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LETTER TO THE EDITOR

A case of eosinophilic granulomatosis with polyangiitis with human immunodeficiency virus as the underlying etiology

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Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare rheumatologic disease classified under anti-neutrophil cytoplasmic antibody-associated disorders, and its etiology has only recently begun to be elucidated. Although there are limited studies in the literature focusing on its etiology, one reported case of torasemide-induced EGPA vasculitis showed disease regression following discontinuation of the treatment.[1]

Herein, we reported a 44-year-old male patient who presented with raised, purple-colored, coalescing-prone rashes on the dorsum of both feet and the left cruris, numbness and pain in the left foot, inability to walk, fatigue, weakness, loss of appetite, and weight loss persisting for two weeks (Figure 1). The patient had a history of bronchial asthma and was receiving treatment. A written informed consent was obtained from the patient.

Prior to skin biopsy, enzyme-linked immunosorbent assay revealed a positive result for anti-HIV (human immunodeficiency virus) antibody, and the patient's HIV status was confirmed via HIV-RNA testing. EGPA can present with peripheral neuropathy, glomerulonephritis, myocarditis, and pulmonary involvement.[2,3] None of these manifestations were present in our patient. The patient's white blood cell count was 16.060/µL, with eosinophils comprising 55% (Table 1). It is well-recognized that the onset of EGPA is driven by the activation of eosinophils in response to a specific trigger.[4,5] With the initiation of antiretroviral therapy (ART), the patient's HIV-RNA levels became undetectable by the end of the first month, and eosinophil levels decreased to 1.4% by the end of the second month. The pathological examination of the skin biopsy from the lesion on the patient's leg revealed eosinophil-rich small vessel vasculitis, with mild ischemic necrosis on the surface. The patient was diagnosed with EGPA following a rheumatology consultation, and methylprednisolone and methotrexate were added to the treatment regimen in the second month of ART. Six months after the initiation of ART, the rashes had completely resolved. The patient remained on methotrexate and methylprednisolone for 24 months, and two months after their discontinuation, eosinophil levels were still within normal limits, with no new lesions observed. The patient will require lifelong HIV treatment.

The reduction in eosinophil levels from 55 to 1.4% with ART, the initiation of remission prior to the introduction of methotrexate and methylprednisolone treatment, and the absence of relapse following the discontinuation of these

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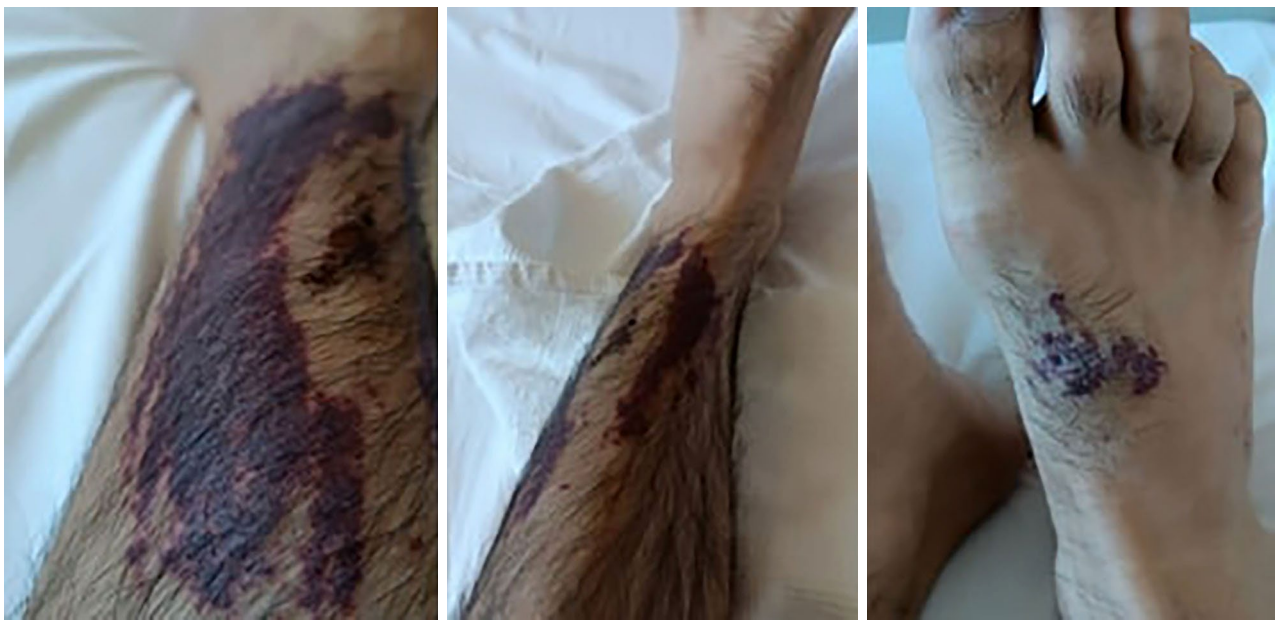

Figure 1. Palpable purpura.

Table 1. Laboratory results

	Before ART	ART (1st month)	ART (2nd month)	ART (3rd month)	ART (6th month)	ART (12th month)
HIV-RNA (copies/mL)	6033	Not detected	Not detected	Not detected	Not detected	Not detected
CD4 (mm^3)	427 (16%)	639 (22%)	975 (36%)	820 (36%)	780 (36%)	680 (38%)
CD8 (mm^3)	1869 (70%)	1710 (59%)	1463 (54%)	1208 (53%)	1105 (51%)	894 (50%)
CD4/CD8	0.23	0.37	0.67	0.68	0.70	0.76
WBC (4-10×10^3/µL)	16060	17180	7160	9630	6660	6520
Eosinophils (0.02-0,5×10^3/µL)	8800 (55%)	9520 (55%)	100 (1.4%)	90 (0.9%)	90 (1.4%)	210 (3.2%)
Hemoglobin (12-16.8 g/dL)	14.9	15.1	15	15.4	14.4	14.4
Platelets (100-400×10^3/µL)	248000	344000	240000	280000	264000	245000
AST (0-50 U/L)	52	41	20	23	24	16
ALT (0-50 U/L)	114	75	34	15	35	28
LDH (0-248 U/L)	466	-	-	-	146	-
GGT (0-55 U/L)	353	152	55	43	82	45
ALP (43-115 U/L)	928	238	82	86	92	88
CRP (0-5 mg/L)	4.3	1.26	0.28	2.1	0.11	0.14

HIV: Human immunodeficiency virus; RNA: Ribonucleic acid; CD: Cluster of differentiation; WBC: White blood cell; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; LDH: Lactate dehydrogenase; GGT: Gamma-glutamyl transferase; ALP: Alkaline phosphatase; CRP: C-reactive protein.

two drugs demonstrate the sustained efficacy of ART and the role of HIV in the etiology of EGPA. This case is the first documented instance in the literature of EGPA with HIV playing a role in its etiology.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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