GENITOURINARY TUBERCULOSIS

Genitourinary tuberculosis (GUTB) is a common site of extrapulmonary TB. (15-20% of extrapulmonary cases of TB.). It may involve the kidneys, ureter, bladder, or genital organs.

Clinical symptoms usually develop 10-15 years after the primary infection.

- Age: The most common age at presentation is 30-45 years.
- Sex: Male-to-female ratio is 5:3.

Only about a quarter of patients with GUTB have a known history of TB; about half of these patients have normal chest radiography findings.

Causative Agents:

- The most common pathogen is M tuberculosis.
- Uncommonly implicated pathogens include the following:
    - Mycobacterium kansasii
    - Mycobacterium fortuitum
    - Mycobacterium bovis
    - Mycobacterium avium-intracellulare
    - Mycobacterium xenopi
    - Mycobacterium celatum

PATHOPHYSIOLOGY:

- **Mycobacterium tuberculosis** bacilli are inhaled through the lungs to the alveoli. They are phagocytosed by polymorphonuclear leukocytes and macrophages. Although most bacilli are initially contained, some are carried to the region's lymph nodes. Eventually, the thoracic duct may deliver mycobacteria to the venous blood; seeding of different organs, including the kidneys, may occur.
- **Multiple granuloma** form at the site of metastatic foci.
- In the kidneys, they are typically bilateral, cortical, and adjacent to the glomeruli. They may remain inactive for decades.

Although both kidneys are seeded, clinically significant disease usually occurs in only one kidney.

- Growing granuloma may erode into the calyceal system, spreading the bacilli to the renal pelvis, ureters, bladder, and other genitourinary organs.
- Depending on the status of the patient's defense mechanisms, fibrosis and strictures may develop with chronic abscess formation.
- Extensive lesions can result in nonfunctioning kidneys (Autonephrectomy).

Ureteral TB is an extension of the disease from the kidneys, generally to the ureterovesical junction. causing ureteral strictures and hydronephrosis.

- The ureter undergoes fibrosis and tends to be shortened and therefore straightened. This change leads to a “golf hole” (gaping) ureteral orifice
- Ureteral stenosis may be complete, causing “autonephrectomy.” Such a kidney is fibrosed and functionless. Under these circumstances, the bladder urine may be normal and symptoms absent.

Bladder TB is secondary to renal TB and usually starts at the ureteral orifice.

- It initially manifests as superficial inflammation with bullous edema and granulation.
- Fibrosis of the ureteral orifice can lead to stricture formation with hydronephrosis or scarification (ie, golf-hole appearance) with vesicoureteral reflux.
- Severe cases involve the entire bladder wall, where deep layers of muscle are eventually replaced by fibrous tissue, thus producing a thick, fibrous bladder.

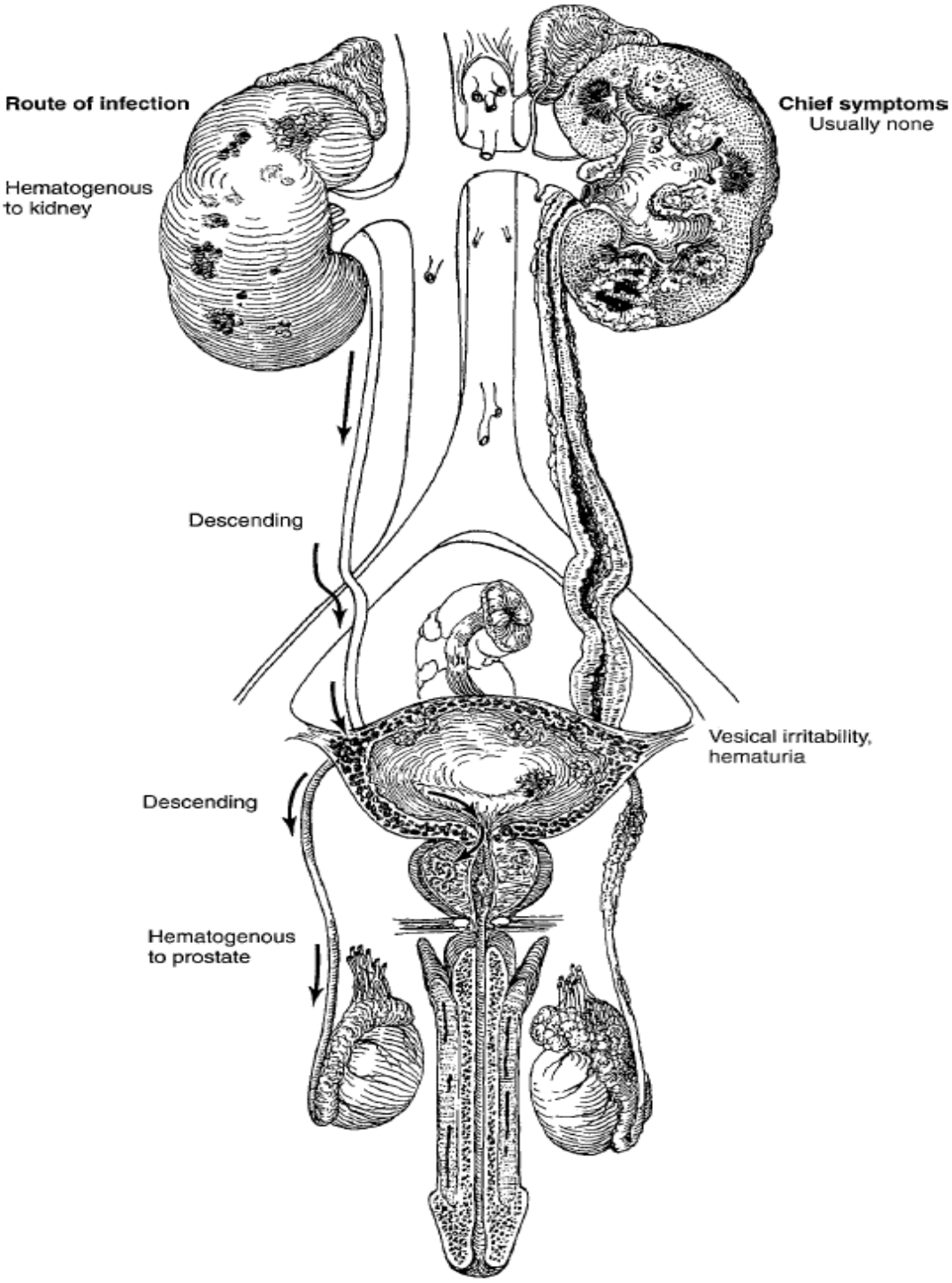
Epididymis TB is always hematogenous in origin and not a direct extension from the bladder.

- If the epididymal infection is extensive and an abscess forms, it may rupture through the scrotal skin, thus establishing a permanent sinus
- The nodular beading of the vas, when it is involved, is a characteristic

physical finding. Infertility may result from bilateral vasal obstruction.

- Involvement of the testis is usually due to direct extension from the epididymis.

Prostate TB is also spread hematogenously, but involvement is rare. The affected prostate is nodular, indurated and not tender to palpation (? Ca Prost.), Severe cases may cavitate and form a perineal sinus



Clinical Findings

Tuberculosis of the genitourinary tract should be considered in the presence of any of the following situations:

1. chronic cystitis that refuses to respond to adequate therapy,
2. the finding of sterile pyuria,
3. gross or microscopic hematuria,
4. a non-tender, enlarged epididymis with a beaded or thickened vas,
5. a chronic draining scrotal sinus,
6. induration or nodulation of the prostate and thickening of one or both seminal vesicles (especially in a young man).

- A history of present or past tuberculosis elsewhere in the body should cause the physician to suspect tuberculosis in the genitourinary tract when signs or symptoms are present.
- There is no classic clinical picture of renal tuberculosis.
- Most symptoms of this disease, even in the most advanced stage, are vesical in origin (cystitis).
- Vague generalized malaise, fatigability, low-grade but persistent fever, and night sweats are some of the nonspecific complaints.
- Active tuberculosis elsewhere in the body is found in less than half of patients with genitourinary tuberculosis.

Investigation:

The diagnosis rests on the demonstration of tubercle bacilli in the urine by culture or positive polymerase chain reaction (PCR).

- Tuberculin skin test
- Complete blood cell count, sedimentation rate, serum chemistry, and C-reactive protein studies are helpful to assess severity of disease, renal function, and response to treatment.
- Serial early morning urine collection for acid-fast smear (at least 3)
- Serial urine cultures (Three to five first morning voided specimens are ideal.) are still considered the criterion standard for evidence of active disease,

The following methods are available:

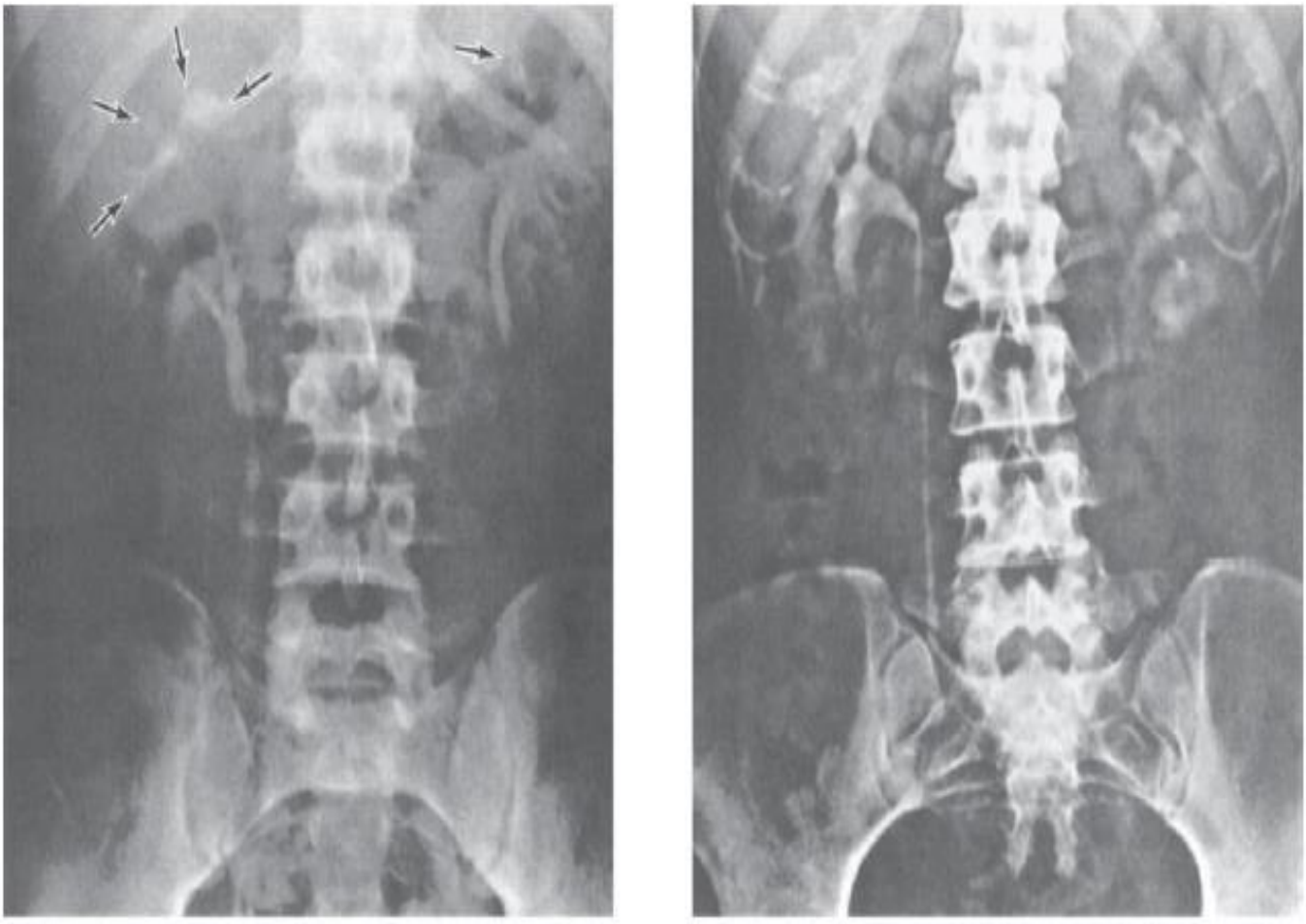
- Solid media: The Lowenstein-Jensen medium takes more than 4 weeks for results.
- Radiometric media: The BACTEC 460 medium takes 2-3 days for results.
- The polymerase chain reaction (PCR) test

Imaging Studies:

- Chest and spine radiographs may show old or active lesions
- (KUB) reveal calcifications in the kidney and ureter in approximately 50% of patients.
- IVP and voiding cystography: are the standard diagnostic imaging studies for renal TB and are suggestive of disease in 88-95% of patients.

The typical IVP changes include:

1. a “moth-eaten” appearance of the involved ulcerate calyces,
2. obliteration of one or more calyces,
3. dilatation of the calyces due to ureteral stenosis from fibrosis,
4. abscess cavities that connect with calyces,
5. single or multiple ureteral strictures, with secondary dilatation, with shortening and therefore straightening of the ureter,
6. absence of function of the kidney due to complete ureteral occlusion and renal destruction (autonephrectomy).



Medical Care: The primary treatment is medical therapy.

Standard treatment is: rifampin (600mg orally once daily), INH (300mg oral once daily), pyrazinamide, and ethambutol for 2 months,

Then: rifampin and INH for 4 more months

Surgical Care: