

## UROGENITAL ANATOMY- HOW IT IS PRONE FOR UROLITHIASIS ?

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**Abstract:** Most recent studies report that the incidence of kidney and ureteral stones has increased significantly in both adults and children. According to the literature, renal calculi formation is a relatively complex process and involves an interplay of various physiochemical and anatomical factors. Almost 50% of the pediatric cases of urolithiasis are idiopathic and the rest due to hypercalciuria (30%) and hyperoxaluria (20%), or rarely due to hyperuricosuria, xanthinuria or hypocitricuria in addition metabolic disorders are also a contributing factor. The predisposition to urolithiasis in all these conditions are also contributed by the intricate anatomical structure and complex development of the urogenital system. The article briefly describes the gross anatomical structure and the development of the urinary tract. Urinary organs develop from intermediate mesenchyme. The intermediate mesenchyme is found longitudinally placed in the trunk, sub-adjacent to somites, at the junction between splanchnopleuric mesoderm and somatopleuric mesoderm. In brief, the ureteral bud developing from the mesonephric duct dilates to form the ampulla which induces the surrounding mesenchymatous tissue i.e. metanephros resulting in the differentiation of mesenchymal agglomerates and their organization in concentrates. The condensate then epithelizes and forms vesicles, which fuse with the ampulla to form a nephron. The various congenital anomalies which can lead to an obstructive uropathy due to hydronephrotic changes causing urinary stasis and, subsequently become a major predisposing factor for urolithiasis. All these have been discussed from an anatomical perspective.

Urolithiasis is a problem that is generally increasing in the tropics as well as in most of the Western countries. There are 2 main types of the urolithiasis the bladder stones in children, and upper urinary tract stones in adults. The former has been decreasing in most developed countries with gradual improvement in levels of nutrition. Reno-ureteral calculosis typical of adult age is more frequent in economically developed countries, with a prevalence rate between 4% and 20%. On the contrary "primitive" vesical calculosis is fairly widespread in Asia, due to malnutrition in the very early years of life. However, the concurrence of a genetic predisposition seems to be crucial for stone formation<sup>1</sup>.

Congenital anomalies and anatomical variations in the urogenital tract can be a major contributing factor in stone formation. It is well known that renal calculi formation is a relatively complex process and involves an interplay of various physiochemical and anatomical factors. Literature shows that almost 50% of the pediatric cases of urolithiasis are idiopathic and the rest due to hypercalciuria (30%) and hyperoxaluria (20%), or rarely due to hyperuricosuria, xanthinuria or hypocitricuria. Hypocitricuria has been quoted as the most important risk factor in the development of idiopathic calcium stones in children. Male children suffer more frequently with urolithiasis<sup>2</sup>. The urinary concentration of stone-forming ions like calcium and oxalate is vital, especially in combination with a deficiency of inhibitors of crystallization-like citrate. In addition, abnormal urinary pH can contribute significantly to stone formation by affecting ion solubility and thereby promoting their crystallization. Metabolic disorders are uncommon etiopathological factors for urolithiasis<sup>3</sup>. Other known predisposing factors for urolithiasis are obstructive uropathy, ureterovesicular reflux, neurogenic bladder, renal foreign body, renal papillary necrosis and UTI. Neonatal renal calculus formation can be subsequent to maternal conditions like hyperparathyroidism, Vitamin D intoxication and diuretic therapy, or due to neonatal diseases like hyperparathyroidism, hypothyroidism, idiopathic hypercalciuria, renal tubular acidosis, inborn errors of metabolism and steroid or diuretic therapy<sup>4</sup>.

In a recent analysis of McKusick's On-line Mendelian Inheritance in Man (OMIM) database, NL was found to be a component of more than 30 genetic disorders, namely familial idiopathic hypercalciuria,

(autosomal dominant) Dent's disease (X-linked recessive), adenine phosphoribosyl-transferase deficiency (autosomal dominant), idiopathic calcium oxalate nephrolithiasis (autosomal dominant/polygenic) and several others, which are either autosomal recessive or X-linked<sup>3</sup> autosomal recessive. The pathogenesis of urolithiasis in these conditions is not well understood. Some of these disorders, especially those inherited via an autosomal recessive or X-linked autosomal recessive pattern, can result in end-stage renal disease. The emphasis on genetic counseling and the possibility of early prenatal diagnosis of congenital malformation have stimulated interest in fetal anatomy<sup>5</sup>.

**The question arises-Is the anatomy of the urinary system such that it could lead to calculi formation?**

Most recent studies report that the incidence of kidney and ureteral stones has increased significantly in both adults and children. Hence to understand the possibility of calculi formation it is necessary to briefly understand the gross structure and development of the urogenital system.

### UROGENITAL SYSTEM

#### Gross Anatomy

The urogenital system comprises of the urinary tract and the reproductive system. The kidneys are essentially regulatory organs which maintain the volume and composition of body fluid by filtration of the blood and selective re absorption or secretion of filtered solutes, the kidneys are retroperitoneal organs (ie located behind the peritoneum) situated on the posterior wall of the abdomen on each side of the vertebral column, at about the level of the twelfth rib. The left kidney is slightly higher in the abdomen than the right, due to the presence of the liver pushing the right kidney down.

The kidneys derive their blood supply directly from the aorta via the renal arteries; blood is returned to the inferior vena cava via the renal veins. Urine (the filtered product containing waste materials and water) excreted from the kidneys passes down the fibromuscular ureters and collects in the bladder. The bladder muscle (the detrusor muscle) is capable of distending to accept urine without increasing the pressure inside; this means that large volumes can be collected (700-1000ml) without high-pressure damage to the renal system.