Urinary tract tuberculosis in a child with late presentation posterior urethral valves

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A case is reported of urinary tract tuberculosis in a 7-year-old boy with a history of late presentation posterior urethral valves. Persistent hydroureteronephrosis after valve ablation at the age of 5 years and a draining urinary fistula from the site where a suprapubic catheter had been inserted prior to valve surgery alerted to the possibility of urinary tract tuberculosis.

Key Words: urogenital tuberculosis, posterior urethral valves, urinary tract infection

Introduction

Extra-pulmonary tuberculosis (TB) is a rare disease in children. A recent publication stated that 27% of all extra-pulmonary TB involves the urogenital system.[1] Urogenital TB has a variable presentation including hematuria, lower urinary tract symptoms (LUTS) with sterile pyuria and, rarely, persistent sinus or fistula drainage.[2,3] Posterior urethral valves (PUV) are typically detected prenatally by ultrasound (US), however, not all women receive an antenatal US, so some late presentations have been reported.[4] We present an unusual case of a young boy with a history of late presentation PUV that subsequently developed urinary tract TB a few years later.

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Case presentation

A 5-year-old boy was referred to the hospital after presenting at a local district hospital with failure to thrive, abdominal distension and incontinence. On presentation he had a urinary tract infection (UTI), and a raised serum creatinine of 205 µmol/L (age adjusted normal range 30 µmol/L-48 µmol/L) and GFR (modified Schwartz formula) of 17 mL/min/1.73m^2. His first US at the referring hospital showed bilateral hydroureteronephrosis. On admission at the referral hospital a voiding cystourethrogram (VCUG) confirmed PUV without evidence of vesicoureteric reflux (VUR),

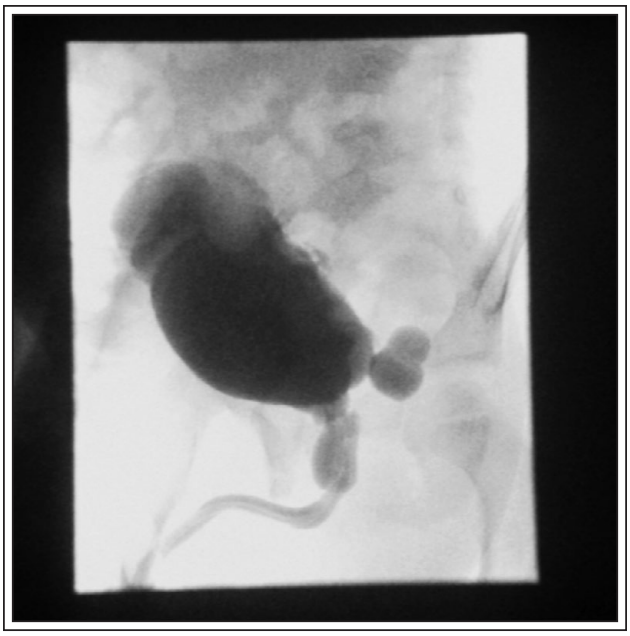

Figure 1. Voiding cysto-urethrogram in the 5-year-old patient showing a dilated posterior urethra with evidence of a posterior urethral valve, and a diverticulum of the posterior bladder wall, but no vesico-ureteric reflux.

Figure 1. The child underwent PUV ablation and a suprapubic catheter was placed and removed before discharge. Approximately 1 month later renal US still showed bilateral hydroureteronephrosis and a non-distended bladder with a thickened wall, Figure 2.

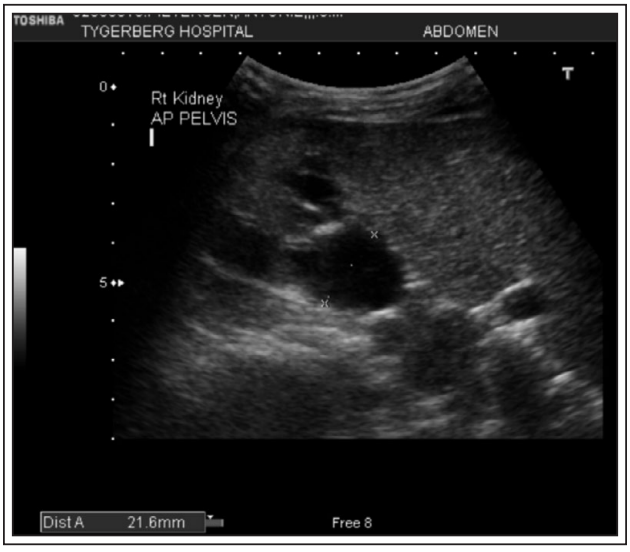

Figure 2. Ultrasound showing hydronephrosis at presentation when the patient was 5 years old.

Multiple follow up renal US were performed and showed improvement but not resolution of the bilateral hydroureteronephrosis.

After discharge, he was lost to follow up despite an attempt by the Social Service department to contact the family.

Two years later (at the age of 7) the child was admitted with complaints of poor weight gain, fever, left-sided abdominal pain and persistent urine leaking from the scar where the suprapubic catheter had been inserted previously. His mother stated that he had been poorly feeding and had been vomiting worms for several days prior to admission. A chest x-ray revealed bilateral hilar adenopathy. Urine dipstick showed evidence of UTI and the patient was started on empiric intravenous cefotaxime, which was followed by intravenous meropenem (aminoglycosides were avoided due to the presence of renal failure). However, after several days of treatment, the patient did not improve and multiple urine cultures revealed sterile pyuria. Renal US revealed bilateral hydroureteronephrosis with turbulent fluid in the left renal pelvis, Figure 3. Further history revealed that the patient had several close contacts that were either previously infected with TB or were being treated for TB. A tuberculin skin test was performed and after 72 hours the area of induration was 16 mm in diameter (> 10 mm in a HIV-negative child is considered positive for TB infection). An early morning urine and a sputum specimen both cultured rifampicine sensitive Mycobacterium tuberculosis.

The patient was started on triple drug therapy (isoniazid, pyrazinamide, rifampin) and remained on this treatment for 9 months. He was hospitalized

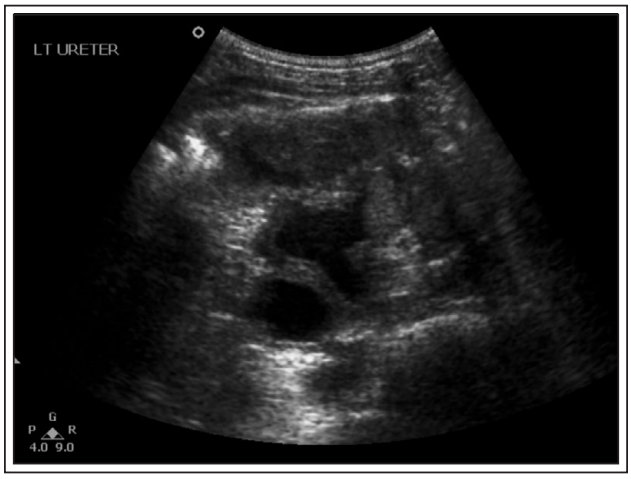

Figure 3. Ultrasound showing hydronephrosis with internal echoes suggesting turbid urine in the renal pelvis when the patient was 7 years old.

for several weeks, with weekly US which showed improvement of the hydroureteronephrosis. Subsequent visits revealed that the child had been compliant with his anti-TB medication and had clinically improved. He remains stable, but has stage 3 chronic renal failure with a serum creatinine of 145 µmol/L and an estimated GFR of 35 mL/min/1.73m^2. He is at present on chronic renal failure medication.

Discussion

We present a case with two uncommon disease entities: late presentation of PUV and childhood urinary tract TB. Typically, PUV are suspected when prenatal US reveals bilateral hydronephrosis and oligohydramnios. Valve ablation via a transurethral approach is the standard of care for these patients. While a majority of valve patients are diagnosed early in life, there are several reports of children who have presented in adolescence and as teenagers with various symptoms including day time incontinence and nocturnal enuresis, voiding pain and UTIs.[4] Management of these patients can be difficult, especially when the detrusor is thickened and fibrotic, causing poor bladder compliance (so called "valve bladder"). The keys to management of these patients are daytime clean intermittent catheterization and night-time continuous catheterization in an effort to prevent the bladder from becoming over-distended.[5]

Urogenital TB in the pediatric population is rare. Approximately 5% of all new TB case are extra-pulmonary in location and of those cases anywhere from 20%-73% is urogenital in origin.[1,6-8] Renal TB results from haematogenous spread from the primary pulmonary source. The presentation of urogenital TB is variable, but often involves LUTS, hematuria and recurrent UTIs. The most common urogenital organ involved is the kidney, but the ureter and bladder can also be involved.[8] The mainstay of treatment is anti-TB medications i.e. rifampin, isoniazid and pyrazinamide for several months. It is also important to continuously monitor these patients for upper tract deterioration by regularly checking laboratory values as well as US imaging of the kidneys to ensure that there is no deterioration with the development of ureteric stenosis.

Conclusion

Persistent drainage from the previous suprapubic tube site led to the diagnosis of renal TB in this child with late presentation posterior urethal valves. Non-resolving hydronephrosis after PUV ablation might be caused by renal or ureteric TB. □

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