>46.0</td>
</tr>
<tr>
<td>Rt loin pain + Hameaturea</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>48.0</td>
</tr>
<tr>
<td>Rt lower abd. Pain + colic</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>50.0</td>
</tr>
<tr>
<td>Rt lower pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>52.0</td>
</tr>
<tr>
<td>Rt. Iliac fossa pain</td>
<td>3</td>
<td>6.0</td>
<td>6.0</td>
<td>58.0</td>
</tr>
<tr>
<td>Rt. Loin pain</td>
<td>8</td>
<td>16.0</td>
<td>16.0</td>
<td>74.0</td>
</tr>
<tr>
<td>Rt. Loin pain + UTI.</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>78.0</td>
</tr>
<tr>
<td>Rt. Loin Pain Haematurea</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>80.0</td>
</tr>
<tr>
<td>Rt. Lower back pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>82.0</td>
</tr>
<tr>
<td>Rt. Lower pelvic pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>84.0</td>
</tr>
<tr>
<td>Rt. Renal Pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>86.0</td>
</tr>
<tr>
<td>Suprabublic pain + umbilical pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>88.0</td>
</tr>
<tr>
<td>Supraublic pain</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>90.0</td>
</tr>
<tr>
<td>Urine Retention</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>92.0</td>
</tr>
<tr>
<td>UTI</td>
<td>4</td>
<td>8.0</td>
<td>8.0</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td><strong>100.0</strong></td>
<td><strong>100.0</strong></td>
<td></td>
</tr>
</tbody>
</table>
Figure (4.3) shows frequency distribution of clinical sign and symptom
Table (4.4) Frequency distribution of patient’s other finding:

<table>
<thead>
<tr>
<th>Other finding</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>H.T.N &amp; Diabetic</td>
<td>6</td>
<td>12.0</td>
<td>12.0</td>
<td>12.0</td>
</tr>
<tr>
<td>Colic</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>16.0</td>
</tr>
<tr>
<td>Diabetic</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>20.0</td>
</tr>
<tr>
<td>Dysurea</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>24.0</td>
</tr>
<tr>
<td>H.T.N</td>
<td>5</td>
<td>10.0</td>
<td>10.0</td>
<td>34.0</td>
</tr>
<tr>
<td>Nausea &amp; vomiting</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>36.0</td>
</tr>
<tr>
<td>No</td>
<td>30</td>
<td>60.0</td>
<td>60.0</td>
<td>96.0</td>
</tr>
<tr>
<td>Stone formation</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>98.0</td>
</tr>
<tr>
<td>U/B stone</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>100.0</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

Figure (4.4) shows frequency distribution of other finding
Table (4.5) Frequency distribution of u/s finding in hydronephrosis pt

<table>
<thead>
<tr>
<th>Us finding</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bil. Hydrorephrosis + moderate Rt. Hydronephros</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Bilateral mild Hydronephrosis</td>
<td>7</td>
<td>14.0</td>
<td>14.0</td>
<td>16.0</td>
</tr>
<tr>
<td>Bilateral renal moderate Hydrorephrosis</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>18.0</td>
</tr>
<tr>
<td>Lt. mild Hydrorephrosis Rt. Moderate Hydrorephr</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>20.0</td>
</tr>
<tr>
<td>Lt. Renal mild Hydronephrosis</td>
<td>9</td>
<td>18.0</td>
<td>18.0</td>
<td>38.0</td>
</tr>
<tr>
<td>Lt. Renal moderate Hydronephrosis</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>42.0</td>
</tr>
<tr>
<td>Lt. renal pelvic stone + upper uretric stone</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>44.0</td>
</tr>
<tr>
<td>Rt. Ectopic kidney with moderate Hydrorephrosis</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>46.0</td>
</tr>
<tr>
<td>Rt. Moderate Hydrorephrosis&amp;Lt. mild Hydronephrosis</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>48.0</td>
</tr>
<tr>
<td>Rt. Renal mild hydronephrosis</td>
<td>15</td>
<td>30.0</td>
<td>30.0</td>
<td>78.0</td>
</tr>
<tr>
<td>Rt. Renal moderate hydronephrosis</td>
<td>7</td>
<td>14.0</td>
<td>14.0</td>
<td>92.0</td>
</tr>
<tr>
<td>Severe L. Hydronephrosis&amp;Hydroureter.</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>94.0</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Count</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>-----------------------------------------</td>
<td>-------</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>Severe Rt Renal Hydronephrosis</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>98.0</td>
</tr>
<tr>
<td>Small Rt. Renal Stone + mild Hydronephrosis</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>50</td>
<td>100.0</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

**Figure (4.5) Frequency distribution of ultrasound**
Table (4.6) frequency distribution of patient’s occupation:

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advocate</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Farmer</td>
<td>5</td>
<td>10.0</td>
<td>10.0</td>
<td>12.0</td>
</tr>
<tr>
<td>House-keeper</td>
<td>14</td>
<td>28.0</td>
<td>28.0</td>
<td>40.0</td>
</tr>
<tr>
<td>Merchant</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>42.0</td>
</tr>
<tr>
<td>No</td>
<td>11</td>
<td>22.0</td>
<td>22.0</td>
<td>64.0</td>
</tr>
<tr>
<td>Officer</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>66.0</td>
</tr>
<tr>
<td>Soldier</td>
<td>3</td>
<td>6.0</td>
<td>6.0</td>
<td>72.0</td>
</tr>
<tr>
<td>Student</td>
<td>10</td>
<td>20.0</td>
<td>20.0</td>
<td>92.0</td>
</tr>
<tr>
<td>Teacher</td>
<td>4</td>
<td>8.0</td>
<td>8.0</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td><strong>100.0</strong></td>
<td><strong>100.0</strong></td>
<td></td>
</tr>
</tbody>
</table>

Figure (4.6) shows frequency distribution of patient’s occupation
Table (4.7) Frequency distribution of causes of hydrenephrosis by ultrasound

<table>
<thead>
<tr>
<th>Final diagnose by ultrasound</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>B.P.H</td>
<td>4</td>
<td>8.0</td>
<td>8.0</td>
<td>8.0</td>
</tr>
<tr>
<td>Bil. Multiple Renal stones</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>12.0</td>
</tr>
<tr>
<td>Cystitis</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>16.0</td>
</tr>
<tr>
<td>It. Renal pelvic stone and upper third Ureteric stone</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>18.0</td>
</tr>
<tr>
<td>Lower ureteric stone</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>20.0</td>
</tr>
<tr>
<td>Lt. Lower ureteric stone</td>
<td>8</td>
<td>16.0</td>
<td>16.0</td>
<td>36.0</td>
</tr>
<tr>
<td>Lt. lower Uterine stone + RT. Renal stone</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>38.0</td>
</tr>
<tr>
<td>Lt. Ovarian cysts.</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>40.0</td>
</tr>
<tr>
<td>Lt. Renal stone</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>42.0</td>
</tr>
<tr>
<td>Psoas Muscle Abscess.</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>44.0</td>
</tr>
<tr>
<td>Reveral flow from distended U/B urethral stricture</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>46.0</td>
</tr>
<tr>
<td>Rt lumbar mass.</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>48.0</td>
</tr>
<tr>
<td>Rt. Ectopic kidney.</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>50.0</td>
</tr>
<tr>
<td>Rt. Lower ureteric stone</td>
<td>17</td>
<td>34.0</td>
<td>34.0</td>
<td>84.0</td>
</tr>
<tr>
<td>Rt. Renal stone</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>88.0</td>
</tr>
<tr>
<td>Rt. Renal stone+Rt. Lower Ureteric Stone</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>90.0</td>
</tr>
<tr>
<td>Small Rt. Renal stone</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>94.0</td>
</tr>
<tr>
<td>Uterine Mass</td>
<td>1</td>
<td>2.0</td>
<td>2.0</td>
<td>96.0</td>
</tr>
<tr>
<td>Vesical stone</td>
<td>2</td>
<td>4.0</td>
<td>4.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Figure (4.7) Frequency distribution of final diagnosed by ultrasound
Table (4.8) chi square test between age distribution and final diagnosis

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Df</th>
<th>Asymp. Sig. (2-sided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Chi-Square</td>
<td>97.780</td>
<td>90</td>
<td>.270</td>
</tr>
<tr>
<td></td>
<td>a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likelihood Ratio</td>
<td>78.807</td>
<td>90</td>
<td>.794</td>
</tr>
<tr>
<td>N of Valid Cases</td>
<td>50</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

a. 114 cells (100.0%) have expected count less than 5. The minimum expected count is .10.

Table (4.9) chi square test between us finding and us final diagnosed

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Df</th>
<th>Asymp. Sig. (2-sided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Chi-Square</td>
<td>3.890</td>
<td>252</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>E2a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likelihood Ratio</td>
<td>138.74</td>
<td>252</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N of Valid Cases</td>
<td>50</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a. 284 cells (99.6%) have expected count less than 5. The minimum expected count is .02.
Table (4.10) chi square test between gender distribution and final diagnosis

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Df</th>
<th>Asymp. Sig. (2-sided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Chi-Square</td>
<td>25.530</td>
<td>18</td>
<td>.111</td>
</tr>
<tr>
<td>Likelihood Ratio</td>
<td>34.133</td>
<td>18</td>
<td>.012</td>
</tr>
<tr>
<td>N of Valid Cases</td>
<td>50</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a. 36 cells (94.7%) have expected count less than 5. The minimum expected count is .44.

Table (4.11) chi square test between gender distribution and us finding

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Df</th>
<th>Asymp. Sig. (2-sided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Chi-Square</td>
<td>16.223</td>
<td>14</td>
<td>.300</td>
</tr>
<tr>
<td>Likelihood Ratio</td>
<td>20.252</td>
<td>14</td>
<td>.122</td>
</tr>
<tr>
<td>N of Valid Cases</td>
<td>50</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a. 28 cells (93.3%) have expected count less than 5. The minimum expected count is .44.
Table (4.12) chi square test between occupation distribution and final diagnosis

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Df</th>
<th>Asymp. Sig. (2-sided)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Chi-Square</td>
<td>1.563E2a</td>
<td>144</td>
<td>.229</td>
</tr>
<tr>
<td>Likelihood Ratio</td>
<td>90.491</td>
<td>144</td>
<td>1.000</td>
</tr>
<tr>
<td>N of Valid Cases</td>
<td>50</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a. 171 cells (100.0%) have expected count less than 5. The minimum expected count is .02.
Chapter Five

Discussion, Conclusion & Recommendations
Chapter Five
Discussion, Conclusion & Recommendations:

5.1 Discussion
This cross sectional descriptive study was done to assess the different causes of hydronephrosis.

Regarding the age group, the study found that the most common age (20 – 30 years) and (31 – 40 years) respectively (22%) for each, followed by less than 20 years and more than 60 years each (16%). The reason for that is the prevalence of urinary stone is more common in the youngest age group from (20 – 40) years old, as literature stated that. And in oldest age group, the presence of hydronephrosis, specially in male due to BPH. This result agree with previous study done by Dr. Suzan Omer; et al(2015) which found that the presence of hydronephrosis common in younges age group. (table 4.1).

Concerning the gender, the study found that the high percentage of hydronephrosis in male more than female (56%) due to BPH disagree with literature and background studies of Suzan Omer, et al( 2015) which found hydronephrosis in female more than male.(table 4.2)

In this study, the most patients present with lion pain (46%). Either right, left or bilateral and sometimes associated with other symptoms. Right lion pain was the most presenting symptoms about(8)( 16%) of patients. Other presenting symptoms are back pain, lower abdominal pain, haematura, suprapubic pain, urine retention, nocturne and dysurea are very rare complaint in only 2% of patients. Table (4.3).

Most of patients had no pathological conditions (30)(60% ) followed by H.T.N diabetic( 6)( 12% ) (table 4.4).
Ultrasound examination showed that the high percentage is right mild hydronephrosis due to ureteric stone (15) (30%) this result agree with the study done by Suzan Omer et al.(2015) and Elsheikh Babikir Ahmed Hamid,(2015) which found hydronephrosis due to ureteric respectively. Followed by left renal mild hydronephrosis (9) (18%), bil mild hydronephrosis and right renal moderate hydronephrosis (7) (14%), table (4.5).

Regarding the patient occupation, the study found that the most patients are housekeepers (14) (28%), followed by no work (11), (22%), student (10) (20%) and farmers (5) (10%). Table (4.6).

Final diagnosed by u/s showed that the most common causes in hydronephrosis is right lower ureteric stone (17) (34%), followed by left lower ureteric stone (8) (16%), and BPH (4) (8%). Table (4.7).
5.2 Conclusion:

The study found that the hydronephrosis was higher incidence in age group(20-40)22% with mild hydronephrosis is most common type. The most common cause of hydronephrosis is right kidney mild hydronephrosis followed by left side hydronephrosis. The study found that the most common cause of hydronephrosis is lower ureteric stone followed by left side ureteric stone and BPH.
5.3 Recommendations:

(1) All patients with urinary tract symptom should be examined by U/S, as a role with other investigations.

(2) The sonographers and sonologists should be in continuous education to develop them by learning the modern information or discovered procedures so as to be up-to-date.

(3) The government should encourage establishing ultrasonographic departments, and should supply primary health centers and hospitals with high quality U/S machines, especially in rural areas.

(4) Further studies should be conducted with more sample volume to assess the cause of hydrenephrosis and to correlate with other investigation such as CTU.
Reference:


Bålens ytanatomy (Superficial anatomy of the trunk). Anca Dragomir, Mats Hjortberg and Godfried M. Romans. Section for human anatomy at the Department of Medical Biology, Uppsala University, Sweden.


Koh JS, Wong MY, Li MK, Foo KT (September 1998). "Idiopathic retroperitoneal fibrosis with bilateral lower ureteric obstruction—a case


Appendex A

Data Sheets:

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Gender</th>
<th>Clinical Diagnosis</th>
<th>Other abnormality</th>
<th>U/S findings</th>
<th>Occupation</th>
<th>Final Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Female</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Female</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Female</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Female</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

63
Appendix B

Images

Image(1) Show classification of hydronephrosis

Image(2) Show moderate & severe hydronephrosis
Image (3) Show Rt kidney severe hydronephrosis

Image (4) Show mild hydronephrosis
Image (5) Show mild hydronephrosis & stone

Image (6) Show mild hydro nephrosis & hydro ureter (ureteric stone)
Image (7) Show duplex & hydronephrosis

Image (8) Show moderate hydro nephrosis & hydro ureter
Image (9) shows moderate hydronephrosis & hydro ureter.

Image (10) shows hydronephrosis & hydro ureter.
Image (11) Show mild hydronephrosis & hydro ureter (ureteric stone)

Image (12) Show mild hydro nephrosis
Image (13) (a,b) Show moderate hydro nephrosis & hydro ureter (ureteric stone)

Image (14) (a,b) Show sever hydro nephrosis
Image (15) enlarged prostate (BPH)

Image (16)(a,b) Show vesical stone
Image (17)(a,b) Show vesical stone