Hydronephrosis

Hydronephrosis: referring to the presence of PCS dilatation and not to the cause of that dilatation.
Obstructive uropathy: structural impedance to the flow of urine anywhere in the urinary tract.
Obstructive nephropathy: renal parenchymal damage that results from an obstruction.
The terms *obstructive uropathy* and *hydronephrosis* should not be used interchangeably.

Causes of Hydronephrosis

(1) Obstructive dilatation, frequently unilateral
Stone, papillary necrosis, clot, tuberculous debris, UPJ obstruction, crossing vessel ,Inflammatory stenosis of the ureter, retroperitoneal Neoplastic infiltration, Primary neoplasm pelvicocalyceal system and ureter ,Bladder neoplasm, Mobile (ptotic) kidney,

(2) Obstructive dilatation, frequently bilateral

- Infravesical urinary obstruction(BPH, Stricture)
- Neuropathic bladder
- Bladder stone, clot in the urinary bladder
- Retroperitoneal fibrosis

(3) Nonobstructive dilatation, frequently unilateral

- Vesicoureteral reflux
- Following relief of obstruction (Residual)
- Pregnancy
- Megacystis-megaureter syndrome

(4) Nonobstructive dilatation, frequently bilateral

- Full bladder
- Urinary tract infection

Diagnostic approach

History
• Severe flank pain suggests a more acute onset of obstruction and, if very sudden in onset, a ureteric stone may well be the cause. Pain induced by a diuresis (e.g. following consumption of alcohol) suggests a possible PUjO.
• Anuria (the symptom of bilateral ureteric obstruction or complete obstruction of a solitary kidney).
• If renal function is impaired, symptoms of renal failure may be present (e.g. nausea, lethargy, anorexia).
• Extrinsic causes of obstruction (e.g. compression of the ureters by retroperitoneal malignancy) usually have a more insidious onset, whereas intrinsic obstruction (ureteric stone) is often present with severe pain of very sudden onset.
• An increase in urine output may be reported by the patient due to poor renal concentrating ability.
• Obstruction in the presence of bacterial urinary tract infection—signs and symptoms of pyelonephritis (flank pain and tenderness, fever) or sepsis.

Examination
• Measure blood pressure—elevated in high pressure chronic retention (HPCR) due to benign prostatic obstruction (caused by fluid overload).
• Bilateral oedema (due to fluid overload).
• Abdominal examination—percuss and palpate for an enlarged bladder.

• DRE (? prostate or rectal cancer) and in women, vaginal examination (?cervical cancer).
• Check serum creatinine to determine the functional effect of the hydronephrosis.

Ultrasound

Ultrasound is a rapid, inexpensive, and reasonably accurate method of detecting hydronephrosis and hydroureter; however, accuracy can depend on the user. Ultrasound generally serves as the preferred screening test to establish the diagnosis of hydronephrosis.

Ultrasound is inferior to other modalities for identifying the presence, source, or duration of obstruction. A chronically obstructed system may remain dilated long after the obstructive process resolves.

IVU findings in renal obstruction

• Persistence of dense nephrogram.
• A delay in filling of the collecting system with contrast material.
• Dilatation of the collecting system.
• An increase in renal size.
• Rupture of fornices (junction between renal papilla and its calyx) with urinary extravasation.
• Ureteric dilatation and tortuosity.
• A standing column of contrast material in the ureter.

Others (CT-Scan, MRI,)

PATHOPHYSIOLOGY OF OBSTRUCTIVE UROPATHY

- Hydronephrosis causes tubular dilation with cellular atrophy and interstitial fibrosis
- Within 7 days, atrophy is seen in the distal nephron
- By 14 days, there is progressive dilation of the distal tubules and atrophy of the proximal tubular epithelial cells.
- At 28 days, there is loss of 50% of the medulla with marked atrophy of the proximal tubules and thinning of the cortex. So obstruction should be relieved as early as possible up to 4-6 weaks
- Glomerular changes are not seen before 28 days of obstruction.
- reduced blood flow :no evidence of microscopic changes in the arterial wall
- Venous drainage is impaired, causing some of the renal damage
- Urine exits the renal pelvis in complete ureteral obstruction by:
  1. extravasation (high pressures)
  2. pyelolymphatic backflow (low-pressure)
  3. pyelovenous backflow (chronic hydronephrosis)
- if one kidney is removed or rendered nonfunctioning by obstruction, the opposite kidney would undergo compensatory hypertrophy
- unilateral ureteral obstruction result in:
  - decreased renal blood flow to the ipsilateral kidney (thromboxanes, angiotensin, endothelin, and mesangial-cell contact)
  - increased blood flow to the unobstructed contralateral kidney (prostaglandins)
- After unilateral ureteral obstruction,
  - First week: there is a bilateral increase in renal mass.
  - Followed by atrophy in the obstructed kidney.
  - Continued hypertrophy in the opposite unobstructed kidney.

Urinary tract infection

Definitions

Urinary tract infection A diagnosis of urinary tract infection (UTI) used to be based on finding $>10^5$ bacteria/ml of urine, whether or not there were associated symptoms of infection.
UTI is currently defined as the inflammatory response of the urothelium to bacterial invasion. This inflammatory response causes a constellation of symptoms (pain, LUTS, systemic symptoms).

Bacteriuria is the presence of bacteria in the urine. Bacteriuria may be asymptomatic or symptomatic. Bacteriuria without pyuria indicates the presence of bacterial colonization of the urine, rather than the presence of active infection ('active' implies an inflammatory response to bacterial invasion of the urothelium).

Pyuria is the presence of white blood cells (more than 5 WBC/ HPF) in the urine implying an inflammatory response of the urothelium to bacterial infection or, in the absence of bacteriuria (Abacterial pyuria), as in carcinoma in situ, TB infection, bladder stones, or other inflammatory conditions.

An uncomplicated UTI is one occurring in a patient with a structurally and functionally normal urinary tract. The majority of such patients are women who respond quickly to a short course of antibiotics.

A complicated UTI is one occurring in the presence of an underlying anatomical or functional abnormality (e.g. functional problems causing incomplete bladder emptying, such as BPH, DSD in spinal cord injury), stones in the kidney or bladder, fistula between bladder and bowel, etc). Most UTIs in men occur in association with a structural or functional abnormality and are therefore defined as complicated UTIs. Complicated UTIs take longer to respond to antibiotic treatment than uncomplicated UTIs, and if there is an underlying anatomical or structural abnormality they will usually recur within days, weeks, or months.

Urinary tract infection may be **isolated, recurrent or unresolved**.

- ***Isolated UTI***: an interval of at least 6 months between infections.
- ***Recurrent UTI***: >2 infections in 6 months, or 3 within 12 months. Recurrent UTI may be due to *reinfection* (i.e. infection by a different bacterium) or *bacterial persistence* (infection by the same organism originating from a focus within the urinary tract).

Bacterial persistence is caused by the presence of bacteria within calculi (e.g. struvite calculi), within a chronically infected prostate (chronic bacterial prostatitis), within an obstructed or atrophic infected kidney, or occurs as a result of a bladder fistula (with bowel or vagina) or urethral diverticulum.

- ***Unresolved infection***: implies inadequate therapy and is caused by natural or acquired bacterial resistance to treatment, infection by different organisms, or rapid reinfection.

Risk factors for bacteriuria

Female sex; increasing age; low oestrogen states (menopause); diabetes mellitus; previous UTI; the institutionalized elderly; indwelling catheters; stone disease (kidney, bladder); genitourinary malformation and voiding dysfunction (including obstruction).

Microbiology of UTI:

Most UTIs are caused by faecal-derived bacteria which are facultative anaerobes (i.e. they can grow under both aerobic and anaerobic conditions)

(1) Uncomplicated UTI

Most UTIs are bacterial in origin. The most common cause is Eschericriia col; (E. coli), a gram-negative bacillus, which accounts for 85% of community acquired and 50% of hospital acquired infection. Other common causative organisms include Staphylococcus saprophyticus and Enterococcus faecalis (also known as Streptococcus faecalis—gram +ve), Proteus mirabilis, and Klebsiella (gram-negative enterobacteriacae).

(2)Complicated UTI

E. coli is responsible for up to 50% of cases. Other causes include Enterococci (e.g. Streptococcus faecalis), Staph. aureus, Staph. epidermidis (gram +ve). Pseudomonas aeruginosa (gram -ve).

Route of infection

(1) Ascending the vast majority of UTIs result from infection ascending retrogradely up the urethra. The bacteria, derived from the large bowel, colonize the perineum, vagina, and distal urethra. They ascend along the urethra to the bladder (increased risk in females as urethra shorter) causing cystitis, and from the bladder they may ascend, via the ureters, to involve the kidneys (pyelonephritis). Reflux is not necessary for infection to ascend to the kidneys, but the presence of reflux will encourage ascending infection,

(2) Haematogenous Uncommon, but is seen with Staph. aureus, Candida fungaemia. and TB.

(3) Lymphatics Seen rarely in inflammatory bowel disease, retroperitoneal abscess.

(4) Direct extention from adjacent organs (intraperitoneal abscess ,VVF , or vesicointestinal fistula)

Factors increasing bacterial virulence

(1) Adhesion factors

Many gram-negative bacteria contain pili on their surface, which aid attachment to urothelial cells of the host. Pili are defined functionally by their ability to mediate hemagglutination (HA) of specific types of erythrocytes.

(2) Avoidance of host defense mechanism

An extracellular capsule reduces immunogenicity and resists phagocytosis (E.coll). M. tuberculosis resists phagocystosis by preventing phagolysosome fusion.

(3) Toxins E. coil species release cytokines which have a direct pathogenic effect on host tissues.

(4) Enzyme production Proteus species produce ureases, which causes breakdown of urea in the urine to ammonia (alkaline urine), which then contributes to disease processes (struvite stone formation).

Host defenses

Factors which protect against UTI are:

1. Mechanical flushing effect of urine through the urinary tract (i.e. antegrade flow of urine).
2. A mucopolysaccharide coating of bladder (Tamm-Horsfall protein) helps prevent bacterial attachment.
3. Low urine pH and high osmolarity reduces bacterial growth.
4. Urinary immunoglobulin (IgA) inhibits bacterial adherence.