

Tuberculosis of the urinary tract

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Genitourinary tuberculosis (GUTB) accounts for 15–20% of extrapulmonary cases of TB. It is secondary and caused by haematogenous spread of tubercle bacilli from the thoracic lymph nodes or the lungs. GUTB occurs as a result of either reinfection or reactivation of old TB granulomas. Blood-borne organisms are deposited close to the glomeruli, causing an inflammatory reaction. Macrophages react and granulomas are formed. If bacterial multiplication goes unchecked, caseous necrosis results in the formation of tubercles. Multiple tubercles coalesce and rupture into the collecting system, causing intermittent tuberculous bacilluria and pyuria. The disease spreads through the collecting system with ulceration initially. When bacterial multiplication is halted by the immune system, sequelae due to fibrosis appear (Figure 82.8). Tubercular obstructing or destructive lesions in the kidneys and ureter responsible for renal function loss. Involvement of the bladder is secondary to renal disease. The disease gradually involves the bladder musculature, which is replaced by fibrous tissue, causing a decrease in the size and capacity of the bladder ('thimble' bladder). Urinary bladder involvement is responsible for urinary frequency, which is the most common symptom of GUTB. Epididymal tuberculosis presents as a painless epididymal nodule, usually involving the tail of the epididymis, or a chronic discharging sinus in the posterior scrotal wall. Patients may present with urinary frequency, colicky flank pain, haematuria and, rarely, fever and constitutional symptoms. They may also present with symptoms suggestive of recurrent UTIs. William 'Bill' Kerr, a Canadian urologist, described his eponymous sign in 1967. and, rarely, calcified tubercular lesions may be misdiagnosed as urinary tract calculi. For microbiological confirmation, at least three consecutive early-morning specimens of urine are examined for acid-fast tubercle bacilli. The gold standard for diagnosis is urine culture. Nucleic acid amplification tests (NAATs) provide rapid diagnosis (within hours). When the diagnosis remains uncertain, bladder biopsy, tissue culture and tissue NAATs may be required. Imaging with CTU may also help and can show early signs such as calyceal distortion and papillary necrosis, hydronephrosis, poor function of renal segments secondary to parenchymal destruction, fibrosis and chronic obstruction. Ureteric strictures and proximal dilatation may also be seen. IVU can pick up the earliest signs of disease activity, such as calyceal distortion. Treatment involves short-course antituberculous therapy (ATT). Rifampicin, isoniazid and pyrazinamide are used sometimes with ethambutol as first-line drugs. The primary aim of therapy is preservation of renal function and avoidance of fibrotic sequelae. Ureteric strictures may require double J (DJ) stenting to preserve function until definitive reconstruction is attempted. Percutaneous nephrostomy (PCN) is recommended in obstructive strictures to achieve prompt decompression. Definitive surgery is usually done 3–6 weeks after starting ATT. The choice of reconstructive procedure depends on the type and location of sequelae. Open surgical repair is generally superior to balloon dilatation for tubercular ureteric strictures. Augmentation enterocystoplasty (usually using

ileum) for small-capacity bladders,

radiological sign seen in an intravenous urogram, caused by microulceration of the calyces
Caseous necrosis Ureteric stricture Thimble bladder: extremely small capacity bladder owing to fibrosis

ureteric reimplantation with or without a Boari flap (bladder tube) for lower ureteric stricture and ileal replacement of the ureter for multiple long ureteric strictures may be required. Nephrectomy is done for major renal lesions with a poorly functioning kidney. Urinary infection in childhood and vesicoureteric reflux All children with UTI must be evaluated for underlying predisposing conditions as recurrent pyelonephritis can cause renal scarring and loss of renal function. UTIs account for 7% of childhood febrile illness. In the age group <3 months, it is more common in males and in the age group >1 year, it is more common in females. Structural and functional abnormalities of the urinary tract such as VUR and posterior urethral valves predispose to UTI. Reflux is considered primary when it is due to an incompetent UVJ and secondary when it is due to increased bladder pressure or outlet obstruction. Presenting symptoms in neonates and infants include febrile illness or sepsis and may not be localised to the urinary tract. The method of urine sampling, especially before toilet training, is crucial and may involve suprapubic aspiration or per urethral catheter collection. A bacterial count of 50 000 colony-forming units per millilitre is generally considered a positive culture result in children, although a lower count from a suprapubic aspirate in a symptomatic child is significant. The most important complication of UTI in a child is renal scarring secondary to renal parenchymal inflammation. US should be performed in all children, and children with recurrent UTIs or a first time UTI with pyelonephritis should be evaluated further. VCUG is the investigation of choice to diagnose reflux and should be performed in high-risk children. Tc dimercaptosuccinic acid (DMSA) radionuclide cortical scan is the best modality to detect parenchymal lesions. VUR is present in approximately 30% of children with UTI and in up to 90% of children with Achille Boari, nineteenth century urological surgeon from Ferrara, Italy, described the technique of a bladder flap in dogs in 1894; it was first performed in a patient in 1936. renal scarring. Renal scarring may cause hypertension in up to 20% and is an important cause of renal failure. The grades of VUR are summarised in Figure 82.9, with grades I-III generally resolving spontaneously. Low-dose nocturnal antibiotic prophylaxis to prevent scar-inducing pyelonephritis is the mainstay of treatment as the majority of reflux cases resolve with time. However, surgery (ureteric reimplantation, periureteric injections of Teflon or collagen) should be considered if episodes of acute pyelonephritis recur despite antibiotic therapy or if severe reflux is accompanied by a surgically correctable malformation such as a paraureteric bladder diverticulum. Summary box 82.3 Infections

Grade 1: Grade 2: Grade 3: reflux into undilated ureter and pelvis moderate dilatation of the ureter, renal pelvis and mild forniceal blunting of the calyces Figure 82.9 Grades of vesicoureteric reflux (courtesy of Nivedita Kekre and Dr Madhuri Sadanala). Grade 4: Grade 5: reflux causing moderate ureteral tortuosity and gross pelvicalyceal dilatation with loss of papillary impressions of the calyces UTI: inflammatory response of the urothelium (host) to invading bacteria ABU: colonisation of urine with bacteria with no evidence of inflammation All pregnant women must be screened in the first trimester for ABU because, untreated, one-third of

these patients will develop UTI GUTB: Always due to either reinfection or reactivation of old tuberculosis Urine examination: sterile pyuria in acidic urine Close monitoring of upper tract during the initial phase of ATT and timely urological intervention may prevent renal loss UTI in children: More common in girls after 1 year of age Sample collection method is important to avoid contamination VUR occurs in 30% of children with a UTI Renal scarring is a possible long-term consequence

Urolithiasis is as old as mankind. The first documented cystolithotomy was described by Sushruta, an ancient Indian surgeon in almost 600 BC. The development of shockwave lithotripsy (SWL) and endourological procedures with multiple efficient energy-generating devices (such as US, pneumatic, electrohydraulic) for stone fragmentation have revolutionised the management of stone disease. Although the incidence of bladder stones has declined progressively owing to the alleviation of poverty and the improvement in basic nutrition, the modern world is witnessing a steady increase in the incidence of renal calculi.

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