

**PROF. M. O. AL-SOHAIBANI**

**\_URINARY TRACT  
OBSTRUCTION, UROLITHIASIS**

- **Urinary Tract Obstruction:**
- **Recognition of obstruction is important since it increase the chance of infection and stone formation. In addition unresolved obstruction almost always lead to permanent renal atrophy fortunately many causes of obstruction are surgically correctable or medically treatable.**
- **It can be sudden or insidious, partial or complete, unilateral or bilateral. It may occur at any level of the urinary tract from the urethra to the renal pelvis.**
- 
- **It can be caused by lesion that are intrinsic to urinary tract or extrinsic lesion that compress the ureter.**

- **Common causes include:**

**Congenital anomalies as posterior urethral valves and urethral stricture, meatal stenosis, bladder neck, obstruction, ureteropelvic junction narrowing or obstruction, severe vesico-ureteral reflux.**

- **Urinary calculi.**

- **Benign prostatic hypertrophy.**

- **Tumors.**

- **Inflammation as prostates, ureteritis, urethritis, retroperitoneal fibrosis.**

- **Sloughed papillae or blood clot.**

- **Normal pregnancy.**

- **Uterine prolapse and cystocele.**

- **Functional disorder and neurogenic bladder spinal cord injury.**

- **Urinary Outflow Obstruction - Renal Stones**
- **Urolithiasis** is calculus formation at any level in the urinary collection system, but most often calculi arise in the kidney. Symptomatic urolithiasis is most common in males. **It is common clinical problem M > F peak age 20-30 years.**

- **ETIOLOGY AND PATHOGENESIS:** About 75% of renal stones are composed of either calcium oxalate or calcium oxalate mixed with calcium phosphate. Another 15-20% are composed of magnesium ammonium phosphate struvite and 10% are either uric acid or cystine stones 1-2%. In all cases, there is an organic matrix of mucoprotein that makes up about 2.5% of the stone by weight, unknown type represents  $\pm 5\%$  .
- The cause of stone formation is often obscure, particularly in the cases of calcium containing stones, 50% of the patients who develop calcium stones have hypercalciuria that is not associated with hypercalcemia. Most in this group absorb calcium from the gut in excessive amounts and promptly excrete in the urine, and some have a primary renal defect of calcium reabsorption. Ten percent of patients have hypercalcemia (due to hyperparathyroidism, vitamin D intoxication, sarcoidosis) and consequent hypercalciuria.

- **The causes of the other types of renal stones are better understood. Magnesium ammonium phosphate (struvite) stones almost always occur in patients with a persistently alkaline urine, owing to urinary tract infections. In particular, the urea-splitting bacteria, such as Proteus vulgaris and the staphylococci, predispose toward urolithiasis. Moreover, bacteria may serve as particular nidi for the formation of any kind of stone. In avitaminosis A, desquamated squames from the metaplastic epithelium of the collecting system act as nidi.**
- **Gout and diseases involving rapid cell turnover, such as the leukemias, lead to high uric acid levels in the urine and the possibility of uric acid stones. Uric acid and cystine stones are seen in acid urine while magnesium ammonium phosphate stone precipitate in alkaline urine.**

- **MORPHOLOGY.** Stones are unilateral in about 80% of patients. Common sites of formation are renal pelvis, calyces and the bladder. Often many stones are found in one kidney. They tend to be small (average diameter 2 to 3 mm) and may be smooth or jagged. Occasionally, progressive accretion of salts leads to the development of branching structures known as **staghorn calculi**, which create a cast of the renal pelvis and calyceal system. These massive stones are usually composed of magnesium ammonium phosphate.
- **Clinical Course.** Stones may be present without producing either symptoms or significant renal damage. This is particularly true with large stones lodged in the renal pelvis. Smaller stones may pass into the ureter, producing a typical intense pain known as renal or ureteral colic, characterized by paroxysms of flank pain radiating toward the groin. Often at this time, there is gross haematuria. The clinical significance of stones lies in their capacity to obstruct urine flow or to produce sufficient trauma to cause ulceration and bleeding. In either case, they predispose to bacterial infection. Fortunately, in most cases the diagnosis is readily made by radiological means.

- **Hydronephrosis is the dilation of the renal pelvis and calyces due to obstruction of urinary outflow causing progressive atrophy of the kidney. The obstruction can occur at any level in the urinary tract. The glomerular function can continue for sometimes even with complete obstruction because the filtrate diffuse back into the interstitium and perirenal spaces where it ultimately return to venous and lymphatic circulation. This continuous filtration causes calyces and pelvic dilatation.**
- **There will increased presence in renal pelvis leading to backflow to collecting ducts and renal cortex causing its atrophy, in addition to compression vascular network of the medulla →↓ plasma fluid. This medullary functional disturbance is reversible. GFR started to diminish later. Obstruction also trigger an interstitial inflammatory interstitial fibrosis.**
- **The most common causes are nodular hyperplasia of the prostate, calculi, or malignant tumors such as cervical or bladder carcinoma.**

- **a) Clinical Cause**
- **Acute obstruction – Pain due to distension of collecting system or renal capsule.**
- **Unilateral, complete or partial hydronephrosis can remain silent for long period since the unaffected kidney maintain adequate function.**
- **Bilateral partial obstruction manifested early by inability to concentrate urine having polyuria and nocturia. Some patients acquire tubular acidosis, renal salt wasting secondary to renal calculi and a typical picture of tubulo-interstitial inflammation with scarring and atrophy of the papillae and medulla. These patients commonly tend to have hypertension massive with having urine rich in NaCl.**
- **Complete bilateral obstruction → oliguria or anuria and is incompatible with long survival unless corrected.**

- **b) Pathology.**

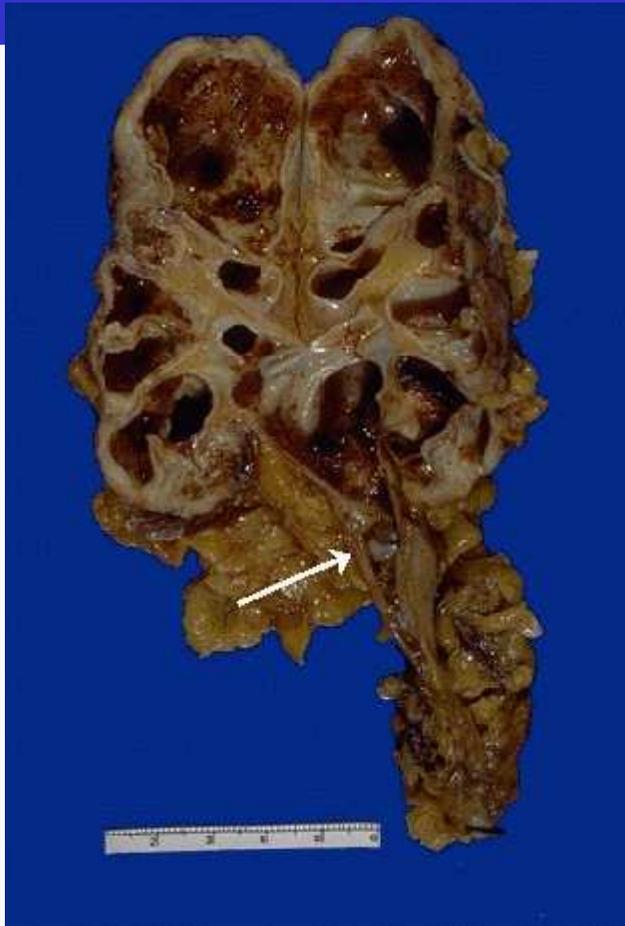
- **1) Macroscopic appearance.** The kidneys may show some degree of enlargement. Cut section shows blunting of the renal calices and cortical thinning. The pelvis is dilated massively and is filled with urine.
- **2) Microscopic appearance.** The tubules may be atrophic, but the glomeruli become hyalinized only in late stages of the disease. Chronic inflammatory cells may be present in the interstitium.
- **c) Treatment** is directed toward the cause of the hydronephrosis. Nephrectomy is performed for some unilateral cases produced by calculi.



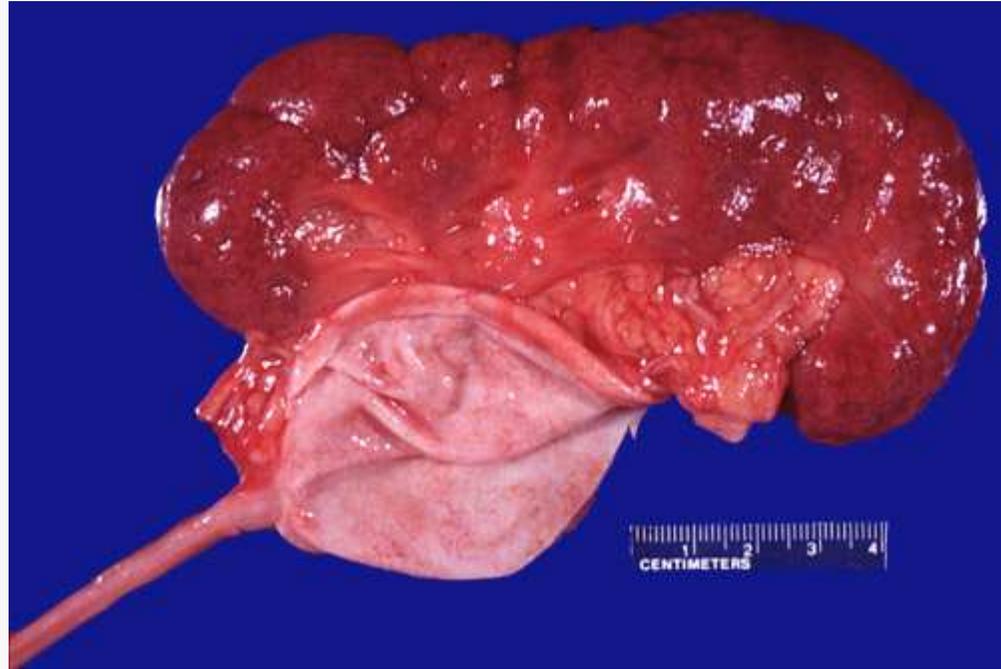
**Sometimes a very large calculus nearly fills the calyceal system, with extensions into calyces that give the appearance of a stag's (deer) horns. Hence, the name "staghorn calculus". Seen here is a horn-like stone extending into a dilated calyx, with nearly unrecognizable overlying renal cortex from severe hydronephrosis and pyelonephritis. Nephrectomy may be performed because the kidney is non-functional and serves only as a source for infection.**



Here is a kidney with much more advanced hydronephrosis in which there is only a thin rim of **remaining renal cortex**. Such a kidney is non-functional and a source for ongoing infection. If this process is unilateral, then the problem originates from the ureteral orifice up to the pelvis. In this case, a large "staghorn" calculus (so named because the prominent projections of the stone into the calyces resemble deer antlers) was present that filled up the pelvis and calyceal system. If this process were bilateral, then the problem would originate in the bladder trigone or urethra (or the prostate around the urethra) or some process (such as a large neoplasm) that could impinge on both ureters.



**The arrow points to the culprit in this case of hydronephrosis--a ureteral calculus caught at the ureteropelvic junction. This kidney demonstrates marked hydronephrosis with nearly complete loss of cortex. Such a kidney would be non-functional. If the other kidney had sufficient function, then renal failure will not ensue. There is sufficient renal reserve capacity that it is possible to survive with half of a normal kidney.**



**There is scarring of this kidney from chronic obstruction and pyelonephritis. The renal pelvis is markedly dilated, but the ureter is not, indicating that the point of obstruction is the ureteropelvic junction.**



**A long-standing obstruction (probably congenital) at the ureteral orifice through which the metal probe passes led to the marked hydroureter and hydronephrosis seen here. In the intravenous urogram below, note the dilation of the right ureter, compared to the normal left ureter. This patient had vesicoureteral reflux. Such obstructive processes increase the risk for urinary tract infection.**



**There is one relatively normal-sized kidney with a granular surface and a few scattered, shallow cortical scars. The other kidney shows atrophy because of renal arterial occlusion. Such a situation can lead to hypertension (Goldblatt kidney).**