The Prostate and Benign Prostatic Hyperplasia

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The Protate

- **u** The prostate is a fibromuscular and glandular organ lying just inferior to the bladder The normal prostate weighs about 20 g and contains the posterior urethra, which is about 2.5 cm in length
- **u** It starts at 12th week fetal life under androgen effect from the fetal testes.
- **u** According to the classification of Lowsley, the prostate consists of 5 lobes: anterior, posterior, median, right lateral, and left lateral. According to McNeal, the prostate has a peripheral zone, a central zone, and a transitional zone; an anterior segment; and a preprostatic sphincteric zone.
- **u** The peripheral zone is (androgen dependent), while the central zone is(non-androgenic dependent); Therefore castration of baby before puberty lead to failure of gland development. So the effect of androgen on the prostate development is documented.

blood supply:

- **q** Arterial:
  - **ü** Inferior vesical artery
  - **ü** Internal pudendal artery
  - **ü** Middle rectal (haemorrhoidal) artery
- **q** Venous:

  The veins from the prostate drain into the periprostatic plexus, which has connections with the deep dorsal vein of the penis and the internal iliac (hypogastric) veins

Nerve supply:

From the sympathetic & parasympathetic nerve plexuses.

Lymphatics:

The lymphatic from the prostate drain into internal iliac(hypogastric), sacral, vesical , and external iliac lymph nodes.

Benign Prostatic Hyperplasia (BPH)

Epidimiology:

- Ø Usually occur in men after 50 years of age (most often between 60 — 70 y)
- Ø It is rare in Negro & very rare in Japanese

Etiology:

- Ø It may be due to discrepancy between Androgen/Estrogen ratio, with ageing the androgen decreases & lead to reversible changes of A/E ratio **à** relative increase in estrogen **à** periurethral gland proliferation
- Ø Cell death & apoptosis is deregulated **à** imbalance between cell proliferation & cell death **à** increase mass

Histology:

- Ø The term BPH refers to well-defined histologic changes characterized by slowly progressive nodular hyperplasia of the periurethral (transitional) zone
- Ø The hyperplasia is made of many components which may be of epithelial type or interglandular (Fibromuscular) stroma.
- Ø The adenoma may be of epithelial type (epithelial T. is dominant), fibrous type (dominant fibrous T.) or mixed fibro muscular adenoma.
- Ø The size of the enlargement that occurs in nodular hyperplasia is unrelated to the degree of obstruction , the smallest prostates may cause the worst outflow obstruction & huge glands may cause no obstruction at all.

Pathophysiology:

I. Urethra: The prostatic urethra is lengthened, sometimes to twice its normal length, there will be compression of the urethra & verumontanum. The urethra may be compressed from side to side or to one side.

II. Bladder: Over time, outflow obstruction leads to characteristic changes in the bladder & upper urinary tract. these changes may be observed cystoscopically & radiologically:

1. Trabeculation: Prominence of the detrusor fibers observed through a cystoscope. It is a manifestation of increased collagen deposition in the bladder wall. This finding is often associated with outflow obstruction but may also be seen in unobstructed bladders (e.g., enuresis, neurogenic bladder dysfunction, idiopathic bladder instability).
2. Cellule formation: Extreme degrees of trabeculation allow the vesical mucosa to be pushed between the muscle fibers of the bladder wall to form small pockets called cellules.
3. Diverticulum formation: Herniation of the vesical mucosa through the detrusor muscle constitutes a bladder diverticulum . Because of stasis of urine within the diverticulum, they are likely to harbor infection, stones, & urothelial cancer. A diverticulum near the ureteric orifice may cause vesicoureteral reflux.
4. bladder calculi: The presence of a bladder stone is a strong evidence of longstanding bladder outflow obstruction. Bladder calculi form most commonly as a result of outflow obstruction, residual urine, stasis, & infection.

III. Upper Tract: With hypertrophy & fibrosis of the detrusor wall, increased work is required to transport the urinary bolus from the ureter into the bladder. This will

lead to dilatation of the upper tract (Bilateral Hydroureteronephrosis). In the early stages, the condition appears radiologically as mild dilatation of the distal segment & elongation & some tortuosity of the ureter.

Cilnical feature:

- ü A decrease in the force of & caliber of the urinary stream is the first & most frequent symptom.
- ü Frequency is the earliest symptom. At first nocturnal, the patient being obliged to get up to micturate twice or more during the night. Freq. occur because the patient can not empty the U.B. completely **à** residual urine **à** the UB. Will full at a quicker rate.
- ü Urgency when the vesical sphincter becomes stretched, a little urine escapes into the normally empty prostatic urethra, causing an intense reflex desire to void.
- ü Hesitancy (difficulty in initiating the stream), patient can not start to urinate.
- ü Acute **U**rinary Retention.
- ü Chronic Retention with overflow incontinence: the patient complains that urine constantly dribbles away.
- ü Sustained bladder outlet obst. Plus hypertrophy or overdistention of the bladder may cause vesicoureteral reflux **à** Hydroureteronephrosis **à** later on produce renal failure.
- ü Haematuria: may be a presenting symptom, a drop of blood at the beginning or end of micturation is not unusual.

Assessment of the patient with LUTS

- **q** Abdominal examination: is usually normal. In patients with chronic retention, a distended bladder will be found on palpation, percussion & sometimes on inspection.
- **q** General physical examination may demonstrate signs of chronic renal impairment with anemia & dehydration.
- **q** The external urinary meatus should be examined to exclude stenosis, & the epididymes are palpated for signs of inflammation
- **q** Rectal examination: in BPH, the posterior surface of the prostate is smooth, convex, & firm. The rectal mucosa can be made to move over the prostate. Hard or nodular prostate gives suspicion of malignancy.
- **q** Neurological examination:
- ü the nervous syst. Is examined to exclude a neurological lesion.
- ü D.M, tabes dorsalis, M.S, Parkinson's disease & other neurological states may mimic prostatic obstion.
- ü examination of perianal sensation & anal tone is useful in detection of an S2 to S4 cauda equina lesion.

Investigations:

- § GUE
- § Blood examination urea, creatinine to assess renal function.
- § PSA (Prostate- Specific Antigen): it is extremely helpful marker in a diagnosis of prostatic carcinoma which may coexist with the BPH, PSA produced by prostatic cells & related to how big the prostate is & carcinoma of prostate. If PSA is high (> 4nmol/L) then transrectal US plus transrectal biopsies should be considered.
- § Imaging: US, IVU, it will tell us the renal function, if there is dilatation or not & in the urinary bladder see any trabeculation, filling defect due to prostatic enlargement, then do post-voiding film, this will show how much residual urine there
- § Urodynamics (uroflowmetery & cystometry): this is done if you suspect an atonic UB or when a clear diagnosis has not been reached.
- § Cystourethroscopy: is the mainstay of diagnosis & should be performed on patients with haematuria. The urethra is inspected first, & the bladder is then examined to

see trabeculation, stone & diverticulum. *It should always be made before prostatectomy.*

Treatment:

I. **Medical Treatment:**

A. **α- adrenergic blocking agents:** The prostate contains smooth muscle that is controlled by α1 - adrenoreceptors. α- blocker agents have **side effects**, such as postural hypotension, tachycardia, palpitations & retrograde ejaculation. e.g. phenoxybenzamine, Prazosin, Alfuzosin, indoramin, Terazosin, Doxazosin, & Tamsulosin.

B. **Androgen suppressants:**

- the rationale for androgen suppression is observation that the embryonic development of the prostate is dependent on the androgen dihydrotestosterone (DHT).
- Testosterone (T) is produced mainly by testes & to a lesser extent by the adrenals, & testosterone enters the prostatic cells & converted into an active component (DHT) by the enzyme 5α-reductase & this enzyme can be inhibited by drugs like Finasteride (5 α- reductase inhibitor).
- It will decrease the size of the prostate by 10 — 20%.
- Median lobe has poor response to androgen suppressants.

II. *Surgical treatment:*

surgical removal of prostate by one of the four approaches which are:

- *Open Simple Prostatectomy:*
  - ***Transvesical** Prostatectomy*
  - ***Retropubic** Prostatectomy (**Millin's** Prostatectomy)*
- *Transurethral Ressection of the Prostate (**TURP**)*
- *Transurethral Incision of the Prostate (**TUIP**)*
- *Minimal Invasive Prostatectomy:*
  - Transurethral microwave therapy (**TUMT**).
  - Transurethral needle ablation (**TUNA**).
  - Photoselective vaporization of the prostate (**PVP**).
  - Holmium Laser Enucleation of the Prostate (**HoLEP**).
  - Holmium Laser Ablation of the Prostate (**HoLAP**).

- The problem with these new methods, is that we can not take a biopsy.

Indications for prostatectomy:

- Acute urinary retention.
- Marked hemorrhage from enlarged prostate.
- Persistent or recurrent UTI.
- Renal impairment as a result of BOO.
- Persistent LUTS (Lower Urinary Tract Symptoms) not responding to medical treatment.

Indications for open prostatectomy:

- Prostate size >75 gm.
- Concomitant symptomatic bladder diverticulum
- Concomitant large, hard bladder stone that cannot be managed transurethrally.
- Marked ankylosis of the hips that prevents proper placement of the patient in the dorsal lithotomy position.

Contraindications for open prostatectomy:

- Small prostate gland.
- Previous prostatectomy.
- Prostate cancer.
- Previous pelvic surgery preventing access to the prostate gland

Post-operative complication of Prostatectomy:

- **Local:**

  Excessive hemorrhage
  Perforation
  Urinary extravasation
  Infection (cystitis, epididymo-orchitis)
  Urge or stress incontinence or both
  Retrograde ejaculation & impotence
  Bladder neck contracture

- **General:**

  DVT
  Pulmonary embolism
  Water intoxication (TUR syndrome)
  Hyponatremia.

Questionnaire for American Urological Association Symptom Score.

	AUA Score					
URINARY SYMPTOMS (SYMPTOM SCORE CRITERIA)	**Not at all**	**Less than 1 time in 5**	**Less than half the time**	**About half the time**	**More than half the time**	**Almost always**
1. Incomplete emptying Over the past month, how often have you had a sensation of not emptying your bladder completely after you finished urinating?	0	1	2	3	4	5
2. Frequency Over the past month, how often have you had to urinate again less than two hours after you finished urinating?	0	1	2	3	4	5
3. Intermittency Over the past month, how often have you found you stopped and started again several times when you urinate?	0	1	2	3	4	5
4. Urgency Over the past month, how often have you found it difficult to postpone urination?	0	1	2	3	4	5
5. Weak stream Over the past month, how often have you had a weak urinary stream?	0	1	2	3	4	5
6. Straining Over the past month, how often have you had to push or strain to begin urination?	0	1	2	3	4	5
	None	1 time	2 times	3 times	4 times	5 or more times
7. Nocturia Over the past month, how many times did you most typically get up to urinate from the time you went to bed at night until the time you got up in the morning?	0	1	2	3	4	5
AUA Symptom Score = sum of questions A1 to A7						

QUALITY OF LIFE DUE TO URINARY PROBLEMS							
	Delighted	Pleased	Mostly satisfied	Mixed—about equally satisfied and unsatisfied	Mostly dissatisfied	Unhappy	Terrible
If you were to spend the rest of your life with your urinary condition just the way it is now, how would you feel about that?	0	1	2	3	4	5	6