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Systemic Hypereosinophilia and Podocyte Injury: Illustration by Focal and Segmental Hyalinosis

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Abstract | **Case Report**

Hypereosinophilic syndrome (HES) is a disorder characterized by blood eosinophilia >500 cells/mm³ associated with visceral involvement in patients without any secondary cause of eosinophilia. Renal involvement during this syndrome is rare. We report the case of a 51-year-old patient hospitalized for a glomerular syndrome secondary to hypereosinophilia. Clinical examination revealed grade 2 hypertension and generalized maculo-erythematous skin lesions. Paraclinical investigations showed proteinuria of 2.25 g/24 h, renal insufficiency with serum creatinine at 23 mg/L, microscopic hematuria, and hypereosinophilia at 17.693 cells/mm³ with a negative etiological work-up. Skin biopsy concluded to eosinophilic dermatitis with granuloma surrounding collagen necrosis. Bone marrow biopsy showed a rich, hyperplastic marrow with hypereosinophilia. Kidney biopsy concluded to focal and segmental hyalinosis (FSH), classic variant, with chronic tubulointerstitial involvement estimated at 15–20% and focal inflammatory infiltrate predominantly lymphocytic without vascular lesions. The patient received high-dose corticosteroid therapy and a nephroprotective treatment. At 6 months, the evolution was marked by the disappearance of proteinuria and stable renal function. Renal involvement during hypereosinophilic syndrome is uncommon. In our case, the clinical and histological findings support focal and segmental hyalinosis. Corticosteroid therapy combined with nephroprotection allowed remission.

Keywords: Hypereosinophilic syndrome, Focal and segmental hyalinosis, Glomerular syndrome, Eosinophilia, Corticosteroids, Proteinuria.

INTRODUCTION

Focal and segmental hyalinosis (FSH) is a podocytopathy leading to loss of the physical integrity of the renal glomerular filtration barrier, and its diagnosis is established by histology. It is most often suspected in the presence of a glomerular syndrome (most commonly an impure nephrotic syndrome). It may be primary or secondary to an underlying disease, and its pathophysiology is complex [1].

Hypereosinophilic syndrome (HES) is a condition characterized by prolonged peripheral blood eosinophilia (greater than 500 cells/mm³) lasting more than 6 months, associated with signs of visceral involvement in patients without parasitic, allergic, or other secondary causes of eosinophilia [2, 3].

Hypereosinophilic syndrome is rare, with an estimated incidence of 0.036/100.000 after exclusion of secondary causes [4]. Various organ involvements may occur in HES (skin, heart, lungs, etc.), but renal involvement is most often rare [2]. We report a case of the classic variant of FSH occurring during hypereosinophilic syndrome.

PATIENT AND OBSERVATION

This is the case of a 51-year-old woman petient with a history of hypertension known for 5 years and poorly followed, managed with Ramipril 5 mg/day. She was hospitalized in the nephrology department for a glomerular syndrome consisting of proteinuria at 2.25 g/24 h, renal insufficiency with plasma creatinine at 28 mg/L, and microscopic hematuria.

Four months before admission, the patient presented with generalized pruritic skin eruptions of sudden onset, evolving in a context of fever with preserved general condition, leading to hospitalization in the dermatology department. Following the discovery of proteinuria at 2.25 g/24 h and renal insufficiency with plasma creatinine at 23 mg/L, the patient was transferred to the nephrology department.

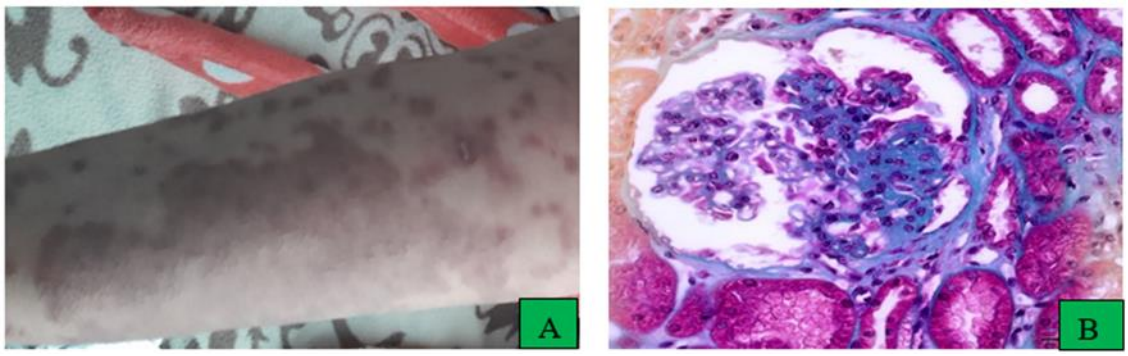
Upon admission, clinical examination revealed a patient in good general condition, afebrile at 36.5°C, with a body mass index of 25 kg/m² and blood pressure of 165/100 mmHg while on Ramipril 5 mg. Diuresis was preserved. Urine dipstick showed +2 protein and +4 blood. Skin examination revealed generalized maculo-erythematous eruptions with scratching lesions. There was no hepatomegaly or splenomegaly, and superficial lymph nodes were not enlarged (Figure A).

From a paraclinical perspective, the complete blood count showed leukocytosis at 23.910 cells/mm³ with hypereosinophilia at 17.693 cells/mm³ (versus 16.560 cells/mm³ four months earlier), the other cell lines were normal. Serum creatinine was 23 mg/L (corresponding to a GFR of 23 ml/min/1.73 m² according to MDRD). Urinalysis showed microscopic hematuria at 20 cells/mm³.

As part of the etiological investigation for hypereosinophilia, stool parasitological examination and serologies for filariasis, trichinellosis, toxocariasis, strongyloidiasis, and ascariasis were performed and were negative. Total IgE levels were elevated at 2.785 IU/ml. Tryptase was negative at 9.8 µg/L. Immunological tests and infectious serologies were also negative. Cardiac evaluation showed no abnormalities with a left ventricular ejection fraction of 58%. A thoraco-abdomino-pelvic computed tomography scan did not reveal deep lymphadenopathy or abdominal or pelvic masses.

Histologically, the skin biopsy showed eosinophilic dermatitis with granuloma around collagen necrosis. Bone marrow biopsy revealed a rich, hyperplastic marrow with hypereosinophilia; no granuloma or malignancy was identified. Renal biopsy concluded to focal and segmental hyalinosis (FSH), classic type, with chronic tubulointerstitial involvement estimated at 15–20% and focal inflammatory infiltrate predominantly lymphocytic without vascular lesions (Figure B).

The patient received high-dose corticosteroid therapy and nephroprotective treatment with Perindopril 10 mg. After 3 months of corticosteroid therapy, proteinuria decreased by more than 50%. At 6 months, proteinuria became negative, and renal function remained stable with plasma creatinine between 15 and 16 mg/L (corresponding to a GFR between 36 and 38 ml/min/1.73 m² according to MDRD). Hematologically and dermatologically, we observed disappearance of hypereosinophilia and skin eruptions.



A: Maculo-erythematous skin eruptions
B: Lesion of focal and segmental hyalinosis; Masson's trichrome staining; high magnification

DISCUSSION

Eosinophilia is an important indicator of various neoplastic and non-neoplastic conditions. Depending on the underlying disease and mechanisms involved, eosinophil infiltration may lead to organ dysfunction, clinical symptoms, or both. Peripheral blood eosinophilia has been classified into mild eosinophilia ($0.5–1.5 \times 10^9$/L), marked ($>1.5 \times 10^9$/L), and massive ($>5.0 \times 10^9$/L). It may be transient, episodic, or persistent (chronic). The term persistent applies to peripheral blood eosinophilia documented on at least two occasions with a minimum interval of four weeks [5]. Our case fulfilled the diagnostic criteria for hypereosinophilic syndrome (HES).

The discovery of peripheral blood hypereosinophilia is a common situation in medical practice and may be associated with a wide range of causes, prompting the search for atopic conditions, drug allergies, parasitic infections, certain immune deficiencies, inflammatory diseases, hematologic disorders, or deep-seated neoplasms. Persistence of hypereosinophilia despite a negative extensive investigation suggests idiopathic hypereosinophilia and raises the diagnostic consideration of HES [6]. In our

patient, the etiological investigation for HES was negative; therefore, the diagnosis of idiopathic HES was retained.

Cutaneous, pulmonary, and cardiac involvement are the most frequent manifestations of hypereosinophilic syndrome, while renal involvement remains rare. Approximately 20% of patients with idiopathic HES develop proteinuria and hypertension [1, 7, 8]. Renal failure is frequent as a preterminal event [2], and some authors have reported dialysis treatment in patients with idiopathic HES [9].

Reported renal manifestations include eosinophilic interstitial nephritis, membranous nephropathy, anti-neutrophil cytoplasmic antibody (ANCA)-positive or non-crescentic glomerulonephritis, and immunotactoid glomerulonephritis, with or without glomerular immune deposits [10, 16].

Date *et al.,* provided renal histopathological findings in autopsied patients with HES. The most frequent renal lesions were interstitial nephritis with eosinophilic infiltrates and tubular atrophy, as well as glomerular lesions (mesangial dilation, hypercellularity, and thickening of the basement membrane) [17]. In a series of 14 patients, Chusid *et al.,*[2] found two cases of glomerular mesangial expansion and thickening of the glomerular basement membrane and one case of eosinophilic infiltrates in the kidney.

In the case report by Motellon [10], renal biopsy revealed glomerular abnormalities such as focal mesangial expansion and focal hyalinosis surrounded by large areas with chronic tubulointerstitial lesions, focal interstitial eosinophilic leukocytes, inflammatory cell infiltrates, and vascular lesions.

In our patient, renal histopathology showed focal and segmental hyalinosis (FSH), classic variant, with chronic tubulointerstitial involvement estimated at 15–20% and focal inflammatory infiltrate predominantly lymphocytic without vascular lesions.

The rapid and sustained improvement in renal function and the reduction in proteinuria after prednisone therapy suggest that glomerulonephritis secondary to HES may not require aggressive treatment such as cytotoxic drugs and plasma exchange, which are traditionally recommended as first-line therapy in other causes of vasculitis and progressive glomerulonephritis [3].

This was the case in our patient, who responded to corticosteroid therapy with a reduction in proteinuria of more than 50% at 3 months, followed by complete remission of proteinuria at 6 months, with stable renal function at 15 mg/L plasma creatinine (GFR 38 ml/min/1.73 m² according to MDRD).

CONCLUSION

Our case shows that focal and segmental hyalinosis is one of the renal manifestations of idiopathic hypereosinophilic syndrome. High-dose corticosteroid therapy with gradual tapering after 6 months, combined with nephroprotective treatment, led to complete remission of proteinuria and stabilization of renal function. This approach may help avoid aggressive treatments such as cytotoxic drugs and plasma exchange, which can have significant adverse consequences.

REFERENCES

1. Séverine Beaudreuil, Antoine Durrbach. Hyalinose segmentaire et focale. Bulletin de l'académie nationale de médecine; Janv-mars 2017, 201(1-3) : (p.47-69) ScienceDirect.
2. Chusid MJ, Dale DC, West BC, *et al.,* Le syndrome hyperéosinophile: analyse de quatorze cas avec revue de la littérature, Médecine, 1975, vol. 54 (p. 1-27) Google ScholarRéférences croisées PubMed.
3. Nathalie Shehwaro *et al.,* Atteinte rénale au cours de syndrome d'hyperéosinophilie. Clinical Kidney Journal, juin 2013, vol 6, numéro 3 (p. 272-276).
4. Klion AD. How I treat hypereosinophilic syndromes. Blood. 2015; 126(9) (p. 1069–1077).
5. Valent, Klion AD, Horny H, *et al.,* Proposition consensuelle contemporaine sur les critères et la classification des troubles éosinophiles et des syndromes apparentés, J Allergy Clin Immunol, 2012, vol. 130 (p. 607-612) Google Scholar Références croisées PubMed.
6. DE Wazieres B. Hypereosinophilie: orientation diagnostique. Rev Prat 1997, vol. 47 (p. 1129-1136) Google Scholar.
7. Spry CJF, Davies J, Tai PC, *et al.,* Caractéristiques cliniques de quinze patients atteints du syndrome hyperéosinophile, Q J Med, 1983, vol. 52 (p. 1-22) Google Scholar Références croisées PubMed.
8. San Jose A, Bosch JA, Candell J, *et al.,*Afeccion cardiaca y hematologica en el sindrome hipereosinofilico idiopatico. Estudio de 12 casos, Med Clin (Bare), 1992, vol. 98 (pg. 161-165) Google Scholar.
9. Garella G, Marra L. Sindrome ipereosinofila e insufficienza renale, Minerva Urol Nefrol, 1990, vol. 42 (pg. 135-136) Google Scholar PubMed.
10. Motellon JL, Bernis C, Garcia-Sanchez A, *et al.,* Implication rénale dans le syndrome hyperéosinophile, Nephrol Dial Transplant, 1995, vol. 10 (p. 401-403) Google Scholar PubMed.
11. Bulucu , Can C, Inal V, *et al.,* Renal involvement in a patient with idiopathic hypereosinophilic syndrome, Clin Nephrol, 2002, vol. 57 (pg. 171-173) Google Scholar Crossref PubMed.
12. Navarro I, Torras J, Goma M, *et al.,*Renal involvement as the first manifestation of hypereosinophilic syndrome: a case report, NDT

Plus, 2009, vol. 2 (pg. 379-381) Google Scholar PubMed.

13. Richardson P, Dickinson G, Nash S, *et al.,*Crescentic glomerulonephritis and eosinophilic interstitial infiltrates in a nephropathy, in a patient with myelodysplastic syndrome, eosinophilia, and trisomy 8, Nephrol Dial Transplant, 2002, vol. 17 (pg. 1336-1338) Google Scholar Crossref PubMed.
14. Choi YJ, Lee JD, Yang KI, *et al.,*Immunotactoid glomerulopathy associated with idiopathic hypereosinophilic syndrome, Am J Nephrol, 1998, vol. 18 (pg. 337-34) Google Scholar Crossref PubMed.
15. Frigui M, Hmida MB, Jallouli M, *et al.,*Membranous glomerulopathy associated with idiopathic hypereosinophilic syndrom, Saudi J Kidney Dis Transpl, 2010, vol. 2 (pg. 320-322) Google Scholar PubMed.
16. Lanfranchi J, Meyrier A, Sachs RN, *et al.,*Blood hypereosinophilia syndrome with cardiac involvement and extramembranous glomerulopathy, Ann Med Interne (Paris), 1986, vol. 137 (pg. 133-137) Google Scholar PubMed.
17. Date A, Parameswaean A, Bhaktaviziam A. Lésions rénales dans les cardiomyopathies oblitérantes : fibrose endomyocardique et endocardite de Loffler, J Pathol, 1983, vol. 140 (p. 113-122) Google Scholar Références croisées PubMed.