CASE REPORT

Obstructive uropathy as initial presentation of genitourinary tuberculosis and masquerading as a postsurgical complication

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SUMMARY

A 46-year-old woman who had a recent total abdominal hysterectomy presented with a 1 month history of lower abdominal pain, 1 week of nausea and vomiting as well as decreased urinary output preceded by a year of significant unintentional weight loss. On renal imaging, she was found to have bilateral hydronephrosis and hydroureters in the setting of bilateral distal ureteric obstruction complicated with acute kidney injury and severe hyperkalaemia requiring haemodialysis. The initial concern was for ureteric injury, a known complication of abdominal hysterectomy procedures, however, a urological intervention, performed 9 months later to relieve the ureteric obstruction, revealed purulent material within the left ureter that was smear positive for acid fast bacilli. A GeneXpert test was positive for *Mycobacterium tuberculosis*. She was diagnosed with genitourinary tuberculosis and responded well to antitubercular treatment and haemodialysis was discontinued after the surgery relieved her ureteric obstruction.

BACKGROUND

Tuberculosis (TB) is a common infectious disease in developing countries and contributes significantly to morbidity and mortality in those regions.¹ In 2015, 10.4 million people were estimated to be newly infected with TB globally, including 26% occurring in the African region.² In Rwanda, 6024 cases of TB were reported in 2014, 14% of which were extrapulmonary infections.³ Even in endemic areas, TB disproportionately impacts individuals with compromised immune systems and can affect almost any organ of the body with a variety of manifestations that can pose a diagnostic dilemma to the physician. Genitourinary (GU) TB occurs at a frequency of 2%–20% of all cases of TB⁴ and can mimic other conditions that cause urinary tract abnormalities. This case report will describe a severe presentation of GU TB masquerading as a postsurgical complication in a female patient.

CASE PRESENTATION

A 46-year-old female presented to our hospital with a chronic history of unintentional and unquantified weight loss and general weakness; and then a more recent 1 week history of abdominal discomfort, bilateral flank pain, non-bloody watery diarrhoea, severe nausea and vomiting with an episode

of haematemesis. A decrease in urinary output over the preceding 5 days was reported. She denied fevers, chills or night sweats. On review of systems, she had no chest pain, shortness of breath, leg or facial swelling, pain with or difficulty voiding urine, change in urine colour, blood in stool or vaginal discharge.

Her medical history included having a cervical polypectomy and uterine leiomyomas for which she had an uncomplicated total abdominal hysterectomy with salpingectomy and right ovarian cystectomy performed a month prior to presentation. She had been recently treated for a urinary tract infection (UTI) caused by *Escherichia coli*. She worked in a healthcare facility attending to health needs of orphans and the elderly. Her age at menarche was 17 years and she had never been pregnant.

On presentation, she was thin and in no acute distress. Her vital signs revealed a temperature of 36.4°C, blood pressure (BP) of 166/104 mm Hg, heart rate of 103 beats per minute (bpm) and respiratory rate of 22 cycles per minute. Her oxygen saturation on room air was 91%. Her ocular and oral examinations were remarkable for pale conjunctivae and dry oral mucous membranes respectively. Cardiovascular exam revealed regular tachycardia with no murmurs or pericardial rub. Her lungs were clear on auscultation, her abdomen was not distended, had a well healed Pfannenstiell incision site and there was suprapubic tenderness without rebound tenderness or guarding; bowel sounds were normal. Perineal exam was unremarkable. She was alert and oriented to time, place and person, had a Glasgow Coma Score of 15/15, pupils were normal sized and reactive and she had no focal neurological deficits. She did not demonstrate asterixis or myoclonus. There was no lower extremity oedema.

INVESTIGATIONS

Initial investigations including a complete blood count revealed white blood cell (WBC) count of $8.0 \times 10^9/L$ with differential showing—neutrophils, 72.9%; lymphocytes, 15.9%; eosinophils, 0.7%; monocytes, 10.3% and basophils, 0.7%; haemoglobin of 8.9 g/dL with mean corpuscular volume (MCV) of 71 and platelet count of $321 \times 10^9/L$. Electrolytes measurements were as follows: urea, 46.5 mmol/L; creatinine, 30.2 mg/dL; potassium, 8.5 mmol/L; sodium, 129 mmol/L and

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chloride, 88 mmol/L. Urinalysis revealed numerous (unquantified) WBCs, with albumin estimation at 0.15 g/L and urine cultures negative for growth. Renal ultrasonography at the time of admission revealed moderate bilateral hydronephrosis (more prominent on the left) without hydroureters and an empty urine bladder. An ECG showed normal sinus rhythm with a heart rate of 100 bpm. There were no T wave abnormalities. HIV test was negative.

DIFFERENTIAL DIAGNOSIS

Initial concerns in the emergency department were for a prerenal acute kidney injury in the setting of gastrointestinal (GI) fluid losses complicated with hyperkalaemia and coincident with partial obstructive uropathy related to her recent abdominal hysterectomy procedure and probably due to bilateral ureteric ligation or injury. Retroperitoneal fibrosis was also considered. Her elevated blood pressure was thought to be related to her renal disease as prior outpatient measurements were within normal range. Her GI symptoms were attributed to uraemia.

TREATMENT

The initial management by the emergency department physicians included administration of 2 L of intravenous fluid (0.9% normal saline) with placement of a urethral catheter to monitor the patient's urine output. She was given treatments for BP control and acute management of her hyperkalaemia. However, her hyperkalaemia remained refractory to medical treatment and her creatinine only decreased from 30.2 mg/dL to 23.2 mg/dL in spite of adequate fluid resuscitation. Within 5 days of admission, she required haemodialysis to correct her persistent hyperkalaemia.

Eight days into her admission, she developed a fever with leucocytosis ($19.3 \times 10^9/L$). Fever workup revealed an *Acinetobacter* UTI that was initially treated with intravenous ceftriaxone 2 g administered twice daily empirically and later switched to oral ciprofloxacin 500 mg taken twice daily when the organism's identification and sensitivities were available. However, despite the treatment, she continued to have intermittent fevers and developed dyspnoea. Chest X-ray showed bilateral pleural effusions. A diagnostic thoracentesis was performed and the pleural fluid obtained appeared grossly bloody. Pleural fluid was only sent for cytology and no malignant cells were seen. No microbiological studies or fluid analysis were performed at the time. Adenosine deaminase test was not performed as it is not available in Rwanda.

After a few weeks of intermittent haemodialysis, markers of kidney function such as serum urea, creatinine and electrolytes improved but the bilateral hydronephrosis persisted despite a normal urine output ($>1 \text{ mL/kg/hour}$). Her pleural effusions resolved. She was discharged home after a 6-week hospital stay on chronic haemodialysis with outpatient urology follow-up.

Seven months following her initial presentation to the hospital, at a follow-up urology clinic visit, an abdominal MRI was ordered and performed which revealed bilateral hydronephrosis and hydroureters with partial obstruction of the distal third of the ureters. There was contrast excreted into the bladder suggesting that the obstruction was partial and there were no enlarged intra-abdominal or pelvic lymph nodes identified. A cystoscopy was then performed revealing turbid urine in the urinary bladder with whitish tissue in the trigone area; however, both orifices of ureters were not visualised, therefore, it was impossible to catheterise the ureters to assess for the cause of the obstruction. No biopsies were taken. A month later, an elective

exploratory laparotomy was performed. During the surgery, it was found that both ureters were dilated, being much worse on the left. While the right ureter was partially patent, the left one was completely stenosed. In the left ureter, a collection of pus that was thought to be the cause of obstruction was subsequently drained after which a left ureteric reimplantation was performed and a JJ ureteric stent inserted. On microbiological testing, the purulent material that was obtained from the left ureter was smear positive for acid fast bacilli (AFB) and a GeneXpert assay returned positive for *Mycobacterium tuberculosis* with no resistance to rifampicin detected.

A definitive diagnosis of urogenital tuberculosis was thereby made and anti-tubercular treatment was initiated as per the national protocol, with 2 months of orally administered rifampicin (600 mg), isoniazid (300 mg), pyrazinamide (1600 mg) and ethambutol (1100 mg) with 25 mg of oral pyridoxine, followed by 4 months of rifampicin (600 mg) and isoniazid (300 mg), also administered with the same dose of pyridoxine.

OUTCOME AND FOLLOW-UP

The immediate post-operative period was remarkable for a post obstructive diuresis (urine output peaked at 4.5 litres per day) which resolved over time. The patient was discharged on post-operative day 10 with normal renal function tests and electrolytes (urea, 4 mmol/L, creatinine, 0.92 mg/dL and potassium, 4.3 mmol/L), had a normal urine output and demonstrated improved but mild residual bilateral hydronephrosis on follow-up renal imaging.

After she was discharged home, she had two subsequent episodes of bacterial pyelonephritis that were successfully treated and her ureteric stent was removed. To date, the patient has completed 5 out of 6 planned months of anti-tubercular treatment without any other events.

DISCUSSION

Extrapulmonary tuberculosis (EPTB) is prevalent worldwide but the exact incidence is difficult to ascertain as it is frequently misdiagnosed or underdiagnosed by clinicians because it mimics many other conditions. The WHO reports that 15% of the 6.4 million TB cases notified globally in 2015 were EPTB.² Data on EPTB in Rwanda is scarce but unpublished data from the Rwanda Ministry of Health TB programme showed that, in 2014, there were 849 cases of EPTB reported. A single centre study conducted in the University Teaching Hospital of Kigali in Rwanda between May 2008 to August 2009, found that of all TB cases, 40% had pulmonary TB, 43% had EPTB and 17% had both.⁵ Genitourinary (GU) TB, in particular, is thought to be the second most common form of extrapulmonary tuberculosis,⁶ and is reported to affect more men than women (2:1 incidence) with obstructive symptoms being the most common presentation.⁴

Extrapulmonary TB usually results from disseminated of infection following inhalation of the bacilli into the lungs and can affect any organ in the body with a wide variety of clinical manifestations.⁷ Urogenital TB develops through haematogenous spread of *M. tuberculosis* from a lung focus to the urinary tract and kidneys, and this can occur months to years following exposure to the bacteria.⁴ Healthcare workers, who practice in TB endemic regions typically with limited infection control infrastructure, as was our patient, are particularly at risk for acquiring the infection from their patients.

The symptoms of GU TB are usually non-specific, and most patients present with urinary tract symptoms or renal failure (a frequency of up to 7.4% has been reported.)⁴ Due to its insidious

nature, patients typically present with symptoms after significant organ damage such as renal failure from ureteric obstruction. GU TB may also present with urolithiasis and as chronic pyelonephritis.⁸ However, there are no specific symptoms that are wholly suggestive of GU TB, and these conditions can result from a myriad of other causes. In our case, the history of pelvic surgery 1 month prior to the development of bilateral hydronephrosis impeded consideration of alternative aetiologies as the treating team assumed that an accidental ligation or injury of both ureters during surgery was the most likely cause of the urinary tract obstruction. However, the delayed onset after her surgery and finding of partial and not complete ureteric obstruction should have raised doubt as to the presumed diagnosis. Furthermore, the finding of 'sterile pyuria' on urinalysis and culture should have raised concern for a chronic infectious process such as TB.

In resource-limited settings, the lack of advanced diagnostic tools such as GeneXpert test and other sensitive molecular based assays for detection of *M.tuberculosis* can make the diagnosis of extrapulmonary TB challenging especially as testing of body fluids including urine for AFB smear and culture are notoriously insensitive. For example, in one study, of 35 patients who had confirmed GU TB, only 13 (37.1%) had positive urine cultures while 33 (94.3%) had a positive urine PCR test.⁹ However, cultures retain the advantage of being amenable to phenotypic

antimycobacterial drug susceptibility testing. In the absence of PCR testing, another diagnostic approach would be to obtain tissue specimens to assess for the disease (by pathological examination for granulomas and/or AFB smears and culture or *M. tuberculosis* PCR testing), but this invasive approach has its own challenges in resource-limited settings including the limited availability of skilled specialists, surgical facilities and equipment and inadequate laboratory infrastructure.

GU TB, because of its insidious progression, thereby becoming symptomatic only when significant organ injury has occurred, is often diagnosed late or misdiagnosed altogether, leading to disastrous renal and reproductive health complications. Auto-nephrectomy,⁸ ureteric strictures,⁴ primary or secondary infertility¹⁰ and nephrocutaneous fistulae⁸ are some of the reported complications of GU TB. Our patient suffered multiple bacterial UTIs probably as a consequence of ureteric strictures with stenosis related to her TB disease. This case highlights the importance of a high index of clinical suspicion for the disease especially in endemic areas, as early diagnosis and initiation of adequate treatment may prevent or reduce the severity of such devastating complications.

Contributors GI, TH both provided direct clinical care to the patient and identified this case as worth sharing. GI, TH, OMM, OO were responsible for conception of the article, literature search, drafting and revision of the article as well as approval of the submitted draft.

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Patient's perspective

I am happy that after several months of undefined illness, a definitive diagnosis is finally made. I did not know that tuberculosis could attack my urinary tract and cause renal problems. As a nun taking care of marginalised, poor and sick people, I had seen many patients with pulmonary tuberculosis but never had I heard of any person who has had this kind of tuberculosis. I am grateful to the whole treating team and I would be happy if my story could help other clinicians and patients around the world.

Learning points

- ▶ In endemic areas, tuberculosis (TB) is a rare but important cause of obstructive uropathy that may mimic other conditions.
- ▶ Recurrent bacterial urinary tract infections are a complication of *Mycobacterium tuberculosis*-associated obstructive uropathy.
- ▶ While urine AFB smear and culture are insensitive for diagnosing genitourinary TB, PCR testing retains high sensitivity.

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