CASE REPORT

Genitourinary tuberculosis: A case report with review of literature

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SUMMARY

The second most frequent extrapulmonary tuberculosis is genitourinary tuberculosis (GUTB). The tuberculosis infection can affect multiple areas of the genitourinary tract. As the presentations of the condition is non-specific, various imaging and laboratory procedures to diagnose GUTB should be considered. As it is difficult to perform a GUTB diagnosis due to the vague presentation in patients, this paper aimed to report and discuss the modalities of GUTB diagnostic investigations and its management. We presented a case of a 31-years-old gentleman with 1 month history of lower urinary tract symptoms, and not responsive to antibiotic treatment. Initial plain radiograph showed opacity at the left lower quadrant. Ultrasound of the kidney, ureters and bladder (KUB) reported a left hydronephrosis and hydroureter. Subsequently, computerised tomography (CT) urography reported left obstructive uropathy. Flexible cystoscopy revealed cystitis changes over the bladder wall with severe trabeculation and turbid urine. Investigation for tuberculosis infection was initiated. Although chest radiograph showed clear lung field with no opacity and tuberculin test was negative, the urine acid fast bacilli staining was finally revealed to be positive. The patient was diagnosed as having GUTB and anti-tuberculous medications were initiated. Patients appearing with chronic urinary symptoms who do not respond to antibiotic treatment should be investigated and treated with caution. Early detection is critical to avoid further damage to the urinary system, and to be followed with proper treatment options which include medical and surgery.

INTRODUCTION

Genitourinary tuberculosis (GUTB) is considered uncommon, but it is the second most frequent and severe form of extrapulmonary tuberculosis.[1,2] It accounts 30 to 40% of extrapulmonary tuberculosis cases and occurs in 2 to 20% of patients with pulmonary tuberculosis.[2] GUTB is more common in developing countries with 15 to 20% cases, compared to developed countries with about 2 to 10%.[2] Human immunodeficiency virus (HIV), chronic renal failure and diabetes mellitus patients have higher risks of contracting GUTB.[3] Clinically, it is important to diagnose GUTB as early as possible as it may prevent further renal destruction.

CASE REPORT

This is a case of a 31-years-old gentleman who was previously healthy. The patient presented with a month history of persistent chronic urinary symptoms, such as, dysuria, increase in frequency and hesitancy. He also complained of left loin pain especially when carrying objects and experienced fever even after he had received three courses of antibiotics from general practitioners. He had no hematuria, no abdominal pain, and did not experience loss of weight and appetite. On examination, he was not cachexic and his lymph nodes were not palpable. There was only mild tenderness over the suprapubic region. The respiratory and neurology assessments were unremarkable, whereas the urine microscopy was positive for leucocyte, protein and blood. The patient had no risk factors for pulmonary tuberculosis such as immunocompromised state or history of exposure to any tuberculosis patients.

Initial plain radiograph showed nonspecific finding. Ultrasound of the kidney, ureters and bladder (KUB) reported left hydronephrosis and hydroureter with focal wall thickening. Subsequently, computerised tomography (CT) urography reported bilateral obstructive uropathy more over the left than the right side. Pooling of contrast was also observed over the left side due to distal obstruction, which can be caused by vesicoureteric lesion with probable differential diagnosis of either mass, soft stone or stricture.

The patient was then investigated for tuberculosis infection. Chest radiograph showed clear lung field with no opacity. The tuberculin test showed 5 mm of induration which was interpreted as negative, as well as the urine culture. Eventually, the urine for acid fast bacilli staining was positive. Unfortunately, histopathology of urinary bladder and Mycobacterium tuberculosis culture of urine were not performed. Consequently, the patient was treated as GUTB-positive. Anti-tuberculous medications were started with Akurit-4 four tablets per day and oral pyridoxine 10 mg daily for 9 months. Repeated urine specimen for acid fast bacilli staining after 1 month on treatment reported negative twice.

During the 6 months of anti-tuberculosis therapy, CT urography was performed to assess the patient's response to treatment. Repeated cystoscopy and retrograde pyelogram showed trabeculated bladder; right ureter opening was wide and golf ball-shaped. From the ultrasound, the bladder was

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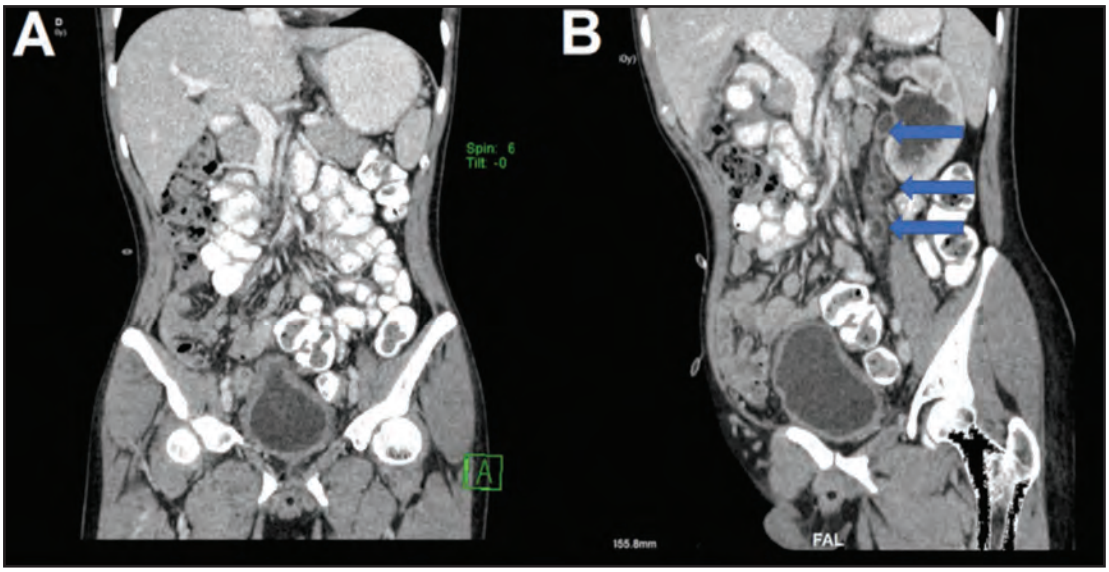

Fig. 1: (A) Image shows abnormal dome-shaped urinary bladder suggestive of neurogenic bladder. (B) The blue arrows in the above picture showed left hydronephrosis.

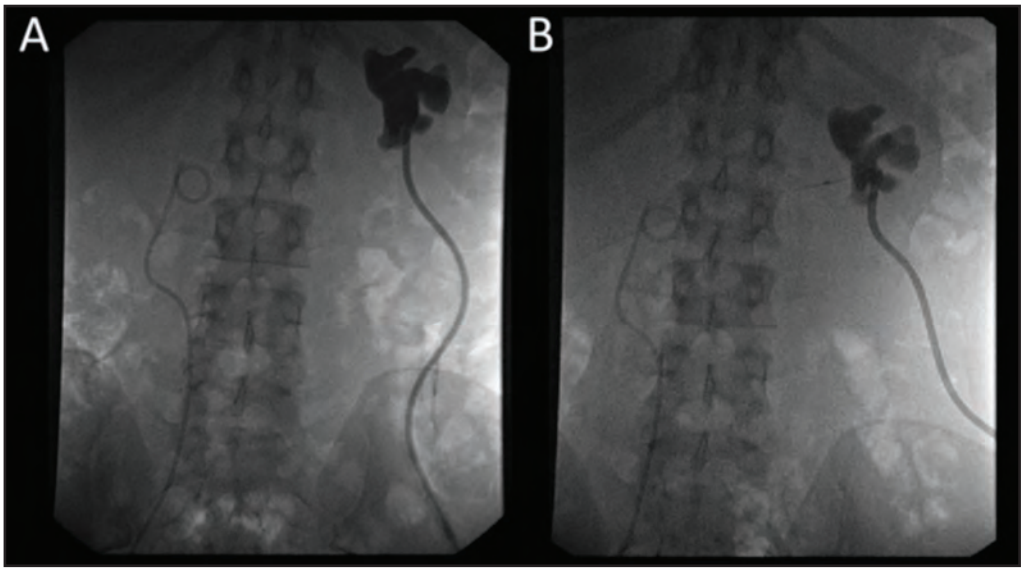

Fig. 2: Left antegrade pyelogram revealed that the patient experienced reducing degree of left hydronephrosis from grade V to grade IV post nephrostomy tube insertion.

found thickened with 430 ml volume which was still within the normal range. Right retrograde pyelogram showed dilated right pelvicalyceal and ureter with kinked proximal right ureter and no ureteric stricture was obvious. However, multiple-length stent was still being used in view of right hydronephrosis. Left retrograde pyelogram was abandoned in view that the left ureter orifice could not be identified during the procedure.

There was a delay in about one month time for left nephrostomy tube in view of the patient having subclinical urinary tract infection. Patient was treated with oral cefuroxime for 1 week. He had nephrostomy tube inserted at the left side. Subsequently, he underwent left antegrade pyelogram which showed left hydronephrosis and hydroureter with abrupt cut off at upper ureter leading to failed antegrade ureteric stenting. This nephrostomy tube is planned for long term. The patient experienced reducing degree of left hydronephrosis post nephrostomy tube insertion (Figure 2). Post-procedure, the patient also developed E. coli ESBL (extended-spectrum beta-lactamases) urosepsis and was treated with intravenous ertapenem for two weeks. White blood cell (WBC) count was initially normal (9 x 10 ^3/uL), but raised to 16 x 10 ^3/uL during the infection and the C-reactive protein (CRP) was high, at 124 mg/L. His CRP later on reduced to 86 mg/L after few days of antibiotic treatment. His previous serum creatinine was 118 umol/L but increased to 155 umol/L during the sepsis episode. But later on, the serum creatinine reduced to 140 umol/L after the infection was controlled. Urine output from

the left nephrostomy tube was also good. No radio-isotope scan was performed to compare both kidney function. We noted that the patient creatinine was increasing in trend from 140 to 200 umol/L in eight months but it remained static after a few months of monitoring. He was planned for a long term left nephrostomy and the tube was changed monthly.

The patient presented eight months later during urology clinic follow up with urinary urgency and acute kidney injury, where serum creatinine increased to 400 umol/L with hyperkalemia but without uremic symptoms. He was still able to pass urine with no abdominal pain and fever. Ultrasound showed echogenic debris within the urinary bladder which may represent cystitis with no significant left obstructive uropathy, and the left nephrostomy tube was in situ. Urine analysis was suggestive of urinary tract infection with WBC in the urine. Complete blood count showed raised WBC 17 x 10 ^3/uL. Urinary catheter was inserted and one litre of urine was drained from the bladder. Urine output from the left nephrostomy tube was around 1.9 L per day. He was treated as neurogenic bladder secondary to GUTB complicated with urinary tract infection. Urine culture grew *Streptococcus, beta-haemolyticus* Group B which is sensitive to penicillin. Intraveneous amoxicillin-clauvenate was started and later changed to oral antibiotic for a duration of one week. He was counselled for clean intermittent self-catherisation (CISC) or suprapubic cystostomy, which he opted for CISC. He was discharged with improved renal profile, where creatinine was reduced to 347 umol/L with good urine output. He was planned to be seen back in the urology clinic after one month but he defaulted urology follow up in view of logistic issues. He subsequently underwent two-weekly changes of urinary catheter in a district hospital. He was also re-diagnosed with hypertension which was probably due to chronic renal disease secondary to GUTB.

DISCUSSION

This case study discussed the diagnosis investigation of GUTB and its management. It is often difficult to infer the diagnosis of GUTB because of the vague presentation in patients. It is important to know the history of previous tuberculous infection either primary pulmonary or extrapulmonary in an individual. There is a latency period that can reach up to 40 years before GUTB is manifested.[2] The symptoms of GUTB are dysuria, nocturia, frequency and chronic urgency of urination; back, flank and suprapubic pain and hematuria. The uncommon symptoms include renal colic, fever, weight loss and night sweat.[4] Patients usually will have abnormal urine microscopy like hematuria, pyuria and albuminuria.[4]

GUTB is caused by the infectious Mycobacterium tuberculosis bacilli which is acquired through inhalation of aerosolised droplet containing the microorganism.[5] The mycobacteria will replicate in alveolar macrophages of the lung, and subsequently metastatically spread hematogenously reaching the genitourinary organ.[1]

GUTB can involve kidneys, ureters, bladder, adrenal gland, male genital, female genitourinary organs and the retroperitoneum.[6] Kidneys can be part of the disseminated infection or localised genitourinary infection which may be present as unilateral or bilateral.[4] The healing process during the course of the infection will result in calcification and fibrosis.[3] This process will result in obstructive uropathy as seen in this patient. Tuberculous infection involving ureters is caused by the spread from kidneys with the ureterovesical junction being the most common site. From the ureteral orifice, this tuberculous infection can extend to the bladder. Further inflammation can cause stricture, rigidity and dilated golf hole-like appearance.[7]

In order to detect M. tuberculosis infection, several diagnostic investigations should be performed. For this patient, the diagnosis was confirmed with urine acid fast bacilli. However, urine acid-fast bacillus smear is often negative, hence might not be reliable as it may be positive for Mycobacterium smegmatis.[4] The diagnosis of GUTB should be made with at least three consecutive early morning urine specimen cultures.[4] Between 10 to 90% of tuberculosis patients have positive urine culture.[3] Polymerase chain reaction (PCR) is also a sensitive and specific diagnostic method for detection of GUTB. Between 25 to 30% of GUTB diagnosis were made from positive culture or histology specimen in combination with M. tuberculosis PCR.[4] A positive tuberculin test supports the diagnosis of tuberculosis, but a negative tuberculin test does not exclude extrapulmonary manifestations.[8]

In GUTB, plain radiograph may show calcifications which occurred in more than 50% of patients.[9] Ultrasound of the urinary tract commonly show involvement of multiple areas in the urinary tract with multiple stages of the disease.[10] For example, the granuloma can be seen as masses and causes distortion of calyces. Cavitations, calcification of kidney and nephrocutaneous fistula can also be seen.[10] CT scan can detect calcifications, distortion of anatomy such as hydronephrosis, renal parenchymal lesions and scars, and the extent of extrarenal infection.[10] Retrograde pyelography may be used to determine the length of the stricture and the severity of the obstruction and dilatation above the stricture.[4] Cystoscopy should be performed to look for cystitis changes and to exclude tumours. Bladder biopsy can be done in patients with tubercles or ulcers distance from a normal ureteral orifice as is needed to exclude carcinoma.[4] Histopathology is also very useful to assist in diagnosing GUTB. In a recent study conducted in Sabah, Malaysia, 52.9% of GUTB cases were confirmed via histopathological assessments.[11] Admittedly, the limitations found in this case is that mycobacterium urine culture and bladder biopsy were not performed. Both assessments are of paramount importance and should be performed in future cases to improve investigation and management for similar conditions.

The treatment for GUTB using anti-tuberculous drugs is generally for 6 months.[1,12] In complicated cases, the treatment may take 9 to 12 months, such as in recurrences of tuberculosis and in immunosuppressive patients. For multidrug resistance tuberculosis (MDR TB) which is defined as resistant to rifampicin and isoniazid, it requires the use of at least four drugs that are selected based on drug susceptibility test. The duration for MDR TB also depends on the susceptibility of the drugs.[12]

Surgical management in GUTB accounts to about 0.5% of urological procedures.[13] Ablative surgery is also one of the treatment available especially in complicated GUTB with abscess and non-functioning kidneys. Examples of ablative surgery are nephrectomy and partial nephrectomy.[4] Reconstructive surgery such as repair of strictures at the lower end of ureter and bladder augmentation are usually performed after at least four weeks of anti-tuberculosis medications.[7] Early ureteral stenting or percutaneous nephrostomy (PCN) in tuberculous ureteral strictures decreases the chance for renal loss and increase the chance for later reconstructive surgery.[14]

CONCLUSION

There should be high suspicion for patient presenting with chronic urinary symptoms and not responding to antibiotics treatment. Early diagnosis is essential to prevent further destruction of the urinary system as multiple modalities of treatments are available nowadays.

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DECLARATION

The authors declare no conflict of interest.

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