

## INTRODUCTION

Urinary stone disease is the third entity following urinary tract infection and prostate diseases among urinary disorders.

Urolithiasis is a common medical problem with a prevalence of approximately 2% to 3% in the general population.

50% of patients with previous urinary stones have a recurrence within 10 years (*Pearle, 2005*).

Management of renal stones includes pharmacotherapy, extra corporeal shock wave lithotripsy (ESWL), percutaneous nephrolithotomy (PCNL) and open surgery (*Miller et al., 2007*).

In 1980, **Dr. Christian Chaussy** of the University of Munich was the first to treat renal stone in humans using a new concept termed extracorporeal shock wave lithotripsy. Using this technology, he determined that patients could have renal or ureteral stones removed without the need of an incision or skin puncture, due to its non-invasive nature ESWL has completely changed therapeutic strategies for urolithiasis, The first lithotripter model (Dornier HM-1™) was soon replaced by the (Dornier HM-2™) IN 1982, and the (Dornier HM-3™) in 1984. The HM-3 was first used in the United States on February 23<sup>rd</sup>, 1984 at Methodist Hospital in Indianapolis. With

technological advances in lithotripter models, ESWL has become the preferred line of treatment for renal & upper ureteric calculi of <2cm in diameter (*Andrade et al., 2006*).

Shock wave lithotripsy (SWL) is an effective, well-established method for treating ureteral calculi.

Investigators have reported that several factors limit the success of SWL for ureteral stones, namely stone size, site, composition, impaction, and stone-induced urinary obstruction.

Ureteral obstruction produces a progressive decrease in renal excretory function. In an acute obstruction, rapid redistribution of renal blood flow from medullary to cortical nephrons occurs resulting in a decrease in glomerular filtration rate and renal plasma flow, reflecting a decrease in both glomerular and tubular function. Obstruction results not only in decreased renal function but also in fairly rapid changes in ureteral peristaltic function.

Hypertrophy of ureteral musculature after only 3 days of obstruction has been observed (*Gee, Kiviat, Invest Urol, 1975*).

Ureteral obstruction results in decreased peristalsis and decreased pressure possibly affecting ureteral stone migration.

## **AIM OF THE WORK**

To assess whether the degree of stone induced hydronephrosis in patients with solitary proximal ureteral stones influences the outcome and clearance rates after SWL.

## ENDOSCOPIC ANATOMY OF THE URETER

**Anatomically**, the ureter is 22 to 30 cm in length and is divided into three portions: the proximal ureter (upper) is the segment that extends from the ureteropelvic junction to the area where the ureter crosses the sacroiliac joint, the middle ureter courses over the bony pelvis and iliac vessels, and the pelvic or distal ureter (lower) extends from the iliac vessels to the bladder (*Narath, 1954*).

**Histologically**, the ureter consists of three distinct layers. The first is an inner mucosal layer of transitional epithelium covered by lamina propria. The inner layer produces mucosal secretions to protect itself from urine. The second or middle layer is muscular and consists of both longitudinal and circular layers of smooth muscle, which help propel urine forward by peristalsis. The outer (adventitial) layer consists of areolar connective tissue and contains nerves, blood vessels and lymphatic vessels (*Narath, 1954*).

**Functionally**, The ureter is a dynamic organ rather than a simple conduit through which urine flows. It conducts urine from the renal papillae to the ureteral orifices in the bladder irrespective of the spatial orientation of the body. However, when the urinary transport system is disturbed, gravity may influence directional flow (*Narath, 1954*).

Just inside the bladder neck is the trigone. It is raised, smooth triangular area with its apex at the bladder neck and its

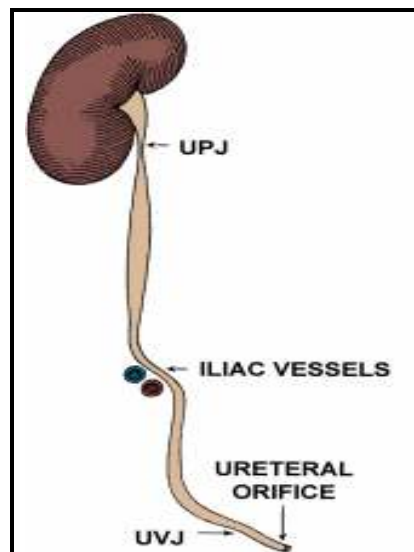
base formed by the interureteric ridge extending between the two ureteral orifices (*Abdel-Razzak, 2006*).

The trigone is the most vascular part of the bladder, and so it is more deeply colored than the rest of the bladder. It is formed by an extension of the longitudinal muscle fibers of the ureters superimposed over the detruser muscle (*Anderson et al., 2007*).

The elevation extending between the ureteral orifices is known as the interureteric ridge or mercier's bar. It is more prominent in males than females; however, it can be identified in almost all bladders. The ureteral orifices are usually symmetrically located along this ridge, 1 to 2 cm from the midline (*Politano, 1972*).

The normal nonrefluxing orifice may have the configuration described as a volcano, a horse shoe, or some other variation. It might be prominent and obvious on endoscopy, or it might be an inconspicuous slit that can be identified only on close examination (*Anderson et al., 2007*).

As the bladder fills, the ureteral orifices are pushed out laterally and the intravesical ureter is compressed making ureteral access more difficult. Alternatively, the ureteral orifice may be quite variable in position and appearance. In an attempt to establish an uniform classification of orifice characteristics, Lyon and colleagues suggested categorizing the ureteral orifice according to these two criteria. They described the orifice as being in position A if it was in the normal medial aspect of the trigone (*Kabalin, 1998*).



**Figure (1):** Anatomical constrictions of the Ureter (*Kabalin, 1998*)

The normal ureter is easily distensible. However, there are three naturally narrow sites within the lumen. The narrowest position is the ureterovesical junction. This requires dilation before introduction of large caliber instruments. The other 2 narrow areas are at the pelvic brim and the ureteropelvic junction. These are relatively wider and are sufficiently dilated with irrigating fluid pressure to allow instrument passage (*Huffman, 1998*).

These areas are identified endoscopically by a slightly stenotic appearance and relative nondistensibility. Furthermore the pelvic brim constriction is at the area of the iliac vessels, which can be seen pulsating behind the ureter as this level is approached. Following this is a relatively straight section where the middle of the ureter lies on the psoas muscle. It is here that the typical stellate appearance of non-distended ureteral lumen can be discerned (*Bagley and Rittenberg 2013*).

These leads to the third constriction at the ureteropelvic junction, which is identified endoscopically as anarrowing in the ureter followed by the wide renal pelvis. A posterolateral lip of mucosa is sometimes seen in this region. It corresponds to the junction of the ureter with the more dependent part of the pelvis and is accentuated with the respiratory movement (*Huffman, 1998*).

The kidney is amobile structure that moves craniocaudally with respiration. This movement is readily apparent endoscopically, because the ureteropelvic region is approached from the relatively fixed ureter. The peristaltic contractions of the ureter, along with the opening and closing of the ureteropelvic junction, can also be observed endoscopically (*Anderson et al., 2007*).

As the kidney lies posteriorly the proximal aspect of the ureter passes posteriorly and laterally over the psoas muscle to enter the renal pelvis. The normal renal pelvis is usually conical on shape, with the apex of the cone leading to the ureteropelvic junction. It may also be more box shaped, with ureteropelvic junction near the lower medial angle. The intrarenal pelvis is often small with short major calices. The extrarenal pelvis, on the other hand, is usually large, and because it lies outside (*Huffman, 1998*).

The renal sinus, the major Caliceal infunibula are by necessity long. As the ureteroscope enters the renal pelvis the first structures to be seen are the ostia of the major calices.

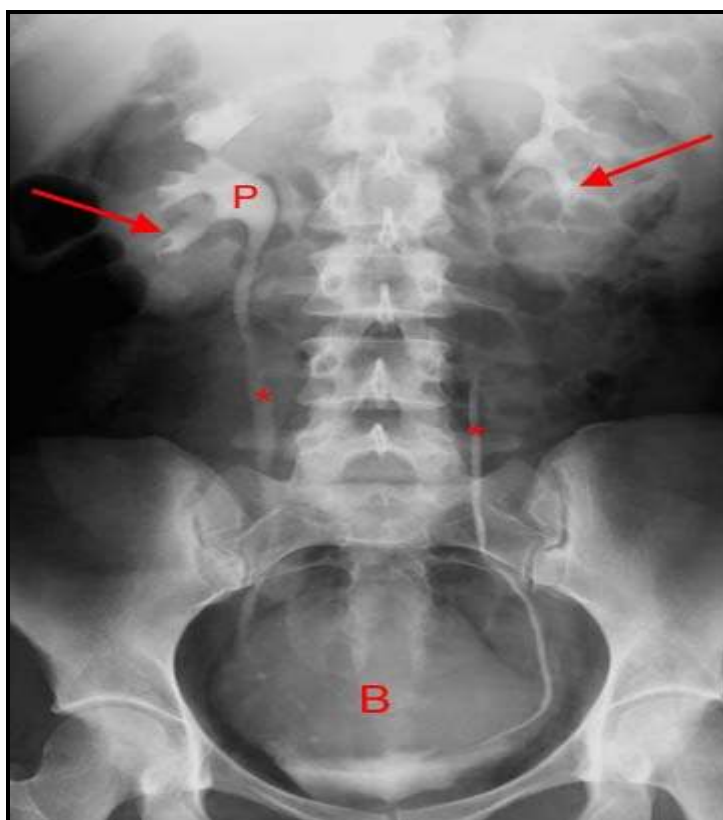
These are circular openings with carinae separating the individual calices along tubular infundibulum connected each ostium to the apex of the major calyx. Which then branches into the minor calices, these are the next structures visible as the ureteroscope enter the infundibulum. The ureteroinfundibular angle represents the angle of deflection necessary for flexible ureteroscope to move from the axis of the upper section of the ureter to the axis of the lower infundibulum (*Bagley and Rittenberg, 2013*).

This was found to be 140 degrees on average; although it may range from 104 to 175 degree. This minor calices can be seen to contract periodically with obliteration of their lumen. This is due to a circular layer of muscle fibers that extends around the base of papilla to help propel urine from the papillary ducts. An inner longitudinal layer of muscle fibers ends where the minor calyx attaches to the renal papilla. The papilla appear endoscopically as bulging discs covered with a pink friable epithelium and surrounded by the paler Caliceal fornix. If more than one papilla projects into the calyx, it is called a compound calyx. The papillae are the apices of the renal pyramids, and as such they receive the papillary ducts of Bellini that drain the pyramids, these ducts can be seen as minute openings which are dilated and more obvious in cases of obstruction (*Huffman, 1998*).

## **RADIOLOGICAL ANATOMY OF THE URETER**

**R**adiologically, the ureter can be divided into upper, middle, and lower segments. The upper ureter extends from the renal pelvis to the upper border of the sacrum. The middle ureter comprises the segment from the upper to the lower border of the sacrum. The lower (distal or pelvic) ureter extends from the lower border of the sacrum to the bladder. Surgically, the ureter is divided by the common iliac artery into abdominal and pelvic portions (*Abdel-Razzak, 2006*).

The renal pelvis is usually opposite L2, with the left side 1 to 2 cm higher. In addition, the kidneys may move up to 4 cm with respiration, so that during inspiration the ureters may become kinked mimicking adhesion or obstruction. Thus the urogram is best obtained in the expiratory phase (*Heyns and Van Gelderen, 1990*).



**Figure (2):** Conventional plain film of the abdomen called a KUB (Kidneys, Ureters, Bladder) obtained following administration of IV contrast for IV urography shows normal collecting Calyces (arrows), renal pelvis (P), ureters (\*) and bladder (B).

The kidney as seen by radiography, is usually 11 to 14 cm in length, with the left side longer than the right by 0.5 cm. This is larger than the actual renal size because of the magnification obtained on the x-ray film and the diuresis associated with the contrast medium (*Anderson et al., 2007*).

Although Calyceal anatomy is usually demonstrated clearly. Its interpretation is still a subject of controversy. In 1901 Brodel published his classic article on renal vasculature

and morphology in which he showed the anterior calices to be more medial than the peripherally situated posterior calices (*Brodel and Gold Berg, 1987*).

This was challenged by Hodson, who offered the view that the anterior row of calices is usually seen more peripherally from the side as cup shaped structure, whereas the posterior papillae are seen more medially and from the end as round concentration of contrast (*Hadson, 1972*).

Anew technique recently introduced allows the reprocessing of standard CT scan to produce three – dimensional reconstructed images of the pelvicaliceal system (*Heyns and Van Gelderen, 1990*). However, this seems to be of limited value in the presence of oblique and lateral x- ray views, ultrasound localization, or the injection of air into the pelvicaliceal system to identify posterior calices.

## PATHOPHYSIOLOGY OF URETERIC OBSTRUCTION

An increase of cytoplasmatic free calcium concentration is one principal mechanism initiating ureteral contraction. It was demonstrated that calcium channel inhibitors counteract the phasic-rhythmic activity in isolated human caliceal segments (Hertle and Nawrath 1984) and in the ureter. (Borghi *et al.*, 1994) smooth muscle in the ureteral wall contracts, in an attempt to resolve obstruction, and becomes spastic if its effort fails. A long isotonic contraction leads to an increased lactic acid production that will irritate type A slow fibers (myelinated) and type C rapid fibers (unmyelinated). These nerve impulses generated travel to medullary segments T11-L2, reaching the central nervous system, where they are specified by location, character, and intensity, which will potentiate the attack.

Endogenous prostaglandin synthesis and calcium influx induce spontaneous rhythmic contractions of the human ureter, which are inhibited by the calcium channel blockers nifedipine and verapamil. This negative effect on ureteral contractility has evoked interest in using calcium channel blockers to facilitate medical-induced stone passage. (Sahin, 1993) three different subtypes of adrenergic receptors have been pharmacologically identified: alpha-1a, alpha-1b, and alpha-1d (Hieble *et al.*, 1995) a heterogeneous distribution of alpha-1 adrenergic

receptor binding sites was detected, with the highest density in the distal ureter (*Sigala et al., 2005*). The distribution of adrenergic receptors throughout the inner and outer smooth muscle of the ureter was highest for alpha-1d, especially in the distal ureter, followed by alpha-1a and alpha-1b adrenergic receptors (*Itoh et al., 2007*). Heterodimers alpha-1a/alpha-1b and alpha-1b/alpha-1d do occur, whereas alpha-1a/alpha-1d adrenergic receptors do not heterodimerize, suggesting a possible regulatory role of alpha-1b. This ability to oligodimerize could influence future drug development (*Uberti et al., 2003*).

The exact pathophysiology of ureteral colic and stone passage is not completely understood. A ureteral stone tends to induce a ureteral inflammatory response by ureteral stone obstruction and ureteral wall tension stimulating prostaglandin.

Synthesis. Prostaglandins have a dilating effect on afferent arterioles resulting in an increased renal blood flow, further increasing ureteropelvic pressure, inflammation, and edema (*Ahmad et al., 1991*). A subsequent increase of smooth muscle contraction impairs propulsive antegrade peristalsis aggravating ureteral obstruction, impaction, and pain (*Yamaguchi et al., 1999*).

# CLASSIFICATION OF URINARY STONES

Urinary stones can be classified according to size, location, X-ray characteristics, etiology of formation, composition, and risk of recurrence (*Kim et al., 2007*).

## 1. Stone size:

Stone size is usually given in one or two dimensions, and stratified into those measuring up to 5, 5-10, 10-20, and > 20 mm in largest diameter.

## 2. Stone location

Stones can be classified according to anatomical position: upper, middle or lower calyx; renal pelvis.

## 3. X-ray characteristics:

Stones can be classified according to plain X-ray appearance (KUB radiography): (Table 1), which varies according to mineral composition (*Kim et al., 2007*).

Non-contrast-enhanced computer tomography (NCCT) can be used to classify stones according to density, inner structure and composition, which can affect treatment decisions.

**Table (1):** X-ray characteristics

Radiopaque	Faint Radiopaque	Radiolucent
Calcium oxalate dihydrate	Magnesium ammonium phosphate	Uric acid
Calcium oxalate monohydrate	Apatite	Ammonium urate
Calcium phosphates	Cystine	Cystine
		2,8-dihydroxyadenine
		Drug-stones

*(Quoted from EAU guidelines on urolithiasis 2014)*

#### 4. Etiology of stone formation:

Stones can be classified into those caused by: infection, or non-infectious causes (infection and non-infection stones); genetic defects; or adverse drug effects what is called (drug stones) *(Yasui et al., 2013)*.

#### 5. Stone composition:

Metabolic aspects are important in stone formation, and metabolic evaluation is required to rule out any disorders. Analysis in relation to metabolic disorders is the basis for further diagnostic and management decisions. Stones are often formed from a mixture of substances *(Yasui et al., 2013)*.