**Intended Learning Objectives (ILOs)**

* Discuss the etiology, types and pathological consequences of urinary calculi.
* Enumerate congenital anomalies of the urinary bladder and discuss their etiology and complications.
* Discuss the etiology, types and pathological consequences of urinary calculi.
* Define cystitis and discuss its etiology and complication.
* Give an account on bilharzial cystitis: its etiology, pathologic features and complications.

**Urinary Calculi**

**Causes:** Stone formation is enhanced by a deficiency in inhibitors of crystal formation in urine (Disturbance in the ratio of colloids-crystalloids).

 **Predisposing factors:**

**1-** Infection: - Change of PH to alkaline.

 - Necrotic material acts as a nidus

2- Obstruction: **→** stagnation of urine **→** infection.

3- **↑** concentration of crystalloids:

 a- Hyperparathyroidism **→↑**Ca & ph in urine.

 b- Excess intake of food rich in oxalate **→** oxaluria.

 c- Familial cystinuria.

 d- **↑**concentration of urine as in hot weather.

4- Urinary bilharziasis:

 - Ova, necrotic tissue & blood clots act as a nucleus.

 - Predispose to secondary infection.

**Types:**

 1- **Calcium oxalate & phosphate stone:** primary, the commonest (70%).

 - in the pelvis & calyces - small - multiple - very hard

 - rough outer surface - dark brown - radio-opaque

 2- **Urate & uric acid stone:** primary (5-10%)

 - usually single - in the pelvis & calyces - in acidic urine

 - hard - smooth outer surface - radiolucent

 3- **Cystine & xanthine stones:** primary -very rare (2%)

 - develop in cases of cystinuria - soft & yellowish green

 4- ***Triple stones* or *struvite stones*:** composed of magnesium ammonium phosphate:

 - Secondary stone, common (15-20%)

 - in the pelvis, calyces &urinary bladder - alkaline urine - rounded

 - white (chalky) - friable - smooth or lamillated outer surface
 -the largest stones occupying large portions of the renal pelvis (staghorn calculi).

 - may form on a nidus of oxalate or urate stone.

 5- Other or Unknown (+ 5)

**Effects and complications:**

**(1) Pain***:* either:
 (a) Fixed dull aching pain (big stone).
 (b) Renal colic results from contraction of the ureteric muscle to move a small stone
 down the ureteric lumen.
**(2) Mechanical irritation***:* Causes:
 (a) Haematuria.

 (b) Mucosal ulceration with consequent stenosis.
 (c) Squamous metaplasia **→** Squamous cell carcinoma of urinary bladder or renal
 pelvis.
 **(3)** **Obstructive effects:** (a) Hypertrophy and dilatation of the bladder. Trabeculation and diverticula
 formation may occur.
 (b) Hydroureter and hydronephrosis.
 (c) Calculus anuria and acute renal failure due to sudden complete obstruction.
  **(4) Infection:**Obstruction by stones facilitates infection with the development of
 cystitis, ureteritis, pyelonephritis, pyoureter and pyonephrosis.

**Comparison between different types of stones**

|  |  |  |  |
| --- | --- | --- | --- |
|  | **CALCIUM OXALATE AND PHOSPHATE (70%)** | **Urate (5-10%)** | **MAGNESIUM AMMONIUM PHOSPHATE (Trible or STRUVITE) (15-20%)** |
| **Type****Composition****Site****Size****Surface** **Consistency** **Colour****In X-ray** | Primary stoneAcidic urine Calcium oxalate & phosphatePelvis & calyces Small, often multipleRough & spikyHardDark brown or black (due to blood pigments)Radio-opaque | Primary stoneAcidic urineUric acid, sod. urate & ammon. UratePelvis & calyces Usually small may be large (staghorn stone)SmoothHardYellow brownRadiotransluscent | Seconday stoneAlkaline urine Magnesium amonium phosphatePelvis, calyces & urinary bladderThe largest stone(staghorn stone)SmoothFriableChalky whiteRadio-opaque |

**Diseases of Urinary Bladder**

**Congenital anomalies:**

**1- Ectopia vesica (exostrophy):**

Anterior wall of urinary bladder & overlying part of anterior abdominal wall are defective.

Complications: inflammation, squamous or glandular metaplasia, carcinoma (transitional, squamous or Adenocarcinoma).

**2- Epispadius:** The urethra opens on the dorsum of the penis.

**3- Hypospadius:** The urethra opens on the ventral surface of the penis.

**4- Patent uracus:** A fistulous tract extends from the bladder to the
 umbilicus discharging urine to outside.

 **Bladder Diverticulae:**

 **Definition:** Local dilatation in the bladder wall forming a pouch.

 **Types:**

1. **Congenital (true):**

 **-** Single
 - Wall contains all layers of bladder.

1. **Acquired (false):**

 **-** Multiple.
 - Occurs in cases of bladder neck obstruction due to **↑** pressure.

 - Wall: mucosa, submucosa & few muscle bundles.

 - Complications: infection, ulceration, stone formation & carc.

 **Cystitis**

**Predisposing factors:**

1- Staises.

2- Inflammation in near-by organs.

3- General disease (DM & septicaemia).

4- Females are more susceptible due to short & wide urethra.

**Causative organisms:**

- Commonly E. coli.

- Other organisms as: strept., staph., gonococci & bacillus pyocyaneous.

**Types:**

\*Acute:

* 1. Characterised by frequency, dysuria, pyuria, suprapubic pain.
	2. Pathology

\*Chronic non-specific: may follow repeated acute attacks or start as such.

 Pathology: it may be associated with:

 1- Leukoplakia 2- Follicular cystitis

 3- **Eosinophilic cystitis**, manifested by infiltration with submucosal eosinophils,
 typically is a nonspecific subacute inflammation but may also be a manifestation

 of a systemic allergic disorder.

 4- Malakoplakia (Michaelis-Gutmann bodies)

Malacoplakia:

This designation refers to a peculiar pattern of vesical inflammatory reaction characterized macroscopically by soft, yellow, slightly raised mucosal plaques 3 to 4 cm in diameter, and histologically by infiltration with large, foamy macrophages mixed with occasional multinucleate giant cells and interspersed lymphocytes. The macrophages have an abundant granular cytoplasm due to phagosomes stuffed with particulate and membranous debris of bacterial origin. In addition, laminated mineralized concretions resulting from deposition of calcium in enlarged lysosomes, known as Michaelis-Gutmann bodies, are typically present within the macrophages and between cells. Similar lesions have been described in the colon, lungs, bones, kidneys, prostate, and epididymis

\*Chronic specific:

 1- Bilharzial cystitis.

 2- Tuberculous cystitis.

**BILHARZIASIS OF THE URINARY BLADDER**

**Pathological Features:**

- It is more common in Upper Egypt and results from Schistosoma haematobium.

- Ova retained in the bladder wall produce granulomatous reaction, mostly in the submucosa; formed of lymphocytes, eosinophils, plasma cells, histiocytes, foreign body giant cells and fibroblasts. The ova seen in this reaction may be living, dead or calcified. This granulomatous reaction ends by fibrosis.

- The frequent sites affected are the trigone, posterior wall and lower ureters.

- Eggs passing through the mucosa produce focal erosion of the epithelium, cystitis and terminal painful haematuria.

- Early lesions consist only of bilharzial cystitis (hyperaemia, inflammatory oedema, petechial haemorrhage and bilharzial granuloma).

- Prolonged and severe infection results in gross lesions in the form of sandy patches, polyps, ulcers and epithelial changes.

**(1) Sandy Patches:** Are common in bilharziasis of the bladder. The mucosa shows dirty yellow patches. The epithelium of the patches is atrophic and covers a fibrotic submucosa packed with heavy deposits of calcified ova.
**(2) Bilharzial Ulcers:** May results from ulceration of the mucosa over an area of sandy patches or tiny ulcers result from extrusion of the ova through the mucosa, they fuse into larger rounded ulcers.

**(3) Bilharzial Polyps:** Are not common in the bladder. They may be single or multiple, but always few in number. The small polyps are sessile, larger ones are pedunculated. A single polyp may enlarge and must be differentiated from polypoid carcinoma.

**(4) Epithelial Changes:**

These changes occur in the urothelium of the urinary bladder in response to bilharzial granuloma and the associated bacterial infection. They are precancerous especially squamous metaplasia.

**(a) *Hyperplasia:***Usually over the fresh bilharzial granuloma.

**(b) *Brunn’s nests:***The hyperplasic epithelium dips in the submucosa forming round or oval nests of transitional epithelium.

**(c) *Cystitis cystica:***Hydropic degeneration in the central cells of Brunn’s nest results in a cyst lined by few layers of transitional epithelium and contain amorphous hyaline debris.

**(d) *Cystitis glandularis:***the lining of cystitis cystica undergoes metaplasia from transitional to columnar epithelium.

**(e)** ***Squamous metaplasia:***

*The urothelium of the urinary bladder is transformed into stratified squamous epithelium. It o*ccurs mostly in the trigone. The area of squamous metaplasia may show extensive keratinization at the surface. It appears as irregular white patches called “Leukoplakia”.

**Complications of Bilharzial Cystitis**:

(1) Microcytic hypochromic anaemia due to persistent hematuria.

(2) Bacterial infection of the bladder wall extends to the perivesical tissue leading to fistulous tracts between the bladder and the skin, rectum or vagina.

(3) Urinary calculi: Due to stasis of urine which helps infection. Infection changes the PH of urine to alkaline and this helps phosphate deposition and formation of phosphate stones. Also ova and epithelial debris form nuclei upon which stone is formed.

 (4) Fibrosis and thickening of the bladder wall with contraction of the bladder and narrowing of the lumen.

(5) Bladder neck obstruction due to fibrosis leading to retention of urine.

(6) Hydroureter and hydronephrosis due to stricture of the bladder neck, ureteric orifices or the ureter itself.

(7) Pyoureter and pyonephrosis.

(8) Uremia and renal failure terminates the life of the patient if hydronephrosis is bilateral.

(9) Carcinoma of the urinary bladder commonly develops on top of squamous metaplasia (squamous cell carcinoma).

**BILHARZIASIS OF THE URETER**

**-** The lesions usually occur in the lower third, but may affect any segment.

**-** Bilharzial granuloma, sandy patches, ulcers, polypi and ureteritis cystica Occur.

- Bilharzjal lesions heal by fibrosis causing ureteric stricture with the development of hydroureter, pyoureter, hydronephrosis, pyonephrosis, Pyelonephritis, renal stones and uraemia.

**Tuberculous cystitis**

* Infection is usually secondary to renal TB, but it may complicate TB of the prostate, seminal vesicles and epididymis in males or fallopiam tubes in females.
* The tubercles are formed in the submucosa and the overlying urothelium becomes ulcerated.