Renal Parenchymal Malakoplakia Presenting as Acute Renal Failure in a Young Woman

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To the Editor: A 26-year-old woman was admitted to the hospital because of a 4-day history of fever (39.3°C), accompanied by red urine, urinary frequency, odynuria, urgency, bilateral flank pain, and 2 days of anuria. The patient had no medical history and never received medical examinations. In the recent 1 month, she felt exhausted due to patient's care. On physical examination, temperature was 39°C. There was percussion pain in bilateral renal regions. Laboratory tests showed that the white blood cell (WBC) count was 21.13 × 10^9/L with 87.7% neutrophils. The serum creatinine (SCr) was increased to 706.5 μmol/L. Serum glucose was 27.5 mmol/L and glycosylated hemoglobin was 8%. Routine urinalysis showed protein (++), glucose (+++), acetone (+), and red blood cell (RBC) full/high power field (HPF) with the normal ones were predominant and WBC 10–20/HPF. Urinary protein was 819.9 mg/d. Urine culture was negative (urine examination after antibiotic treatment). Blood culture grew *Escherichia coli*.

Renal ultrasonography revealed the left kidney of 15.8 cm × 6.1 cm × 6.9 cm and the right kidney of 12.9 cm × 6.5 cm × 7.1 cm. A computerized tomography (CT) scan showed that both kidneys, especially the left kidney, were enlarged and hypoperfused, and there were multiple flaky low-density shadows in the left kidney.

The first renal biopsy was performed. No positive findings were found by immunofluorescence staining. Under light microscopy, no positive findings were found in glomeruli. Renal interstitium was massively expanded by a cellular infiltration, including some foamy eosinophilic macrophages (von Hansemann cells). A few of round cytoplasmic periodic acid–Schiff-positive basophilic Michaelis–Gutmann (M-G) bodies were observed [Figure 1]. Under electron microscopy, the interstitium was infiltrated by ample phagocytes containing membranous phagolysosome.

The patient was treated with insulin and intravenous antibiotics (7-day meropenem followed by levofloxacin). After 7-day antibiotic therapy, the body temperature returned to normal. SCr decreased to 84.7 μmol/L and fasting sugar to 5.2 mmol/L after 2 weeks treatment.

The patient remained on antibiotic therapy with levofloxacin. The second renal biopsy was done 8 weeks later. Compared to the first pathological results, the number of von Hansemann cells in the interstitium was decreased but inflammatory cell infiltration in the interstitium and tubular injury did not improve significantly [Figure 1]. Laboratory tests showed SCr 86.9 μmol/L; urinalysis showed protein (−), glucose (−), RBC 0–2/HPF, and WBC 4–12/HPF. Renal ultrasonography revealed shrinkage of bilateral kidneys (the left kidney 12.9 cm × 4.5 cm × 4.8 cm and the right kidney 11.2 cm × 5.0 cm × 4.5 cm). Advanced CT revealed

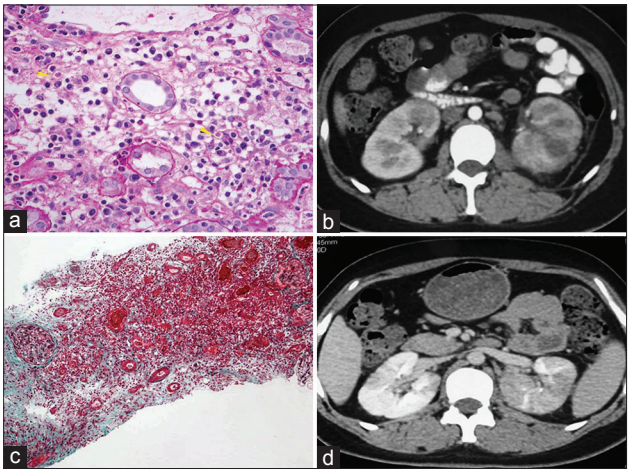

Figure 1: (a) Photomicrograph of the first renal biopsy. Kidney biopsy specimen shows diffuse interstitial inflammation and Michaelis–Gutmann bodies (arrows) (periodic acid–Schiff staining, Original magnification ×400). (b) Abdominal computerized tomography shows bilaterally enlarged kidney with multiple flaky low-density shadows. (c) Photomicrograph of the second renal biopsy showing renal interstitium is massively expanded by a large number of inflammatory cell infiltration (Masson staining, Original magnification ×100). (d) Follow-up advanced computerized tomography after 8 weeks of antibiotic therapy shows that the kidneys are smaller in size and the left kidney is more hypoperfused than the right kidney.

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that the kidneys were still enlarged but shrank than before, with multiple flaky low-density areas in the parenchyma.

In view of these inflammatory changes in renal interstitium, prednisone (30 mg/d) and cyclophosphamide (50 mg/d) were commenced and reduced gradually for 6 months. Levofloxacin treatment lasted for 6 months simultaneously. Her renal function has remained normal at routine follow-up for 17 months.

Renal parenchymal malakoplakia is a rare cause of acute renal failure. It is usually associated with an immunocompromised status such as diabetes, alcohol abuse, and immunosuppression. Renal manifestations included flank pain, hematuria, repeated urinary infection and protein, white cells and red cells in the urine, and with or without renal dysfunction. Under CT images, the kidney is enlarged and renal parenchyma had inhomogeneous low-density masses.[1]

Histopathologically, it is characterized by von Hansemann cell and M-G bodies. Under electron microscopy, there are different stages of phagolysosomes and bacterial fragments in foamy macrophages.[2]

Currently, malakoplakia is thought to be associated with infection. *E. coli* is the most common. Therefore, agents targeting Gram-negative bacteria with high bioavailability in macrophages are most commonly chosen, such as quinolones and sulfamido groups.

Some scholars pointed out that in malakoplakia, macrophage dysfunction and persistent antigens within cells cause progressive delivery of cytokines, resulting in renal inflammation and further injury.[3] If the early interstitial injury is not controlled, renal injury continues to deteriorate and even processes to interstitial fibrosis even though bacteria are removed.[4] To date, there are only very few case reports concerning antibiotics plus corticosteroids for treatment of renal malakoplakia.[5] In the present case, although renal function was recovered after antibiotics treatment, repeated renal biopsy revealed significant infiltration of inflammatory cells in renal interstitium. Therefore, corticosteroids and cyclophosphamide were given to inhibit interstitial inflammation and improve the prognosis. The patient's renal function has remained normal at routine follow-up. Suppression of the inflammation with immunomodulators would, therefore, seem logical and is not contrary to the present treatments, emphasizing the differences in pathogenesis outlined above.

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Conflicts of interest

There are no conflicts of interest.

References

1. Kobayashi A, Utsunomiya Y, Kono M, Ito Y, Yamamoto I, Osaka N, *et al.* Malakoplakia of the kidney. Am J Kidney Dis 2008;51:326-30. doi: 10.1053/j.ajkd.2007.08.029.
2. Keitel E, Pêgas KL, do Nascimento Bittar AE, dos Santos AF, da Cas Porto F, Cambruzzi E. Diffuse parenchymal form of malakoplakia in renal transplant recipient: A case report. Clin Nephrol 2014;81:435-9. doi: 10.5414/CN107506.
3. Hyun KH, Shin HD, Kim DH. Malakoplakia in a healthy young female patient. Korean J Intern Med 2013;28:475-80. doi: 10.3904/kjim.2013.28.4.475.
4. Tam VK, Kung WH, Li R, Chan KW. Renal parenchymal malacoplakia: A rare cause of ARF with a review of recent literature. Am J Kidney Dis 2003;41:E13-7. doi: 10.1016/S0272-6386(03)00367-6.
5. Mannan AA, Kahvic M, Singh NG, Abu Sara Y, Bharati C. An unusual case of extensive epididymotesticular malakoplakia in a diabetic patient. Int Urol Nephrol 2010;42:569-73. doi: 10.1007/s11255-009-9674-2.