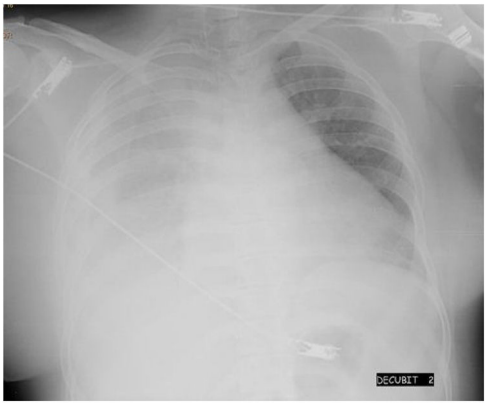

Figure 1 – Massive right pleural effusion.

pain and dyspnoea. We decided to definitively discontinue the PD.

Although there are numerous published cases of PRES in patients with renal failure, many of them on haemodialysis, few cases have been reported in patients on PD. Virtually all of them had seizures as an initial symptom, and volume overload and poor compliance with dialysis were the main contributing or aggravating factors.[5,6] In a review of the literature we did not find any cases in which PL was the triggering cause of PRES.

Conservative treatment of PL is effective in approximately 50% of patients.[5] In the case we present here, the severity of the symptoms and the suspicion of a possible diaphragmatic defect led us to definitively discontinue PD. Progressive ultrafiltration through intensification of the haemodialysis programme achieved resolution of the PRES and PL.

REFERENCES

1. Dang MH, Mathew M, Raj R. Pleuroperitoneal Leak as an Uncommon Cause of Pleural Effusion in Peritoneal Dialysis: A Case Report and Literature Review. Case Rep Nephrol. 2020;2020:8832080, http://dx.doi.org/10.1155/2020/8832080.
2. Nomoto Y, Suga T, Nakajima K, Sakai H, Osawa G, Ota K, et al. Acute hydrothorax in continuous ambulatory peritoneal dialysis–a collaborative study of 161 centers. Am J Nephrol. 1989;9:363–7.
3. Díaz R, del Peso G, Rodríguez M, Fernández B, Ossorio M, Bajo MA, et al. Comunicación pleuro-peritoneal en pacientes en diálisis peritoneal. Experiencia en un centro y revisión de la literatura. Nefrologia. 2011;31:213–7.
4. Kennedy C, McCarthy C, Alken S, McWilliams J, Morgan RK, Denton M, et al. Pleuroperitoneal leak complicating peritoneal dialysis: a case series. Int J Nephrol. 2011;2011:526753, http://dx.doi.org/10.4061/2011/526753.
5. Moreiras M, Fernández F, Azkárate Ramírez N, Nájera de la Garza W, Martín-Báez I, Hernansanz-Pérez M. Diálisis peritoneal: ¿un factor de riesgo o de protección para la encefalopatía posterior reversible (PRES)? Revisión de la literatura. Nefrología. 2018;38:136–40.
6. Kaneko S, Hirai K, Minato S, Yanai K, Mutsuyoshi Y, Ishii H, et al. A case of posterior reversible encephalopathy syndrome in a patient undergoing automated peritoneal dialysis. CEN Case Rep. 2019;8:178–82.

Montserrat Picazo Sánchez*, Marc Cuxart Pérez, Rosa Masdeu Jufré, Maria Cufí Vallmajor

Fundacio Salut Emporda, Figueres, Girona, Spain

* *Corresponding author.*
E-mail address: mpicazo@salutemporda.cat (M. Picazo Sánchez).

2013-2514/© 2022 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
https://doi.org/10.1016/j.nefroe.2024.01.004

Inmunoglobulin-mediated glomerulonephritis and mixed cryoglobulinemia as a form of presentation of visceral leishmaniasis in a patient with HIV

Glomerulonefritis inmunomediada y crioglobulinemia mixta como forma de presentación de leishmaniasis visceral en paciente con VIH

Dear Editor:

Leishmaniasis is a zoonosis caused by a protozoan parasite of the genus *Leishmania*.[1] Risk factors include malnutrition, immunosuppression treatment and co-infection with the human immunodeficiency virus (HIV), the latter being the most frequent association, occurring in most cases in a torpid and recurrent form.[2,3] Renal involvement at both glomerular

DOI of original article:
https://doi.org/10.1016/j.nefro.2022.04.003.

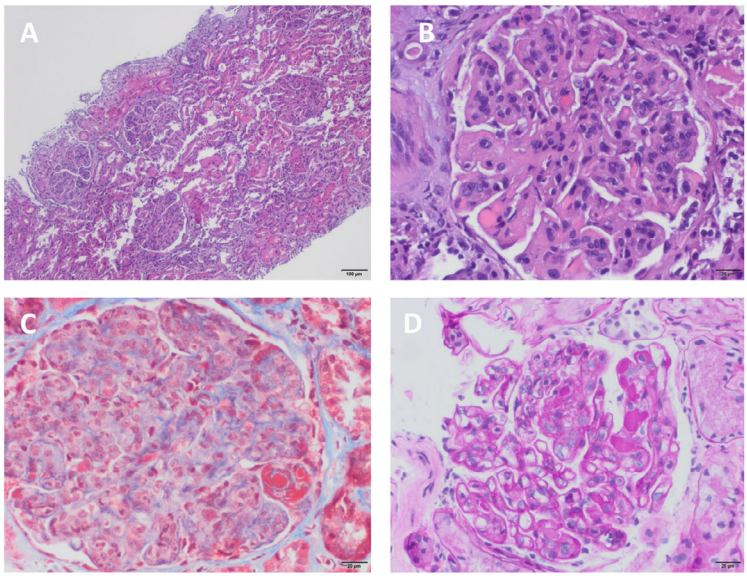

Fig. 1 – Renal biopsy. (A) Mesangial and intracapillary hypercellularity with polylobulated appearance of the glomerular tuft and membranoproliferative changes, showing a "puzzle piece" appearance with the presence of hyaline pseudothrombi (H–E, ×4). (B) Mesangial and intracapillary hypercellularity with presence of inflammatory infiltrate and membranoproliferative pattern. Capillary thickening with double contour images and hyaline pseudothrombi (H–E, ×20). (C) Membranoproliferative changes and mesangial hypercellularity with intense red staining of hyaline pseudothrombi (Masson's trichrome, ×20). (D) Capillaries with thickened and rigid appearance, with double-contour or railroad track images (PAS, ×10).

and tubular level has been described: it is usually mild and it cures with control of the infection.[4,5] Here we present the case of a 45-year-old man with a medical history of hepatitis B virus infection in 2004 and HIV infection with normal CD4 levels (undetectable viral load) undergoing treatment with abacavir/lamivudine and doravirine. He had a history of opportunistic infection due to esophageal candidiasis and *Pneumocystis jirovecii* pneumonia. In addition, he had thrombophilia due to protein S deficiency on the anticoagulant acenocouramarol due to a history of deep vein thrombosis and pulmonary thromboembolism.

The patient presented in the emergency room for dyspnea on moderate exertion and abdominal pain in the right hypochondrium for 2 weeks. He also presented with intermittent moderate fever for 3 months, arthralgias and nonspecific skin lesions on the upper limbs. Physical exam revealed hypertension (BP 157/103 mmHg, HR 80 lpm), predominantly bimalleolar edema and respiratory auscultation with bibasilar crackles. Analytics highlight deterioration of renal function, with serum creatinine of 2.42 m/dl (previous renal function was normal), hypocomplementemia of C3 and C4, microangiopathic hemolytic anemia with positive direct Coombs test, leukopenia, thrombocytopenia and polyclonal hypergammaglobulinemia. Urinary sediment showed dysmorphic red blood cells and proteinuria with an albumin/creatinine ratio of 1,537.6 mg/g. Antinuclear and anti-DNA antibodies were negative. Abdominal ultrasound showed normal-size kidneys and hepatosplenomegaly. Viral load for HIV and hepatitis B virus on serology were undetectable, with hepatitis C virus being negative. Cryoglobulins were positive, with a cryoprecipitate of 40% and immunofixation with data compatible with cryoglobulinemia type 2.

A percutaneous renal biopsy was performed, and 41 glomeruli were obtained, one of them with global glomerulosclerosis; the rest showed mesangial and endocapillary proliferation, with capillaries with double contour morphology. No crescents were seen. Tubular atrophy and interstitial fibrosis of 20%. Moderate lymphocytic infiltration. The immunofluorescence study showed mesangial and glomerular subendothelial deposition of IgM, IgG and C3. The diagnosis was glomerulonephritis with membranoproliferative pattern type I or glomerulonephritis mediated by immunocomplexes with membranoproliferative pattern (Fig. 1). Given the hypersplenism, it was decided to perform a splenic biopsy, showing abundant basophilic intracellular pathogens with the appearance of *Leishmania* (Fig. 2). In view of these results, treatment was started with liposomal amphotericin B at a dose adjusted to the glomerular filtration rate of 3 mg/kg/day. After 3 months of outpatient treatment the patient was asymptomatic, with improvement of renal function with creatinine of 1.2 mg/dl and without microhematuria and proteinuria.

The renal involvement described by *Leishmania* is very heterogeneous. The infection itself, the hemodynamic alterations derived from the disease (anemia, hypotension, hypovolemia), and even the treatment directed to the infection (amphotericin B) favor the development of renal lesions.[6] Renal involvement by *Leishmania* is rare but is produced by the formation of autoantibodies and immunocomplexes, which lead to the activation of cytotoxic T cells and adhesion molecules.[7] In our case, the patient started with deterioration of renal function with hematuria and proteinuria, hypocomplementemia and positive serum cryoglobulins. Serology for hepatitis C virus was negative, so it was suspected that another disease was associated cryoglobulinemia. A splenic

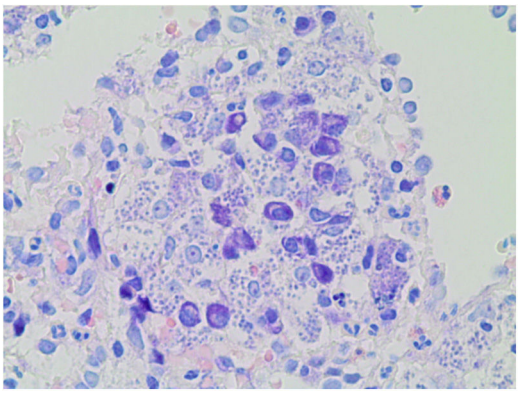

Fig. 2 – Splenic biopsy, Giemsa staining. Microorganisms corresponding to *Leishmania* with rounded or oval morphology inside the cytoplasm of macrophages/histiocytes.

and renal biopsy was performed at the same time, with the splenic biopsy providing the diagnosis of leishmaniasis and the renal biopsy the diagnosis of immune-mediated glomerulonephritis associated with this infectious disease. In a patient with constitutional symptoms, pancytopenia and renal lesion, clinician's should consider leishmaniasis. The rapid initiation of specific therapy with amphotericin B for opportunistic infection by *Leishmania* led to a good evolution of the patient.

REFERENCES

1. 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(06):370–7.
2. de Vries HJC, Reedijk SH, Schallig HDFH. Cutaneous leishmaniasis: recent developments in diagnosis and management. Am J Clin Dermatol. 2015;16(2):99–109.
3. Laguna F, Adrados M, Alvar J, Soriano V, Valencia ME, Moreno V, et al. Visceral leishmaniasis in patients infected with the human immunodeficiency virus. Eur J Clin Microbiol Infect Dis. 1997;16(12):898–903.
4. Libório AB, Rocha NA, Oliveira MJC, Franco LFLG, Aguiar GBR, Pimentel RS, et al. Acute kidney injury in children with visceral leishmaniasis. Pediatr Infect Dis J. 2012;31(5):451–4.
5. Dutra M, Martinelli R, de Carvalho EM, Rodrigues LE, Brito E, Rocha H. Renal involvement in visceral leishmaniasis. Am J Kidney Dis. 1985;6(1):22–7.
6. Clementi A, Battaglia G, Floris M, Castellino P, Ronco C, Cruz DN. Renal involvement in leishmaniasis: a review of the literature. Clin Kidney J. 2011;4(3):147–52.
7. Nylén S, Sacks D. Interleukin-10 and the pathogenesis of human visceral leishmaniasis. Trends Immunol. 2007;28(9):378–84.

Cristina Andrades Gómez, Marina Sánchez-Agesta Martínez*, Alicia Luna Aguilera, Gema Montilla Cosano, Maria Mercedes Toro Ramos

Servicio de Nefrología, Hospital Universitario Virgen del Rocío, Sevilla, Spain

* *Corresponding author.*
E-mail address: marsanagesta@gmail.com (M.S.-A. Martínez).

2013-2514/© 2022 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
https://doi.org/10.1016/j.nefroe.2024.01.008

The need for renal biopsy in oncology patients on check-point inhibitors check-point inhibitors: New triggers for extracapillary glomerulonephritis extracapillary glomerulonephritis

La necesidad de la biopsia renal en paciente oncológico con inhibidores de check-point: nuevos trigger para glomerulonefritis extracapilar

Dear Editor,

Checkpoint inhibitors (CPI) have revolutionised cancer treatment. They act by inhibiting T-lymphocyte (TL) receptors, cytotoxic T-lymphocyte antigen 4 (CTLA4) or programmed cell death 1 (PD1) receptors or their ligands, triggering TL dysregulation and hyperactivation, which causes immune-related adverse events (irAE). In the kidneys, the most common is immune-mediated interstitial nephritis, but there is also

DOI of original article:
https://doi.org/10.1016/j.nefro.2022.04.008.