Arch Iran Med. April 2026;29(4): 260-263

doi:10.34172/aim.35173

http://journalaim.com

ARCHIVES OF IRANIAN MEDICINE

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Case Report

Glomerulonephritis Following Visceral Leishmaniasis; A Case Report

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Abstract

Visceral leishmaniosis (VL) is an endemic systemic infection in various parts of Iran. Renal failure is often seen in VL which is usually mild and related to interstitial nephritis, as glomerular involvement is rarely found. This study reports a case of a 20-year-old Iranian male who presented with myalgia, arthralgia, chills, cough, and a significant weight loss of 20 kg over the past two months. In addition, the patient reported a past history of ITP from two months ago which was treated with prednisolone 35 mg daily & a history of animal contact (cat). On physical examination, he had petechiae, purpura over the limbs and trunk, and a significantly enlarged palpable spleen. On laboratory evaluations, we found a platelet count of 6000/μL, impaired liver function tests, serum creatinine level of 1.5 mg/dL, blood urea nitrogen level of 21 mg/dL, proteinuria on 24-hour urine collection analysis, positive results for antinuclear antibodies, and a low level of C3 complement. Microscopic examination of PBS demonstrated thrombocytopenia, no schistocyte and the identification of Leishman bodies was positive. Bone marrow biopsy showed intracellular Leishman parasites. After correction of thrombocytopenia, a kidney biopsy was performed that revealed mesangial proliferative glomerulonephritis. Accordingly, the patient was treated with liposomal amphotericin B for 21 days, following which his condition improved significantly. This case report discusses the association between glomerulonephritis and VL and focuses on endemic infections as a potential underlying cause of manifested renal disease.

Keywords: Glomerulonephritis, Kala-azar, Mesangial proliferative glomerulonephritis, Visceral leishmaniosis

Cite this article as: Yaghoubi F, Dalil D, Tavakoli F, Mirzaian E, Iranzadeh S. Glomerulonephritis following visceral leishmaniasis; a case report. Arch Iran Med 2026;29(4): 260-263. doi:10.34172/aim.35173

Received: September 26, 2025, **Revised:** February 20, 2026, **Accepted:** February 28, 2026, **ePublished:** April 1, 2026

Introduction

Visceral leishmaniasis (VL) is an endemic systemic infection in various parts of Iran. More than 20 species of Leishmania are responsible for the 3 main clinical syndromes of cutaneous leishmaniasis, mucocutaneous leishmaniasis and VL. Iran has a subtropical climate that provides optimal conditions for the survival and reproduction of the mosquito and many other arthropod vectors. Three forms of leishmaniasis have been identified in Iran. The first recorded case of VL in Iran was reported from the Mazandaran province in 1949, and then other geographical areas were reported based on microscopic or molecular confirmation.[1] In Iran and throughout the Middle East, *L. infantum* is the main cause of VL and usually infects children in endemic areas where dogs (*Canis familiaris*) are considered the primary reservoir of the disease.[2]

This life-threatening vector-borne parasitic disease can present with several clinical presentations, from asymptomatic to severe forms. Classically, it is characterized by intermittent fever, general discomfort, weight loss, and hepatosplenomegaly. However, clinical features may not be readily apparent due to multi-organ involvement. This in turn can make it difficult to make a correct diagnosis and subsequently manage the patient.

VL can significantly affect kidney function, potentially leading to increased complications and mortality. Affected individuals may experience proteinuria, hematuria, leukocyturia, and occasionally suffer from acute kidney injury. Generally, kidney involvement is not severe and resolves as the infection is treated.[3] The most frequently observed tissue change in affected kidneys is interstitial nephritis. Although rare, there have been instances of proliferative glomerulonephritis reported in some cases.[4] Here, the authors present a case of VL in a 20-year-old Iranian male patient with glomerulonephritis.

Case Report

A 20-year-old Iranian male presented to Shariati Hospital, Tehran, Iran, with chief complaints of myalgia, arthralgia, chills, and cough. His medical history was significant for immune thrombocytopenic purpura (ITP), which was diagnosed two months previously. Following treatment, the patient was discharged with a platelet count of 110,000/μL and daily administration of 35 mg prednisolone. The patient was residing in Tehran, Iran, and reported keeping a domestic cat. He also reported recent travel to northern regions of Iran, areas recognized as endemic for VL.

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Moreover, the patient reported a significant weight loss of 20 kg over the past two months.

On physical examination, the patient was conscious, alert, and oriented toward time, place, and person. His vital signs were as follows: body temperature, 38.8°C; respiratory rate, 26 breaths/min; pulse rate, 95 beats/min; blood pressure, 120/80 mmHg; and O_2 saturation, 90% on room air. He had petechiae and purpura over the limbs and trunk along with a significantly enlarged palpable spleen. Fine crackles were detected on pulmonary auscultation. The rest of the physical examinations yielded results that were within the normal limits.

Initial blood tests revealed a white blood cell (WBC) count of 1800/μL, platelet count of 6000/μL, impaired liver function test (LFT) results, and elevated inflammatory marker levels. Table 1 presents all the laboratory findings on patient admission. Urine analysis revealed 2+ proteinuria, 0-1 WBCs/high-power field, and 0-1 RBCs/high-power field with no nitrites. Blood and urine cultures were negative. The RT-PCR test result for COVID-19 was positive. Sepsis evaluation was also performed.

Serological tests for viral hepatitis, cytomegalovirus, human immunodeficiency virus (HIV), Toxoplasma, and Brucella were negative. The interferon-gamma release assay (IGRA) was negative for *Mycobacterium tuberculosis* infection. The malaria smear test result was negative. Further laboratory investigations revealed positive results for the antinuclear antibodies (ANA) test, a low level of C3 complement, and negative results for other serological tests of autoimmune diseases.

Chest CT scan revealed diffuse bilateral interstitial reticulonodular infiltrations. Abdominopelvic ultrasound revealed moderate splenomegaly, measuring 80 × 190 mm, while the renal dimensions appeared within the normal range. Echocardiography revealed an ejection fraction of 55%, moderate pericardial effusion, and absence of vegetation. The patient was initiated on a three-day course of intravenous methylprednisolone pulse therapy, followed by a five-day regimen of remdesivir, meropenem, and vancomycin.

Based on the CT scan report, bronchoscopy was performed and a bronchoalveolar lavage (BAL) specimen was sent to the laboratory. BAL culture was negative for microorganisms. Microscopic examination of peripheral blood smear (PBS) revealed thrombocytopenia and no schistocytes, and the identification of Leishman bodies was positive. Bone marrow biopsy (BMB) revealed hypercellular bone marrow with erythroid hyperplasia and megaloblastic erythroid precursors as well as Leishman bodies in one macrophage (Figure 1). Whole-body PET (positron emission tomography) scan findings were normal. Chromosomal analysis was normal, and genetic studies did not show mutations in BCR-ABL1 and JAK2-V617F.

The patient's symptoms, in conjunction with elevated ANA and low levels of C3 complement and proteinuria (24-hour urine collection demonstrated a volume of 5.2 L, protein excretion of 2390 mg, and creatinine excretion of 1989 mg), raised suspicion of systemic lupus erythematosus (SLE). However, renal biopsy was contraindicated because of thrombocytopenia. After an observed increase in the platelet count, renal biopsy revealed mesangial proliferative glomerulonephritis (Figure 2).

Table 1. Laboratory Findings on Patient Admission

Parameter	Result	Reference	Unit	Parameter	Result	Reference	Unit
WBC	1.8	4.1–10.1	1000/μL	Cr	1.5	0.7-1.3	mg/dL
Hb	11.5	12–16	g/dL	BUN	21	7-18.6	mg/dL
Platelet	6	150–400	1000 /μL	Na	136	135-148	mEq/L
Reticulocyte	0.5	0.5- 2.5	%	K	4.4	3.5-5.5	mEq/L
INR	1.09	>1.1	-	Ca	6.6	8.6-10.2	mg/dL
AST	340	<31	U/L	P	3.7	2.5-4.5	mg/dL
ALT	126	<31	U/L	SI	74	60-70	μg/dL
Total protein	6.1	6.0-8.3	g/dL	TIBC	385	240-450	μg/dL
Albumin	3.1	3.4-5.4	g/dL	Vitamin B12	683	197-771	pg/mL
Total Bili	0.9	0.1–1.2	mg/dL	Folic acid	10.8	3.1-17.5	ng/ml
ALP	458	70–360	U/L	ESR	40	<15	mm/h
ACE	211	13.3-63.9	mg/L	CRP	115	<6.0	mg/L
ANA	1:160	<1:80	-	C3	69	90-180	mg/dL
Anti-ds-DNA	33.6	<50	IU/mL	C4	30	10-40	mg/dL
Urine analysis					**24-hour urine collection analysis**		
SG	1.018	RBC	0-1/hpf	**Parameter**	**Result**	**Reference**	**Unit**
Protein	2+	WBC	0-1/hpf	Volume	5200	800-2000	mL
Glucose	Negative	Bacteria	Not seen	Protein	2390	<150	mg
Nitrite	Negative	Epithelial cell	Not seen	Cr	1989	950-3000	mg

WBC: White Blood Cell, Hb: Hemoglobin, INR: International Normalized Ratio, AST: Aspartate Aminotransferase, ALT: Alanine Transaminase, Bili: Bilirubin, ALP: Alkaline Phosphatase, ACE: Angiotensin-Converting Enzyme, ANA: Antinuclear Antibodies, Cr: Creatinine, BUN: Blood Urea Nitrogen, SI: Serum Iron, TIBC: Total Iron Binding Capacity, C3: Complement Component 3, C4: Complement Component 4, SG: Specific Gravity.

Based on these findings, the patient was treated with a weekly dose of 4 mg/kg liposomal amphotericin B for three weeks, followed by oral voriconazole 200 mg twice daily. After 2 weeks, the patient exhibited notable clinical improvement, accompanied by favorable shifts in laboratory parameters. Following completion of the anti-leishmania regimen, all test results returned to the normal range.

Discussion

VL, a long-term parasitic infection, has been recognized as an opportunistic microbial infection and is commonly observed in HIV patients, [5,6] bone marrow transplant recipients, and individuals undergoing solid-organ transplants, including kidney transplants.[7] In other words, immunosuppressed states may accelerate VL progression and lead to severe complications.[8] The immune response against Leishmania primarily involves CD4+ T helper cells, interferon γ, dendritic cells, and macrophages. Research indicates that VL infection may trigger T helper 2 lymphocytes to produce interleukins (IL)-4 and IL-10. These interleukins are thought to stimulate B cells, resulting in the production of a diverse range of antibodies linked to glomerular damage.[4,9] The resulting antibodies could potentially cause glomerular injury through either the formation of immune complexes within the glomeruli or by directly depositing in the tissue. Immunosuppressive treatments that interfere with these components may facilitate VL development.[10] The immunosuppressive drugs administered to the patient in this case clearly contributed to his susceptibility to opportunistic infections.

VL manifests in various ways, and kidney involvement can be a significant complication of VL, increasing the morbidity and mortality.[8] Renal damage in VL can affect interstitial, glomerular, and vascular structures;[11] cause interstitial nephritis;[11,12] glomerular sclerosis;[13] and membranoproliferative glomerulonephritis,[8,11-13] potentially resulting in nephritic syndrome or acute/chronic renal failure.[8] In immunocompromised individuals, direct parasitic invasion of the kidney tissue is particularly damaging. Previously, several cases of VL-associated renal damage have been reported both in immunocompetent and immunosuppressed patients.[14-17] The patient discussed in this case report had a history of travelling to a region where leishmaniasis is endemic. The authors suggest exploring potential links between VL and glomerulonephritis when investigating the underlying causes of the latter in similar cases.

VL can be diagnosed using various laboratory methods. These include identifying the parasite in affected tissue samples using light microscopy, culture techniques, or animal inoculation; detecting parasite DNA in tissue specimens; employing immunodiagnostic approaches such as identifying parasite antigens or antibodies; or assessing Leishmania-specific cell-mediated immune responses.[18] In our patient, fever, significant weight loss, animal contact, and clinical findings were suggestive of VL. Moreover, suspected VL was supported without the presence of any other infection, autoimmune disease, or underlying malignancy. The definite diagnosis of VL was established by identification of intracellular Leishman bodies (amastigotes) in bone marrow aspiration.

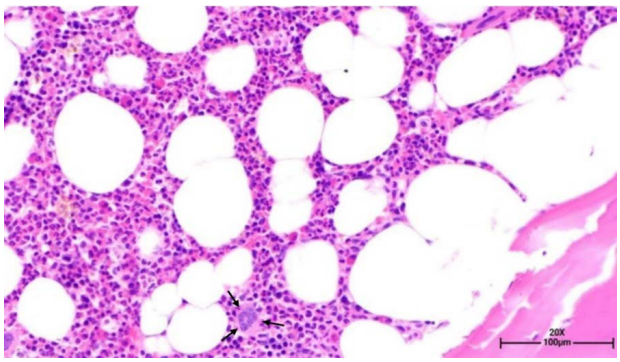

Figure 1. Microscopic Examination of Bone Marrow Biopsy Shows Mildly Hypercellular Marrow with Increased Number of Megakaryocytes and Leishman Bodies in One Macrophage (H&E, 200X).

Conclusion

To summarize, we underline the necessity of employing an approach based on etiology, rather than one focused on syndromes, when identifying new instances of glomerulonephritis to avoid inappropriate treatment. Infections that are endemic to certain areas can be the underlying cause of apparent kidney disorders. The researchers highlight the importance of performing tests to rule out infectious sources, especially parasitic infections in areas where they are common, such as schistosomiasis,

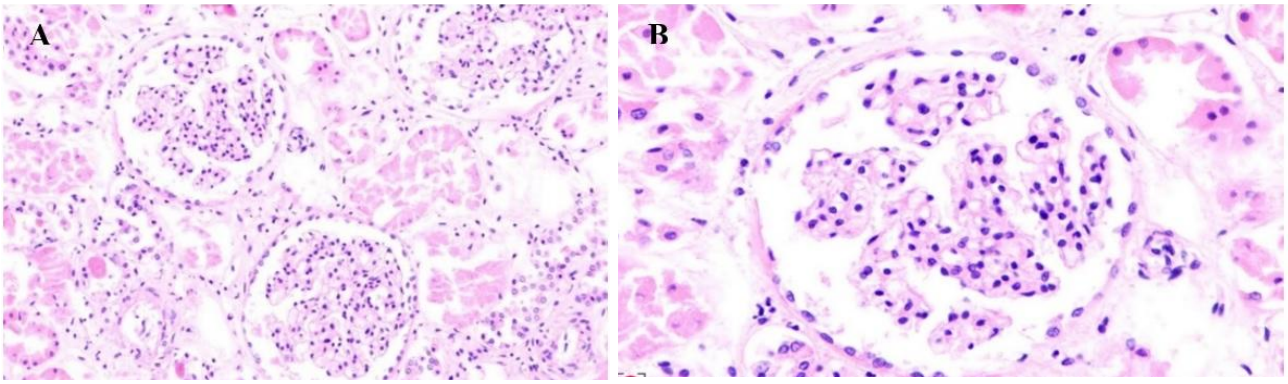

Figure 2. Glomeruli Show Mild Mesangial Matrix Expansion and Hypercellularity (H&E, **A:** 200X and **B:** 400X).

malaria, and leishmaniasis, when managing patients with glomerulonephritis.

Acknowledgments
The authors thank the patient and his family for their cooperation.

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Competing Interests
The authors declare that they have no conflicts of interest.

Ethical Approval
The Research Ethics Committee of Tehran University of Medical Sciences approved all procedures performed in the current study with the approval number: IR.TUMS.MEDICINE.REC.1403.310. The patient expressed gave his informed consent through a written form.

Funding
The authors received no financial support for this study.

References

1. Shirzadi MR, Esfahania SB, Mohebalia M, Ershadia MR, Gharachorlo F, Razavia MR, et al. Epidemiological status of leishmaniasis in the Islamic Republic of Iran, 1983-2012. East Mediterr Health J 2015;21(10):736-42. doi:10.26719/2015.21.10.736
2. Mohebali M, Edrissian G, Akhoundi B, Shirzadi M, Hassanpour G, Behkar A, et al. Visceral leishmaniasis in Iran: an update on epidemiological features from 2013 to 2022. Iran J Parasitol 2023;18(3):279-93. doi:10.18502/ijpa.v18i3.13751
3. Suankratay C, Suwanpimolkul G, Wilde H, Siriyasatien P. Autochthonous visceral leishmaniasis in a human immunodeficiency virus (HIV)-infected patient: the first in Thailand and review of the literature. Am J Trop Med Hyg 2010;82(1):4-8. doi:10.4269/ajtmh.2010.09-0434
4. Liberopoulos E, Kei A, Apostolou F, Elisaf M. Autoimmune manifestations in patients with visceral leishmaniasis. J Microbiol Immunol Infect 2013;46(4):302-5. doi:10.1016/j.jmii.2012.01.016
5. Fernández-Guerrero ML, Aguado JM, Buzón L, Barros C, Montalbán C, Martín T, et al. Visceral leishmaniasis in immunocompromised hosts. Am J Med 1987;83(6):1098-102. doi:10.1016/0002-9343(87)90948-x
6. Clevenbergh P, Okome MN, Benoit S, Bendini JC, De Salvador F, Elbeze M, et al. Acute renal failure as initial presentation of visceral leishmaniasis in an HIV-1-infected patient. Scand J Infect Dis 2002;34(7):546-7. doi:10.1080/00365540232020857
7. Simon I, Wissing KM, Del Marmol V, Antinori S, Remmelink M, Nilufer Broeders E, et al. Recurrent leishmaniasis in kidney transplant recipients: report of 2 cases and systematic review of the literature. Transpl Infect Dis 2011;13(4):397-406. doi:10.1111/j.1399-3062.2011.00598.x
8. da Silva Junior GB, Barros EJ, de Francesco Daher E. Kidney involvement in leishmaniasis--a review. Braz J Infect Dis 2014;18(4):434-40. doi:10.1016/j.bjid.2013.11.013
9. Goto H, das Graças Prianti M. Immunoactivation and immunopathogeny during active visceral leishmaniasis. Rev Inst Med Trop Sao Paulo 2009;51(5):241-6. doi:10.1590/s0036-46652009000500002
10. Bogdan C. Leishmaniasis in rheumatology, haematology and oncology: epidemiological, immunological and clinical aspects and caveats. Ann Rheum Dis 2012;71 Suppl 2: i60-6. doi:10.1136/annrheumdis-2011-200596
11. Ortiz M, Mon C, Herrero JC, Oliet A, Rodríguez I, Ortega O, et al. Glomerulonephritis and cryoglobulinemia: first manifestation of visceral leishmaniasis. Clin Nephrol 2015;83(6):370-7. doi:10.5414/cn108195
12. Duarte MI, Silva MR, Goto H, Nicodemo EL, Amato Neto V. Interstitial nephritis in human kala-azar. Trans R Soc Trop Med Hyg 1983;77(4):531-7. doi:10.1016/0035-9203(83)90131-1
13. Leblond V, Beaufils H, Ginsburg C, Bricaire F, Danis M, Deray G, et al. Collapsing focal segmental glomerulosclerosis associated with visceral leishmaniasis. Nephrol Dial Transplant 1994;9(9):1353. doi:10.1093/ndt/9.9.1353a
14. Efstratiadis G, Boura E, Giamalis P, Mandala E, Leontsini M, Tsiaousis G, et al. Renal involvement in a patient with visceral leishmaniasis. Nephrol Dial Transplant 2006;21(1):235-6. doi:10.1093/ndt/gfi157
15. Rollino C, Bellis D, Beltrame G, Basolo B, Montemagno A, Bucolo S, et al. Acute renal failure in leishmaniasis. Nephrol Dial Transplant 2003;18(9):1950-1. doi:10.1093/ndt/gfg267
16. Alex S, Criado C, Fernández-Guerrero ML, de Górgolas M, Petkov V, Garcia Perez A, et al. Nephrotic syndrome complicating chronic visceral leishmaniasis: re-emergence in patients with AIDS. Clin Nephrol 2008;70(1):65-8. doi:10.5414/cnp70065
17. Enríquez R, Sirvent AE, Padilla S, Toro P, Sánchez M, Millán I. Membranoproliferative glomerulonephritis due to visceral leishmaniasis in an HIV patient. Am J Case Rep 2015;16:8-11. doi:10.12659/ajcr.892641
18. Sundar S, Rai M. Laboratory diagnosis of visceral leishmaniasis. Clin Diagn Lab Immunol 2002;9(5):951-8. doi:10.1128/cdli.9.5.951-958.2002