INTERNAL MEDICINE

□ CASE REPORT □

Use of Corticosteroids for Urinary Tuberculosis Patients at Risk of Developing Ureteral Obstruction

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Abstract

A 77-year-old man with urinary tuberculosis developed post renal anuria two days after starting an anti-tuberculosis drug regimen. He had bilateral hydronephrosis, and his right kidney was radiologically diagnosed to be non-functioning. A transurethral catheter was placed in the left ureter. No improvement in the ureteral stricture was noted during the initial three weeks of treatment; however, the stricture did thereafter improve after the commencement of oral prednisolone. In cases of urinary tuberculosis, ureteral stricture can deteriorate and result in ureteral obstruction during anti-tuberculosis treatment. Pre-emptive administration of corticosteroids may be beneficial for preventing such stricture in patients with a pre-existing ureteral lesion.

Key words: urinary tuberculosis, ureteral stricture, corticosteroid

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Introduction

The urinary system is a relatively uncommon site of tuberculosis, accounting for 1-12% of all of the extrapulmonary tuberculosis cases in Europe and 1.9% in Japan (1, 2). Ureteral stricture is a major complication of urinary tuberculosis, occurring in more than 50% of these patients (3). The guidelines of the European Association of Urology state that corticosteroids can be added to anti-tuberculosis chemotherapy if the ureteral stricture doesn't improve within three weeks (4). However, evidence supporting the effectiveness of corticosteroids in this patient population is limited. We herein report a case where the initiation of anti-tuberculosis drugs acutely worsened ureteral constriction, resulting in the need for emergent catheterization due to anuria, which was mitigated after the administration of steroids.

Case Report

A 77-year-old Japanese man with a 1-month history of low back pain and 2 days of fever was referred to the department of infectious diseases at our hospital because his urine smear was positive for acid-fast bacilli (AFB). Ten months prior to this referral, the patient had experienced dysuria, which was easily relieved by the administration of tamsulosin. The patient was otherwise healthy and was independent in his activities of daily life. A physical examination showed that the patient was 156 cm tall and weighed 47.6 kg, thin but with a good general appearance, and had a body temperature of 37.2℃ and stable vital signs. A lower back examination revealed knocking pain on the left costovertebral angle. The laboratory test results showed him to have leucopenia (white blood cell count 2,500/μL), mild anemia (hemoglobin 11.4 g/dL), renal dysfunction (serum creatinine 1.54 mg/dL), and mild inflammatory response (C reactive protein 5.02 mg/dL and erythrocyte sedimentation rate 31 mm/hr). A urinalysis revealed sterile pyuria, and a polymerase chain reaction (PCR) test for *Mycobacterium tuberculosis* showed positive findings in the urine as well as gastric aspirate samples. A sputum smear was negative for AFB. Drug-sensitive *M. tuberculosis* was isolated from both the urine and sputum samples. Chest X-rays showed multiple nodular lesions on the left lung field (Fig. 1). Chest

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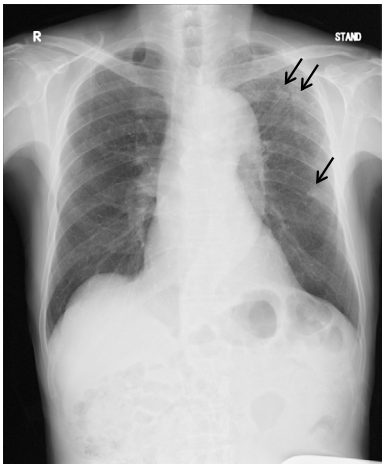

Figure 1. Chest X-ray on the day of admission. Chest X-ray showed nodular lesions (arrows) in the left lung field.

computed tomography (CT) showed multiple centrilobular nodules (Fig. 2a), and abdominal CT showed bilateral hydronephrosis: dilatation of both ureters; severe cortical thinning of the right kidney, indicating a complete loss of function; and calcification of the bladder wall (Fig. 2b-d).

The patient was diagnosed with both urinary and pulmonary tuberculosis. Anti-tuberculosis treatment with isoniazid 300 mg/day, rifampicin 450 mg/day, ethambutol 750 mg/day, and pyrazinamide 1.2 g/day was initiated from the day following admission. On the 6th hospital day, the patient developed anuria with a fever of 39℃, and his serum creatinine levels rose to 4.21 mg/dL (Fig. 3a). We attempted to insert a double J stent into the left ureter, but were unsuccessful. A smaller-bore catheter was therefore placed to relieve the obstruction, but it could not reach the pelvis of the left kidney (Fig. 3b).

Since no signs of improvement in the ureteral stricture were noted during the 3 weeks of anti-tuberculosis treatment, we started the patient on 60 mg/day oral prednisolone on the 27th hospital day. Two days later, urine began leaking from both the catheter in the left ureter as well as in the bladder. This indicated improvement in the ureteral stricture (Fig. 3c). Subsequently, we removed the ureteral catheter 8 days after the initiation of prednisolone. Prednisolone was continued for a further 2 weeks before being tapered to 10 mg/week. Although the patient complained of frequent urination, probably due to a shrunk bladder, there was no sign of re-occlusion thereafter (Fig. 3d). Anti-tuberculosis treatment was continued for nine months in total.

Discussion

Urinary tuberculosis is caused by the spread of *M. tuberculosis* through the blood stream from a primary pulmonary infection (4). Because a diagnosis is often delayed and the narrow lumen of the ureteral tract is anatomically vulnerable to obstruction, a majority of patients present with urinary tract complications, such as a non-functioning hydronephrotic kidney, ureteral stricture, and a shrunk bladder (5, 6). Surgery is reportedly required in more than half of urinary tuberculosis patients (7). A controlled trial showed that patients who underwent early ureteral stenting or percutaneous nephrostomy had a better chance of avoiding nephrectomy than those in whom stenting or nephrostomy was delayed (8). However, such invasive treatment is undesirable and often problematic, especially among elderly patients.

Several studies have shown that the administration of corticosteroids improves outcome of certain types of extrapulmonary tuberculosis, such as tuberculous meningitis and tuberculous pericarditis (9). However, to our knowledge, only two published observational studies have described the potential effects of corticosteroids in treating ureteral stricture in cases of urinary tuberculosis. According to Horne's study, when 29 patients with ureteral stricture were treated with additional corticosteroids, 21 (72%) did not require any surgical intervention (10). Bennai et al. treated 16 tuberculous ureteric stricture patients with concomitant use of corticosteroids, and 8 did not require urological intervention (11). The European Association of Urology guidelines suggest the use of corticosteroids only in cases where ureteral stricture does not improve within three weeks (4). However, we believe that earlier administration of corticosteroids can be considered as a treatment option to prevent the progression of ureteral stricture after the initiation of anti-tuberculosis treatment. Furthermore, no previous reports have so far described any details regarding the dose or duration of corticosteroid administration for urinary tuberculosis. Findings from more case reports on the successful use of combined corticosteroid treatment will therefore be of great value.

Patients with urinary tract tuberculosis are at high risk of developing anuria during anti-tuberculosis treatment, especially if they have a history of urinary obstruction and bilateral dilated ureter, as we experienced in the presented case. In a previously published report, of 37 urinary tuberculosis cases without urinary obstruction at the start of treatment, 6 developed ureteric obstruction during treatment (10). An as-yet unknown immune-pathological mechanism may be involved in ureteral stricture. Paradoxical reactions frequently occur from a few days to a month after initiation of anti-tuberculosis drug administration and are believed to be induced by antigen release from the destroyed bacterial bodies (12, 13). Similar immune responses may have worsened the pre-existing stricture in the present case, resulting in postrenal acute renal failure. Careful evaluation of the urinary tract is always necessary when urinary tuberculosis patients start receiving anti-tuberculosis treatment.

In conclusion, in cases of urinary tuberculosis, ureteral stricture can deteriorate during anti-tuberculosis treatment. In the present case, ureteral stricture was relieved drastically after starting an oral corticosteroid regimen. Pre-emptive administration of corticosteroids may be effective in preventing further deterioration of the ureteral stricture in patients with a pre-existing ureteral lesion.

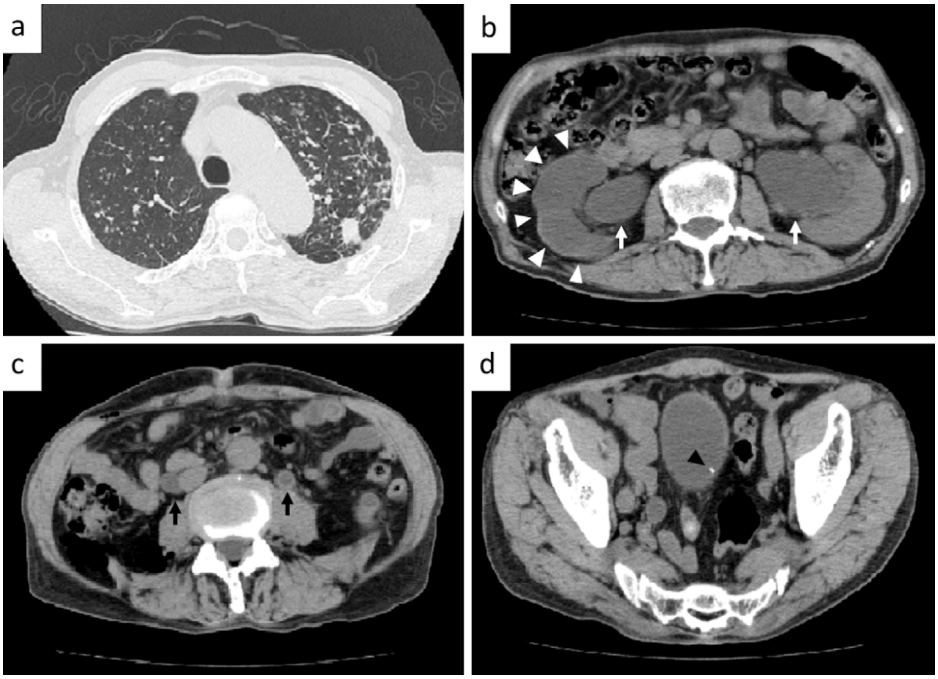

Figure 2. **Chest and abdominal CT on admission day. Chest CT (a) showed multiple centrilobular nodules. Abdominal CT showed bilateral hydronephrosis (white arrows), cortical thinning of the right kidney (white triangles) (b), dilatation of both ureters (black arrows) (c), and the calcification of the bladder wall (black triangle) (d).**

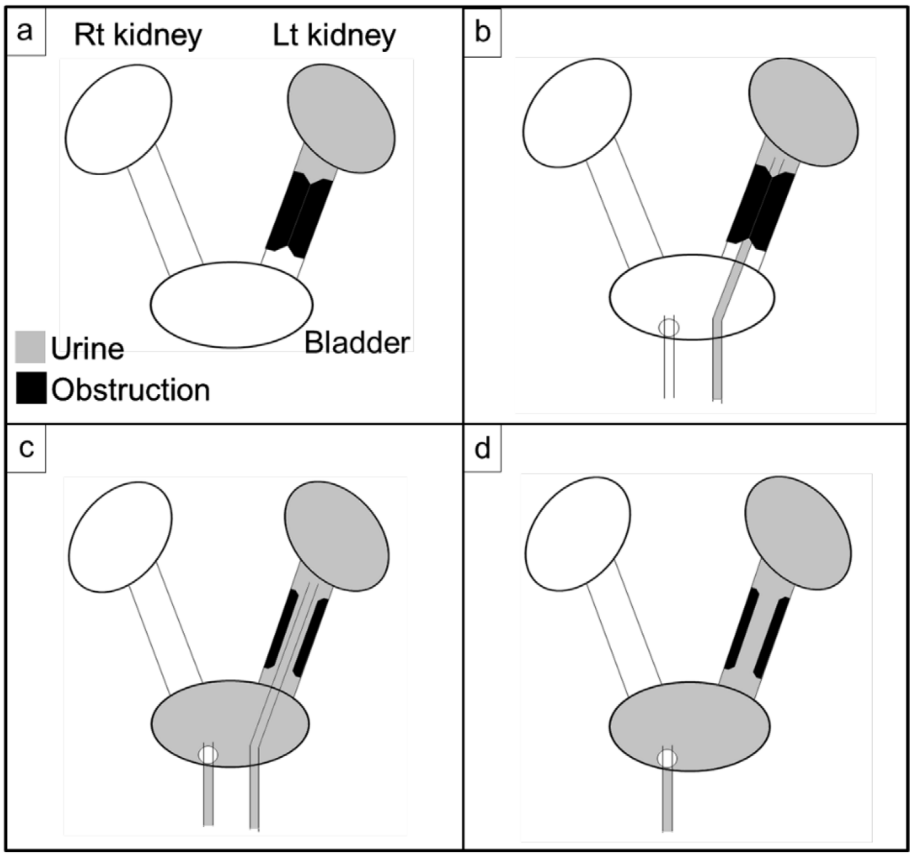

Figure 3. **Schematic illustration of urinary tract. (a) On Day 6, the patient suddenly developed anuria due to the deterioration of the left ureteral stricture. (b) A smaller-bore catheter was inserted into the middle of the left ureter, resolving the postrenal acute renal failure. However, no urine output was recognized from the balloon catheter. (c) On Day 27, corticosteroid administration was initiated. Urine started leaking from the balloon catheter, indicating the improvement of the ureteral stricture. (d) On Day 35, the ureteral catheter was removed.**

The authors state that they have no Conflict of Interest (COI).

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