

CASE REPORT

Renal tract tuberculosis in a young man with chronic flank pain and lower urinary tract symptoms

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Abstract

We present here a case of a 37-year old white male, of African origin, who presented on numerous occasions with lower urinary tract symptoms and flank pain. He eventually was admitted with acute on chronic renal failure.

Inpatient investigations demonstrated bilateral ureteric obstruction secondary to stricture disease. A nephrostomy was placed to the functioning right kidney but antegrade ureteric stenting was impossible. Rigid cystoscopy demonstrated a low-capacity bladder with copious debris. Early morning urine samples, eventually demonstrated mycobacterium, alongside bladder biopsy

samples which demonstrated caseous granulomas. The patient was treated, under nephrology and TB physicians with quadruple anti-TB therapy with considerable improvements in his renal function.

This case was noteworthy because: the patient had never had pulmonary TB, we were successfully able to demonstrate positive urine cultures and the outcome was positive.

We compare this case to the available literature and reflect upon the delays in diagnosis and potential improvements to management.

Key words

tuberculosis; obstructive uropathy; renal tract tuberculosis; ureteric stricture disease

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Introduction

Lower urinary tract symptoms (LUTS) such as poor flow, urinary frequency and nocturia are very common complaints in urology outpatient departments.

Flank pain, or 'renal colic' is a common presentation to Emergency departments. However, the prevalence of these presenting complaints does not guarantee the ultimate diagnosis is straight - forward.

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We present a case of a 37 - year old male with a 4 year history of progressively worsening LUTS and left flank pain. Amongst other factors, the diagnosis was delayed by failing to keep a broad differential diagnosis or thinking beyond the common causes.

This case also serves as a reminder that the prevalence of tuberculosis is increasing in Europe. The effects of renal tract TB can be widespread and severe. Given its insidious onset and that a history of primary, pulmonary TB may not be noted, the diagnosis can be delayed or missed. This case was not only interesting and unusual, but also a useful reflective exercise about our own practice

Case Presentation

Mr C initially presented to the urology outpatient department in 2010, aged 33. He was a Portuguese - speaking white gentleman, born in Mozambique who had moved to the UK, via Portugal, in 2008. He worked as a chef at a nearby airport. He had never smoked, does not drink alcohol and eats a balanced diet. His wife and 3 children still live in Mozambique. He reported a past medical history of only 'an enlarged prostate', he had never been hospitalised before, nor had he ever knowingly had tuberculosis or any chest complaint.

Mr C had been referred by his GP with hesitancy, poor flow, and urinary frequency; passing urine 10 times per day and 4 times at night. Following this initial consultation a flexible cystoscopy was planned but the patient never attended and was lost to follow - up. Over a year later he presented to the emergency department, on two separate occasions, with left flank pain. He reported these two episodes were typical of flank pain he regularly experienced and for which he took ibuprofen regularly. On both occasions he had a CTKUB but was not admitted. A clinic appointment was arranged in February 2012 as a result of the CT findings (discussed later). From clinic, urine cultures and serology for schistosomiasis were sent and a follow - up ultrasound and flexible cystoscopy arranged but again the patient did not attend. Finally, in November 2013 Mr C was again re - referred to urology clinic with worsening LUTS although he still felt systemically well. Blood results on the day demonstrated a creatinine of 716µmol/L and urea of 30.8mmol/L, he was admitted for further investigations as an inpatient.



Figure 1: Coronal non - contrast CT from 1 year prior to hospital admission. Performed by the emergency department for renal colic. The left kidney has an unusual appearance with thin cortex and large central hypodense area and lobulated margin. In addition there is nonspecific increased density or calcification of the bladder wall

Investigations

The two CTKUBs in 2012 had demonstrated left sided Pelvi - calyceal dilatation, without a dilated ureter and marked thinning of the left renal cortex. On the right, the kidney cortex appeared normal but a dilated pelvi - calyceal system and ureter were noted. The second scan also noted possible circumferential calcification of the bladder wall (**Figure 1**). A follow - up USSKUB confirmed the findings but demonstrated an apparently normal Doppler blood flow in both renal parenchyma.

Urinalysis in the emergency department had demonstrated microscopic haematuria and in clinic had demonstrated leucocytes, without nitrites, but routine urine cultures had grown no pathogens.

A flexible cystoscopy, performed on the day of admission demonstrated a stricture in the penile urethra 2cm from the urethral orifice which the cystoscope was unable to pass. Urine flow testing demonstrated very poor stream (Qmax 4.9ml/s, average 2.7ml/s).

An ultrasound during admission showed an enlarged, 14cm right kidney with pelvicalyceal dilatation and a 4.8 cm 'cystic area' with 'sloughing of the pyramids'. The left kidney was again noted to have very thin cortex and appear cystic.



Figure 2: *a.* Coronal CT sections performed after nephrostomy and attempted retrograde stent insertion. Note the dilated renal pelvis of the right kidney. *b.* Note how the distal ureter is swollen yet no contrast is able to pass to the empty bladder - demonstrating severe stricture disease

Treatment

On presentation, the urethra was dilated and a urethral catheter placed, the residual volume was only 65ml. Later, a right nephrostomy was inserted to protect the hydronephrotic right kidney. Fluid optimisation and IV hydration was performed with Mr C entering a post-obstructive diuresis phase, producing up to 8,000ml/day. An antegrade stent could not be passed during this procedure, with obstruction noted at the right vesico-ureteric junction. CT images with contrast were obtained following the procedure, demonstrating dilated renal pelvis and right ureter, but no passage of contrast through to the urinary bladder (**Figure 2a**, **Figure 2b**). Once the nephrostomy was in place, there was zero urine output via the urethral catheter confirming a non-functioning left renal unit.

Retrograde attempts to identify and stent the right ureteric orifice were unsuccessful with the bladder wall noted to be inflamed with copious debris in the bladder. Subsequent histology of bladder biopsies demonstrated caseous granuloma.

During admission, creatinine values fell, before plateauing at 400µmol/L. Multiple investigations during a prolonged inpatient stay included a positive TB Elispot test and 3 separate early-morning urines (EMU). After 9 days incubation one of these EMU

grew fully-sensitive *Mycobacterium tuberculosis*.

Mr C was commenced on quadruple TB therapy (Rifampicin, Isoniazid, Ethambutol and Pyrazinamide) for 8 weeks then reduced to triple therapy (Rifampicin, Isoniazid and Ethambutol) to complete 1 year. High-dose prednisolone was commenced to aid renal function, alongside appropriate bone and stomach protection.

Outcome and follow-up

6 months after discharge Mr C remains well and compliant with his medication. He copes well with a right-sided nephrostomy and has returned to work. His visual acuity had deteriorated such that ethambutol was stopped, his vision has subsequently recovered. His creatinine count has fallen to 274µmol/L and CRP normalised.

A recent nephrostogram failed to demonstrate any passage of contrast in to the urinary bladder suggesting stenting may still be impossible. Unfortunately, his attendance to appointments is not reliable, we hope to perform further urodynamics assessment of his bladder prior to definitive reconstruction of the urinary tract.

Discussion

Tuberculosis (TB) remains a large global healthcare



problem. The incidence of disseminated and extra-pulmonary disease is rising¹. After peripheral lymphadenopathy, genito-urinary TB is the most common form of extra-pulmonary TB. 8-15% of patients with pulmonary TB develop infection in the genito-urinary system². Mycobacteria bacilli reach distant organs by haematogenous spread, TB of the urinary tract then takes a descending route of infection³. Genito-urinary effects of TB can be broad and varied including renal calcification, necrosis, stricture disease throughout the urothelium and bladder contractures^{3,4}. Renal failure can, therefore, be caused by damage to the renal parenchyma itself, or by obstructive uropathy.

In this case stricture disease was evident in the penile urethra, right vesico-ureteric junction and can be assumed to have been present in the left renal unit causing complete non-functionality. Mr C was hospitalised before his kidney function was irreversibly damaged. Although no such cases have been found in recent Western medical literature, two similar cases have been published in Brazilian journals, with less successful outcomes. One reports a 33-year old man with 3 years intermittent history of flank pain presenting in renal failure, despite best care he remains on lifelong haemodialysis⁵. The second reports a 32-year old female with recent history of peritoneal TB who, despite 6 months anti-tuberculosis therapy, presented in acute renal failure secondary to TB which was ultimately fatal⁶. Unlike our case - in both of these cases a clear history of pulmonary TB was elicited and indeed these presented in areas where TB is endemic.

Also satisfying within our case is that we were able to demonstrate positive mycobacterium urine cultures. A Moroccan review of 109 urogenital TB cases found bacilli in only 41 (38%) of their patients,⁷ indeed these two aforementioned published reports were unable to demonstrate positive urine cultures.

On retrospective analysis of this case we considered clues that may have led to earlier diagnosis. Initial urinalysis had demonstrated sterile pyuria and microscopic haematuria, a common finding in urinary tract TB along with acidic urine⁸. Irritative lower urinary tract symptoms, which fail to improve with routine antibiotics should raise the possibility of TB⁹.

Radiological investigations can also demonstrate signs suggestive of TB. USS, CT and IVU can all be helpfully used, with CTIVU the most sensitive at identifying all manifestations of renal tuberculosis¹⁰. Signs progress from papillary necrosis in early disease to multifocal strictures and mural thickening prior to endstage progressive hydronephrosis, parenchymal thinning and dystrophic calcification. Even the non-contrast CTs performed on Mr C in 2012, showing abnormal left kidney size, should perhaps have been reviewed in a multi-disciplinary meeting. This case serves as a reminder of the importance of strong communication within and between departments, and coordinated strategies to reliably follow-up patients; all the more important as modern healthcare becomes more sub-specialised. 

Conflicts of interest

The authors declared no conflicts of interest.

Περίληψη

Παρουσιάζουμε την περίπτωση ενός 37 χρόνου άνδρα, Αφρικανικής καταγωγής, που παρουσιάστηκε στα εξωτερικά ιατρεία με συμπτώματα του κατώτερου ουροποιητικού και οσφυϊκό πόνο. Η εισαγωγή του έγινε με κλινική εικόνα οξείας σε έδαφος χρόνιας νεφρικής ανεπάρκειας. Κατά την παραμονή του στην Κλινική οι διάφορες εξετάσεις ανέδειξαν αμφοτερόπλευρη απόφραξη των ουρητήρων και τοποθετήθηκε νεφροστομία στον πιο λειτουργικό δεξιό νεφρό καθώς η τοποθέτηση JJ stent δεν ήταν δυνατή. Η άκαμπτη κυστεοσκόπηση ανέδειξε ουροδόχο κύστη περιορισμένης χωρητικότητας ενώ στα δείγματα πρωινών ούρων και στην παθολογοανατομική εξέταση βιοψίας της κύστης απεδείχθη φυματίωση του ουροποιητικού. Ο ασθενής έλαβε τετραπλή αντι-φυματική αγωγή και υπήρξε σημαντική βελτίωση της νεφρικής του λειτουργίας. Η περίπτωση αυτή είναι ενδιαφέρουσα καθώς ο συγκεκριμένος ασθενής δεν είχε ποτέ νοσήσει με πνευμονική νόσο ενώ ταυτόχρονα θελήσαμε να αναδείξουμε την πάθηση ώστε να βελτιωθεί ο τρόπος με τον οποίο αντιμετωπίζονται παρόμοια περιστατικά.



Λέξεις ευρετηριασμού

**φυματίωση ουροποιητικού,
αποφρακτική ουροπάθεια,
στένωση ουρητήρων**

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