Clinical Images in Nephrology and Dialysis

Hypereosinophilia in a Patient with Kidney Allograft Dysfunction on Hemodialysis

Inès Dufour,[1] *Selda Aydin*,[2] *and Eric Goffin*[1]

Kidney360 5: 1780–1781, 2024. doi: https://doi.org/10.34067/KID.0000000582

Case Description

A 55-year-old woman with kidney failure due to primary FSGS received a kidney transplant in 1989. Chronic allograft dysfunction required KRT, and the patient was started on home hemodialysis in October 2020. In February 2022, she presented with isolated macroscopic hematuria for three consecutive days. Urinalysis ruled out urinary tract infection. Abdominal computed tomography scan showed uncomplicated kidney allograft atrophy. Cystoscopy and gynecologic examination were normal. In April 2022, routine monthly biology revealed severe hypereosinophilia (peak eosinophil count: 3.33×10^9/L [normal, $<0.5\times10^9$/L]), total white blood cells: 12.1×10^9/L (normal, $4–10\times10^9$/L), and acute phase response (C-reactive protein: 41 mg/L, normal <5 mg/L). No new medications had recently been introduced; the regular treatment regimen included cyclosporin, aspirin, irbesartan, atenolol, atorvastatin, omeprazole, sodium zirconium cyclosilicate, and darbepoetin alfa. The patient had no history of allergies. Stool ova and parasite test and anti-neutrophil cytoplasmic antibody were all negative; lymphocyte immunophenotyping and IgE concentration were normal. ^{18}F-fluorodeoxyglucose positron emission tomography-computed tomography showed intense hyperactivity of the kidney allograft cortex associated with several locoregional lymphadenopathies, compatible with an inflammatory phenomenon within the graft, such as an acute rejection (Figure 1A). Graft nephrectomy was subsequently performed. Histopathology showed significant interstitial inflammation with lymphocyte and eosinophil infiltration (Figure 1B), severe glomerulitis (g3), peritubular capillaritis (ptc3), and glomerular basement membrane double contours (cg1) (Figure 1C). C4d staining was positive in peritubular capillaries. These findings were consistent with a diagnosis of eosinophil-rich chronic active antibody-mediated rejection. Blood eosinophil count normalized 2 weeks after surgery.

Discussion

Eosinophilia (eosinophil count $\geq500/\mu$l) is observed in patients on hemodialysis, often due to parasitic infections, dialyzer reactions, or malignancies. In this report, we present a case of hypereosinophilia induced by kidney allograft rejection.

Association between peripheral blood eosinophilia and episodes of graft rejection was initially reported in the 1980s. More recent studies have confirmed those findings, proposing eosinophilia as a biomarker for acute rejection.[1] Rise of blood eosinophil count in early post-transplant months has even been proposed as a hallmark of graft rejection.[2]

Eosinophils are now considered true immunoregulatory cells having some roles in antigen presentation; T-cell regulation; B-cell priming; as well as regulation of dendritic and mast cells, basophils, and neutrophils. Eosinophils have been linked to acute allograft rejection induced by Th2-type CD4$^+$ cells, which are effector cells in the alternative pathway implicated in acute rejection.[3] Cytokines, such as IL-5, released by activated T lymphocytes are responsible for the eosinophil recruitment in acute rejection. In experimental mouse heart transplant, acute rejection mediated by Th2 cells was characterized by a marked eosinophil infiltrate in the allograft.

In summary, this case confirms that antibody-mediated rejection should be considered in the differential diagnosis of hypereosinophilia in a transplanted patient, even after kidney graft failure and hemodialysis resumption.

Teaching Points

- Hypereosinophilia is observed in patients on hemodialysis, often due to parasitic infections, dialyzer reactions, or malignancies.

[1]Division of Nephrology, Cliniques universitaires Saint-Luc, Brussels, Belgium
[2]Pathology Department, Cliniques universitaires Saint-Luc, Brussels, Belgium

Correspondence: Dr. Inès Dufour, email: ines.dufour@saintluc.uclouvain.be

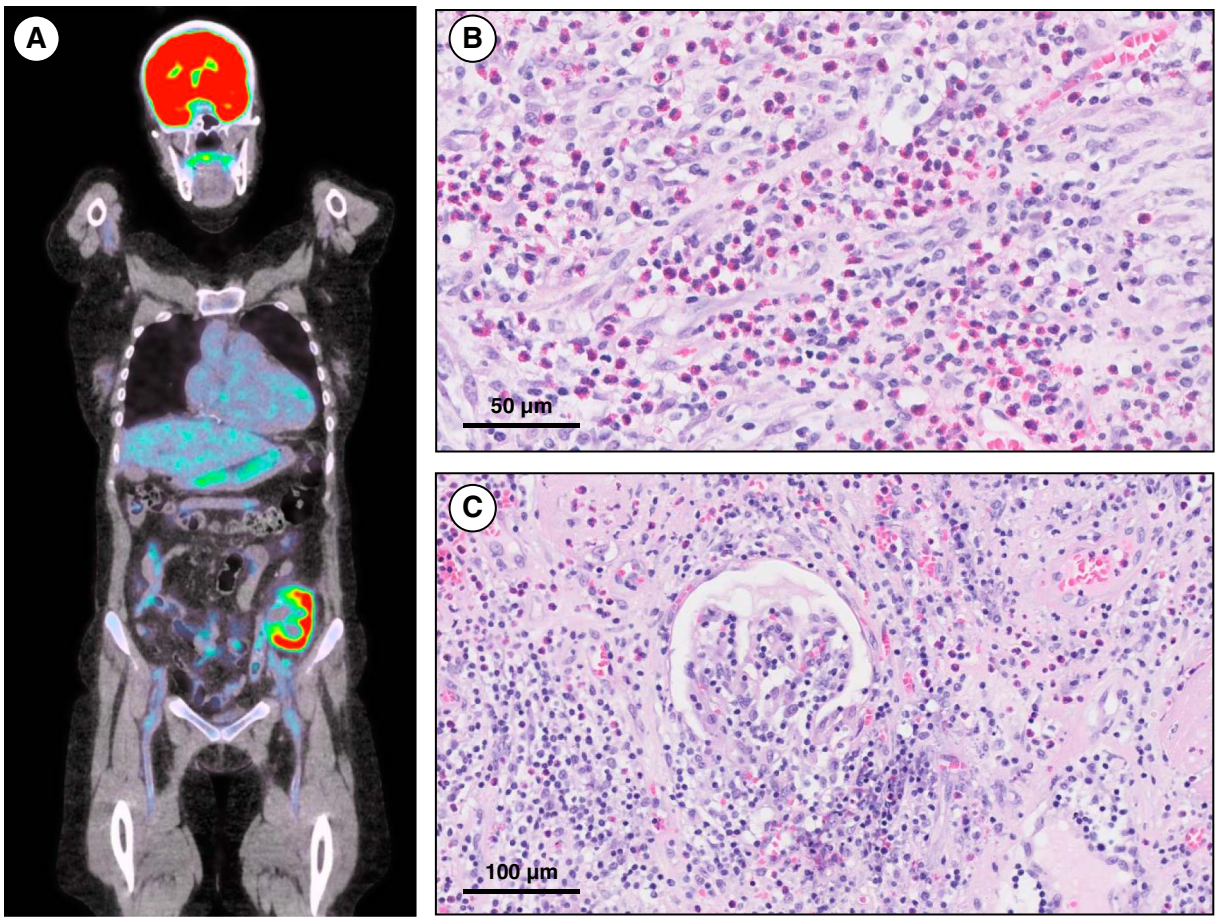

Figure 1. Antibody-mediated rejection of the kidney allograft. (A) ^{18}F-FDG-PET/CT showed hypermetabolism of the renal allograft cortex with a mean SUV of 4.6 (normal <1.6). (B) Kidney allograft biopsy showed an infiltrate of the parenchyma by a large number of eosinophils (hematoxylin and eosin stain; bar=50 μm). (C) Overview of cABMR, with signs of glomerulitis and peritubular capillaritis, with Banff scoring g3 cg1 mm0 t3 ct3 i3 ci3 ti3 i-IFTA3 v1 cv2 aah0 ptc3. Bar=100 μm. cABMR, chronic active antibody-mediated rejection; ^{18}F-FDG-PET/CT, ^{18}F-fluorodeoxyglucose positron emission tomography-computed tomography; SUV, standardized uptake value.

- Allograft rejection should be considered in the differential diagnosis of hypereosinophilia in a patient who underwent transplant, even after kidney graft failure and hemodialysis resumption.

Disclosures

Disclosure forms, as provided by each author, are available with the online version of the article at http://links.lww.com/KN9/A677.

Funding

None.

Acknowledgments

The patient provided informed consent for the publication of this case report.

Author Contributions

Conceptualization: Selda Aydin, Inès Dufour, Eric Goffin.
Data curation: Selda Aydin, Inès Dufour, Eric Goffin.
Writing – original draft: Inès Dufour, Eric Goffin.
Writing – review & editing: Selda Aydin, Inès Dufour, Eric Goffin.

References

1. Wang GY, Li H, Liu W, et al. Elevated blood eosinophil count is a valuable biomarker for predicting late acute cellular rejection after liver transplantation. *Transpl Proc.* 2013;45(3):1198–1200. doi: 10.1016/j.transproceed.2012.10.008
2. Almirall J, Campistol JM, Sole M, Andreu J, Revert L. Blood and graft eosinophilia as a rejection index in kidney transplant. *Nephron.* 1993;65(2):304–309. doi:10.1159/000187493
3. Goldman M, Le Moine A, Braun M, Flamand V, Abramowicz D. A role for eosinophils in transplant rejection. *Trends Immunol.* 2001; 22(5):247–251. doi:10.1016/s1471-4906(01)01893-2