American Journal of Infectious Diseases

Case Report

An Unusual Case of Leptospirosis Presenting as Glomerulonephritis With Crescents

Felipe Guedes Bezerra[1], Ana Flávia de Holanda Veloso[2], Yago Sucupira Amaral[1], Thiago de Paula Pessoa Franco Silva[2] and Anaiara Lucena Queiroz[1]

[1]Department of Nephrology, Walter Cantídio University Hospital, Ceara Federal University, Brazil
[2]Department of Clinical Medicine, Walter Cantídio University Hospital, Ceara Federal University, Brazil

Article history
Received: 14-05-2024
Revised: 27-05-2024
Accepted: 06-12-2024

Corresponding Author:
Felipe Guedes Bezerra
Department of Nephrology, Walter Cantídio University Hospital, Ceara Federal University, Brazil
Email: felipeguedesbezerra@gmail.com

Abstract: Leptospirosis is a zoonosis caused by the spirochete Leptospira, which commonly affects the kidneys. Varied renal findings, most commonly tubulointerstitial nephritis, are found in human kidney biopsies and in vivo models. Remarkably, glomeruli are usually spared in this disease. However, some cases report evidence of glomerular damage. We describe a case of leptospirosis with fibroepithelial crescents, a rare histopathological finding.

Keywords: Leptospirosis, Glomerulonephritis, Crescentic Glomerulonephritis, Fibroepithelial Crescents, Acute Kidney Injury (AKI)

Introduction

Leptospirosis is a common zoonosis caused by the spirochete Leptospira, first described by Adolf Weil in 1886. Animal or environmental exposure is the main mode of transmission to humans, primarily through contact with urine-contaminated soil or water with injured skin or mucous membranes. Risk factors for infection include practicing certain recreational activities, living in or traveling to flood-prone areas, and occupational exposure to infected animals (Muthuppalaniappan et al., 2018).

The spectrum of illness ranges from self-limited or asymptomatic infection to multisystem organ dysfunction and failure (Andrade et al., 2008).

We describe a case of leptospirosis with fibroepithelial crescents, a rare histopathological finding.

Case Presentation

A 23-year-old male event producer presented to the emergency department of a hospital in Manaus, in the Brazilian state of Amazonas, with a 7-day history of abdominal pain and watery diarrhea. He was previously well and claimed that he had landed in the city 10 days prior, but had never been to the Amazon region before. Furthermore, he had a past medical history of hospitalization during infancy for Henoch-Schonlein purpura, but abandoned follow-up after being discharged. He denied alcohol consumption, smoking, drug abuse, or drinking herbal teas. He denied having any allergies. After initial workup, he was discharged from the emergency department with a prescription for moxifloxacin and anthelmintic therapy. Subsequently, he reported resolution of the diarrhea and returned from Manaus to his hometown in Fortaleza, Brazil.

After 7 days, however, he presented to the emergency department in Fortaleza, reporting fever, hematuria, and bilateral calf tenderness. He had a normal urine output and denied jaundice, pale stools, respiratory symptoms, or bleeding. Physical examination was notable for pallor and mild abdominal tenderness, with no other findings. Initial laboratory exams revealed high creatinine, high liver transaminase and alkaline phosphatase levels, anemia, thrombocytopenia, and mild hypokalemia. Urinalysis confirmed hematuria (20 RBC/HPF) and proteinuria (1+), with a 24-hour urine protein test result of 270 mg/day.

Eventually, the patient was transferred to our hospital for the continuation of care in a specialized department. At admission, he was clinically well, had normal urine output, and had no need for renal replacement therapy.

An abdominal Doppler ultrasound was performed, showing mild liver and spleen enlargement. Aside from increased parenchymal echogenicity, renal ultrasound showed no remarkable findings.

A kidney biopsy was performed, revealing 19 glomeruli, three of which displayed global sclerosis. Crescents were observed in two glomeruli (Figure 1), and the interstitium displayed moderate tubulointerstitial nephritis, consisting of a mononuclear inflammatory infiltrate (Figure 2). Immunofluorescence was notable for granular C3 deposits (+3), IgA (+), IgM (+2), and IgG (+) on the capillary loops.

DOI: 10.3844/ajidsp.2026.1.3

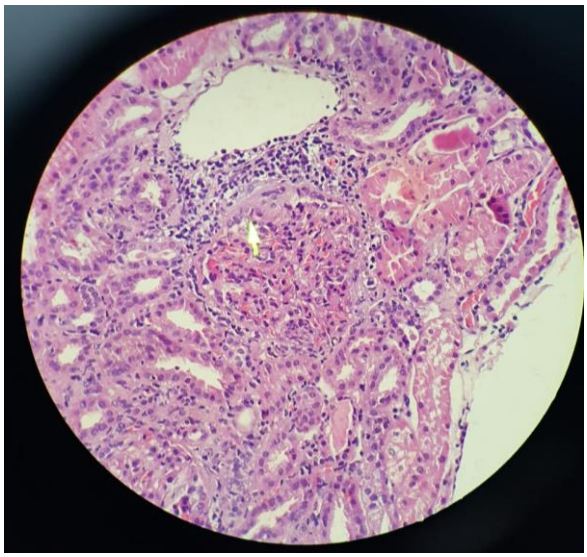

Fig. 1: Kidney biopsy examination. Light microscopy shows a fibroepithelial crescent in one glomerulus (green arrow)

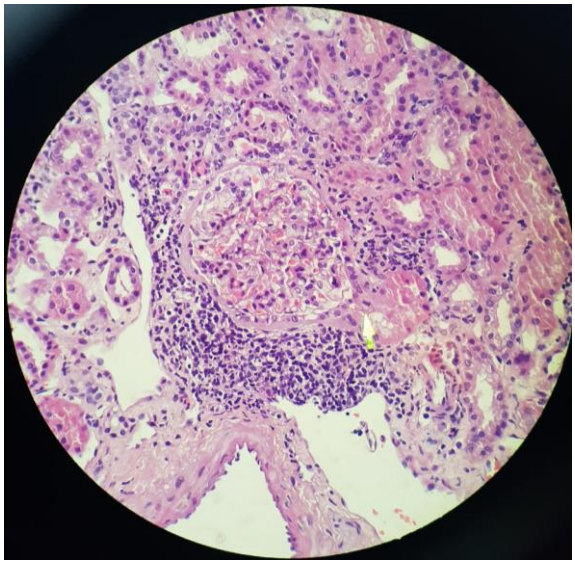

Fig. 2: Kidney biopsy examination. Light microscopy shows a glomerulus with mononuclear inflammatory infiltrate (green arrow)

Additionally, serological tests returned positive for leptospira immunoglobulin M, and microscopic agglutination testing confirmed the diagnosis. Tests for hepatitis B and C, human immunodeficiency virus, cytomegalovirus, toxoplasmosis, malaria, schistosomiasis, and yellow fever returned negative. Plasma C3 and C4 levels were normal. Testing for ANCA and ANA returned negative.

After 2 weeks of supportive care, serum creatinine and liver enzyme levels improved substantially. He was clinically well and asymptomatic when discharged, and had normal laboratory results on the follow-up visit 1 month later.

Discussion

Renal findings in leptospirosis are most commonly microscopic hematuria, proteinuria, and nonoliguric Acute Kidney Injury (AKI) (Andrade et al., 2008). Electrolyte disturbances, including hyponatremia, hypomagnesemia, and hypokalemia, may also occur because of proximal tubule injury (Geraldo et al., 2013; Seguro and Andrade, 2013). Approximately 10-85% of infected patients develop AKI. Among these patients, 30% require hemodialysis, and the mortality rate varies between 4-20% (Yang, 2016). A retrospective analysis revealed that 63.8% of the patients completely recovered renal function within 90 days, but 10.3% persisted with some degree of renal dysfunction (Covic, 2003).

In affected patients, renal histopathology most often shows tubulointerstitial nephritis in conjunction with lymphocyte infiltrates, tubular necrosis, and interstitial edema (Yang et al., 2001; Yang, 2007). However, glomeruli are usually spared in humans and in vivo models, mostly demonstrating areas of podocyte fusion and endothelial cell swelling (De Brito, 2018).

After hematogenous dissemination, Leptospira reaches the kidney and causes glomerular injury. On the second day, peritubular capillaritis is observed, followed by migration of the spirochete to the interstitium, resulting in interstitial swelling within seven days (Andrade et al., 2008). Geraldo et al. propose that leptospira migration through the capillary wall may cause transient mesangial cell proliferation (Geraldo et al., 2013; De Brito, 2018). which is supported by Silva et al. in a case report of mesangial proliferative glomerulonephritis (Silva et al., 2016). In 2021, Arshad et al. observed cellular crescents in a young male with leptospirosis and AKI, followed by clinical improvement after antibiotic therapy and intravenous corticosteroids (Arshad and Chauhan, 2021).

The findings in our case are uncommon because the renal biopsy revealed fibroepithelial crescents in conjunction with tubulointerstitial inflammatory infiltrates. Additionally, immunofluorescence analysis suggested late postinfectious glomerulonephritis. As mentioned previously, our patient rapidly recovered renal function spontaneously without the need for therapy other than supportive care.

Conclusion

Recognizing leptospirosis is a challenge, especially for physicians in non-endemic areas. Considering the variety of clinical, laboratory, and histopathological findings, a high index of suspicion is required to diagnose this disease. Moreover, many aspects of renal involvement in Leptospira infections are not completely understood. Our case illustrates the presence of fibroepithelial crescents in the renal biopsy specimen of an infected patient, highlighting the importance of understanding this disease for future therapeutic and prognostic advancements.

Acknowledgment

Thank you to the publisher for their support in the publication of this research article. We are grateful for the resources and platform provided by the publisher, which have enabled us to share our findings with a wider audience. We appreciate the efforts of the editorial team in reviewing and editing our work, and we are thankful for the opportunity to contribute to the field of research through this publication.

Funding Information

The authors have not received any financial support or funding to report.

Authors Contributions

Felipe Guedes Bezerra: Took care of the patient during his hospital stay and wrote the case description.

Ana Flávia de Holanda Veloso: Wrote the scientific part of the paper.

Yago Sucupira Amaral: Responsible for the literature research and management of the case.

Thiago de Paula Pessoa Franco Silva: Organized the present study and led the search of the Journal publication.

Anaiara Lucena Queiroz: Coordinator of the Nephrology Wing of the Teaching Hospital and attending physician of the patient.

Ethics

This article is original and contains unpublished material. The corresponding author confirms that all of the other authors have read and approved the manuscript and no ethical issues involved.

References

Andrade, L., de Francesco Daher, E., & Seguro, A. C. (2008). Leptospiral Nephropathy. *Seminars in Nephrology*, *28*(4), 383–394. https://doi.org/10.1016/j.semnephrol.2008.04.008

Arshad, A. R., & Chauhan, M. (2021). Leptospirosis with Crescentic Glomerulonephritis. *Journal of Pakistan Society Internal Medicine*, *2*(3), 247–249. https://doi.org/10.70302/jpsim.v2i3.2157

Covic, A., Goldsmith, D. J. A., Gusbeth-Tatomir, P., Seica, A., & Covic, M. (2003). A retrospective 5-year study in Moldova of acute renal failure due to leptospirosis: 58 cases and a review of the literature. *Nephrology Dialysis Transplantation*, *18*(6), 1128–1134. https://doi.org/10.1093/ndt/gfg095

De Brito, T., Silva, A. M. G. da, & Abreu, P. A. E. (2018). Pathology and pathogenesis of human leptospirosis: a commented review. *Revista Do Instituto de Medicina Tropical de São Paulo*, *60*, e23. https://doi.org/10.1590/s1678-9946201860023

Geraldo Bezerra da, S. J., & De Francesco, D. E. (2013). Tropical diseases-associated kidney injury. *Rev Da Soc Bras Clínica Médica*, *11*(2), 155–164.

Muthuppalaniappan, V. M., Rajakariar, R., & Blunden, M. J. (2018). Leptospirosis presenting as haemolytic uraemic syndrome: a case report. *BMC Nephrology*, *19*(1), 20. https://doi.org/10.1186/s12882-018-0817-5

Seguro, A. C., & Andrade, L. (2013). Pathophysiology of Leptospirosis. *Shock*, *39*(Supplements), 17–23. https://doi.org/10.1097/shk.0b013e31828fae49

Silva, F., Brandão, M., & Esteves, A. (2016). A Rare Presentation of Leptospirosis. *European Journal of Case Reports in Internal Medicine*, *3*(6), 000447. https://doi.org/10.12890/2016_000447

Yang, C., Wu, M., & Pan, M. (2001). Leptospirosis renal disease. *Nephrology Dialysis Transplantation*, *16*(suppl 5), 73–77. https://doi.org/10.1093/ndt/16.suppl_5.73

Yang, C.-W. (2007). Leptospirosis renal disease: Understanding the initiation by Toll-like receptors. *Kidney International*, *72*(8), 918–925. https://doi.org/10.1038/sj.ki.5002393

Yang, L. (2016). Acute Kidney Injury in Asia. *Kidney Dis*, *2*(3), 95–102. https://doi.org/10.1159/000441887