Case Report
Systemic lupus erythematosus accompanying with renal tuberculosis: a case report

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Abstract: A 26-year-old woman, with a six-year history of well-controlled systemic lupus erythematosus (SLE), complained of urinary frequency and urgency. After failure of commonly-used antibiotic therapy, mycobacterium tuberculosis was cultured from her urine and renal tuberculosis (TB) was diagnosed. However, she underwent right nephrectomy after the combination therapies of prednisone for SLE and anti-tuberculosis treatment for renal TB failed. To our knowledge, SLE accompanying renal TB is rare, and such a rapid deterioration in renal function has never been reported.

Keywords: Systemic lupus erythematosus, renal tuberculosis, non-functioning kidney

Introduction
Patients with SLE are prone to infections, either because of intrinsic immunologic deficits or therapy with immunosuppressive agents [1]. The prevalence of tuberculosis (TB), ranging from 9.3% to 20.0%, is higher for patients with SLE compared with general population [2]. Furthermore, the genitourinary system, following the pleural and lymphatic systems, is the third most frequent extra-pulmonary location of TB, in which the kidney is the most affected site [3]. Here we describe a rare patient simultaneously suffering from SLE and renal TB and analyze the characteristics of SLE accompanied by renal TB.

Case
A 26-year-old woman, diagnosed with SLE six years ago, was well-controlled with oral prednisone (10 mg/day). However, two years ago, she was hospitalized with a one-month history of urinary frequency and urgency. Urinalysis showed pyuria and her symptoms were not relieved with the usual antibiotic administration. Following Ziehl-Neelsen stain was positive for acid-fast bacilli and mycobacterium tuberculosis was cultured from the urine. Urinary tract TB was diagnosed and anti-tuberculosis therapy with oral isoniazid, rifapentine, ethambutol, and pyrazinamide was initiated.

Although her physical condition improved, pyuria and microscopic hematuria were detected occasionally on her regular follow-up. One year ago, decreasing renal function in the right kidney made further treatment necessary. On admission, the patient did not have malaise, and physical examination revealed no tenderness or percussion tenderness in renal region. On laboratory test for SLE, antinuclear antibodies were positive (1:100, homogeneous pattern) and anti-dsDNA antibodies were negative, while serum C3 was low and C4 was within reference range. Other investigations were normal, with blood urea nitrogen of 4.17 mmol/L, serum creatinine of 90.0 umol/L, and negative for urinalysis and urine culture. Abdominal enhanced computed tomography (CT) revealed that: 1) parenchyma of right renal was thinned out and weakly enhanced, with multiple cystic transformations (Figure 1); 2) right ureter was dilated, with wall thickening (Figure 2). Radionuclide renal dynamic imaging revealed severely impaired right-sided renal function (GFR = 10.8 ml/min) (Figure 3).

Combined with her medical history, diagnoses of right-sided non-functioning kidney, right renal
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TB, and SLE were established, and laparoscope nephrectomy was performed. Pathological examination showed: 1) granulomatous inflammation, Ziehl-Neelsen stain was negative and mycobacterium tuberculosis DNA fragments were detected in TB-PCR; 2) renal parenchyma was atrophy; 3) partial glomerulus was sclerosis.

To prevent triggering of SLE activity and dissemination of TB, 100 mg of hydrocortisone succinate and 0.3 mg of isoniazid were intravenously administered once a day, for the day of operation and the following two days. Then a combination of prednisone and anti-tuberculosis medication were administered orally for maintenance therapy for one year. On her last visit, except for prescription of oral prednisone, she was in good condition and all the laboratory tests were in normal range.

Discussion

Like other immunocompromised subjects, characteristics of TB in SLE patients were: higher incidence rate; more frequent extra-pulmonary involvement; and more extensive pulmonary involvement [4]. Actually, renal TB is an uncommon cause of renal failure, which is characterized by potentially preventable and treatable; however, management became challenging if TB encountered with SLE [5].

The diagnosis of renal TB is often delayed as myriad clinical and radiological manifestations. Many symptoms are similar to that of conventional bacterial cystitis and suspicion is aroused only when there is no response to antibiotics [6]. Besides, renal TB and SLE interact through complicated ways, and both usually presents with manifestations of persistent fever, night sweat, etc., all of which make identification of renal TB in a SLE patient challenging [6].

For renal TB, sensitivities of 42.7% and 69.1% were demonstrated by detection of acid-fast bacilli in urinalysis and intravenous urography, respectively [7]. CT, with advantages of showing renal parenchymal cavity, mass and scarring, local parenchymal thinning, and strictures of infundibula [8], is the mainstay for investigating urinary TB, and a sensitivity of 84.3% has been reported [7].

For classical detection methods, Tuberculin Skin Testing (TST) is limited by low sensitivity in patients with an immunocompromised condition and low specificity due to cross-reactivity. Interferon-gamma Release Assays (IGRAs), with advantages of repeatability without booster effect and being rarely influenced by prior BCG (Bacillus Calmette-Guerin) vaccinations, are superior to TST for immunosuppressed conditions [9]. However, the actual diagnosis would be difficult and challenging, therefore, a dozen of clinical suspicions could be relatively important, particularly for those with intrinsic immunologic defects and immunosuppressive therapies.

Combination therapy is recommended for the treatment of TB accompanied with SLE [9]. Our patient suffered from renal TB after a well-controlled history of SLE. Though diagnosis and anti-tuberculosis therapy initiation were prompt, her right kidney was destroyed within only one year period; to our knowledge, this has never been reported before.

To explain this, the following inferring must be taken into consideration. Firstly, SLE is characterized by immunologic and genetic alterations,
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Figure 3. Radionuclide renal dynamic imaging showed right severely impaired renal function (GFR = 10.8 ml/min).
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including deficit in complement, impaired phagocytosis, and functional deficits in T-cell activity [6]. Secondly, immunosuppressive agents, such as azathioprine, cyclophosphamide, or mycophenolate, decrease T and B lymphocytes counts, and the widespread use of corticosteroids blocks T-cell proliferation and antigen-specific immune response, which producing significant impairment of cellular immunity [6]. Both increase the susceptibility to infection of mycobacterium tuberculosis [6]. Furthermore, no matter it is useful, isoniazid prophylaxis (INHP) has been recommended for SLE patients receiving long-term steroid treatment, and propagating INH resistance cannot be excluded [10].

At present, the incidence of extra-pulmonary TB is increasing, mainly as a result of HIV infections, intrinsic immunologic defects, and increasing immunosuppressive therapies [3]. Besides, more patients show serious drug resistance, which undoubtedly make treatment more difficult [3]. Therefore, SLE should be controlled in an advisable immunosuppressive dose, and the strategy of directly observed treatment short course (DOTS) is recommended for all anti-tubercular therapy [10].

Conclusion

TB accompanying with SLE is rare, and such a rapid disease progression, as seen in this patient, has never been reported to our knowledge. As clinical features and laboratory investigations can coexist in both diseases and the presentation may be variable. Treatment remains difficult and no clear guidelines have been summarized for the management of TB accompanying with SLE. Therefore, in some degree, a sensible immunosuppressive therapy and early diagnosis of TB in SLE are critical.

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Disclosure of conflict of interest

None.

Abbreviations

SLE, Systemic lupus erythematosus; TB, tuberculosis.

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