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Case Report
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Multisystem Eosinophilic Vasculitis with Cutaneous, Renal, and Enteric Involvement: A Case Report

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Abstract

A 59-year-old male electrician presented to the Emergency Department with hematuria, severe asthenia, and an erythematous rash with desquamative lesions involving all photo-exposed areas, including the forehead, face, scalp, upper and lower limbs, and hands, which had appeared one week earlier. Laboratory tests revealed normochromic anemia requiring transfusion, marked eosinophilia (2,500/mm^3), and severe acute kidney injury with creatinine of 16 mg/dL and BUN of 165 mg/dL, associated with metabolic acidosis. Although no peripheral edema was present, the patient showed mild dyspnea and tachypnea.

Renal ultrasound showed increased cortical echogenicity suggestive of parenchymal damage, while chest CT demonstrated left lower lobe atelectasis and small mediastinal lymphadenopathy. The patient was admitted to the Internal Medicine ward for further evaluation. Autoimmune and rheumatologic screening, including C-ANCA, P-ANCA, ANA, and anti-dsDNA antibodies, was negative.

Following nephrology consultation, hydration therapy led to partial improvement of renal function, after which a renal biopsy was performed. Histological findings were consistent with membranoproliferative glomerulonephritis, showing enlarged glomeruli, mesangial hypercellularity, and flocculocapsular adhesions. Dermatologic evaluation with skin biopsy revealed lichenoid dermatitis with neutrophilic and eosinophilic infiltration. Due to persistent diarrhea, endoscopic evaluation with colonic biopsy was carried out, demonstrating normal mucous glands with marked eosinophilic infiltration of the lamina propria.

Based on multisystem involvement cutaneous, renal, and colonic the final diagnosis was eosinophilic vasculitis. High-dose corticosteroid therapy with prednisone (1 mg/kg/day) resulted in rapid clinical and laboratory improvement, with resolution of systemic symptoms and normalization of eosinophil counts. Corticosteroids were gradually tapered during follow-up without the need for immunosuppressive or biologic therapy.

Keywords: Hypereosinophilia; Eosinophilic vasculitis; Acute renal failure; Glomerulonephritis

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Introduction

Hypereosinophilia represents a heterogeneous clinical condition defined by a persistent increase in peripheral blood eosinophils exceeding 1,500 cells/mm^3 and may be associated with tissue infiltration and organ damage. Current clinical guidelines emphasize the importance of distinguishing between secondary (reactive) eosinophilia, primary (clonal or neoplastic) eosinophilia, and idiopathic hypereosinophilic syndromes, as management and prognosis differ significantly among these entities. A comprehensive diagnostic workup is therefore required, including exclusion of allergic, infectious, drug-induced, autoimmune, and hematologic causes, as well as careful assessment of eosinophil-related end-organ involvement.

Among the systemic diseases associated with hypereosinophilia, eosinophilic vasculitides represent a rare but clinically significant group of disorders characterized by vascular inflammation accompanied by eosinophil-rich infiltrates. According to the Revised International Chapel Hill Consensus Conference 2012 (CHCC2012), vasculitides are classified based on the size of the predominantly affected vessels and defined by clinicopathological features [1]. Within this

Table 1: Serial laboratory parameters from Emergency Department presentation through follow-up, showing improvement in renal function and eosinophilia after initiation of therapy.

	Emergency Room Entrance	After a week	After two weeks	After four weeks
Creatinine	16.2 mg\dl	7 mg\dl	6.5 mg\dl	2 mg\dl
Urea	165 mg\dl	145 mg\dl	110 mg\dl	70 mg\dl
Sodium	147 mmol/l	145 mmol\l	138	135
Potassium	5.3 mmol\l	4.9 mmol\l	4.7	4.4
Calciium	8.1 mg\dl	8.5 mmol\l	8.6	9
Hemoglobin	6.3 gr\dl	7.5 gr\dl	8 gr\dl	9 gr\dl
Neutrofhil granulocytes	0.577	0.59	0.613	0.684
Basofhils	0.004	0.01	0.012	0.013
Eosinofhils	0.287	0.24	0.16	0.08
Limphocytes	0.06	0.08	0.132	0.14
Monocytes	0.072	0.08	0.083	0.083

framework, Eosinophilic Granulomatosis with Polyangiitis (EGPA) is categorized as a small-vessel necrotizing vasculitis associated with asthma, eosinophilia, and extravascular granulomas; however, the CHCC2012 also acknowledges that eosinophilic vasculitis may present with variable phenotypes and incomplete or atypical clinical features.

The diagnosis of eosinophilic vasculitis relies on the integration of clinical manifestations, peripheral eosinophilia, laboratory findings, and, most importantly, histopathological evidence of eosinophil-rich inflammation involving the vessel wall or surrounding tissues. ANCA positivity, although helpful when present, is not mandatory for diagnosis and is absent in a substantial proportion of cases. Histological confirmation from affected organs remains the gold standard, particularly in atypical presentations lacking classic respiratory features. In general, EGPA (formerly eosinophilic granulomatosis with polyangiitis, Churg–Strauss syndrome) is not diagnosed when the key defining criteria are absent, even if an eosinophil-rich vasculitis is present.

In other words, the presence of eosinophils within the vessel wall alone is not sufficient to establish a diagnosis of EGPA.

This case illustrates a rare and diagnostically challenging presentation of eosinophilic vasculitis with severe multisystem involvement, highlighting the critical role of a structured diagnostic approach guided by current recommendations on hypereosinophilia and by the CHCC2012 classification system.

Case Presentation

A 59-year-old male electrician was admitted to the emergency room with frank hematuria, severe fatigue, an erythematous rash, and scaling lesions in all areas of the body exposed in the photo (Figure 1) the frontal region, upper limbs, legs, hands, face, and scalp, which had appeared a week earlier. Blood tests performed in the emergency room revealed normochromic anemia requiring transfusion, marked eosinophilia (2500 mm), and severe renal failure with a creatinine of 16 and a BUN of 165. Blood gas analysis revealed metabolic acidosis. He had no renal edema but mild dyspnea with an increased respiratory rate.

In the emergency room, he underwent renal ultrasound and chest CT without contrast medium. Renal ultrasound revealed the kidneys with cortical hyperechogenicity, showing signs of distress.

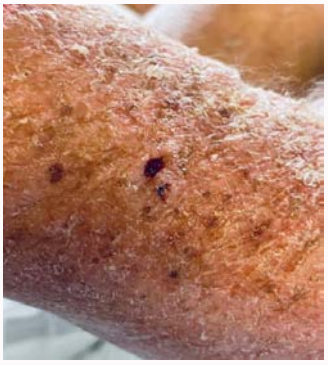

Figure 1: Exfoliative skin lesions with small papules.

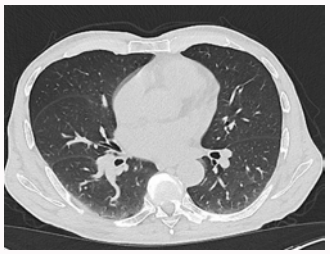

Figure 2: Slight disventilatory aspects in the left lower lobe with ground glass at the bases.

There were no fluid deposits, the bladder had thickened walls, and the prostate was at the upper volumetric limits with an inhomogeneous echotexture.

A chest CT scan showed ventilatory phenomena in the left lower lobe and some peri centimeter mediastinal lymph node nodules (Figure 2).

He was therefore admitted to the Internal Medicine Department for further diagnostic workup.

During the first few days of hospitalization, he underwent all rheumatological screening, which showed negative C-ANCA and P-ANCA, negative ANA and DNA antibody.

Upon the advice of the nephrologist consultant, he began rehydration therapy with a gradual reduction in renal insufficiency

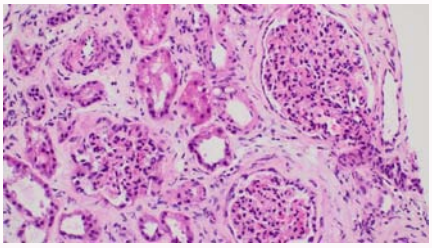

Figure 3: Enlarged glomeruli, increased mesengial cellularity, presence of flocculocapsular adhesion.

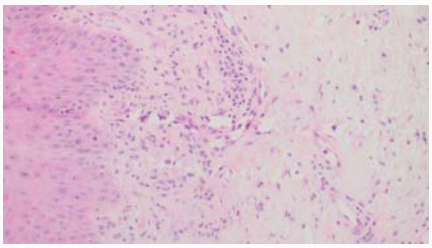

Figure 4: Skin tissue covered by thickened, orthokeratotic epidermis which in the dermis is the site of inflammatory infiltrates of lichenoid keratinocytes and eosinophils in the dermis.

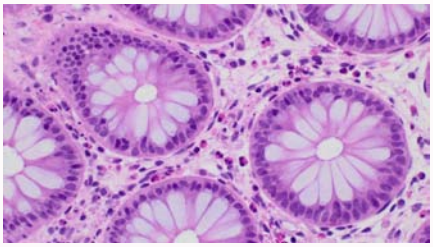

Figure 5: At high magnification, glands with a normal component of goblet cells and numerous eosinophilic granulocytes in the tunica propria are observed.

values until the decision was made to perform a renal biopsy by the interventional radiologist. The pathologist's report was consistent with membranoproliferative glomerulonephritis with enlarged glomeruli, increased mesengial cellularity, and the presence of flocculocapsular adhesion (Figure 3).

A dermatological consultation was performed, which revealed lichenoid dermatitis and neutrophilic and eosinophilic keratinocytes in the dermis (Figure 4).

He presented with several bouts of diarrhea, so after stool cultures and a negative parasitology test and a negative Clostridium difficile toxin test, it was decided to submit the patient to an endoscopic examination. A third biopsy was performed, revealing glands with a normal goblet cell composition and numerous eosinophilic granulocytes in the tunica propria (Figure 5).

The final diagnosis was eosinophilic vasculitis involving the skin, kidneys, and colon.

He was therefore placed on corticosteroid therapy with prednisone 1 mg/kg/day, which rapidly reduced inflammation and eosinophil counts, resulting in remission of systemic symptoms and normalization of laboratory values. After an outpatient follow-up, the corticosteroids were gradually reduced without the need for immunosuppressants or biologics.

Discussion

The clinical case described falls within the spectrum of eosinophilic vasculitides, a heterogeneous and relatively rare group of systemic inflammatory disorders characterized by eosinophilic tissue infiltration and multisystem organ damage. These conditions often pose significant diagnostic challenges, particularly in the early stages, due to clinical overlap with autoimmune, infectious, neoplastic, and drug- or toxin-induced diseases (Table 1) [1-5].

The patient presented with an acute and severe onset, characterized by hematuria and rapidly progressive renal failure, associated with extensive photosensitive cutaneous manifestations and marked systemic symptoms. The presence of severe hypereosinophilia from the time of Emergency Department admission represents a key diagnostic clue suggestive of an eosinophil-mediated disease, while simultaneously necessitating a careful process of diagnostic exclusion. In particular, the negativity of autoimmune screening tests (ANCA, ANA, anti-dsDNA antibodies), the absence of documented infections, and the lack of diagnostic criteria for a primary hypereosinophilic syndrome helped to narrow the differential diagnosis [2].

A highly distinctive feature of this case is the histologically documented multisystem involvement. Renal biopsy revealed membranoproliferative glomerulonephritis, a finding that is uncommon in eosinophilic vasculitis, while both skin and colonic biopsies confirmed significant eosinophilic infiltration, further supporting the diagnosis of systemic vasculitis. The concomitant presence of photosensitive cutaneous involvement, severe renal impairment, and gastrointestinal manifestations makes this clinical presentation particularly rare and of considerable scientific interest [5-7].

It Is Referred to as Eosinophilic Vasculitis Rather Than EGPA For Five Reasons

The Absence of Asthma or Typical Allergic Disease: EGPA is strongly characterized by asthma (almost constant) and chronic rhinitis/sinusitis.

If the patient has never had asthma, the diagnosis of EGPA becomes unlikely.

Lack of the Classic Three-Phase Evolution:

- Allergic phase
- Eosinophilic phase (organ infiltration)
- Systemic vasculitic phase

If this disease course is not recognizable, EGPA is generally excluded.

Absence of Typical Organ Involvement:

- In EGPA, the following are common:
- Peripheral neuropathy (mononeuritis multiplex)
- Pulmonary involvement
- Eosinophilic cardiomyopathy
- Glomerulonephritis

If the vasculitis is limited (e.g., cutaneous only), the term eosinophilic vasculitis is preferred.

Histology Not Suggestive of EGPA:

In EGPA, one would expect:

- Extravascular granulomas
- Fibrinoid necrosis
- Eosinophil-rich infiltrates

If only eosinophils are present without granulomas, the diagnosis remains more generic.

ANCA Negativity (Not Decisive, But Contributory: Approximately 30% to 40% of EGPA cases are ANCA-positive.

Negative ANCA does not exclude EGPA, but in the absence of the typical clinical picture it supports an alternative diagnosis.

Finally, the rapid and complete response to corticosteroid therapy, achieved without the need for immunosuppressive or biologic agents, represents an additional distinctive element, highlighting the importance of early diagnosis and a targeted therapeutic approach [8,9]. This case contributes to expanding current knowledge on atypical presentations of eosinophilic vasculitis and underscores the value of histopathological confirmation in the diagnostic pathway. The Revised International Chapel Hill Consensus Conference 2012 (CHCC2012) divides vasculitides based on combinations of features that separate different forms of vasculitis into definable categories. [1]. Our case is a presentation of ANCA-negative vasculitis of small and medium-sized vessels with hypereosinophilia but without airway involvement. Having ANCA-negative vasculitis with eosinophilia is a rare presentation. This condition has not been added to the relevant consensus definition of vasculitis despite its importance and significance.

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