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Lecture- 1

Bladder

Objectives:

1. To revise briefly the anatomy, physiolgy, embryology, and histology of the urinary bladder.
2. To illustrate the presentation, diagnosis and treatment of congenital diseases of the bladder.
3. To illustrate the presentation, diagnosis and treatment of some acquired diseases of the bladder.

Anatomy:

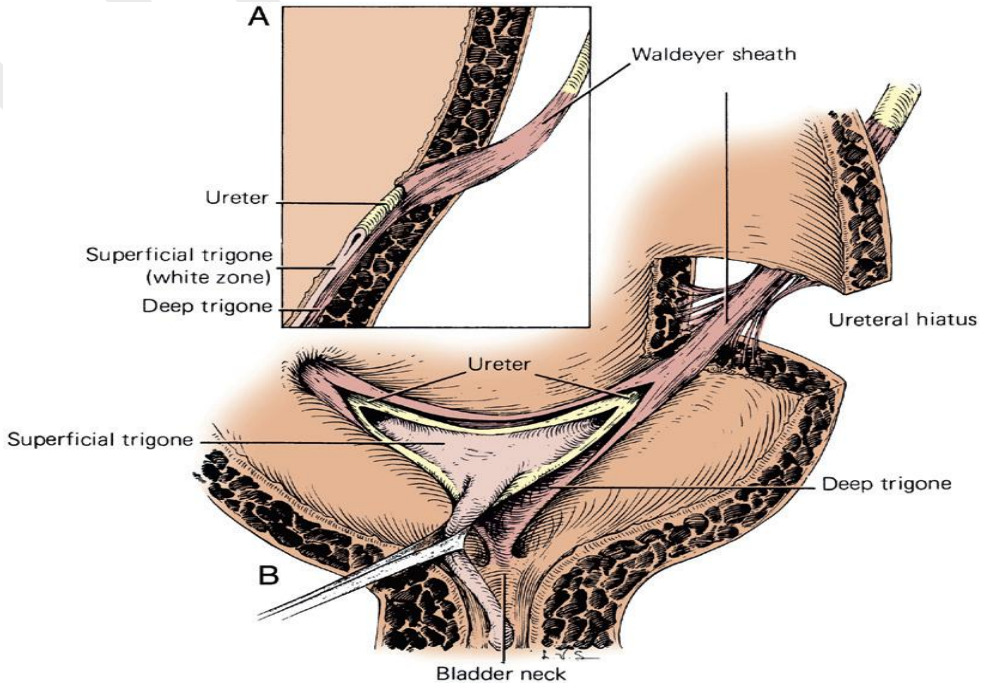
Hollow muscular organ, has storage and emptying function. When filled, the bladder is ovoid shape& has a capacity of approximately 500 mL. When empty it is tetrahedral and lies behind the symphysis pubis.

The sphincteric unit consist of:

- **Involuntary internal sphincter :** composed of smooth muscles & situated at the balder neck,supplied with α receptors.
- **Voluntary external sphincter:** composed of striated muscles & lies distal to the verrumontanum at the apex of prostate.

Extending from the dome of the bladder to the umbilicus is a fibrous cord, the median umbilical ligament, which represent the obliterated urachus.

The ureters enter the bladder posteroinferiorly in an oblique manner & at these points they are about 5cm apart. The orifices situated at the extremities of the crescent-shaped interureteric ridge that form the proximal border of the trigone, are about 2.5cm apart. The trigone occupies the area between the ridge & the bladder neck.



Relationships of the bladder :

- Male--- posteriorly: seminal vesicles, vasa defferentia, ureters & rectum.
- Female --- posteriorly: uterus & vagina.
  The dome & posterior surface of the bladder are covered by peritoneum.

Blood supply

Arteries:

1. Superior & inferior vesical arteries which arise from the anterior trunk of the internal iliac (hypogastric) artery.
2. Small branches from the obturator & inferior gluteal arteries.
3. In females, the uterine & vaginal arteries also send branches to the bladder.

Veins:

The bladder is surrounded by a rich plexus of veins that empties into the internal iliac (hypogastric) veins.

Lymphatics

The bladder drains into the vesical, external iliac, internal iliac & common iliac lymph nodes.

Innervations

-Efferent (motor):

- **Parasympathetic**: originates in the S_2-S_4 segments, its cholinergic postganglioinic fibers supply both the bladder & sphincter.
- **Sympathic nerves:** originates at T_{10}- L_2 segments & innervate the smooth muscles of the bladder base, internal sphincter & proximal urethra.
- **Somatic motor:** originate in S_2-S_3 & travel to the external sphincter via the pudendal nerve.

-Afferent (sensory):

- **Somatic afferents:** are carried by the pudendal nerves to the respective spinal areas.
- **Visceral afferents:** carried by the sympathetic & parasympathetic nerves to the respective spinal areas.

Histology

The wall is composed of:

1. **Mucosa;** of transitional epithelium (3-7 cell layer).
2. **Submucosa.**
3. **Detrusor muscle;** made of mixture of smoth muscle fibers arranged at random in longitudinal, circular and spiral manner without any layer formation or specific orientation except close to the internal meatus , where the muscles assume 3 definite layer; inner longitudinal, middle circular, & outer longitudinal.

Embryology

The bladder arises from urogenital sinus which is the ant**er**ior portion of the cloaca (which is the caudal part of the allantois).

The urogenital sinus receives the mesonephric ducts. The absorbed mesoderm of the mesonephric ducts will later be differentiated as the trigonal structure, which is the only mesodermal inclusion in the endodermal vesicourethral unit.

So, the bladder is derived from the urogenital sins (endoderm) except the trigone which is derived from the mesonephric ducts (mesoderm).

The allantois usually is obliterated at the level of the umbilicus by the 15th week.

The bladder then starts to descend by the 18th week. As it descends, its apex become stretched & narrowed, & it pulls on the already obliterated allantois, now called **the urachus**.

By the 20th week, the bladder is well separated from the umbilicus, & the stretched urachus become the middle (median) umbilical ligament.

Congenital Anomalies of the bladder

I- Bladder Extrophy (Ectopia Vesicae)

It is complete ventral defect of the urogenital sinus & the overlying skeletal system (defective development of the anterior bladder & lower abdominal wall, leaving the posterior bladder wall lying exposed on the abdomen). So the urine spurts onto the abdominal wall from the ureteric orifices.

1. Classical extrophy:

Occur in 1:50 000 births. 3:1 male predominance.

The bladder & urethra are opened dorsally, almost always associated with epispadias.

The penis is short & the clitoris is bifid.

2. Cloacal extrophy (10%):

Result from failure of the urorectal septum to descend.

Occur in 1:200 000 births. About equally in male & females.

Embryology:

The basic defect is in abnormal overdevelopment of the cloacal membrane. Which prevents medial migration of the mesenchymal tissue and proper lower abdominal wall development.

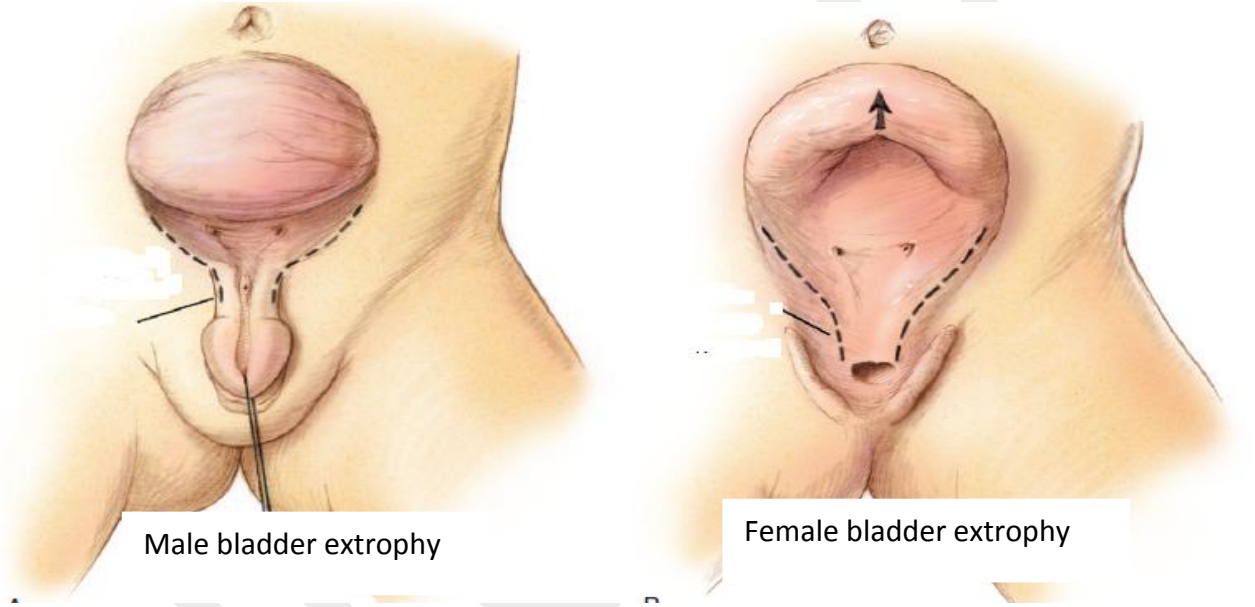
Associated anomalles:

- **Bone defects**: Diastasis (widening) of the symphysis pubis due to outward rotation of the pelvic bones along the sacroiliac joints & the child "waddles like a Duck" .
- **Musclofascial defects**: umbilical hernia, inguinal hernia, & abnormal pelvic floor.
- **Genetal defects:**
  - Male:- short, broad penis with lateral splaying of the corporal cavernosa & short urethral plate.
  - Female: Bifid clitoris, stenotic vaginal orifice, normal caliber but shorter than normal vaginal canal, and vaginal prolapse.
- **Urinary tract defects**: There is lateral displacement of the orifices, & the ureters enter the bladder with little or no obliquity. Therefore, reflux in the closed exstrophy bladder occurs in 100% of cases, & subsequent surgery is usually required at the time of the bladder neck reconstruction.

- **GIT defect**: Anteriorly diplaced anus, rectal prolapse and abnormal anal sphincter may contribute to incontinence.

Diagnosis:

- **Prenatal:** By ultrasonography, typical features include:-
  (1) absence of bladder filling, (2) a low set umbilicus, (3) widening pubis ramus, (4) diminutive genitalia, and (5) a lower abdominal mass that increase in sizes as the pregnancy progresses and as the intra- abdominal viscera increases in sizes.
  The main reason for the prenatal diagnosis of bladder exstrophy is so that the parents can be counseled regarding the risks and benefits and other aspects of the condition & for delivery of the baby in a specialized exstrophy center where immediate reconstruction of the exstrophy can occur.
- **Postnatal**: it is a clinical diagnosis.



Male bladder extrophy

Female bladder extrophy

Management:

Should be surgically treated.

Care until surgery includes:

- The umbilical cord should be tied with 2-0 silk close to the abdominal wall so that the umbilical clamp does not traumatize the delicate mucosa and cause excoriation of the bladder surface.
- The bladder can be covered with a nonadherent film of plastic wrap (e.g. Saran wrap) to prevent sticking of the bladder muscosa to clothing or diapers.
- Each time the diaper is changed the plastic wrap should be removed, the bladder surface irrigated with sterile saline, and clean plastic wrap placed over the bladder surface area.

Types of surgery:

1- One stage repair: for selected cases.

2- Staged repair: Modern Staged Reconstruction of Bladder Exstrophy; it is 3 stages:

- ***In neonate:*** closure of the bladder, posterior urethra, & abdominal wall, usually with pelvic osteotomy (cutting the bone to correct deformity)
- ***At 6-12 months:*** Epispadias repair.

- ***At 4-5 years:*** bladder neck reconstruction (Young – Dees – Leadbetter procedure) & antireflux surgery (ureteric reimplantation) done when the bladder capacity is adequate & children can participate in voiding protocols.

3- Bladder augmentation or urinary diversion: when the bladder capacity is too small (less than 80ml).

Complications

Trauma to the bladder mucosa can result in:

1- Squamous metaplasia 2- Cystitis cystica 3- Adenocarcinoma (the most common type of malignancy in bladder extrophy. 4- Squamous cell carcinoma.

II- Urachal anomalies:

Patent urachus and persistence of part of the urachus as cysts or pseudodiverticulum result from failure of fibrosis of the cranial embryonic bladder segment.

Urachal formation is directly related to bladder descent.

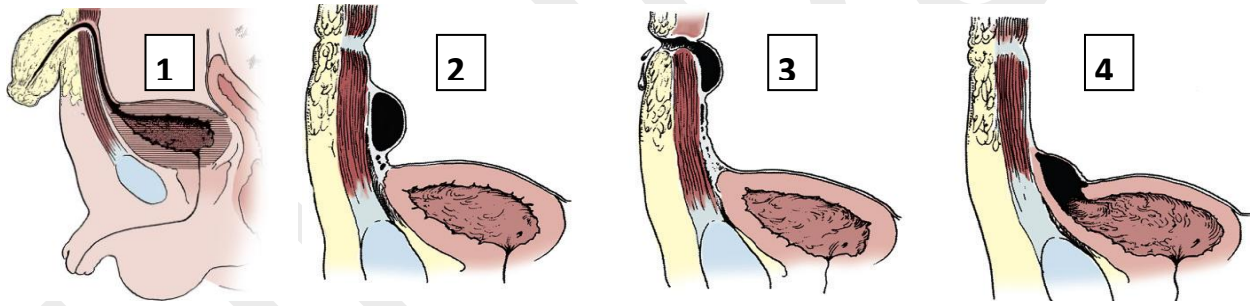
Clinichal features: Four different urachal anomalies have been described:

1-Patent urachus (50%): urine drains consistently from the umbilicus.

2- Urachal cyst (30%): low midline mass, present more commonly in adults than in infants or children.

3- **Sinus** (15%): causing a continuously draining sinus.

4- Vesicourachal diverticulum (3%to 5%): are usually nonsymptomatic.



Complications:

1-Adenocarcinoma: may occur in a urachal cyst particularly at its vesical extremity, & tends to invade the tissues beneath the anterior abdominal wall. It may be seen cystoscopically.

2- stones: may develop in cyst of the urachus. These can be identified on a plain x-ray film.

3- **Infection:** The most common organisms cultured from the umbilical drainage include staphylococcus aureus.

Investigations:

Imaging possibilities include VCUG, ultrasound, sinogram & CT scan.

Treatment:

1- Asymptomatic remnants are managed with physical and sonographic examination because **spontaneous resolution with nonoperative management is likely with remnants in patients younger than 6 months.** However, if symptoms persist or the remnant fails to resolve after age 6 months, excision of the urachus, which lies on the peritoneal surface (preperitoneal), is recommended.
2- If adenocarcinma is present, radical resection is required.

Prognosis:

Unless other serious congenital anomalies are present, the prognosis is good. The complication of adenocarcinma offers a poor prognosis.

III- Congenital bladder diverticulum:

- Usually present during childhood, with a peak incidence in younger than 10 yrs.
- Are usually solitary, occur almost exclusively in boys, & located lat. & post. to the ureteral orifice.
- The primary causation appears to be a congential weakness at the level of the ureterovesical junction and not outlet obstruction.
- Most often present with urinary tract infection, but hematuria, abdominal pain, urinary retention or an abdominal mass may be present as well.
- Are often found in children with generalized connective tissue diseases such as Ehlers-Danlos, Williams Elfin –Facies, or Menkes' syndromes.

Investigation:

- VCUG is the gold standard; with anterior- posterior, oblique, and lateral images, provides information about anatomy, location, size, associated vesicoureteric reflux, and, importantly, emptying of the diverticulum with voiding.
- It can be suspected during ultrasound examination, especially if the bladder is viewed in different filling stages.
- Cystoscopy: it is found in smooth –walled bladders and not associated with significant trabeculation on cystoscopic examination.
- On histologic examination, the diverticulum wall is composed of mucosa, subepithelial connective tissue or lamina propria, scattered thin muscle fibers, and an adventitial layer.

Treatment: Symptomatic diverticula, especially in conjunction with VUR, should be treated surgically.

IV- Contracture of the bladder neck.

Is common cause of vesico ureteral reflux, vesical diverticulum, a large bladder capacity & syndrome of irritable bladder with enuresis.

Diagnosis: VCUG& endoscopic observation and urodynamic study.

Treatment: Suprapubic bladder neck revision or transurethral resection.

Note: Revision of the bladder neck in female should be avoided because may cause incontinence & in young boys may lead to bladder neck incompetence causing retrograde ejaculation & infertility.

V-Bladder Hypoplasia, Bladder Agenesis, &Bladder Duplication: are all rare anomalies .

Acquired disease of the bladder

A-Interstitial cystitis (Hunner's urlcer)

It is primarily a disease of middle-aged women. It is characterized by fibrosis of the vesical wall, with consequent loss of bladder capacity.

Frequency, urgency, & pelvic pain with bladder distention are the principal symptoms.

Etiology : Unknown.

-Theories: increased epithelial permeability, autoimmune, neurogenic & endocrine.
So that interstitial cystitis is a neuroimmunoendocrine disorder.

Classification:

1- **Non ulcerative** (more common): occur in younger to middle aged.

2- **Ulcerative:** in middle aged to older.

Clinical features:

1- suprapublic pain is usually marked when the bladder is full.

2- gross haematuria : occasionally.

3-Suprapublic tenderness may be noted.

Diagnosis:

Not usually adhered to the National Institute of Diabetes and Digestive and kidney Disease (NIDDK) required criteria which include:

1- Glomerulations (submucosal petechiae) or Hunnerʹs ulcer on cystoscopy, and

2-pain associated with the bladder or urinary urgency.

Differential diagnosis:

1-UTI 2- Vaginitis 3- bladder tumour,4- radiation \chemical cystitis 5- Eosinophilic\ TB cystitis 6- Bladder calculi.

Treatment:

There is a 50% incidence of temporary remission unrelated to therapy, with a mean duration of 8 months. Modalities of treatment according to the severity include:

- Conservative therapy: patient education, dietary manipulation, nonprescription analgesics, & pelvic floor relaxation techniques.
- Oral medication: Tricycllc Antidepressant, sodium pentosan polysulfate, & Analgesics.
- Intravesical treatment: Sliver nitrate, Dimethyllsulfoxide, Heparin, & pentosan polysulfate .
- Hydrodistention: Diagnostic & therapeutic.
- Neuromodulation.
- Surgical therapy: Transurethral resection of a Hunnerʹsulcer, Augmentation cystoplasty, & Urinary diversion with or without cystourethrectomy.

B- Schistosomiasis (bilharzia)

Urinary schistosomiasis is caused by a trematode (or fluke) called Schistoma haematobium. It occurs in Africa, Egypt, and the Middle East.

Egyptian physicians of the twelfth dynasty (1900 BC) recognized hematuria as the cardinal sign and symptom of this disease. Theodore Bilharz, a German pathologist working in Cairo in 1852, first described worm pairs in mesenteric veins at autopsy and linked these to eggs found in human excreta.

Pathogenesis

Fresh water snails release the infective form of the parasite (cercariae), which can penetrate skin, and migrate to the liver (as schistosomules), where they mature. Adult flukes couple, migrate to vesical veins, and lay eggs (containing miracidia larvae), which leave the body by penetrating the bladder and entering the urine.

The disease has two stages:

1- active (when adult worms are actively laying eggs) and

2- inactive (when the adult has died, and there is a reaction to the remaining eggs).

Presentation

- The first clinical sign is,swimmer's itch, a local inflammatory response.

- Other early manifestations include Katayama fever, a generalized allergic reaction, which includes fever, urticaria, lymphadenopathy, hepatosplenomegaly, and eosinophilia.
- Active inflammation results in haematuria, frequency, and terminal dysuria.

Investigation

Midday urine specimen; bladder and rectal biopsies may contain eggs (distinguished by having a terminal spine). finding eggs is the gold standard for diagnosis of active infection.

Serology tests: that combine a FAST-ELISA followed by Western blot analysis. In cases where the diagnosis is highly suspected but eggs are not present, the serologic tests can be useful, but do not distinguish between acute and chronic disease. Antibody titers can remain positive even after curative treatment.

Radiography: is an important diagnostic tool in the evaluation of sequelae and complications of urinary schistosomiasis.

- **Plain radiograph:** to diagnose calcification in the bladder (like fetal head in the pelvis;is pathognomonic of chronic urinary schistosomiasis) Ureteral calcification is typically mural, and the ureter is dilated. This differs from the calcification seen in tuberculosis, which forms a cast of a nondilated ureter.
- **IVU** may show a calcified, contracted bladder, Hydroureter, hydronephrosis, nonfunctioning kidney, ureteral stenosis, and bladder and ureteral filling defects caused by polypoid lesions
- **Ultrasonography**: detect focal thickening of the bladder wall and polypoid lesionsof the urinary tract. It also detects hydroureter, hydronephrosis, and heavily calcified patches
- Computer tomography (CT) scans have the advantage of being able to detect both obstructive uropathy and calcified lesions in the urinary tract and colon, giving them advantage over the intravenous urogram
- Vesicocystourethrography:It indicates the presence of vesicoureteral reflux, which occurs in 25% of infected ureters.

Cystoscopy identifies polypoid lesions, sandy patches, ulcers (weeping ulcers that bleed when the bladder is deflated), tumours, strictures and also assess the bladder capacity.

Treatment

I- Medical treatment:

1- Praziquantel: Active against all types of schistosomiasis, orally given, 40 mg/kg as single dose or 20mg/kg 3 times in one day.
2- Metrifonate: Active against S. hematobium only, orally given, 7.5-10 mg/kg (600 maximum dose) once & then repeated twice at 2 week interval.
3- Oxamniquine: Active against S. mansoni only, orally given, 12-15 mg/kg once only.
4- Niridazole: Active against S. hematobium and S. mansoni only, orally given, 25 mg/kg (maximum 1.5 gm) daily in 2 divided doses for 7 days.
5- Antimonial drugs: These includes (stibophen, tarter emetic) which are toxic and no longer used in the treatment of S. hematobium.

II- General measures:

1- Antibiotics or urinary antiseptics are needed to control secondary infection(salmonella typhi & paratyphi).
2- Supportive treatment in the form of iron, vitamins and high calorie diet in selected cases.

III- Treatment of complication:

1- Ureteric stricture: resection and ureteroneocystostomy.
2- Bladder neck contracture: transurethral ant. Commissurotomy or suprapubic Y-V plasty.
3- Weeping ulcer: partial cystectomy
4- Small bladder: augmentation cystoplasty.
5- Urethral stricture: optical urethrotomy.
6- Squamous cell carcinoma: Cystectomy and urinary diversion.