

Introduction

Worldwide urolithiasis prevalence varies from 4-17 cases per 1000 inhabitants. It affects 12% of the world population. It can create pain, hematuria, infection, fever, nausea, vomiting and even uraemia. The goal of treating urinary calculi is to achieve complete stone clearance with minimal morbidity for the patient (*Kijvikai et al., 2012*).

Many theories have been proposed for the pathogenesis of urolithiasis, including nucleation theory, the crystal inhibitor theory or the crystal retention theory. Moreover there are a number of diseases associated with stone formation (i.e. hyperparathyroidism, renal tubular acidosis, cystinuria, hyperoxaluria, intestinal malabsorptive conditions) as well as medications (i.e. calcium supplements, vitamin D, triamterene, indinavir). Predisposing factors for stone formation include anatomical abnormalities (i.e. ureteral strictures, vesico-ureteral reflux, ureteropelvic stenosis, extrinsic ureteral compression and ureterocele among others). Calculus location and size, expressed in millimeters on its two major axes, continue to be important factors for deciding which therapeutic option to utilize. The contemporary guidelines state that stones 0.5 cm or less in diameter have a 80% chance of spontaneous passage, 0.5 cm stones have the chance of 20-50%, and for stones greater than 0.5 cm the patient should consult urologist (*Stefanos et al., 2011*).

Although most ureteral stones pass spontaneously, the pain and cost associated with repeated episodes of renal colic are substantial (*Dellabella et al., 2009*).

Current standard treatment for urolithiasis follows the non-invasive to more invasive spectrum depending on the size of the stone and the location within the ureter, Watchful waiting with conservative analgesic therapy is the first-line in urolithiasis treatment. Non-steroidal anti-inflammatory medication, such as ketorolac and diclofenac, have the possible advantage of decreasing ureteral smooth muscle tone, thereby directly treating the mechanism by which pain is thought to occur namely, ureteral spasm (*Joe Miller et al., 2013*).

On the more invasive end of the spectrum, therapies such as single shock wave lithotripsy, endoscopy, and finally open surgical stone extraction have been the traditional second and third line treatments for urolithiasis. Although there have been vast improvement in the last 20 years with the above mentioned minimally invasive procedures for ureteral stones, there has also been a significant increase in treatment costs (*Seitz et al 2009*).

The costs that Dellabella et al is referencing are not only financial, rather the overall cost to the body in terms of surgical infection, post intervention pain, and other possible post-surgical sequelae (*Dellabella et al., 2009*).

In 2004, Sigala et al published a paper entitled evidence for the presence of alpha-1 adrenergic receptors subtypes in the human ureter, showing increased density of alpha-1_a adrenergic receptors in the smooth muscle of the ureter (*Sigala et al., 2004*).

The implication of their research is crucial in terms of urolithiasis treatment. Urolithiasis migration down the ureter is modulated by the sympathetic nervous system via the alpha-1 adrenergic receptors and the movement of the stone is facilitated by peristaltic movement of the tubular ureter (*Sigala et al., 2004*).

The pain pathway of urolithiasis arises from the increased intraureteral pressure and peristaltic muscle movements in the presence of urolithiasis. Thus, sympathetic alpha adrenergic antagonists such as tamsulosin might have the ability to inhibit basal tone, peristaltic amplitude and frequency, dilating the urethral lumen and decreasing intraureteral pressure, thereby increasing the rate of fluid transport and ultimate facilitation of the passage of the stone (*Sun et al., 2009*).

Aim of work

The purpose of this study is to outline the current role of Tamsulosin as an adjuvant treatment after SWL for patients with renal and upper ureteric stones.

Renal Anatomy

General Anatomy:

The kidneys are paired organs lying retroperitoneal on the posterior abdominal wall. Each kidney is of a characteristic shape, having a superior and an inferior pole, a convex border placed laterally, and a concave medial border. The medial border has a marked depression, the hilum, containing the renal vessels and the renal pelvis (*Drake et al., 2007*).

Position of the Kidneys:

The kidneys lie on the posterior abdominal wall, against the psoas major muscles; their longitudinal axis parallels the oblique course of the psoas (**Fig.1**). Moreover, since the psoas major muscle has a shape of a cone, the kidneys also are dorsal and inclined on the longitudinal axis. Therefore, the superior poles are more medial and more posterior than the inferior poles. As the hilar region is rotated anteriorly on the psoas muscle, the lateral borders of both kidneys are posteriorly positioned. It means that the kidneys are angled 30 to 50° behind the frontal (coronal) plane (**Fig. 2**) (*Drake et al., 2007*).

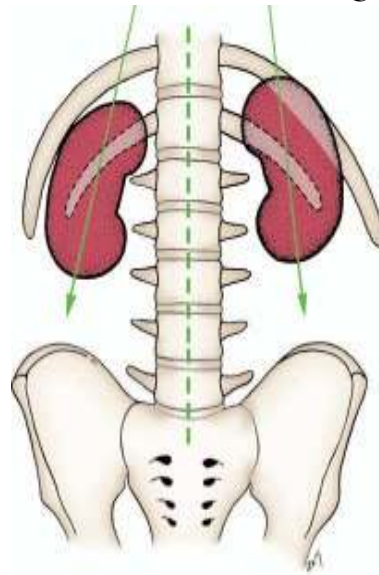


Figure (1): Anterior view of the kidneys in relation to the skeleton, shows that the longitudinal axis of the kidneys are oblique (*Sampaio, 2000*).

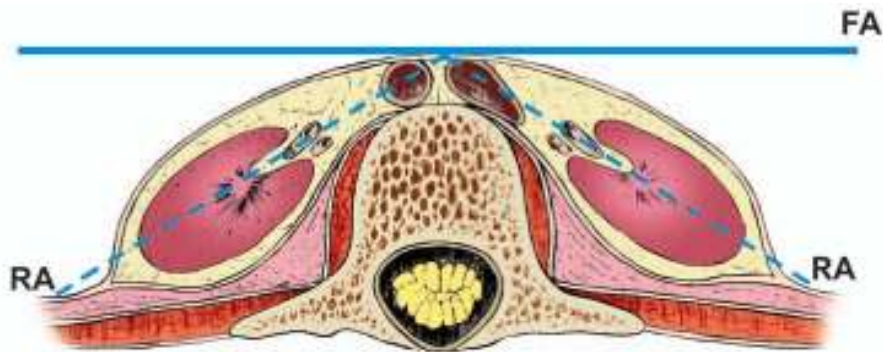


Figure (2): Superior view of a transverse section of the kidneys at the level of the 2nd lumbar vertebra (*Sampaio, 2000*).

Perirenal Coverings:

The kidney surface is enclosed in a continuous covering of fibrous tissue called the “renal capsule” or “true renal capsule”. Each kidney within its capsule is surrounded by a mass

of adipose tissue called the perirenal fat (**Fig.2&4**). The perirenal fat is enclosed by the renal fascia (so-called fibrous renal fascia of Gerota's fascia). The renal fascia is enclosed anteriorly and posteriorly by another layer of adipose tissue, which varies in thickness, called the pararenal fat (**Fig. 4**) (*Sampaio, 2000*).

The renal fascia comprises a posterior layer (a well-defined and strong structure) and an anterior layer, which is a more delicate structure that tends to adhere to the peritoneum (**Fig. 2 & 4**). The anterior and posterior layers of the renal fascia (Gerota's fascia) subdivide the retroperitoneal space in three potential compartments (**Fig. 3**).

1. The posterior pararenal space, which contains only fat.
2. The intermediate perirenal space, which contains the suprarenal glands, kidneys and proximal ureters, together with the perirenal fat.
3. The anterior pararenal space, which unlike the posterior and intermediate spaces, extends across the midline from one side of the abdomen to the other. This space contains the ascending and descending colon, the duodenal loop and the pancreas.

(*Sampaio, 2000*)

Inferiorly, the layers of the renal fascia end weakly fusing around the ureter. Superiorly, the two layers of the renal

fascia fuse above the suprarenal gland and end fused with the infra-diaphragmatic fascia. An additional fascial layer separates the suprarenal gland from the kidney. Laterally, the two layers of the renal fascia fuse behind the ascending and descending colons. Medially, the posterior fascial layer is fused with the fascia of the spine muscles. The anterior fascial layer merges into the connective tissue of the great vessels (Aorta and IVC) (**Fig. 2&4**). These anatomic descriptions of the renal fascia show that right and left perirenal spaces are potentially separated, and therefore, it is exceptional that a haematoma, urinoma or perirenal abscess involves the contralateral perirenal space(**Fig.4**) (*Drake et al., 2007*).

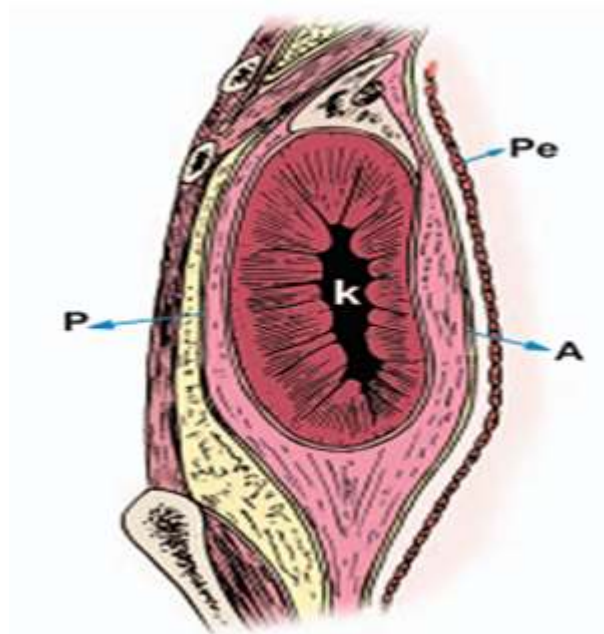


Figure (3): Lateral view of a longitudinal section through the retroperitoneum, reveals the posterior (*P*) and the anterior (*A*) layers of the renal fascia. *Pe*= peritoneum; *K* = kidney (*Sampaio, 2000*).

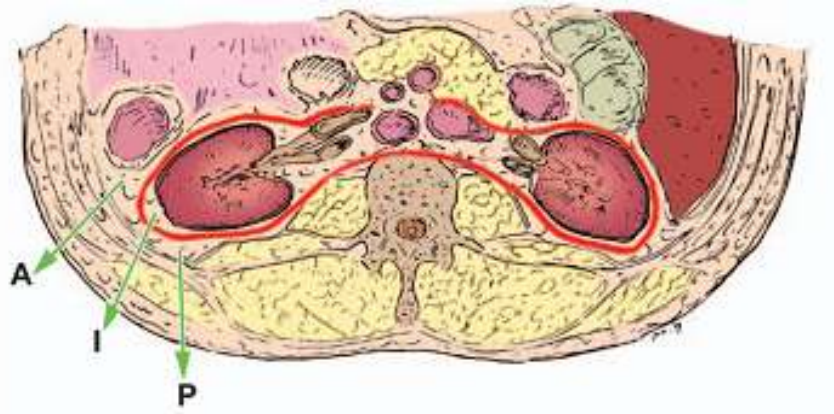


Figure (4): Superior view of a transverse section of the kidneys at the level of the 2nd lumbar vertebra shows the three compartments of the retroperitoneal space (*Sampaio, 2000*).

Posteriorly, the superior pole of each kidney rests against the diaphragm and the tips of the 11th and 12th ribs. Deep to this, the underlying pleura attaches to the 11th rib.

The anterior surface of the right kidney is associated with the liver superiorly, the curve of the duodenum over the mid portion and the ascending colon inferior and medially. On the right side, the colon often covers the lower half of the kidney medially. The anterior surface of the upper pole of the left kidney is covered by the spleen superiorly and just the tail of the pancreas medially as well as by, the splenic flexure of the colon; the anteromedial surface of the entire left kidney is covered by the descending colon. A retrorenal colon can be seen on either side in 1–10% of percutaneous cases depending on patient positioning; it is more common when the patient is in the prone position (*Sampaio, 1996*).

The kidney can be divided into anterior and posterior segments. The plane of division for these segments rests 30–50° posterior to the frontal plane of division for the body as a whole owing to the rotation of the renal axis anteriorly by the psoas major muscle (**Fig. 5**) (*Kaye, 1984*).

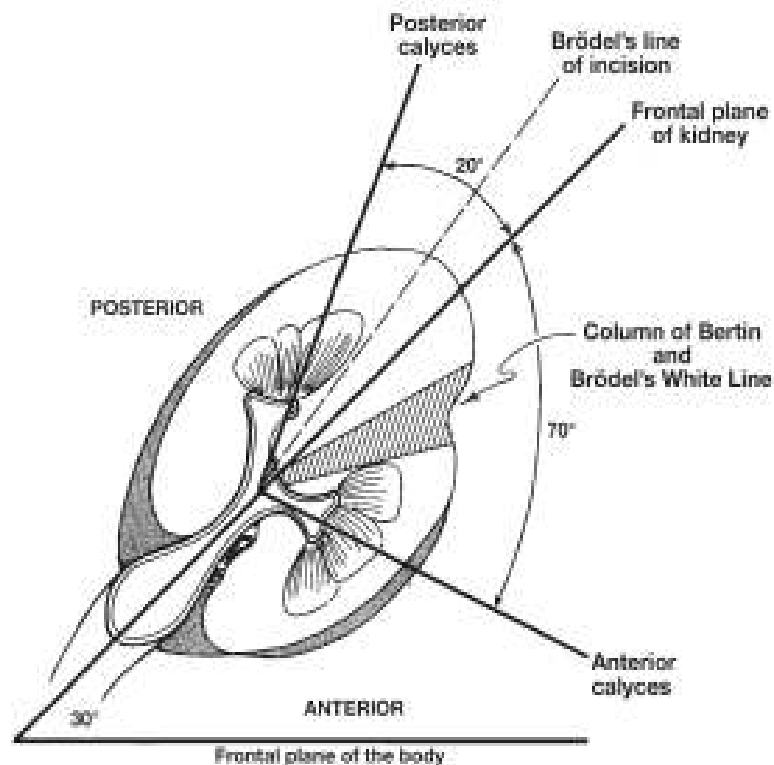


Figure (5): Left kidney viewed from above showing anterior calyces projecting 70° and posterior calyces 20° from the frontal plane of the kidney, as in a classic Brodel-type kidney (*Quoted from Louis Eichel, urinary stone disease 2007*).

Anatomy of 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 (*Srinivasa et al., 2009*).

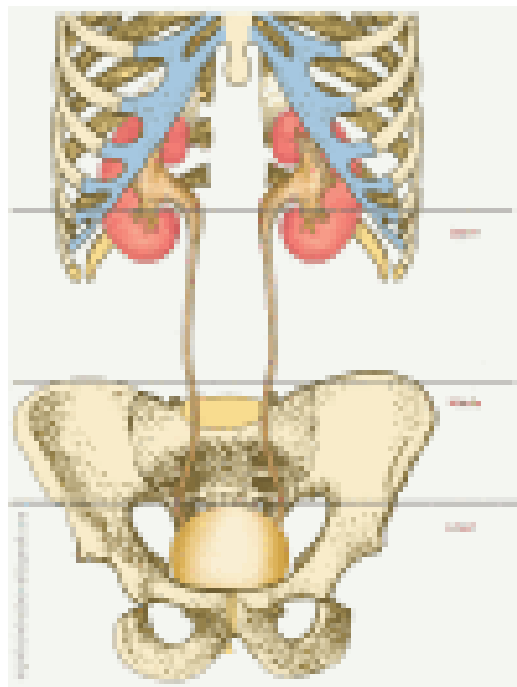


Figure (6): Anatomic division of the ureter (*Srinivasa et al., 2009*).

The terminal portion of the ureter may be subdivided further into the juxtavesical, intramural, and submucosal portions. The surgeon must pay special attention to the gonadal and iliac

vessels, as they cross the ureter at the posterior and anterior levels respectively, descending into the pelvis (*Srinivasa et al., 2009*).

The left ureteropelvic junction is posterior to the pancreas and ligament of Treitz. The inferior mesenteric artery and sigmoidal vessels cross in front of the left ureter at its inferior pole. On the right side, the ureter lies posterior to the duodenum and just lateral to the inferior vena cava, with the right colic and ileocolic vessels crossing in front. Due to this protection, injuries to the ureter are typically accompanied by significant collateral damage and management is related to the severity of associated injuries (*Srinivasa et al., 2009*).

The ureter's blood supply comes from the ureteral artery, which runs longitudinally along the ureter and lacks collateral flow in 80% of patients. The upper third of the ureteral artery is supplied by the aorta and renal artery, while branches of the iliac, lumbar and vesicular arteries supply the middle and lower thirds of the ureter. In the abdomen the blood supply is medial, while in the pelvis the blood supply is lateral with the richest blood supply to the pelvic ureter. From a surgical standpoint, knowledge of the vascular supply to the ureter is crucial prior to any manipulation and subsequent repair. This tenuous blood supply must be considered when dealing with complex repairs of significant injuries and strict adherence to the principles of ureteral repair can prevent complications such as leak, renal injury and in some cases, death (*Tezval et al., 2007*).

The ureters are richly innervated by nerves that travel alongside the blood vessels. The primary sensation to the ureters is provided by nerves that come from T12-L2 segments of the spinal cord. Thus pain may be referred to the dermatomes of T12-L2, namely the back and sides of the abdomen, the scrotum (males) or labia majora (females) and upper part of the front of the thigh (*Drake et al., 2007*).

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. No continuous lymph channels extend from the kidney to the bladder. Lymphatic drainage from the ureter drains to regional lymph nodes including the common iliac, external iliac and hypogastric lymph nodes (*Lowe et al., 2005*).

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, 1998*).

Three major functions are attributed to the renal pelvis and ureters: absorption, dynamics, and tonus. Absorption is minimal and unaffected by repair of the ureter and its consequent function. The dynamics reflect the synchronous and progressive contractile movement of the ureter away from the ureteropelvic junction (UPJ) to the ureter-vesical orifice, produced by the intrinsic automaticity of the ureteral musculature (*Narath, 1940*).

Tonus of the ureter is the degree of contraction that the ureteral wall assumes for a given rate and volume of urinary output. Tonus initiates detrusor action at a certain volume, thus perpetrating the cyclical undulations. When a ureter is damaged by penetrating or blunt trauma, peristalsis beyond the injury ceases. Tonus is decreased in the ureter, proximal to the injury, due to stretching from the increased volume of urine in this segment. This increased volume of urine is the result of detrusor action being halted at the damaged (inert) segment of the ureter. Thus, urine volume, diuresis and distention are the main modulators of peristalsis along with the sympathetic and parasympathetic nervous system; however, prostaglandins and tachykinins also play a role (*Pumphrey et al., 2002*).

Classification of renal stones

Kidney stones are typically classified by their location and chemical composition.

Chemical composition:

○ Calcium-containing stones

By far, the most common type of kidney stones worldwide contains calcium. For example, calcium-containing stones represent about 80% of all cases in the United States; these typically contain calcium oxalate either alone or in combination with calcium phosphate in the form of apatite or brushite (*Vijaya et al., 2013*).

Factors that promote the precipitation of oxalate crystals in the urine, such as primary hyperoxaluria, are associated with the development of calcium oxalate stones (*Vijaya et al., 2013*).

The formation of calcium phosphate stones is associated with conditions such as hyperparathyroidism and renal tubular acidosis (*Vijaya et al., 2013*).

○ Struvite stones

About 10–15% of urinary calculi are composed of struvite (ammonium magnesium phosphate, $\text{NH}_4 \text{MgPO}_4 \cdot 6\text{H}_2\text{O}$). Struvite stones (also known as "infection stones",